The Wiley Handbook of Eating Disorders

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Volume 1
Basic Concepts and Foundational Research

Edited by

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A project this size can tax anyone's sanity and patience. You need time away from the work. Linda's grandchildren, Sabrina, Nathan, Izzy, and Lydia, were always a source of joy and relaxation as was Zeppelin. Finally, and perhaps most importantly, her husband, Jim Keeler, provided input on the process, ideas, and unending support for the complaining, the editing day after day (including weekends), and the distracted conversation. Thanks to one and all.

Michael would like to thank, first and foremost, his wife, Mary Suydam, who supported this project in so many ways as her husband wrestled with it and with the all too flexible meaning of "retirement." And somehow words such as "thanks" and "gratitude" and "deep" seem necessary but insufficient for what Michael would like to say in appreciation for the following friends and colleagues: Ted Mason, Jim Keeler, Sarah Murnen, Gail McVey, Margo Maine, Beth McGilley, Michael Strober, Catriona Galloway, Laura Hill, Carolyn Costin, Bruce Martin, Kathryn and Michael Dean, and, of course, The Hamster Music Regulars: Ann Cooke, Amy Baker Dennis, and Craig Johnson.

Last (speaking linearly), and foremost (speaking curvilinearly), Michael thanks Linda for the opportunity to work closely yet again with someone who is so accomplished as a researcher, theorist, writer, editor, mentor, critical thinker, and a scholar. It has indeed "been a long and fruitful collaboration," and perhaps someday Michael will be able to fully honor it by understanding that "perfect is the enemy of good."

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Volume 2
Assessment, Prevention, Treatment, Policy, and Future Directions

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Despite Linda's retirement, her colleagues from Kenyon have continued to provide support ranging from good ideas to sympathy. In particular, Sarah Murnen, Dana Krieg, Mary Suydam, and Judy Smith were always ready to talk.

A project this size can tax anyone's sanity and patience. You need time away from the work. Linda's grandchildren, Sabrina, Nathan, Izzy, and Lydia, were always a source of joy and relaxation as was Zeppelin. Finally, and perhaps most importantly, her husband, Jim Keeler, provided input on the process, ideas, and unending support for the complaining, the editing day after day (including weekends), and the distracted conversation. Thanks to one and all.

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Last (speaking linearly), and foremost (speaking curvilinearly), Michael thanks Linda for the opportunity to work closely yet again with someone who is so accomplished as a researcher, theorist, writer, editor, mentor, critical thinker, and a scholar. It has indeed "been a long and fruitful collaboration," and perhaps someday Michael will be able to fully honor it by understanding that "perfect is the enemy of good."

Section I
Definitions and History

1

Body Image, Disordered Eating, and Eating Disorders: Connections and Disconnects

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As we worked on this two-volume handbook, the field of psychiatric diagnosis in general and of eating disorders (EDs) in particular continued to be marked by conflict, controversy, and change. The development and publication of the fifth edition of the American Psychiatric Association's (2013) *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*) has been accompanied by considerable discussion and criticism (Frances & Widiger, 2012). Indeed, the National Institutes of Health and its subsidiary, the National Institute of Mental Health (NIMH), raised serious questions about *DSM-5*. NIMH actually announced plans to use a multidimensional system of marking mental illness rather than the categorical approach favored by *DSM-5* (Insel, 2013; Insel et al., 2010).

Under the criteria employed in *DSM-IV* (American Psychiatric Association, 1994) and *DSM-IV-TR* (American Psychiatric Association, 2000), some 40–60% (i.e., approximately half) of the clinically diagnosed cases of eating disorders were classified as "Eating Disorders – Not Otherwise Specified" (ED-NOS; Thomas & Vartanian, 2011, chap. 4). This catchall category included a diverse group of people with widely variant symptoms; its usefulness in guiding treatment strategy was virtually nil. So, *DSM-5* has changed the categorization scheme somewhat. For example, *DSM-5* has a specific category for binge eating disorder (BED). There is also a code for "Other Specified Feeding or Eating Disorder" (OSFED). This category, which includes forms of anorexia nervosa (AN), bulimia nervosa (BN), and BED that do not quite meet the full criteria, is meant to bring clarity to the earlier ED-NOS group. The OSFED category also defines purging disorder and night eating syndrome as eating disorders. However, none of the OSFED syndromes are as well developed in terms of symptomology, etiology, epidemiology, or differential diagnosis as AN, BN, or BED. A residual catchall category, now named "Unspecified Feeding or Eating Disorder" (UFED), is still available for clinicians who are convinced that a "disorder" of "feeding and eating" is present but who opt not to specify a particular form of OSFED (*DSM-5*, 2013, p. 354).

Furthermore, there is substantial disagreement about the nature and implications of models of ED. In particular, there has been considerable debate about what exactly the two dominant paradigms, the biopsychiatric and sociocultural approaches, are proposing, which type of

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explanation should be emphasized, and how these might be integrated (Becker, Keel, Anderson-Fye, & Thomas, 2004; Klump, Bulik, Kaye, Treasure, & Tyson, 2009; Levine & Smolak, 2014; Striegel-Moore & Bulik, 2007; Strober & Johnson, 2012; see also Chapter 67). The more biopsychiatric approaches have tended to downplay the eating and body image symptoms emphasized in *DSM-5* (American Psychiatric Association, 2013). Instead, they focus on cognitive and personality features of ED clients that seem to have a genetic or neural circuitry basis. This allows biopsychiatric theorists to minimize the role of the cultural influences, such as peers and media, that the sociocultural proponents emphasize.

There is, then, much disagreement among ED experts about some core issues. However, there is nearly unanimous agreement on at least one issue. EDs are serious illnesses. They significantly interfere with daily functioning. They have a high mortality rate. And for a substantial percentage of clients, they are chronic. Furthermore, EDs are qualitatively different from the type of body dissatisfaction that was referred to as "normative" (Rodin, Silberstein, & Striegel-Moore, 1985) or the type of dieting engaged in at some time by the majority of adolescent and adult U.S. females (Neumark-Sztainer, Wall, Larson, Eisenberg, & Loth, 2011). Yet, these more typical forms of body image, weight and shape concerns, and eating dysfunction are important predictors of the onset of and recovery from ED (Jacobi & Fittig, 2010; see also Chapters 22, 24, & 54).

The overarching purpose of this chapter is to provide a brief exploration of the relationships among body image problems, disordered eating, and EDs. This discussion is intended to provide background and context for this two-volume *Handbook*. The chapter contains three major sections. The first focuses on the role of body image and disordered eating as definitional criteria for and core features of EDs. The second section considers body image and disordered eating as risk factors for EDs. Finally, the question of causes of body image dysfunction and disordered eating is given some attention. Throughout the chapter, we give some consideration to why body image and disordered eating perhaps should not be considered critical to our understanding of EDs.

Negative Body Image and Disordered Eating as Criteria

The *DSM-5* section on Feeding and Eating Disorders begins with the statement: "Feeding and eating disorders are characterized by a persistent disturbance of eating or eating-related behavior that results in the altered consumption or absorption of food and that significantly impairs physical health or psychosocial functioning" (p. 329). Thus, in keeping with the American Psychiatric Association's definition of a "mental disorder" in *DSM-5* (American Psychiatric Association, 2013, p. 20), some aspect of eating that is not culturally normative or approved, and that is disabling and/or distressing, is a symptom or sign of every eating disorder listed in the *DSM-5*. Disordered eating ranges from eating nonnutritive substances (pica) to severe food restriction (as in AN or avoidant/restrictive food intake disorder) to binge eating (as in BED or BN). An eating disorder is "persistent," so in pica and rumination disorder, the disordered eating symptom must be present for at least a month. In BN and BED, the binge eating and purging behaviors must continue for 3 months. However, the severe food restriction that marks both AN and avoidant/restrictive food intake disorder (ARFID) and results in physical symptoms is not time delineated in *DSM-5*. This difference likely acknowledges that some forms of disordered eating occur in non-eating-disordered populations and become "abnormal" only when they are more severe, intense, and/or

dangerous. While food restriction, in the form of calorie restrictive dieting, is fairly common in the general population, it does not have the concomitant physical damage associated with AN and ARFID. Thus, the *DSM-5* criteria are designed to differentiate less severe disordered eating from pathology.

Body image disturbance, on the other hand, is part of the criteria for only AN and BN. Both BN and AN are marked by body image issues surrounding weight and shape. AN symptoms also include disturbances in the perception of one's body. Outside of the OSFED subtypes pertaining to AN and BN, other diagnostic categories within the *DSM-5* Feeding and Eating Disorders do not feature disturbances in body image, thereby emphasizing the connections between BN and AN. Though AN and BN are certainly distinct categories, there is more crossover by sufferers between these two than of either one with any other psychiatric disorder (see Chapter 54). Furthermore, AN is the preeminent diagnosis of the feeding and eating disorders in that its diagnosis "trumps" all others. Thus, AN must be eliminated as a diagnosis before other feeding and eating disorders can be identified.

Most of the research discussed in these volumes focuses on AN and BN with some consideration of BED. Again, all three of these disorders are marked by disordered eating. Indeed, both BED and BN are defined by episodes of binge eating. Nevertheless, it appears that BN and AN are more closely related than BN and BED are. AN and BN both involve weight and shape concerns. The "three essential features of anorexia nervosa" (*DSM-5*: American Psychiatric Association, 2013, p. 339) all focus on disordered eating and disturbed body image. The same is true of the core components of BN. Thus, given the symptomology of EDs, any etiological model needs to explain disturbances in body image and eating.

Of course, the emphasis on symptoms assumes endorsement of at least the nosological approach of the *DSM-5*. The Director of the NIMH has rejected that approach and has instead called for the development of a biologically based nosology of mental illness, emphasizing neural circuits, genetics, and cognitive patterns (Insel, 2013). Furthermore, this approach, dubbed the Research Domain Criteria (RDoC), will offer analysis of functions. In RDoC, diagnostic categories would reflect clusters of neurological, genetic, and cognitive features rather than the symptoms that provide definitional criteria in *DSM-5*. Currently, the research is not available to support this type of cluster analysis much less to ascertain the value and effectiveness of the RDoC approach for treatment and prevention. Nonetheless, the RDoC approach does call for research emphasizing components of eating disorders other than the disordered eating and body image featured in *DSM-5*.

Body Image and Disordered Eating as Risk Factors

All of the research on ED risk factors is based on the assumption that EDs are the disorders described in *DSM*. Risk factor research consistently shows that body image and disordered eating are perhaps the best predictors of the development of EDs. Among adolescent girls, over a dozen longitudinal studies indicate that weight and shape concerns predict the onset of EDs (Wertheim, Paxton, & Blaney, 2009; see also Chapter 22). Several prospective studies have also indicated that naturalistic dieting predicts both the onset and the worsening of EDs (Neumark-Sztainer, Wall, Guo, Story, Haines, & Eisenberg, 2006; Stice, Cameron, Killen, & Hayward, 1999), though experimental data indicate that some types of food restriction are likely beneficial rather than risky (see Chapter 24). There are also data indicating that fussy or

restrictive eating during childhood is related to the later development of EDs (Marchi & Cohen, 1990).

Body image concerns focusing on weight and shape are common, particularly among adolescent girls and women (Wertheim et al., 2009; see also Chapter 22). Even among girl children in the United States, Australia, and other developed countries, a substantial minority (perhaps 40% or so) report body dissatisfaction. Similarly, large percentages of girls and women report using weight control methods, with exercise and calorie restriction being particularly popular and more extreme methods such as purging being less common (Neumark-Sztainer & Hannan, 2000; Wertheim et al., 2009). Yet, as many theorists have argued, only a small minority of women develop EDs (Striegel-Moore & Bulik, 2007).

It may be the case that some of the difference between normative and pathological weight and shape concerns is primarily quantitative (Gordon, Holm-Denoma, Crosby, & Wonderlich, 2010). The body dissatisfaction experienced by people with an ED may be more multifaceted and intense (Williamson, Gleaves, & Stewart, 2005). It is this possibility that allows some measures that emphasize (The McKnight Investigators, 2003) or incorporate (Mond et al., 2008) weight and shape concerns to serve as effective screens for EDs. However, there is also some consensus that there is a qualitative difference between normative and pathological body concerns. In his transdiagnostic theory of eating disorders, Fairburn (2008) explicitly distinguishes between widely occurring body shape dissatisfaction and the essence of the eating disorders. Specifically, Fairburn argues that AN and BN and "most cases of eating disorder NOS share a distinctive 'core psychopathology' that is cognitive in nature. This psychopathology is the overevaluation of shape and weight and their control as the most important domain of life by which to define and determine the worthiness of one's self" (p. 12).

Theorists often suggest that personality or cognitive factors might form this qualitative difference (Nunn, Lask, & Frampton, 2011; Striegel-Moore & Builik, 2007; Strober & Johnson, 2012). These characteristics are assumed to be genetically based and more or less hard-wired into the neural circuits. In other words, those most likely to develop EDs are people who are fundamentally different and at risk for psychopathology early in life. Experiences such as trauma, particularly perhaps sexual abuse, may exacerbate these tendencies (Thompson & Wonderlich, 2004; see also Chapter 34). These models minimize the role of sociocultural influences, suggesting that the messages provide only content for symptom expression rather than creating and sustaining psychopathology and pathophysiology per se. From this perspective, body shape concerns are just an outlet for the rigidity, perfectionism, and harm avoidance that are at the heart of EDs (Kaye, Bailer, & Klabunde, 2012; Strober & Peris, 2011).

This is an interesting argument and one that certainly seems consistent with the RDoC approach (Insel, 2009, 2013; Insel et al., 2010). Nonetheless, there is no evidence that any particular personality, cognitive, or traumatic variables are specific to EDs. There is not even clear evidence that any of these are genetically caused, although there is little doubt that trauma and habitual patterns of behavior each sculpt neural circuits (Charney, Deutsch, Southwick, Krystal, & Friedman, 1995). On the other hand, it is not surprising that body image concerns and disordered eating are specific risk factors for EDs, given their role as symptoms within the *DSM-5* and given the continuous or dimensional nature of drive for thinness, fear of fat, and inappropriate compensatory behaviors (Gordon et al., 2010; Williamson et al., 2005). One question, then, is how important are specific risk factors to understanding and treating EDs? This is a recurring issue throughout the *Handbook*.

Causes of Body Image Dysfunction and Disordered Eating

The *Handbook* is about EDs. Yet, many pages focus on body image concerns and disordered eating, particularly in Section V, dealing with risk factors. This is where the research actually is. Samples in such research are typically community-based rather than clinical. Researchers who use sophisticated statistical techniques to test multifaceted theories, such as hierarchical linear modeling or structural equation modeling, need large samples of often 200 or more participants. EDs, particularly AN, are low-frequency disorders (see Chapters 5–7), so it may take years to recruit a sufficiently large sample from clinical settings. Furthermore, the clinical participants are likely to have undergone treatment. Finally, by definition, clinical samples are already suffering from EDs. This makes them useless for the type of prospective research needed to identify causal relationships (Kraemer et al., 1997). In general, clinical samples are most likely to be useful in experimental designs, particularly focusing on treatment, relapse, and recovery.

Much of the risk factor research, then, is derived from community-based samples, particularly of White college women. College women are a sample of convenience for many researchers, but they also are a group particularly at risk for the development of EDs, especially BN. So they are appropriate. There is also prospective research using child and adolescent samples (Dohnt & Tiggemann, 2006; Neumark-Sztainer et al., 2006). Although there are data delineating the frequency of EDs, body image concerns, and disordered eating among various U.S. ethnic groups and in a variety of countries, there are few prospective studies of these groups (see Chapters 23 & 25).

While there certainly are limits on the risk factor data, researchers have been able to identify important variables using both experimental and longitudinal designs. Internalization of the thin ideal, rooted in sociocultural messages about beauty, gender, and self-control, may be important (see Chapter 21). The messages themselves, coming from peers, family, and media, also appear to play an important role in developing body image concerns and disordered eating (see Chapters 26, 29, & 31). More specific messages from more narrowly defined cultures, such as certain sports, may also be influential (see Chapter 35).

Some of the cultural messages reflect the vilification of fatness in countries such as Canada, Great Britain, and the United States. Even very young children are aware that body fat is undesirable and unacceptable. They associate a range of negative characteristics with fatness and express a preference for thinner friends (Harriger, Calogero, Witherington, & Smith, 2010; Holub, 2008). Thus, research often shows that actual body mass index (BMI) or perceived weight status predicts body dissatisfaction or disordered eating. However, messages concerning the thin ideal are so strong and pervasive that normal and below-average-weight women often suffer from body dissatisfaction and disordered eating.

It is also important to recognize that body dissatisfaction and disordered eating are gendered behaviors (Rodin et al., 1985; Smolak & Murnen, 2004; Striegel-Moore & Bulik, 2007; see also Chapters 19, 27, & 37). Women and girls are more likely than men and boys to be concerned about being thin and are more likely to engage in behaviors aimed at achieving thinness. Not surprisingly, then, girls and women are substantially more likely to develop the EDs of AN and BN that are based in overvaluation of weight and shape, negative body image, and caloric restriction. This is true throughout the lifespan (see Chapter 36). Macrocultural factors, such as gender and ethnicity roles, shape the messages that individuals receive. For example, women are more likely to be sexualized in the media (American Psychological Association, Task Force on the Sexualization of Girls, 2010; see also Chapters 27 & 29), reinforcing the belief that the appearance of women's bodies is especially important. Men and

women also have different experiences with sexual violence, experiences that have been shown to be related to body image, disordered eating, and EDs (see Chapters 27 & 34).

There is some research linking genetic influences to body image and disordered eating (Culbert, Burt, McGue, Iacono, & Klump, 2009; Suisman et al., 2012; see also Chapter 28). However, these data are principally concurrent and so cannot clearly establish genetic diversity as a risk factor. Furthermore, it is not clear at this time whether a genetic influence actually directly affects body dissatisfaction or disordered eating, or instead reflects another correlated characteristic such as social comparison tendencies.

Conclusions

Both volumes of *The Wiley Handbook of Eating Disorders* are devoted to examining, enhancing, and challenging, in constructive ways, our understanding of the eating disorders. We strongly believe that in accordance with current data from risk factor research, as well as current trends in classification, diagnosis, and transdiagnostic theory, any attempts to understand, prevent, and treat eating disorders must acknowledge the relationships among body image concerns, disordered eating, and EDs. Across the past 30–35 years the American Psychiatric Association, in collaboration with the American Psychological Association, has fashioned a series of *DSM*s that have included body image disturbances as part of the diagnostic criteria for AN and BN. Although most research uses the reigning version of the *DSM*, it is noteworthy that some aspects of body image disturbance are also key diagnostic features of eating disorders in the 10th edition of the World Health Organization's (1992/1993) *International Classification of Diseases* (*ICD-10*), in effect since the early 1990s.

The central roles of negative ("distorted") body image and disordered eating in the definition and classification of eating disorders complicate the relationship between body image and disordered eating as risk factors versus symptoms of EDs. For example, it remains unknown when and under what circumstances weight and shape concerns stop being a risk factor and become a symptom. This in turn has significance for a number of phenomena addressed in this book, such as the classification of EDNOS and OSFED, the epidemiology of disordered eating, the distinction between prevention and treatment, and the meanings of recovery.

The overarching goal of this *Handbook* is to provide the background information people need to work in the ED field. Our superordinate principle in organizing this *Handbook* is that a wide variety of people, including students in many different disciplines, etiological researchers, prevention program designers, therapists, social policy advocates, and friends and family of sufferers, deserve very research-oriented, state-of-the-art information. We hope that readers from anthropology, cultural studies, dietetics, mass communications, sports science, neuroscience, psychiatry, psychology, psychopharmacology, public health, and social work will find this information so compelling that they will join the effort to discover the causes and cures for EDs. These disorders, more fatal than most other psychiatric problems, have already robbed us of enough people.

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2

History of Anorexia Nervosa

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The term *anorexia nervosa* (AN) has been in use for the past 140 years (Gull, 1874). This designation has been criticized as a misnomer, because the syndrome does not necessarily involve a lack of appetite. Hilde Bruch (1973) suggested that the German term "Magersucht" described better the distinctive psychopathological feature, an "addiction" to extreme thinness, termed variously "drive towards emaciation" (Selvini Palazzoli, 1963/1974) and "relentless pursuit of thinness" (Bruch, 1965), or, more adequately in stressing fear, "l'idée fixe d'obésité" (Charcot; cited by Janet, 1907), "weight phobia" (Crisp, 1970), or "morbid fear of being fat" (Russell, 1970). These terminological issues reflect how much AN has been disputed in history, and also how much its history is still in dispute.

Knowledge of the history of AN helps put into perspective current controversies, which in some respects repeat past debates. History shows how some insights are over and over again forgotten or repudiated for ideological reasons. Finally, history helps put into focus both the influence of cultural context and our own biases. After considering the nature of evidence in historical research on syndromes, I will provide an overview of the history of extreme fasting and AN, and then propose ways in which the ideological and theoretical preferences of doctors influenced their clinical reports. Then I will summarize some possible sociocultural factors influencing actual changes in eating disorders (EDs) prior to their identification as medical disorders, as well as how medical conceptualizations have secondarily influenced the nature of EDs. For brevity's sake, I will refer to the principal secondary sources that contain references to the primary historical sources.

Methodological and Ideological Intricacies of Writing the History of a Syndrome

Writing the history of a psychiatric syndrome mainly serves two purposes: Legitimize the writer's present-day conception and learn about the syndrome itself, the discipline, or society. Syndrome histories may focus on the history of medical ideas, termed conceptual history by

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Berrios (1996), or they may reconstruct historical changes in the actual occurrence and form of the syndrome. Conceptual histories can legitimize the current state of knowledge by heralding founders and writing a history of the progress of knowledge. Such histories may also remind contemporary researchers and practitioners of forgotten insights into the nature and treatment of the syndrome, or point to past mistakes. "Natural" histories can be used to demonstrate that a newly defined syndrome has always existed, thereby justifying the new category. This strategy was used by Charcot when he analyzed medieval reports on witches using his modern concept of hysteria (Charcot & Richer, 1887). Histories that demonstrate the historical constancy of a syndrome may also be used to argue for its somatic nature. Histories of the variability of a syndrome may decry possibly responsible societal developments, or justify the call for the allocation of resources to new services and positions.

The history of AN has served all of these purposes, and no history can escape present-day implications. Therefore this chapter both spells out its methodological assumptions and argues for present-day implications. The epistemological stance toward psychological disorders and their history taken here may be termed realist and lies between idealist or postmodern positions and positivist or naturalist positions.[1] Psychological disorders need to be described at the psychological level, because they are defined as disorders of thinking, feeling, and acting. They are influenced to varying degrees by biological and social factors. They are assumed to exist independently of the observing scientist, but are influenced by the observer. Observations, on the other hand, are influenced by the observers' theoretical and practical perspectives, which in turn depend on their personality, professional orientation, and wider cultural context. Both patient and observer share a cultural and natural environment, which influences the nature of the patients' suffering and their illness behavior, as well as the observers' systems of significance and practice.

These assumptions have implications for what is considered valid evidence when writing the history of a syndrome. When writing a conceptual history of the profession's thinking about a syndrome, it is sufficient to ensure that the sample of publications used is representative. Depending on the historical distance, concepts and even words used in the sources will be different from today's usage and therefore need to be reconstructed. Writing the history of a syndrome is more difficult than a conceptual history, because its object (a syndrome) is one step more remote from us. Both the historical "distance" to the phenomena of interest, that is, the afflicted individuals, their thoughts, feelings, and actions, as well as the historical distance from the sources through which we gain access to psychological inflictions, need to be bridged. Thus, sources need to be critically evaluated not only in terms of trustworthiness, which is influenced by the writers' interests and purpose in writing, but also in terms of the writers' mindset. What was noteworthy and what was reportable versus what was taken for granted or deemed insignificant may differ significantly from what we are interested in today. Therefore the historian has to reconstruct the historical writers' mindset to interpolate what they may have actually seen. This may be close to impossible for individual writers' psychology, but might be approximated in terms of cultural and professional mindsets. This is easier the more similar the historical period is to ours and the more we know about it.

The nature of AN renders writing its history even more difficult, because typically there is a denial of illness and therefore also of emaciation, and, to circumvent social pressure to eat and gain weight, there is also secrecy regarding motives for eating little and moving much. Even nowadays this may pose a problem for diagnosis. This motivates some authors (Keel & Klump,

[1] I use the term "psychological disorders" and not "psychiatric disorders" here because I wish to refer to psychological phenomena independent of their medical classification.

2003; Treasure & Campbell, 1994) to suggest deleting the central organizing motive of AN from the diagnostic criteria. This would, however, imply giving up understanding the specific psychopathological core of the syndrome. Nevertheless, nowadays a combination of empathic interviewing and clinical observation makes diagnostic judgment possible at least over the course of some time. Historical writers, however, who did not have the modern concept of AN, had a hard time seeing and reporting in print those observations that we need to identify a case of AN (see Chapter 8).

Given these intricate methodological issues, the history of AN can try only to draw a more or less probable picture by attempting to fill the gaps. The two major ways to do this are by reconstructing the observers' worldview and interests and by using those indirect diagnostic signs of AN that still allow the making of a probable differential diagnosis even in the absence of any explicit report of weight phobia.

History of Extreme Fasting and Miraculous Abstinence from Food

Most research on the history of eating disorders (EDs) was published in the 1980s. The most comprehensive work on extreme fasting and food refusal was published by Vandereycken and van Deth (1994), while the best-informed source concerning the history of overeating was published by Ziolko and Schrader (1985; see also Chapter 3). Two relatively distinct historical types of intentional abstention from food were identified: ascetic-mystic fasting and miraculous survival without food.

Ascetic-Mystic Extreme Fasting

Ascetic-mystic fasting is motivated by religious ideas about the duality of body and spirit. Fasting aims at purifying the spirit by mortifying (i.e., subduing or deadening) the body. Fasting is only one among other ascetic practices that serve to liberate the mind from its dependency on bodily needs for food, drink, warmth, rest, freedom from pain, and freedom from longing for sensual pleasures. In addition, extreme fasting facilitates entering states of ecstasy and visions, which makes it a highly valued practice in mystic cults. These practices are reported, for example, by early Christian monks (Bemporad, 1997), as well as by medieval Chinese Daoist priestesses (Lo, Hsu, & Vandereycken, 2012).

Apart from a few isolated medieval reports of young women who survived without eating and who were understood as being possessed by the devil (Habermas, 1986; Skrabanek, 1983; Vandereycken & van Deth, 1994), in the period leading up to the Reformation (12th to 15th centuries) reports on cases of extreme fasting are dominated by ascetic-mystic fasting in the context of the religious practices of pious people. Bell (1985) described a type of late medieval, typically Italian female saint who was famous for her extreme fasting, often living only on the host, immersed in ascetic practices, and experiencing mystic revelations. Saint Catherine of Siena is the most well known. Speaking of "holy anorexia," Bell likens many of those saints' practices to those of modern day anorexics. The striking similarities are extreme fasting that goes beyond normal ascetic practices; self-denial as a reaction of girls to adolescent sexual maturation; social withdrawal; and stubborn refusal to eat once the religious establishment, doctors, or the family begin to exert pressure.

Despite these similarities, differences abound (Bynum, 1987). Fasters intentionally cultivated and practiced their fasting religiously in a religious context, imitating the models of Christ and

saints such as St Francis of Assisi (Davidson, 1999), and it was apparent to them and often to others that asceticism produced mystical experiences that brought them closer to God. Also they were offered institutional roles by the Church hierarchy if only they moderated their fasting, which some of them accepted (Bynum, 1987). Their focus was more on not eating rather than on losing volume and weight. The Church did everything to prevent these young women from gaining fame for being able to live for a long time without eating in their lifetime, because "living saints" (Zarri, 1980) undermined the Church's authority. However, if they did succeed in avoiding trial for demonic possession by ceding to ecclesiastical powers, some of the "holy anorexics" did play quite influential roles in their time. With the Reformation's questioning of miracles and abolition of saints, the Catholic Counter-Reformation (mid-16th to mid-17th century) restricted the criteria for miracles and canonization of saints. The historical traces of ascetic-mystic fasters changed from hagiographies to records of the inquisition (Zarri, 1980) and to medical publications, producing an altogether different appearance of extreme food restraint.

Secularized Miraculous Fasting

From the 16th century onwards the reported pattern of not eating changes in several respects. Not eating was embedded in physical illness and disability, often resulting from apparent hysterical conversion symptoms. These women were of socially modest background, passive, often still lived with their parents (not in a monastery), and parents mediated the contact with the secular public. None evidenced ascetic practices, although the young women themselves were very religious and were often sought after as holy women by the public. It is difficult to tell to what degree this relatively sudden historical change is due to the change from religious, Catholic to secular, medical sources, and to the change from mostly Italian to mostly Germanic sources. It appears that the new characteristic of not eating and pretended survival without eating was exceptional in Italy. This is seen in the work of two Italian medical writers who mention only two such Italian cases, but report at length on many German and Dutch cases of the 16th century (Habermas, 1990, 2005; van Deth & Vandereycken, 1992).

However, phenomena did not change as abruptly as the change of sources suggests. Secularization was a very gradual process also in Protestant areas, as historical anthropologist Waltraud Pulz (2007) has shown. She analyzed in detail seven of the ten 16th century cases known from German and Dutch areas, drawing on a rich array of mostly medical historical sources. The more detailed the stories, the more complex each appears, showing that secularization and the abolition of belief in miracles and saints was not a sudden effect of the Reformation and Counter-Reformation of the 16th century. Rather, secularization and abolition of supernatural beliefs was a long process stretching between the 16th and 20th centuries. Actually the process started even earlier, because in the 15th century the Catholic Church had already started refraining from officially acknowledging miraculous cases of not eating for extended periods of time (and also mystical experiences).

Similar cases of "miraculous fasting" were reported in northwest Europe and also in the United States, especially in the late 18th and 19th centuries. These included Molly Fancher in Brooklyn and Sarah Jacobs in England (Brumberg, 1988), and Anne Marie Kinker in northern Germany (Habermas, 1990). The English language secondary literature on this period has mostly been limited to English, U.S., and Canadian cases (Brumberg, 1988; Keel & Klump, 2003). Early 20th century case reports from Catholic areas like Belgium (Louise Lateau), Italy (Palma Maria d'Oria; for both see Hammond, 1879), and Bavaria (Therese Neumann; Seidl, 2008) focus somewhat more on traditional religious signs such as stigmata and mystic experiences. What most of these

women appear to lack is the active ascetic zeal of ascetic-mystic fasters of earlier centuries. Instead there is a stress on passive suffering and, often, normal body weight! What unites these reports is the contention of living without food (being nurtured solely and miraculously by faith in God's grace), which is contested by medical and juridical professionals in the service of both ecclesiastical and worldly powers.

Reconstructing the History of Anorexia Nervosa

This section focuses on the reconstruction of the history of the phenomenon now called "anorexia nervosa" (AN). The history of medical *conceptions* of AN, in contrast, will be discussed in the subsequent section. For retrospective identification of cases I will use today's concept of AN, especially a fear of an abundance of body mass, which is the central organizing motive for the symptomatic actions aiming at achieving or maintaining an underweight body, such as eating food that promises little weight gain; physical and mental overactivity despite underweight conditions; denial of underweight and illness; and secretive actions to protect the person's liberty to fast. It is weight phobia and the ensuing denial of emaciation and of illness that differentiates AN from what Hilde Bruch (1973) termed "secondary anorexia," that is, other forms of psychogenic malnutrition and fasting, as can be seen in depression, delusions of food poisoning, food phobic states, hysterical vomiting, *globus hystericus*, and gastric complaints. In none of these states is there a fear of normal body weight; emaciation is a consequence of various other motives for not eating.

If the current definition of AN is used for writing a history of the syndrome, if historical descriptions are taken at face value, and if only English language historical publications are analyzed, then it appears that AN was first described in the 1930s, by the British doctors Young in 1931, Ryle in 1936, and Nicolle in 1938.[2] A few years later U.S. doctors of internal medicine, influenced by psychoanalysis and interested in psychosomatics, published several articles also mentioning the fear of weighing too much as a motive in specific cases (McCullough & Tupper, 1940; Rahmann, 1939; Waller, Kaufmann, & Deutsch, 1940).

This picture implies that prior to 1930 the term AN was applied to describe patients who nowadays would not be diagnosed with AN. Yet, this has not been established and, even if it were, it would need to be explained why the same term was used so homogenously for such different patients. Nevertheless, given the strong preference for a nonpsychological, somatic definition of psychiatric diseases, it appears that the dominant view of the history of AN in the anglophone literature is that we should define AN more broadly than we presently do to include any psychogenic food restriction, or at least any extreme fasting. This would mean that the history of AN begins with Lasègue in 1874 and Gull in 1873, and would imply that weight phobia became a new component in the 1930s in a malady that otherwise is 60 years (Casper, 1983; Russell, 1985) or even centuries older (Keel & Klump, 2003).

If, however, articles and books published in languages other than English are also included in the sample of sources, weight phobia emerges as a symptom in clinical descriptions of cases of AN over half a century earlier. Searching German, French, and Italian psychiatric and medical journals available between 1850 and 1950, I found weight phobia described in cases in several reports starting in 1878. The list in Table 2.1 summarizes

[2]For the sake of brevity, unless otherwise noted, in this section and the subsequent section the references for case histories and other medical sources on anorexia published between 1870 and 1945 are listed in Habermas (1989, 1992a).

Table 2.1 Clinicians describing weight phobia in case presentations in German, French, and Italian psychiatric and medical journals and books between 1878 and 1945.

Year	*Author(s)*	*Country of Residence*	*Language of Publication*
1878	Rist	Switzerland	Italian
1883	Charcot (Féré)	France[a]	French
1892	Féré	France[a]	French
	Gungl & Stichl	Austria	German
	Wallet	France[a]	French
1894	Brissaud & Souques	France[a]	French
1898	Janet	France[a]	French
	Kissel	Russia	French (German, Russian)
1900	Ling	Estonia	German
1902	Ebstein	Germany	German
	Raymond	France[a]	French
1905	Girou	France	French
1909	Bérillon	France	French
1910	Tarrius	France	French
1911	Régis	France	French
1912	Noguès	France	French
	Schnyder	Switzerland	French
1914	Raimbault	France	French
1922	Lévi	France	French
1924	Möller	Denmark	German
1925	Souques	France[a]	French
1926	Faber	Denmark	German
	Ziehen	Germany	German
1927	Lafora	Spain	Spanish
1930	Aurimond	France	French
1931	Young	Britain	English
1932	Schottky	Germany	German
	Steinitz, & Thau	Germany	German
1936	Benedek	Germany	German
	Bergmann	Germany	German
	Ryle	Britain	English
1937	Krause & Müller	Germany	German
	Kylne	?	German
1938	Nicolle	Britain	English
1939	Leibbrand	Germany	German
	Rahmann	USA	English
	Trefzer	Switzerland	German
1940	McCullough & Tupper	USA	English
	Waller et al.	USA	English
1941	Deutsch	USA	English
	Falta	Germany	German
	Wissler	Germany	German
1942	Feuchtinger	Germany	German
1943	Accornero	Italy	Italian
1944	Binswanger	Switzerland	German

Note. References and more details are provided in Habermas (1989, 1992a), except for Charcot (1883), which I found recently thanks to Gelfand (2000). Starting in the 1920s, German references are oversampled because of intensive research for a chapter on anorexia nervosa in Germany between 1916 and 1945 (Habermas, 1994, chap. 9).
[a] Worked at the Salpêtrière in Paris.

earlier lists in Habermas (1989, 1992a). Starting in the 1880s, many more texts mention the desire to lose weight as the motive for extreme fasting in AN, without providing case descriptions (Habermas, 1989, 1992a). The relatively extensive case reports by Brissaud and Souques (1894), Janet (1902), Schnyder (1912), and Binswanger (1944; see Hirschmüller, 2003) provide ample evidence that these patients psychologically resembled current patients with AN.

A summary of a lecture by Charcot that is not included in my previous publications demonstrates the observation of weight phobia in the last quarter of the 19th century. Even this report on an 18-year-old young woman, summarized by Féré and Levillain (1883), includes the typical psychological aspects of AN. She had consulted Lasègue and Cornil before coming to Charcot in the summer of 1882. "It was a case of psychic anorexia. The patient systematically and energetically refused all food. … She weighed 29 kg [editor's note: approximately 64 lbs] … For her, the beauty ideal was defined by excessive skinniness (*maigreur*)." Charcot goes on to describe an extended struggle with the patient over any food she was to eat, as "the *idée fixe* not to eat [was] ever present. At times she hid the food in her towel, her pockets, her stockings, sometimes she even tried to keep it in her mouth… She entered the water cure clinic of Passy in a state of great agitation. Her skin was cold and viscous; she was incessantly tormented by the idea of gaining weight (*grossir*)." Half a year later, after being treated by water cure and a strict alimentary regimen, the patient was released with a body weight of 40 kg (88 lbs). After returning home, the young woman soon lost weight again. Her mother refused to put her back in the hospital, and the patient died within weeks (Charcot, 1883, p. 4).

Charles Féré and Fernand Levillain's (1883) summary of another of Charcot's lectures in the same year basically confirms Lasègue's clinical description of 1873, stressing the lack of insight and the subjective gain from abstinence as well as overactivity, but falling short of mentioning anything similar to weight phobia both in the general description and in the case of a 21-year-old patient treated in 1881. However, Féré's own three-page case report published nine years later again stresses the conscious project of losing weight, in this case to attract the attention of a man, over which at some point the girl lost control. This 15-year-old had laced her corset very tightly and secretly wore a strictly tied linen belt under her blouse. She did everything to prove that she was not ill.

Thus, because the reporting of weight phobia in anorexic patients began in the last quarter of the 19th century in French, German, and Italian language publications, the timing of the historical emergence of weight phobia in AN needs to be moved back 50 years, at least in regard to these three linguistic areas. However, if one does insist on the current diagnostic understanding of AN, the problem remains that there were other publications of alleged cases of AN that did not report weight phobia, including the two publications credited for introducing the concept of AN (Gull, 1874; Lasègue, 1873).

A critical approach to historical sources suggests that they need not be taken at face value. The writers' interests, categories, and notions of what is worthy of reporting, as well as the journals' policies and audiences, need to be taken into account when inferring from a source what historical reality may have looked like. The next section explores some of these factors in more detail. The one to be mentioned here is that people with AN tended to hide their motivation to fast, including the fact that they were intentionally reducing their body weight by fasting and possibly by overactivity and purging. Such secretive behavior was noted frequently in the literature. If historical writers were not expecting to find weight phobia, it was even more difficult to detect. Therefore it was often not reported,

although it may have been present in the patients. Following Bruch (1973), I argue that two interrelated types of indirect specific evidence for AN are a denial of illness and a denial of emaciation despite a state of severe malnourishment. This was mentioned by Lasègue in 1873, but not so explicitly by Gull in 1874. Nevertheless, if indirect evidence such as denial of illness or overactivity is taken into account, some of the case reports that do not mention weight phobia can be argued nevertheless probably to describe actual cases of AN (Habermas, 1989, 1992a). Confirming the existence of weight phobia starting in the second half of the 19th century, Queen Elizabeth of Austria suffered from an intense fear of becoming overweight in the early 1860s, which she countered by strict fasting and physical exercise resulting in severe emaciation (Vandereycken & Abatzi, 1996).

From the 1930s onwards, publications started reporting case series with more than 20 patients. The number of publications began to increase more rapidly in the 1960s and 1970s. Available epidemiological evidence suggests that in Europe and North America the prevalence and incidence of AN increased between 1940 and 1990, especially in the 1960s and 1970s, probably reaching a stable level since 1990 (Hoek, 2006; see also Chapter 5).

Before moving on to the conceptual history of AN, two counterarguments against this view of the history of the phenomenon of AN need to be considered. One concerns the historical boundary between AN and religiously motivated and interpreted fasting, the other the boundary of Western AN with non-Western forms of food refusal (see Chapters 6 & 7). The only explicit reference in the historical literature on AN to religiously interpreted extreme fasting and surviving without food can be found in Brugnoli's report on two cases of possible AN from 1875. He refers to the illuminated Pope Lambertini's reform of the criteria for canonization in the 18th century and to the appendix to the Pope's major text on canonization, which was written by Beccari, a medical doctor who discussed natural versus supernatural causes of not eating (Habermas, 1992b). The few historical case reports of anorexic-like behaviors with religious motivation appear not to be actual cases of AN, with the exception of the one patient described in 1912 by Schnyder (Habermas, 2005). There appears to be a relatively clear difference between religious motivations for ascetic fasting and the motivation for fasting in AN.

Cross-cultural comparison of forms of EDs, especially between Western and more traditional societies, might provide indirect evidence on the history of AN, if it is assumed that present-day traditional societies somewhat simulate earlier Western societies. Putting aside the weaknesses of this assumption for the sake of the argument, Lee's (1995) finding that the majority of anorexic patients in Hong Kong in the 1980s lacked weight phobia but presented otherwise comparable clinical features might be seen as a confirmation of AN without weight phobia. Accordingly, Lee argued for dropping weight phobia from the diagnostic criteria to render them, as he thought, more culture-neutral. However, systematic comparisons between samples of underweight patients with and without weight phobia do indicate that people with very low body weight who have EDs with an absence of weight phobia show less severe symptomatology (Carter & Bewell-Weiss, 2011) and have a better course (Lee, Chan, & Hsu, 2003; Strober, Freeman, & Morrell, 1999) than patients with AN. Crow et al. (2012) recently found that underweight eating-disordered patients without weight phobia had a higher mortality rate than those with weight phobia. However, weight phobia was assessed by questionnaire, so it is not clear whether self-reported absence of weight phobia can be seen as authentic or as a sign of denial of illness. Actually, AN (defined by weight phobia) has been increasing and atypical forms of EDs decreasing in frequency in the 1990s and 2000s in Hong Kong (Lee, Ng, Kwok, & Fung, 2010).

The Influence of Medical Thinking on the Description of Anorexia Nervosa

The history of theories about AN begins with the introduction of the terms *anorexie hystérique* in 1873 by Charles Lasègue, and *anorexia nervosa* in 1874 by Gull. The differences between the French and British traditions already show in these first two reports, inasmuch as Gull reports mostly physical and behavioral aspects, whereas Lasègue pays more attention to the psychological aspects of his patients.

As strange as it may sound to us now, a historical precondition for understanding extreme fasting as a psychological disorder was the firm conviction that humans could not survive extended periods of time without eating. The physiology of eating and digestion was advancing in the second half of the 19th century, but some experts still believed that not eating does not necessarily lead to weight loss and physical collapse. Thus, Empereur (1876) still maintained that hysterical patients could live without eating! In fact, in the late 19th and early 20th centuries intensive research on food intake and elimination in psychiatric patients in highly controlled environments over weeks and months still reflected doubts in regard to the possibility of not losing weight when not eating (Gilles de la Tourette & Cathélineau, 1890; Janet, 1926, pp. 159 ff.). On the other hand, Italian physiologist Luciani published a book on fasting in 1889, reporting his research on Succi, one of many male hunger artists of the time. The change in attitude is exemplified by Luciani's severe and condescending criticism of a doctor who had written a report on the survival without food of the fasting maid Anna Garbero only 60 years earlier.

Analysis of the nationality, medical discipline, theoretical orientation, and professional interest of those historical doctors who described weight phobia in anorexic cases or probable cases of AN before the end of World War II enables us to identify some specific influences that helped these physicians see and report what nowadays are considered the defining clinical features of AN. For example, three factors explain why the French were so advanced in understanding AN and who within each national tradition was most predisposed to describe AN. These factors are: (a) a belief in the value of considering in detail the symptoms and history of each patient; (b) attention to psychological aspects of the patient and her subjective experience; and (c) an interest in nutrition. These three factors practically exclude British doctors and favor French psychiatrists, and, to a far lesser degree, German doctors, especially of internal medicine. Some doctors who did meet these criteria nevertheless did not publish case reports in which we can identify AN. They were either not interested in the neuroses or did not work in hospitals, in which it was most probable to see anorexic patients (for more detail see Habermas, 1991). In addition, as documented in Table 2.1, Charcot's discovery of weight phobia in the early 1880s paved the way for other psychiatrists working at the Salpêtrière to author most of the early case reports that described weight phobia, like Féré (1892), Wallet (1892), Brissaud and Souques (1894), and Janet (1898).

Both the opinions of influential teachers and basic convictions about the nature of illness have strongly influenced how AN has been conceptualized and described up to this day. The German doctor Simmonds described an endocrinological disorder in 1916, which for the next three decades dominated the interpretation of unexplained underweight. A preference for somatic explanations in medicine reinforced this trend. Surprisingly, those medical doctors who did believe in the role of psychological factors in disease, that is, proponents of psychosomatic medicine in the 1930s to 1950s, also tended to overlook the voluntary nature of fasting in AN, and instead attempted to explain weight loss with physical factors. A detailed analysis of German

publications on AN between 1918 and 1945 (Habermas, 1994, chap. 9) reveals that in addition to the misapplication of the concept of Simmond's disease, it was actually a belief in a "holistic medicine" that hindered doctors' acknowledgement of the psychological nature of AN. They chose AN as a prime example of how psychological factors could influence the body and cause disease *without* voluntary actions directly affecting the body. The main proponent of psychosomatic medicine, von Bergmann, and experts on the psychoanalysis of psychosomatic disorders all insisted on the endocrinological nature of AN. Only Viktor von Weizsäcker, who pursued an anthropological conception of medicine, understood the primarily psychological nature of AN and of intentional fasting as the cause of weight loss. An analysis of the editorial policies for accepting or rejecting manuscripts by the U.S. journal *Psychosomatic Medicine* demonstrates that this was not an isolated German phenomenon of the 1930s. Rather, the aim to belong to the medical mainstream by being as scientific as possible led to a focus on the somatic aspects of AN in the 1960s and 1970s (Mizrachi, 2002).

Finally, in the case of Nazi Germany, political influences played a role in isolating the medical literature from the international discussion. Furthermore, quasi-intentional self-destruction could be dangerous in a Nazi Germany that systematically murdered psychiatric and handicapped patients, which made it by far safer for patients to have their AN treated as a somatic illness.

Primary and Secondary Historical Influences on the Emergence and Form of Modern Eating Disorders

Primary Historical Influences

Modern AN must have emerged some time in the middle of the 19th century, while the modern form of bulimia nervosa (BN) with weight-concerns emerged over half a century later (Habermas, 1989, 1992a; see also Chapter 3). AN increased drastically in the 1960s and 1970s, whereas the increase in BN occurred after its medical definition in the 1980s and 1990s. Several historical developments have typically been seen as responsible for the emergence of and increase in these modern EDs. The most popular explanation is the female body ideal, which became thinner in the 20th century, especially in the 1960s. However, there have been other periods in history in which slimness was the moral or fashionable ideal. Consequently, other factors may have played a role (Gordon, 1990; Habermas, 1990). These include:

- The absence of famines in Europe, beginning about 1850, which rendered fasting a way to distinguish oneself from others.
- The de-ritualization and individualization of eating. Norms regarding eating began to shift in the late 19th century from regulating the form and amount of eating in social situations to prescribing long-term health and aesthetic effects for the individual body.
- The dominant cultural technique for adapting one's body to the socially prescribed form shifted in the second half of the 19th century from using external devices temporarily applied to the body (e.g., a corset) to manipulate the person's social appearance, to actions aiming at long-term internal modifications of the naked body (e.g., exercising and dieting for weight control).

The latter two changes can be conceptualized in terms of an extension of the theory of the process of civilization. Elias (1939/1982) described a civilizing process between 1600 and

1900 that was marked by an increase in self-control of impulses and emotions for the benefit of foreseeing the long-term effects of one's actions. Elias conceived this in terms of the diffusion of courteous manners that were then internalized so that violations of manners evoked shame. In the course of the 19th century, self-control was extended to the private satisfaction of sexual bodily needs and aggressive impulses. As Wouters (2007) points out, the increase in self-restriction described by Elias changed into a process of informalization of affect controls in the course of the 20th century. This loosening of manners and of social codes of emotional expression, conduct, and status liberalized especially sexual but also food-related behavioral standards, only to subject them to even stricter norms regulating their long-term outcome. This general societal development describes historical changes in cultural techniques for shaping and disciplining the body as well as for eating.

Elias explained the civilizing process in terms of the necessity to increasingly foresee results of one's actions on others due to the growing interconnectedness of people through trade and communication. Given the relative exclusion of women from the public sphere in the West, in the 19th century their integration into societal systems of communication via schooling, work, and mobility accelerated more than that of men, putting additional pressure on them to acquire cultural techniques of affect control to be able to participate in these impersonal systems of communication (Habermas, 1990). Learning how to diet so as to control one's body weight can be understood as an exemplary socializing practice in the transition to adulthood, training women to look ahead for the consequences of their actions. It is probably no coincidence that in decades of women's emancipation ideals of female appearance became slimmer and allowed less external aids, as was the case in the 1920s and the 1960s.

But AN is not identical to normative dieting; it is an exaggeration, a caricature. Apparently, the normative adolescent socializing practice of dieting offers a totalizing defense against the threats of puberty, which come from within in the form of sexual urges and from without in the form of (male) objectification of girls' bodies (Selvini Palazzoli, 1963/1974). AN is even more effective than early adolescent asceticism (Anna Freud, 1936) in defending against all sensual pleasure, because it establishes an emaciated body that physically is less capable of experiencing sensual pleasures (Bruch, 1973; Crisp, 1980).

Secondary Influences on the Psychiatric Conception of an Eating Disorder

Why did BN emerge later than AN? Sociologically, BN can be seen as a failure to internalize the normative practice of dieting. In this regard BN can be interpreted as an ethnic disorder as conceptualized by George Devereux (1956/2000; see also Gordon, 1990), because it (a) focuses on culturally central preoccupations; (b) uses important cultural techniques for defensive purposes; (c) negatively defines central social norms, in this case the norm of controlling one's body weight without impulsive breakthroughs; and (d) serves as a cultural model of misconduct (Linton, 1936), that is, as a model for how to be mentally ill in a culturally accepted way. Thus, I suggest that BN was introduced in Western societies successfully as a new psychiatric disorder once the cultural technique of dieting for weight control had been sufficiently established to expect the majority of female adolescents to comply with it (Habermas, 1994). Consequently, BN is a disorder that from its beginning also relied on the mechanism of imitation (Gordon, 1990). In the most straightforward sense this involved imitating the technique of self-induced vomiting from others to compensate for impulsive binge eating (Habermas, 1992c).

Anorexia nervosa, in contrast, was for a long time "invented" individually anew by each anorexic adolescent. According to Bruch (1973), AN offers, or at least historically offered, the psychological reward of uniqueness and superiority. Only when the concept of AN became known among adolescents could they start imitating this somewhat glamorized disease. For this historical change in the mechanisms of AN, Bruch (1973) invented the term "Me-too-anorexics." However, I believe that the main thrust of the imitation of AN produced BN rather than AN. Possibly only in the past decade did AN become a disorder in which imitation also played a homogenizing role by way of pro-anorexia ("pro-ana") websites (see Chapter 29) and the socializing effect of specialized eating disorder units (Vandereycken, 2011).

Conclusions and Future Directions

The history of AN has several lessons to offer. It shows how malleable conceptions of a psychiatric disease are due to professionals' major convictions about the nature of mental disorders. In the case of AN, ignorance of patients' subjective point of view and a naturalistic somatic prejudice obscured the understanding and adequate recording of AN in the past, and these factors continue to trouble the field to this day (Strober & Johnson, 2012). This shows in proposals to drop weight phobia as a diagnostic criterion, because it relies on patients' subjectivity and on psychological, but not somatic criteria. A similar preference for somatic criteria for defining psychological disorders must have motivated Keel and Klump (2003) to suggest that AN was a historically invariant disease once it is defined without reference to psychological criteria, only to conclude that genetic factors play a more dominant role than social influences in AN. Given that genes interact with environmental and social conditions (see Chapters 28 & 67), this conclusion reveals an inadequate preconception that psychiatric disorders need to be defined in somatic terms.

In contrast, a methodologically informed approach to the history of EDs that critically takes into account the available historical evidence suggests not only that psychological criteria are indispensable for defining the specific disorder of AN, but also that AN as well as BN are historically new syndromes. This, of course, in no way precludes genetic dispositions and somatic maintaining and aggravating conditions in AN.

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3

The History of Bulimia Nervosa

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In his important and definitive paper on the history of bulimia nervosa (BN), Gerald Russell (1997) commented that pursuing that history is roughly comparable to chasing a will-o'-the-wisp. The reasons for this are at least in part semantic. The term "bulimia" has one set of meanings (ravenous hunger, eating to extreme excess) that reaches back at least to the Romans, while "bulimia nervosa," which refers to a syndrome of binge eating and purging that is organized around a concern with weight and shape, is something that seems peculiarly modern and gender-linked. By way of contrast, the earlier historical cases of bulimia appear to be equally or randomly associated with gender.

This chapter will first briefly summarize the accounts of BN that predated the first descriptions of the modern syndrome, which began to emerge in the 1960s. It will stress the rather inchoate nature of early descriptions of the syndrome, as well as the prolific number of terms that were used to characterize it. The first modern descriptions of the syndrome, which appeared in continental Europe in the 1960s, will then be addressed. Following this, a detailed discussion of the discovery of BN in the United States and UK in the 1970s will be presented, along with the recognition and documentation by key observers that the prevalence of the syndrome had increased, that it was predominant in females, and that its incidence was a hidden though perhaps epidemic phenomenon. Finally, the chapter will take note of developments in treatment of the bulimic syndrome since its recognition as an independent diagnosis, as well as recent trends in epidemiology and conceptions of etiology.

Early History

In contrast to anorexia nervosa (AN), which was clearly defined as a medical and psychiatric entity in the 1870s, BN was not formally named until 1979 in a classic paper by Gerald Russell, and then in the *DSM-III* (American Psychiatric Association, 1980). But, as previously noted, the term "bulimia" is in fact not a new one, nor was Russell the first to

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describe the syndrome in modern times. The term may have its origin in the Hebrew word *boolmot*—and in the Greek equivalent *bulimy*—described in the Talmud as "ravenous hunger" (Kaplan & Garfinkel, 1984).

The most familiar and widely discussed ancient precursor of bulimia was the well-known gorging and vomiting behavior of the Roman aristocracy, most dramatically portrayed in Seneca's famous description: "They eat in order to vomit and vomit in order to eat." In perhaps the only effort to deal with Roman "bulimia" analytically, Crichton (1996) investigated historical accounts of the behaviors of the Roman emperors Claudius and Vitellus. Both men drank excessively and at least one, Vitellus, engaged in morbid gluttony and was obese. Given the absence of any reference to concerns with shape and weight in these men, Crichton was reluctant to attribute BN to the emperors and suggested rather that their behaviors were an extreme manifestation of a class-based indulgence in overeating. However, he did feel that the habitual nature of the behavior, and particularly the vomiting, justified seeing it as some sort of precursor of the modern disorder.

Parry-Jones and Parry-Jones (1991) reviewed a wide range of archival literature in Europe and the UK and cited 12 cases from the 15th to the beginning of the 20th century. Many terms were used to describe the syndrome of ravenous appetite and eating, including *bolismo*, *boulime*, *dissolute appetita*, *canina fames*, and *bovina*, among others. However, perhaps because the level of clinical description did not emphasize psychiatric features and possibly because these cases reflected something else altogether, few if any of these cases are recognizable as the modern syndrome of BN and most were probably of infectious origin.

In a wide-ranging historical article first published in German in 1976, the psychiatrist Horst-Ulfert Ziolko (1996) described the long history of a syndrome of ravenous appetite. Ziolko was convinced that these instances were indeed precursors of the modern syndrome, which he had renamed (prior to the general adoption of Russell's terminology several years later) "hyperorexia nervosa." In making this argument, Ziolko emphasized that the core of the syndrome was the appetitive "binge-eating," to use more modern terminology. Unlike the modern disorder, these older case histories did not reflect a largely female syndrome, nor did they place the overeating in a context of food restriction or the pursuit of thinness. Ziolko doubted that the latter was anything but the superficial clothing in which an ancient syndrome was dressed. That is an interesting but highly debatable argument. As Russell (1997) pointed out, BN is understood as a pattern of binge eating and compensation (typically purging) that is driven by a fear of weight gain that occurs mostly in women and rarely in men. To Russell, the fear of weight gain and skewed sex ratio are not superficial characteristics, but central to the modern understanding of the disorder. It is ravenous appetite and overeating, and not the bingeing-purging cycle driven by a fear of fatness, that is typical of the older historical instances.

As Russell (1997), Casper (1983), and Keel and Klump (2003) have cogently argued, BN is most likely a modern disorder and one that did not begin to appear to clinicians in significant numbers until the 1960s or 1970s. This is in sharp contrast to the kindred disorder AN (see Chapter 2), and it is of great interest that descriptions of bulimic episodes and weight concerns were generally absent (with one minor exception) in the seminal writings of Gull and Lasègue about AN in the 1870s. In fact, with a few exceptions, there was little discussion of overeating and purging in AN prior to the 1960s. One notable exception was the case of Ellen West in the 1920s, a pseudonym for a patient whose lengthy and literary case history of AN was published by Binswanger (1944/1958) and provided a detailed description of bulimic episodes, along with a characteristic weight obsession. Another case, noted by Russell (1997), was Pierre Janet's 1903 report on "Nadia," a young

woman who suffered from weight obsessions and various dimensions of disordered eating. Noted psychiatrist Albert Stunkard (1990) also resurrected some work from 1932 by a German analyst, Wulff, who wrote about a number of cases of binge eating that had an "addictive character." Such behaviors were also noted by Fenichel (1945), and a very dramatic case of binge-eating disorder (the story of Laura) was published by the analyst Robert Lindner (1954).

The absence of references to the bulimic syndrome in the cases of AN described by Gull and Lasègue is a notable omission. Tilman Habermas (1989) speculated that weight concerns may have been hidden by 19th century patients in their clinical encounters with physicians, out of motives such as shame, modesty, and the like. Russell (1997), however, is highly skeptical about Habermas' argument and doubts that clinicians as perceptive and thorough as Gull and Lasègue would have missed such a phenomenon had it occurred.

Interestingly, Casper (1983) determined that, when cases of AN began to appear in the American psychoanalytic literature in the 1940s, weight concerns were rarely mentioned. Features of the bulimic syndrome remained generally absent, but there were some exceptions. For example, instances of binge eating were noted by the Harvard psychiatrist John Nemiah (1950) in his report on 30 cases of AN. In general, the appearance of binge eating and purging in people who did not show the drastic weight loss of AN began about the same time that the phenomenon of bulimic symptoms appeared with increasing frequency in clinical accounts of AN. This adds complexity and some confusion to the historical picture, and it took some 20 years, beginning in 1960 and culminating in Russell's paper in 1979, for the distinction between the syndrome of bulimia in AN and the independent syndrome of BN to be clarified.

From 1960 to 1972: First Descriptions of the Modern BN Syndrome

It was not until the 1960s that bulimia began to be described as a distinctive syndrome that deserved clinical attention in its own right. In the first full-length 20th century monograph on AN, U.S. psychiatrists Bliss and Branch (1960) mentioned a number of cases of binge eating and vomiting among their many patients. In a comprehensive review of sources little familiar to English-speaking readers, Vandereycken (1994) took note of a paper by Paul Abely and colleagues (1963) presented to the French Société Medico-Psychologique in 1963 that described a syndrome of "pathological hyperorexias" in females (in 3 years, 15 cases had been noted). Most of these women had been attempting to control weight with appetite suppressants, which had become commonly used at the time. Abely et al. did not emphasize vomiting or other efforts to compensate for the overeating, but they did note that these "food addictions" had developed in young women paradoxically preoccupied with their weight.

Later in the decade, the aforementioned German psychiatrist Ziolko (1966, 1967, as cited in Vandereycken, 1994) published two short papers focused on one patient that described a pattern that to Ziolko's thinking was essentially the opposite of AN. Ten years later, Ziolko (1976, p. 525) described 70 cases of what appeared to be an unmistakable syndrome:

> This hyperorexia nervosa is, according to our experiences so far, found exclusively in young girls and women; it generally starts during postpuberty, and is characterized by (episodically or constantly) increased, greedy, compulsive food intake. It is mostly attended by a failing feeling of satiation; there often exists vomiting and mostly amenorrhea. Frequently an increase in weight occurs.

Ziolko also noted the frequency of depression and the negative impact that the syndrome had on social relationships. The paper did not appear in English until almost 20 years later (Ziolko, 1994). As Vandereycken (1994) pointed out, had Ziolko originally published his work in English, it is possible that we may have been using his terminology today.

A remarkable publication (although little noticed because it was published in Spanish in a Chilean Journal in 1972) was that of Otto Zoerr-Degers (1994). This work offered the first complete description of what we now call BN. Based on a small number of in-depth case studies, the author argued cogently that what we now call BN must be recognized a distinct psychiatric disorder and carefully distinguished it from other kindred disorders. Zoerr-Degers emphasized that, while the syndrome was most closely tied to AN, the normal weight of the patients as well as the centrality of overeating at the start of the syndrome made it something unique. Furthermore, he made the unusual point that the body image distortions in BN, while centering on thinness, also included in many instances a desire for greater fullness in certain parts of the body. There is a general perception that the body is misshapen and therefore unattractive. This makes for a close kinship with what we now call body dysmorphic disorder (BDD; American Psychiatric Association, 2013). Finally, the reason for use of the term "oral perversion" in the title was that Doerr-Zegers compared the behavior with food in bulimics to sexual behavior in the perversions, in which an appetitive behavior is isolated from its interpersonal context and magnified to the point of all-consuming obsession.

From 1972 to 1980: The Emergence of BN as an Independent Syndrome

The decade of 1972 to 1982 was a critical one in the emergence of BN.[1] First, the syndrome was well described in the English-speaking countries by two clinicians working in very different contexts, the U.S. psychologist Marlene Boskind-White (then Boskind-Lodahl) and the British psychiatrist Gerald Russell. The syndrome was also clearly recognized by European writers, but because their works were published in German and French, respectively, they were little known to an English-speaking audience (Celerier, 1977, cited in Vandereycken, 1994; Igoin, 1979; Ziolko, 1994). Second, the new syndrome was shown to have an extensive, although hidden, prevalence, to the extent that it was often described as an epidemic. These discoveries were largely a result of research conducted by the U.S. psychologist Craig Johnson and the Oxford-based psychiatrist Christopher Fairburn. Third, the rudiments of treatment methods were explored by Boskind-White and Fairburn, with the first experimenting with group and psychoeducational approaches, and the second developing elements of a cognitive-behavioral methodology that was later to become the gold standard of treatment (see Chapters 18 & 56). Fourth, the syndrome was named independently of AN in the *DSM-III* (American Psychiatric Association, 1980), although with the designation "bulimia," not "bulimia nervosa." And finally, the early 1980s saw some initial efforts at using medications in the treatment of bulimia, with successful initial trials of antidepressant reported by Pope and Hudson at McLean Hospital in Massachusetts and Timothy Walsh and colleagues at Columbia University.

[1]In the following discussions of the work of Boskind-White, Russell, Johnson, and Fairburn, considerable use is made of material from interviews that the author conducted with these clinicians/researchers on the dates indicated. The first instance of such material is noted as a personal communication in each case.

The Contribution of Marlene Boskind-White (Boskind-Lodahl)

It is often taken for granted that Russell's 1979 paper, which offered a systematic and detailed description of what he argued was a disorder deserving of a distinct diagnosis, was the definitive first formulation of BN. Nevertheless, it should be acknowledged that the first publication in English on BN came from Boskind-White (Boskind-Lodahl, 1976), then an obscure graduate student in counseling psychology at Cornell University. Boskind-White's discussion was cast to a considerable extent in terms of feminist psychology, and her paper was published in a journal of feminist studies (*Signs*) little known in the psychiatric community. Nevertheless, her paper ultimately became widely cited and was influential. In contrast, by the time Russell published his paper in a mainstream psychiatric journal, he was already well established as an expert on AN, so his paper immediately commanded a great deal of professional attention.

It was during the early stages of her training at Cornell University to become a counseling psychologist that Boskind-White first encountered a woman with the syndrome of bulimia. She was impressed that this female undergraduate had the body image concerns and anxiety about gaining weight that were typical of AN, but was of normal weight and appearance. Similar to what happened to many observers in the 1970s, within weeks after seeing this first patient, she encountered a second female student with the same essential problem, and then another. She decided to post a notice in a college newspaper asking those who were seeking help with similar problems to come forward. She was stunned to find that well over 100 students within this one college campus responded. Boskind-White was struck by the isolation of these students, each of whom tended to think that her struggles with food were unique.

Boskind-White searched the literature and found very little that was of direct use in understanding these women, although she was impressed by the clinical writings of Hilde Bruch on such issues as the tremendous deficits in self-esteem suffered by anorexic patients, and by the studies of voluntary starvation done at the University of Minnesota, which showed that many presumably normal men had become bulimic after a period of stringent food deprivation (Keys, Brozek, Henschel, Mickelson, & Taylor, 1950). These findings convinced her that there were both psychological and psychobiological roots to the problems of her clients. However, she found singularly unhelpful the traditional psychoanalytic approaches to AN, in particular what she characterized as "bizarre" theories of the desire for oral impregnation as well as the notion that AN represented a rejection of femininity (Waller, Kaufman, & Deutsch, 1940). Specifically, she felt strongly that quite the opposite was true for her bulimic patients, who showed an exaggerated yearning to fulfill stereotypical feminine ideals of appearance and to be perfect in the eyes of men.

These ideas were central in the feminist formulations that became the crux of her first paper (Boskind-Lodahl, 1976). Boskind-White's personal experiences had led her to become deeply involved in the feminist upheaval of the late 1960s and early 1970s, and exposure to the ideas of Betty Friedan (1963) had been a revelation to her. At the center of the paper published in *Signs* was the idea that the psychological problems driving women to adopt bulimic behavior patterns could not be understood outside of the context of the socialization of women to a dependent and subservient role, one in which the status of men was glorified and seen as the standard of respect and power. Thus the low self-esteem and sensitivity to rejection that was so central in the psychology of bulimic women were exaggerations of the typical consequences of female socialization. The profound sense of disempowerment that affected eating-disordered women was merely the far end of a normative experience for women in contemporary culture (see Chapters 19, 27, 43, & 58). While family experiences

were undoubtedly important for individual patients, Boskind-White argued that the cultural structure of sex roles was paramount and alone could account for why this new eating disorder (ED) had become so common.

Boskind-White initially experimented with an eclectic experiential group therapy and continued to do so throughout her clinical career, during which she published a widely read book that went through several editions (Boskind-White & White, 1983, 2000). She published one preliminary study that evaluated the effectiveness of the approach, and commented in interpreting the results that the disorder was more intractable to treatment than she had initially thought (White & Boskind-White, 1981). Nevertheless, throughout her career she maintained the perspective that BN, which she had called bulimarexia, should not be understood as a disease but rather as a self-damaging habit pattern that people acquired to resolve socially induced feelings of low self-esteem. She acknowledged that AN, with which she gained experience in later clinical work, needed to be understood quite differently, specifically because of the pathological implications of low body weight. Interestingly, her view of BN as a mild form of AN contrasted quite sharply with the initial formulations of Gerald Russell, whose first paper on BN described the syndrome as an *ominous* variant of AN (Russell, 1979). However, in a 25-year retrospective on BN, Russell (2004) conceded that his early view of the severity of BN was incorrect, as the disorder was far more responsive to treatment than AN.

The Work of Gerald Russell

By the time Russell came across BN in the 1970s, he had already become a leading figure in the world on the treatment of AN. His training at the Maudsley hospital in London was directed by the esteemed psychiatrist Aubrey Lewis, who embraced phenomenology, biology, and social factors in the understanding of psychiatric disorders. In the 1960s Russell conducted painstaking laboratory research on the metabolism of AN patients and became an expert on the hospital treatment of anorexic patients (Russell, 1970), as well as their variable outcome following weight restoration (Russell, 1977).

Russell, like many others, found the intractable vomiting that some anorexic patients manifested a very puzzling and troubling phenomenon. In 1972 he saw a patient who exhibited these symptoms but who was of normal body weight. Over the next few years, he continued to see more such patients, first at the rate of one or two a year, but in accelerating numbers in the second half of the 1970s. Other than their normal weight, he also noticed some significant differences from AN patients: all had normal menstruation and were sexually active. But what was the cause of the vomiting? Russell was almost embarrassed to say in retrospect that it came as something of a revelation to him when a patient told him with some hesitation that the reason that she vomited was the huge amount of food that she had ingested and her fears about the threat that her eating binge posed for her weight. As Russell commented, all this may seem obvious today, but suddenly for him the essence of BN had been revealed: a pattern of binge eating and vomiting that is centered around a patient's anxiety about weight gain or "fear of fatness." These three components together defined the new syndrome.

Russell felt that he had seen enough cases – 30 (28 females, 2 males) to be exact – to formalize his findings in a publication that described a new syndrome (Russell, 1979). He knew that the binge eating and vomiting pattern was akin to AN, but yet it was distinct, mainly because of the normal body weight, the clear-cut physiological difference of the presence of menstruation, and the presence of a normal sexual life. So, Russell decided that the syndrome could be described as a "variant" of AN, distinct but related. However, because of the apparent

intractability of the symptoms (Fairburn's efforts with cognitive-behavioral therapy (CBT) were just being formulated – see below), especially the vomiting, Russell's exact subtitle was "An Ominous Variant of Anorexia Nervosa."

Russell's 1979 paper has been described as a "classic" (Frampton, 2013; Palmer, 1998). The tone and discourse contrasted sharply with Boskind-Lodahl's psychodynamic, developmental, and feminist explanation of the symptoms. Russell's paper achieves a careful, detailed, and nuanced description of the symptoms, the typical course of the illness, the variations in weight histories among patients, and so forth. For example, Russell devoted a full page and a half to variations in the behavior and experience of self-induced vomiting among his patients. He also vividly described binge eating and its variations, along with the intense preoccupation with food that such behavior triggers, both before and after episodes. One factor Russell emphasized was the profound sense of shame felt by patients about both their eating behavior and their efforts to undo it, something that makes the persistence and intractability of the syndrome all the more remarkable.

Russell reported that some 80% of the patients in his sample had either experienced an earlier episode of outright AN or milder weight loss without amenorrhea (Russell dubbed the latter syndrome a "cryptic episode" of AN). These patterns, coupled with the frequent occurrence of bulimic behavior in AN, gave Russell further confirmation of the kinship of the syndrome he was describing with AN. Russell also discussed in detail the depression experienced by the great majority of BN patients. They typically experienced feelings of gloom, suicidal thoughts, concentration impairments, and significant irritability. The outward appearance of BN patients, which was quite normal, often masked their severe feelings of despair.

The Strange Tale of the *DSM-III* Diagnosis

Through Boskind-Lodahl and Russell, as well as predecessors such as Ziolko and Doerr-Zegers, the diagnosis of BN ultimately crystallized. Vandereycken (1994) characterized this rather complex history as the transition from a growing awareness of a symptom in the 1960s (binge eating and purging) to a syndrome by the end of the 1970s. Clinicians in France in the 1990s also began to describe more fully an independent bulimic syndrome, one that was related to AN and yet distinct from it (Celerier, 1977, cited in Vandereycken, 1994). Igoin (1979), in particular, made the astute observation that the syndrome of bulimia was more common than AN, and yet less common than obesity.

Given that the *DSM-III* was published in 1980, the timing seemed perfect for the appearance of a separate diagnosis for BN. However, there was an interesting twist to the story, one that seems unexpected in retrospect. The *DSM-III* diagnosis of "bulimia" – not "bulimia nervosa" – identified a syndrome of binge eating and "compensation" (it was noted that this was most often self-induced vomiting), but made no reference to the overinvestment in body weight and shape. These psychological components, which were so central in the clinical descriptions made by Boskind-Lodahl and Russell, were omitted, and entered the diagnostic criteria only when a diagnosis of "bulimia nervosa" appeared in *DSM-III-R* (American Psychiatric Association, 1987).

Exactly why this happened is unclear, but there is a plausible explanation. In the late 1950s, the well-known U.S. psychiatrist and obesity expert Albert Stunkard (1959) had described a syndrome of binge eating, as well as a related "night-eating syndrome," that was typically, although not necessarily, found in obese patients (see Chapter 4). In the 1970s others took up the subject of binge eating, particularly those with a neurological orientation who were attempting to relieve the syndrome with medication. For example, Green and Rau (1974)

described three types of patients in whom binge eating occurs: obese, anorexic, and those of normal weight. The latter group struggled against powerful, compulsive desires to eat, which sometimes broke though in the form of eating binges, typically followed by efforts totally to avoid food. However, vomiting was not described by Green and Rau. The authors suspected that binge eating was a seizure-like behavior – they garnered some evidence of spiking from EEGs – and treated their patients with the anticonvulsant phenytoin.

Three years later a second publication, co-authored by Stunkard, appeared on the use of phenytoin to treat binge eating in a sample of 19 patients (Wermuth, Davis, Hollister, & Stunkard, 1977). While treatment effects were modest, the important element of this paper was its definition of a binge-eating syndrome that almost literally prefigured the diagnostic criteria for bulimia in the *DSM-III* (American Psychiatric Association, 1980):

1 Impulsive, unpredictable, episodic, uncontrolled, and rapid ingestion of large quantities of food over a relatively short period of time.
2 Termination of the episode only when a point of physical discomfort has been reached (e.g., abdominal pain, and feelings of nausea or distention). Self-induced vomiting supported but was not required for diagnosis.
3 Subsequent feelings of guilt, remorse or self-contempt.
4 A frequency of at least one binge per week with no binge-free intervals of longer than 3 weeks in the previous year.

Interestingly, Stunkard participated on the *DSM-III* committee, chaired by psychiatrist Katherine Halmi, that formulated the new diagnosis of bulimia. This may account in large part for the fact that the *DSM-III* (1980) diagnostic criteria are essentially the same as those used by Wermuth et al. (1977). Thus, to reiterate, although by the late 1970s the idea of BN as a distinct diagnosis from AN had clearly reached the "critical mass" to be included in the manual, it was not until the publication of the American Psychiatric Association's *DSM-III-R* in 1987 that the diagnosis of BN entered the lexicon and included as a criterion Russell's and Boskind-White's emphasis on shape and weight concern. That, in turn, was probably a result of both an active debate in the field about the *DSM-III* criteria, as well as growing evidence from Fairburn's studies in the 1980s that dealing with shape and weight concerns via cognitive-behavioral therapy had a significant impact on clinical improvement (Fairburn, 2008; see also Chapter 18). The "shape and weight concern" criterion has remained part of the diagnosis of BN in subsequent versions of the *DSM* (see Chapter 9).

Developments After 1980: Emergence of the Awareness that BN was "Epidemic"

Following publication of *DSM-III* (American Psychiatric Association, 1980) and widespread discussion of BN in the popular press, self-referrals for treatment increased sharply at centers in North America (Soundy, Lucas, Suman, & Melton, 1995). This took many observers by surprise, and the notion began to take hold that a silent "epidemic" of EDs, but mainly BN, had been proliferating in the community for an unknown amount of time. The term "epidemic" has been faulted for its sensationalist overtones, but if we use it in its conventional epidemiological sense of "a greater prevalence than expected" and omit more specific attributes

such as "contagion," it appears to be applicable to the findings about BN. It took two researchers, Craig Johnson (then of Chicago) and Christopher Fairburn (of Oxford), originally unknown to each other, to conduct studies that validated the previously unknown or at least undemonstrated extent of BN. And this work turned out to be critical to the emergence of a scientific field of EDs, which stemmed from a series of international conferences that began in the early 1980s.

While completing a postdoctoral fellowship at Yale University in the late 1970s, Johnson had been impressed when one of his supervisors, the widely known psychiatrist Theodore Lidz, observed that the small number of patients being admitted with symptoms such as binge eating, vomiting, and self-mutilation might represent a new syndrome. When Johnson accepted a position as the head of a new research center for AN at the Michael Reese Hospital in Chicago, he and some enthusiastic graduate students began exploring the prevalence of what we now know as BN by interviewing people living in the North Shore area of Chicago. Fortuitously, three national women's magazines heard about the study and wrote articles about the "binge-purge" syndrome, offering (without his permission) Johnson's phone number in Chicago. Within 3 weeks he received over 5,000 phone calls. The follow-up questionnaires to these constituted the first large-scale descriptive study of BN in the community in the United States (Johnson, Stuckey, Lewis, & Schwartz, 1982). The results showed a high degree of consistency, with most respondents reporting shame, secrecy, and a sense of social isolation and suffering in silence for a number of years without any treatment.

Johnson's report on his findings at a conference on adolescent psychiatry held at Johns Hopkins University in 1980 was met with considerable skepticism. How could such an unknown condition be so widespread? However, he received some vindication when he presented a similar paper at a conference in Toronto in 1981, which was immediately followed by a presentation from Fairburn of virtually identical results from Oxford, UK. Fairburn had undertaken psychiatric training at the University of Edinburgh, Scotland, between 1975 and 1978 (C. G. Fairburn, personal communication, April, 2006). In 1976 he was asked to see a patient with "anorexia nervosa," but it was apparent from her normal weight and appearance that she did not have AN. Although she remarked that it was amazing that she was able to maintain the weight that she did, given how much she ate, she soon made that understandable when she confessed to vomiting after her eating binges.

Both Fairburn and his supervisors were perplexed by this presentation and could not unearth anything in the literature to account for it. Then the now familiar pattern emerged of seeing an increasing number of patients who binged and purged and yet were of more or less normal weight. Fairburn began to take some exploratory steps to devise a treatment. He noticed that all of his patients were extremely conscious of their weight and desired to reduce it further through imposition of a rigorous set of dietary rules. When they allowed themselves to eat to a point of excess, they had broken the rules and thrown all caution to the winds. They then forced themselves to vomit to counter the excessive intake. A similar pattern of restraint followed by its opposite had been studied and analyzed by C. Peter Herman, Janet Polivy, and others at the University of Toronto under the rubric of the psychology of "restrained eating" (Herman & Mack, 1975). Fairburn concluded that, if the rigid dietary rules of these patients, along with their body image preoccupations, could be modified, then the bulimic pattern could be broken. This was the basis of the cognitive-behavioral treatment (CBT) for which Fairburn became well known and the efficacy of which was demonstrated much later in rigorous treatment trials (see Chapter 56).

Fairburn was interested in epidemiology and was convinced from his experience that there must be thousands of such patients "out there," since his experience had been in a limited geographic area. Unlike Johnson, whose involvement with the media was essentially accidental, Fairburn sought to use media as an avenue for inquiry. A notice in *British Cosmopolitan*, which participated reluctantly because of editorial skepticism about the condition, yielded a thousand replies within a very short time. This became the basis of the first British publication addressing a large sample with BN (Fairburn & Cooper, 1982). Fairburn wanted to broaden the investigation, so he helped create a BBC television program on BN that was screened in 1981. To counter the bias inherent in inquiring about the disorder via women's magazines, the program featured among other things, self-descriptions of the disorder by one female and one male subject (the program also featured Gerald Russell and another well-known UK eating disorder specialist, Hubert Lacey; see Chapter 12). The response was so overwhelming that special methods of mail and telephone management were needed to manage the deluge. The resulting sample was the basis of a study published in *The British Journal of Psychiatry* (Cooper & Fairburn, 1983). Despite the inclusion of a male sufferer in the program, the response continued to be 100% female, thus confirming the impression that, like AN, BN was strongly tied to gender.

Intensive Investigation and Treatment of BN

The new interest in what the *DSM-III* (1980) labeled bulimia, along with the ongoing challenge of AN, spurred the founding in 1982 of the *International Journal of Eating Disorders*, with Craig Johnson as its first editor. Theander (2002) has shown that publications on EDs surged in the 1980s, led by a dramatic increase in publications on bulimia, with a special interest in such areas as epidemiology, comorbidity, and treatment. Meanwhile, Fairburn continued to develop the specifics of a cognitive-behavioral approach to treatment and published a formal manual in 1985. He also began a series of treatment studies, which utilized small samples at first but became multicenter trials in the 1990s (Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000). In general these studies concluded that CBT was an effective method for over 50% of patients. However, there were still some shortcomings, as a certain number of patients dropped out of the trials and a minority of those who completed the trials did not improve significantly. Nevertheless, CBT was adopted by the British National Institute for Clinical Excellence (NICE; now the National Institute for Health and Care Excellence) guidelines as the standard for the treatment of BN. An incidental finding of these studies was that interpersonal psychotherapy (IPT; see Chapter 62), which was originally included as a control, was also an effective treatment.

Meanwhile, in the early 1980s, a totally different approach emerged. Pope and Hudson (1982) at McLean Hospital in Massachusetts published data showing that the tricyclic antidepressant imipramine has significant positive clinical effects on BN. Two years later Pope and Hudson (1984) published a book that made an extensive argument that BN was one expression of an underlying syndrome of biological depression. The basis of this argument was not only the positive response of BN patients to tricyclic antidepressants, but also the extensive comorbidities that BN patients had in common with depressed patients, particularly certain anxiety disorders as well as impulse and addictive disorders (see Chapters 15 & 54). The emergence of the selective serotonin reuptake inhibitor (SSRI) medications in the late 1980s, with their more benign side-effect profile, made them the medication of choice for BN, and one, fluoxetine, became and continues to be the only drug for BN approved by the U.S. Food and Drug Administration (FDA) (see Chapter 59). While the initial enthusiasm for the use of antidepressants was strong, subsequent assessments have not shown them to be as effective as

was first thought. However, antidepressants are still considered part of the therapeutic armamentarium in the treatment of BN, and one study showed that the SSRIs are quite helpful in the treatment of patients who did not improve after psychotherapy (Walsh et al., 2000).

Two significant developments after the 1980s were genetic studies (see Chapter 28) and Fairburn's (2008) continued improvements in the CBT approach that he originated. Genetic studies have revealed a substantial heritability for BN and suggestive evidence for specific chromosomal locations of predisposing genes, although many questions remain about the specific mechanisms of the genetic contribution. As for CBT, the enhanced method (called CBT-E) incorporates the emotional and interpersonal dimensions of the disorder. Fairburn hopes that the general template of this method will have a "transdiagnostic" impact, that is, that it will be applicable for other eating disorders, including AN and EDNOS (see Chapter 11).

Conclusions and Future Directions

Epidemiological studies (Kendler et al., 1991; Lucas & Soundy, 1993) indicate that, of those who reported having had BN, only a few said that this disorder began before the late 1960s. To reiterate, then, unlike AN, which has a long history (see Chapter 2), BN appears to be a recent phenomenon. In their thorough review of historical research, Keel and Klump (2003) suggested that BN is more strongly influenced by cultural factors than is AN. One such factor is intensification of the idealization of thinness for women that began in the 1960s (Garner & Garfinkel, 1980). Another is the increasing availability of low-cost, carbohydrate-rich foods that began after World War II (Casper, 1983) and intensified with the subsequent proliferation of industrially processed, highly calorie-dense foods—seen as one cause of the steep increase in overweight in the general population (Kessler, 2010).

Even if there is some truth in accounts such as Ziolko's (1996), which argue that bulimia has an ancient history, any explanation of the modern phenomenon of BN must take into account its gender disparity. Although some community studies such as the National Comorbidity Survey (Hudson, Hiripi, Pope, & Kessler, 2007) have suggested a larger than expected male prevalence, the fact remains that the majority of individuals with BN are female. While cultural factors such as the differential idealization of thinness and changing role expectations among women have been invoked to explain this disparity (Gordon, 2000), it needs to be emphasized that the relatively small percentage of the general population who develop BN signals that these forces trigger BN only in a subgroup, perhaps from a combination of genetic, hormonal, and environmental vulnerabilities (Klump et al., 2012; Lehoux & Howe, 2007).

Meanwhile, there has actually been some evidence for a decline in the incidence of BN, at least in a small number of studies conducted between 2000 and 2010 (Keel, Heatherton, Dorer, Joiner, & Zalta, 2005). Should subsequent research confirm that BN declined after 2000, despite its surge in the 1970s, this will have to be accounted for. Nevertheless, the findings from the National Comorbidity Survey (Hudson et al., 2007) indicate an overall lifetime prevalence of 1% among post-16-year-olds in the United States, a substantial figure. There is also evidence that BN is emerging more frequently in younger adolescents than previously (Favaro, Caregaro, Tenconi, Bosello, & Santonastaso, 2009), a trend of considerable concern. Clearly BN remains a substantial public health problem, and the advances in treatment noted in the present chapter represent important beginnings. More

research is needed, particularly on hitherto little-explored areas such as how particular foods and dieting may affect the physiology of vulnerable individuals. And, as with all EDs, efforts at prevention are needed, as is work on early intervention to prevent chronicity (see Chapters 41 to 49).

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4

History of BED and ED-NOS

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The understanding of eating disorders (EDs) has advanced tremendously over the past four decades. The evolution of this expanding knowledge has been so rapid that the *Diagnostic and Statistical Manual of Mental Disorders* (e.g., *DSM-IV-TR*; American Psychiatric Association, 2000) has necessarily lagged in accurately capturing the clinical presentations of the many individuals who present for ED treatment but do not fit clear criteria for anorexia nervosa (AN) or bulimia nervosa (BN). As a consequence, an increasing number of individuals with markedly different clinical presentations have fallen into a residual ED category, which has challenged clinical communication, treatment planning, and basic research (Thomas, Vartanian, & Brownell, 2009).

What follows is the history of this complex, heterogeneous diagnosis and how it has undergone important changes with each revision of the *DSM*, reflecting the field's increased appreciation of diagnostic diversity. Before we begin, meet Julie and John and follow the trajectory of their clinical diagnoses with each *DSM* iteration. As you read, keep in mind the following questions: Which specific diagnostic criteria do each of these individuals meet? What are their overarching *DSM* diagnoses? How might our classification system facilitate their clinical detection and optimize their treatment?

Two Cases

Julie

Julie is a 26-year-old woman who presents to her local outpatient psychiatric clinic with vague concerns that she might have an eating disorder (ED). She explains that, despite feeling as though her life is "objectively great" (she has a prominent teaching position, a supportive family, and a loving fiancé), she feels "miserable and out of control" around food. She indicates that she has become increasingly preoccupied with her weight,

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declaring that she "needs to lose 5 pounds in order to fit into the perfect wedding dress." She reports that what were initially "just passing thoughts" soon devolved into obsessive ruminations that plagued her "every minute of every day."

Because Julie fears gaining weight and returning to her highest weight of 160 pounds (after she had gained the "freshman 15" in college), she attempts to do whatever she can to control it. To that end, she restricts her caloric intake to approximately 700–1,000 calories per day, and she exercises (i.e., running, elliptical machine, lifting weights) up to 3 hours per day. She explains that her compulsive physical activity is geared toward controlling her weight in general, rather than compensating for any specific eating episode. She further laments that, despite her best efforts, she has maintained an average BMI (23.9 kg/m^2). Weighing 148 pounds (67 kg) at a height of 5′6″ (1.68 m), Julie considers herself to be "grossly obese." She also worries that her weight loss efforts are constantly thwarted by her subjective binge episodes, which occur once every 2 weeks and consist of rapidly consuming "three spoonfuls of ice cream and three cookies" while feeling like she cannot stop eating. These episodes are sometimes (though not always) followed by self-induced vomiting. She denies using ipecac, laxatives, diuretics, or diet pills.

Julie fears that her relationships with family and friends have "deteriorated" because of the time she spends worrying about her weight, and she also notices that her performance at work has suffered because she is "constantly" distracted by negative body image thoughts. Her eyes fill with tears of regret as she relates that her symptoms have "ruined [her] life." She becomes even more despondent when she intimates that it is "ridiculous" that she is consumed by these symptoms after she has read on various clinical websites that she does not meet full diagnostic criteria for anorexia nervosa (AN) or bulimia nervosa (BN), tearfully relating that "[she] can't even be a real anorexic." She continues to menstruate notwithstanding her low calorie intake, and hopes to get pregnant after her wedding, though she worries about the amount of weight she will gain if she conceives. She is seeking treatment so that she can "stop worrying about my weight" and "just enjoy my wedding day."

John

John is a 65-year-old married male with type II diabetes who comes to an outpatient ED specialty clinic hoping to "gain control" over his binge eating, fearing that this behavior may be threatening his cardiovascular health. He explains that he is quite capable of making "radical" changes in his life, citing his 15-year sobriety from alcohol as an example of his strong will and determination. Specifically, he reports that for the past 10 months he has been binge eating most days of the week—up to 3 times per day. During each of his binges, which occur even when he is not hungry, he rapidly ingests anywhere from 3,000 to 6,000 calories of high-carbohydrate snacks, the equivalent of 1 to 2 dozen glazed donuts. Because he reports not getting full and feeling "completely out of control," he is limited only by his supply of available food.

John notes feeling "repulsed and guilty" after each binge and has to inject increased boluses of insulin in order to control his blood sugar. He reports that he does not engage in any compensatory behavior following his binges, denying excessive exercise, self-induced vomiting, ipecac, laxatives, or diuretics. He says he is "satisfied" with his current

weight of 175 pounds (79.4 kg) (representing a slightly overweight BMI of 27.4 kg/m^2 at a height of 5′7″ [1.70 m]) and does not restrict his caloric intake in any way. John notes that his life is so consumed with binge eating that he is unable to make time for his friends and family and thus fears that he will lose their support. He presents to treatment so that he can "stop bingeing and hopefully avoid having a heart attack."

Evolution of *DSM* Diagnoses: The Feighner Criteria

In the mid-20th century, psychiatric practice in the United States was largely dominated by the influence of Freudian psychoanalytic theory about unconscious processes. Use of diagnoses was viewed disdainfully by psychiatrists, with many arguing that such diagnoses could be injurious to patients (Kendler, Muñoz, & Murphy, 2010). As a result, there was little reliability in the diagnostic criteria, methodologies, or treatment standards employed by psychiatrists (Wilson & Fairburn, 1993). In an effort to promote the use of empirically supported diagnostic criteria, John Feighner and colleagues published the "Diagnostic Criteria for Use in Psychiatric Research" in the January 1972 issue of the *Archives of General Psychiatry* (Feighner et al., 1972). This publication proposed Research and Diagnostic Criteria (RDC) for 14 psychiatric disorders, including AN (see Chapters 2 & 8) as the sole ED described in this set. These criteria served as the impetus for operationalizing psychiatric diagnoses in future iterations of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders*, beginning with publication of the *DSM-III* in 1980. According to the RDC, neither Julie nor John would meet criteria for AN, given that neither exhibit anorexia with accompanying weight loss of at least 25% of original body weight (Criterion B).

DSM-III

The publication of *DSM-III* (American Psychiatric Association, 1980) revolutionized the understanding of EDs by introducing a more systematic method of diagnosis and classification. The advent of this new psychiatric taxonomy provided an operationalized language for both clinicians and researchers that led to enhanced clinical assessment, improved treatment formulation, and a surge in further clinical investigations of EDs (Thomas et al., 2009).

DSM-III identified two main EDs, AN (see Chapter 2) and BN (see Chapter 3), and one feeding disorder (pica, or the persistent eating of nonnutritive substances). To capture the remaining clinical cases that did not fall neatly into these predefined, established diagnoses, *DSM-III* identified a residual category for the first time in ED classification. According to *DSM-III*, the diagnosis of *atypical eating disorder* was reserved as a "residual category for eating disorders that cannot be adequately classified in any of the previous categories [AN, BN, or pica]" (American Psychiatric Association, 1980, p. 73). Indeed, atypical eating disorder was described in only one sentence, and it received very little empirical attention as it was thought to be rare in comparison to the formal EDs (Ash & Piazza, 1995; Kutcher, Whitehouse, & Freeman, 1985).

Under the *DSM-III* classification scheme, both Julie and John would be diagnosed as having an atypical eating disorder, given that their symptoms do not fall within one of the already established diagnoses, despite obvious clinical impairment. Julie meets some of the *DSM-III* criteria for AN (i.e., expressing an intense fear of becoming obese and exhibiting a disturbance of body image), but ultimately she does not fulfill the diagnosis because she does not exhibit weight loss of at least 25% of her original body weight and her BMI falls in the normal range. Furthermore, she meets none of the *DSM-III* criteria for BN. Interestingly, John almost meets the criteria for BN in *DSM-III*, including recurrent episodes of binge eating (Criterion A), awareness that the eating pattern is abnormal (Criterion C), depressed mood and self-deprecating thoughts following eating binges (Criterion D), and the fact that the bulimic episodes are not due to AN (Criterion E). However, he falls short of meeting criterion B, which describes various facets and sequelae of binge eating. He meets none of the *DSM-III* criteria for AN. It is noteworthy how two individuals with such radically different clinical presentations fall into the same residual diagnostic category, a theme that continued into *DSM-III-R* and *-IV*.

DSM-III-R

With the 1987 transition to *DSM-III-R*, the *DSM-III* category *atypical eating disorder* became known as *eating disorder not otherwise specified (ED-NOS)* (American Psychiatric Association, 1987). *DSM-III-R* also revised the diagnostic criteria for AN and BN in order to improve diagnostic reliability by including objective thresholds for each diagnosis (e.g., revising the suggested AN weight cutoff from 75% of original body weight to 85% of that expected; adding requirements that individuals with BN binge eat twice per week and also report compensatory behaviors). This increased emphasis on objective cut points represented a major shift from the historical, anecdotal evidence that had suffused prior iterations of the *DSM* (Clinton & Glant, 1992; Parry-Jones & Parry-Jones, 1994). However, enhanced reliability came at the price of potential validity: A growing number of patients with clinical impairment did not meet these stricter criteria (Kurth, Krahn, Nairn, & Drewnowski, 1995; Thomas et al., 2009).

In order to partially acknowledge the increasing heterogeneity of the residual category, as documented by clinicians in the field, ED-NOS in *DSM-III-R* was expanded to include three examples describing possible clinical presentations (Mizes & Sloan, 1998). The first example featured an individual who was not underweight and who did not binge eat, but who engaged in self-induced vomiting because of a fear of gaining weight. The second ED-NOS example was an individual who exhibited all the criterion features of AN except for amenorrhea. The third example included an individual who met all the diagnostic features of BN except for the frequency of twice weekly binges for at least 3 months, which had been added to the BN diagnostic criteria in the *DSM-III-R* to reflect current clinical thinking and research (American Psychiatric Association, 1987; Clinton & Glant, 1992).

Applying the criteria in *DSM-III-R*, both Julie and John, given their degree of clinical impairment, would still fall into the expanded ED-NOS category, though neither would be characterized by any of the three example presentations. Interestingly, because of the revisions made to the BN diagnostic criteria to include inappropriate compensatory behaviors to prevent weight again, John moves further from meeting criteria for this diagnosis: he does not engage in compensatory behaviors and is not preoccupied with his body shape or weight (American Psychiatric Association, 1987).

DSM-IV and *DSM-IV-TR*

Given the increasing heterogeneity and number of individuals diagnosed with ED-NOS (Ash & Piazza, 1995), the subsequent edition and text revision of *DSM—DSM-IV* (American Psychiatric Association, 1994) and *DSM-IV-TR* (American Psychiatric Association, 2000)—expanded the ED-NOS diagnostic category to include six prototypical clinical presentations. One of these was binge-eating disorder (BED), which, though initially described decades earlier (Stunkard, 1959), was not officially added to *DSM* until 1994, when it was described with specific diagnostic criteria in the appendix as a possible disorder warranting further study (Mizes & Sloan, 1998; see also Chapter 10). Of note, there were no changes made to the ED-NOS diagnostic criteria with the transition from *DSM-IV* to *DSM-IV-TR*.

Indeed, BED was first identified in the medical literature by Stunkard (1959), when he described the binge behavior of a particular subgroup of obese individuals as having an "orgiastic quality, [during which] enormous amounts of food may be consumed in relatively short periods" (Stunkard, 1959, p. 289). He further noted that binge eating episodes in this subgroup predominantly occurred during periods of life stress, suggesting that a specific life event could trigger binge eating. In this way, the binge eating episodes themselves were conceptualized as oftentimes having unconscious significance that remained to be elucidated in treatment (Stunkard, 1959).

Since this publication, many others have noted that a number of these individuals are found in weight-loss programs (Thomas et al., 2014), and that they exhibit both psychological distress and physical health problems, including an increased risk for weight gain and the development of obesity (Devlin, 2007; Gormally, Black, Daston, & Rardin, 1982). Epidemiological studies conducted during the era of *DSM-IV* revealed that individuals meeting the provisional criteria for BED represented the most common ED presentation in the general population (Hudson, Hiripi, Pope, & Kessler, 2007). These findings served as a catalyst for an enormous amount of research focusing on potential etiological factors of BED, its various treatment approaches, and a number of further epidemiological studies to elaborate and understand the diagnosis (Striegel-Moore & Franko, 2008).

Despite the mental health disturbances and physical implications evidenced by those with BED, the diagnosis itself was initially met with both criticism and controversy, given the limited evidence available in support of specific diagnostic criteria, its potential overlap with BN (especially the nonpurging subtype), and the notion that its inclusion in the *DSM* as a stand-alone diagnosis might trigger a false epidemic (Frances & Widiger, 2012). According to *DSM-IV* and *DSM-IV-TR*, the hallmark feature of BED is recurrent episodes of binge eating (eating a substantially large amount of food within a discrete period of time while feeling out of control) occurring an average of at least twice weekly for at least 6 months, without any compensatory behavior to prevent potential weight gain (Criteria A, D, & E). Furthermore, patients must experience substantial distress regarding their binge eating (Criterion C) and exhibit at least three of the following five behaviors and/or feelings during or associated with binge episodes: eating more rapidly than usual; eating until uncomfortably full; eating copious amounts of food even when not physically hungry; eating alone because of embarrassment over how much food one is ingesting; and feeling disgusted, depressed, or guilty after overeating (American Psychiatric Association, 1994, 2000).

Research to date provides moderate support for these five additional criteria associated with binge eating episodes, which underscores the degree of psychological impairment experienced while binge eating (Latner & Clyne, 2008). Revisiting the case of John, he would clearly meet

criteria for BED according to the *DSM-IV* and *DSM-IV-TR* proposed research criteria. His presentation to his local outpatient EDs specialty clinic with a strong desire to stop binge eating is a marker of the level of distress he is experiencing (Criterion C). He further reports that he has been binge eating up to 3 times per day for the past 10 months, thus satisfying Criterion D. During each binge, he reports that he ingests anywhere between 3,000 to 6,000 calories, all the while feeling "completely out of control," satisfying both Criteria A(1) and A(2). He also describes his eating binges as being rapid [Criterion B(1)] and occurring when he is not hungry [Criterion B(3)], and he reports feeling "repulsed and guilty" after each episode [Criterion B(5)], thereby meeting Criterion B. Lastly, because John does not engage in any type of compensatory behavior to prevent weight gain, he also meets Criterion E, which differentiates his clinical presentation from that of BN. John's new diagnosis of BED (rather than an unnamed example of ED-NOS) under *DSM-IV* makes it more likely that his problem will be recognized by clinicians and that he will receive specialized treatment for his condition.

In addition to the inclusion of BED as a specific clinical example of ED-NOS under *DSM-IV* and *DSM-IV-TR*, two other new clinical examples were added and the other three clinical examples included in the *DSM-III-R* were retained. One of the new examples was an individual who repeatedly chews and spits out, but does not swallow, large amounts of food, which was thought to be a common symptom that had, to date, been quite neglected in research studies (American Psychiatric Association, 1994, 2000; Guarda et al., 2004; McCutcheon & Nolan, 1995). Patients who engage in this behavior report that they do so in order to enjoy the taste of foods that they generally forbid themselves from eating, or to consume larger quantities of food than they would typically ingest, and thereby attempt to avoid any weight gain by spitting it out (Mitchell, Pyle, Hatsukami, & Eckert, 1988). Because some prevalence studies performed on patients with BN found that up to 65% of patients had exhibited this behavior during the course of their illness, its inclusion as a variant of ED-NOS in *DSM-IV* was intended to acknowledge and stimulate further understanding of the implications of this phenomenon (Guarda et al., 2004). The other new *DSM-IV* ED-NOS example comprised individuals who met all the criteria for AN but who—despite significant weight loss—continued to exhibit a weight in the normal range. Interestingly, recognition of this common ED-NOS presentation coincided with the increase in the mean body weight of the U.S. population and with the emergence of the U.S. obesity epidemic during the same period (Flegal, Carroll, Kuczmarski, & Johnson, 1998). Despite the inclusion of three new prototypical ED-NOS presentations in *DSM-IV* and *DSM-IV-TR*, Julie would still be most appropriately diagnosed with ED-NOS, but would not be specifically captured by any of the clinical examples provided.

DSM-5

In the 19-year interval between *DSM-IV* (American Psychiatric Association, 1994) and *DSM-5* (American Psychiatric Association, 2013), ED-NOS moved for the first time from supporting cast to center stage. Due to an increasing diversity of cases and/or simple improvements in clinical detection, ED-NOS grew to represent 40 to 60% of patients presenting to ED specialty clinics (Thomas et al., 2009), and up to 90% of ED cases in general psychiatric settings (Zimmerman, Francione-Witt, Chelminski, Young, & Tortolani, 2008). Importantly, an emerging body of evidence suggested that the eating pathology, general psychopathology, and physical health consequences of ED-NOS were often commensurate with those of full-syndrome EDs (Thomas et al., 2009). Empowered by access to online health information and

given voice through social media, criteria-savvy e-patients began raising concerns than *DSM-IV* inadvertently contributed to a hierarchy of EDs that invalidated the burden of suffering associated with ED-NOS. According to one patient, "Anorexia is like Saks [Fifth Avenue], bulimia is Target, and binge eating disorder is Wal-Mart" (Sterling, 2011, p. 2).

Thus, a major task of the *DSM-5* Work Group was to reduce the preponderance of ED-NOS by revising diagnostic criteria to better reflect the important critiques of clinicians, researchers, and patients, as well as emerging clinical realities. In order to achieve this, criteria for the major eating disorders (i.e., AN and BN) were relaxed (see Chapters 3, 8 & 9), and new diagnoses (e.g., Avoidant/Restrictive Food Intake Disorder, BED) were added. Specifically, BED moved from the *DSM-IV* Appendix (where it had first appeared as a diagnosis proposed for further study) to the main body of the manual, where it became an officially recognized ED (American Psychiatric Association, 2013). Although the formal inclusion of BED met with some controversy in the popular media (Frances, 2013), several lines of research highlighting the syndrome's diagnostic reliability, associated impairment, and familial clustering supported its official recognition (Striegel-Moore & Franko, 2008; see also Chapter 10).

The specific criteria for BED remained very much the same from *DSM-IV* to *DSM-5*, with two minor exceptions. First, consistent with emerging data suggesting that individuals who had episodes of binge eating less often than twice per week exhibited commensurate eating pathology to those with the full-blown diagnosis (Wilson & Sysko, 2009), the binge frequency criterion was reduced to just once per week. Second, to parallel the duration required for binge eating and purging in BN, the syndrome was required to be present for just 3 months (rather than 6, as in *DSM-IV*). Note that John, whose binge eating had occurred several times per day for nearly a year, would easily meet this lower bar.

Available data from research conducted in the 5 years prior to the publication of *DSM-5* (2013) suggested that, although proposed diagnostic changes would significantly reduce the preponderance of ED-NOS, a substantial number would still fall into a residual category (Machado, Goncalves, & Hoek, 2013; Thomas et al., 2015). Reflecting *DSM*-wide changes in nomenclature, this group was renamed yet again in *DSM-5*, going from ED-NOS to *other specified feeding or eating disorder* (OSFED). To further support the detection and treatment of individuals who have an ED that does not meet all of the criteria for AN, BN, ARFID (Avoidant/Restrictive Food Intake Disorder), or BED, prototypical OSFED presentations were given formal names (rather than Arabic numerals) in *DSM-5*. *Atypical anorexi*a comprises individuals who meet all criteria for AN except for not having a substantially low weight. *Subthreshold BN* includes those who exhibit BN-like features of limited frequency and/or duration. Similarly, *subthreshold BED* includes those who engage in binge eating less often than once per week, and/or for less than 3 months. *Purging disorder*, which had recently been described by Keel, Haedt, and Edler (2005), comprises individuals who exhibit purging behaviors after consuming small amounts of food. *Night eating syndrome*, first described by Stunkard (Stunkard, Grace, & Wolff, 1955) and later refined by Allison and colleagues (Allison et al., 2010), includes individuals who consume a large proportion of their calories at night, either through eating at least 25% of calories after finishing dinner (i.e., evening hyperphagia) or waking up in the middle of the night to eat (i.e., nocturnal ingestions). Although these OSFED examples were meant to be mutually exclusive, early studies of diagnostic reliability suggested that there was some degree of overlap among them (Thomas et al., 2015). In contrast, the prototypical ED-NOS presentation of chewing/spitting, featured in *DSM-IV*, was eliminated due to recognition that the behavior is not typically a stand-alone problem, but rather co-occurs alongside more widely recognized *DSM* ED presentations (Kovacs, Mahon, & Palmer, 2002).

Individuals with heterogeneous eating disorder presentations who do not meet criteria for the OSFED examples described above, now receive a diagnosis of *other OSFED*. Although available data suggest that individuals will rarely fall into this doubly-residual category (Thomas et al., 2015), note that Julie—whose presentation does not fit neatly into any of the OSFED examples—would receive this *other OSFED* diagnosis. Finally, patients for whom there is not sufficient information to confer a formal *DSM-5* ED diagnosis (e.g., upon presentation to an emergency room), now receive a diagnosis of *unspecified feeding or eating disorder*. Furthermore, if the history of ED-NOS and BED has taught us anything, it is that once clinicians and researchers think they have planned for all possible diagnostic contingencies, individuals with unique new presentations will begin appearing at our clinics and enrolling in our studies.

Conclusions and Future Directions

For more than 30 years and now through five versions, the American Psychiatric Association's *DSM* (1980, 1987, 1994, 2000, and 2013) has recognized EDs of clinical severity that do not meet the criteria for one of the formal ED diagnoses. This residual diagnostic category was originally introduced as *atypical eating disorder* in *DSM-III*, renamed ED-NOS in *DSM-III-R* and both versions of *DSM-IV*, and ultimately retitled OSFED in *DSM-5*. Some presentations—such as BED—have made the leap from residual to formally recognized ED. Other remaining constituents of this leftover category are defined somewhat problematically by criteria that are absent rather than present. For example, atypical AN may be challenging to distinguish from normative dieting in epidemiological studies without objective measures such as low weight. In contrast, purging disorder has captured the imagination of the research community, which has identified unique biomarkers (e.g., neuroendocrine response to a test meal) (Keel, Wolfe, Liddle, De Young, & Jimerson, 2007) and established a level of predictive validity (e.g., outcome and mortality) comparable to those for officially recognized EDs (Koch, Quadflieg, & Fichter, 2013). The net effect is that many OSFED presentations have little in common with one another and have differing levels of research support.

The explosion of ED research in the past half-century has helped researchers and clinicians develop a richer and more nuanced understanding of EDs, which in turn has led to their increased detection and the development of innovative treatment strategies. As the field's diagnostic acumen has increased, the ED-NOS category has expanded to include an increasing number of diverse example presentations. To keep up with the increasing rapidity of scientific advances, the American Psychiatric Association intends for *DSM-5* to be a "living document" in which diagnostic criteria are revised as soon as convincing incremental data become available (e.g., *DSM* 5.1, 5.2, etc.), much like contemporary software packages (Kraemer, 2013, p. 412). Looking even farther into the future, continued reliance on discrete ED diagnoses may prove to be too clinically limiting. Some have proposed moving toward dimensional models of classification based on continuous measures of ED psychopathology (Beumont, Garner, & Touyz, 1994; Williamson, Gleaves, & Stewart, 2005), comorbid psychopathology, or underlying neurobiology (Wildes & Marcus, 2013). In particular, Wildes and Marcus (2013) have suggested combining all three of these dimensions to create a comprehensive classification system with maximal clinical utility: continuous measures of eating pathology (e.g., body weight, binge frequency) could direct treatment intensity (e.g., inpatient versus outpatient); measures of comorbid psychopathology (e.g., anxiety) and personality (e.g., impulsivity) could

identify maintenance factors to target in psychotherapy; and measures of neurobiology could guide pharmacologic management. Overall, adopting a more dimensional approach, coupled with a greater understanding of the genetics and clinical neuroscience of these disorders as informed by the NIMH Research Domain Criteria (Insel et al., 2010), may obviate the need for this clinically significant, but heterogeneous, residual diagnosis in *DSM-6* and beyond.

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Section II
Epidemiology

5

Prevalence and Incidence of Eating Disorders in Western Societies

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This chapter reviews the prevalence and incidence of eating disorders (anorexia nervosa, bulimia nervosa, binge eating disorder, and eating disorder not otherwise specified) with a focus on Western societies, specifically the United States, Canada, Europe, and Australia. Prevalence refers to the percentage of individuals affected by a condition in a given population. This can be reported as the percentage who are currently ill, also called "point prevalence" (Keel, Heatherton, Dorer, Joiner, & Zalta, 2006); the percentage ill over a specified window of time (5-year prevalence in Hoek et al., 1995); or the percentage who have been ill at any time in their life, called "lifetime prevalence" (Hudson, Hiripi, Pope, & Kessler, 2007). Prevalence counts cases of illness regardless of onset and, thus, combines both existing and new-onset cases in estimates. This is important for understanding the public health impact of a problem and informs the need for prevention and treatment. Prevalence estimates also are useful for examining differences in illness risk across demographic groups (e.g., comparisons between women and men or between different racial/ethnic subgroups within a population).

In contrast to prevalence estimates, incidence rates reflect the number of new cases of an illness per 100,000 people per year, also expressed as cases per 100,000 person years. Incidence rates are important for determining whether an illness is becoming more (or less) common in a population over time. This question is particularly relevant for eating disorders (EDs) because etiological theories have emphasized the contributions of the historically recent idealization of thinness in Western cultures to the development of EDs.

Data regarding the prevalence or incidence of EDs are often collected via interviews in which the interviewer makes a diagnosis according to defined criteria. An alternative approach involves the application of diagnostic algorithms to data collected either by interview or self-report questionnaires. While diagnostic algorithms ensure reliable use of rules in forming diagnoses they do not permit the use of clinical judgment. Most studies reviewed in this chapter have relied on some form of diagnostic interview, and, thus, findings represent cases identified from diagnostic interviews unless otherwise indicated.

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Epidemiological Studies

Anorexia Nervosa

Sir William Gull (1874) first described the syndrome in 1874 after observing adolescent females who experienced significant weight loss without medical explanation (see Chapter 2). It is one of the best-known EDs but may be the least common. The *Diagnostic and Statistical Manual, Fourth Edition-Text Revision* (*DSM-IV-TR*; American Psychiatric Association, 2000) states that lifetime prevalence of anorexia nervosa (AN) is 0.5% in women. Consistent with this, an 8-year prospective study of adolescent females in the United States found a lifetime prevalence of 0.6% by age 20 years (Stice, Marti, Shaw, & Jaconis, 2009). Notably, AN has a mean (SD) age of onset of 18.9 (0.8) years (Hudson et al., 2007), and women at the age of 20 may still be in the period of peak risk for AN. Other epidemiological studies have found higher lifetime prevalence estimates for AN of around 1% in adult women both in Europe (Preti et al., 2009) and in the United States (Hudson et al., 2007), with estimates of approximately 2% in Australia (Wade, Bergin, Tiggemann, Bulik, & Fairburn, 2006), Italy (Favaro, Ferrara, & Santanastaso, 2003), and Finland (Keski-Rahkonen et al., 2007). Lifetime prevalence increases to 3% when using a broad definition of AN (Baker, Mitchell, Neale, & Kendler, 2010), which likely includes those with AN and related eating disorder not otherwise specified (ED-NOS; see Chapters 4 & 11).

Prevalence does not appear to differ by ethnic group in Western cultures (Marques et al., 2011), but does differ by gender (American Psychiatric Association, 2000; see also Chapter 27). The *DSM-IV-TR* indicates that women are 10 times more likely to meet criteria for AN than are men. In accordance, no cases of AN were identified in men in a study of six countries in Europe (Preti et al., 2009). In contrast, the National Comorbidity Study Replication (NCS-R) study found that lifetime AN prevalence in men was approximately one third of that found in women (0.3% vs. 0.9%) (Hudson et al., 2007). A study of adolescents (up to age 18) in the United States found both girls and boys had a lifetime prevalence of 0.3% (Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011). Taken together, gender differences may be more dramatic in Europe compared to the United States; however, no study has directly investigated this possibility, and the inclusion of a wider range of ED diagnoses may lessen the magnitude of gender differences.

While up to 3% of women may experience broadly defined AN in their lifetimes, at any given point in time, fewer individuals meet criteria for AN because the age at which individuals are evaluated may occur before the onset of illness or after remission from illness. For example, though Favaro and colleagues (2003) reported a lifetime prevalence of 2.0%, the point prevalence of AN in women was 0.3%. Twelve-month prevalence estimates ranged from approximately 0.01% to 0.06% in the United States (Marques et al., 2011) and Europe (Preti et al., 2009), with some epidemiological studies finding no (0) cases of AN over a 12-month period in adults (Gauvin, Steiger, & Brodeur, 2009; Hudson et al., 2007). In contrast to studies in adults, studies in adolescents and college women find higher point and 12-month prevalences, ranging from 0.1 to 0.7% (Isomaa, Isomaa, Marttunen, Kaltiala-Heino, & Björkqvist, 2009; Machado, Gonçalves, & Hoek, 2013; Swanson et al., 2011). These differences in point prevalence may reflect differences in age such that adolescents are in the period of peak risk, whereas the majority of adult women are past peak risk and may have achieved remission. Alternatively, differences in point prevalence may reflect cohort effects such that the prevalence of AN has increased with each successive generation.

Across the 20th century, AN incidence appears to have increased (Hoek & van Hoeken, 2003; Keel & Klump, 2003). Hoek and van Hoeken (2003) reported that in the 1970s incidence rates stabilized, a conclusion supported in a recent review (Smink, van Hoeken, & Hoek, 2012). Indeed, AN incidence rates in primary care have been stable from 1994 to 2000 in the United Kingdom, using a primary care database for case identification (Currin, Schmidt, Treasure, & Jick, 2005), and from 1985–89 to 1995–99 in The Netherlands (van Son, van Hoeken, Bartelds, van Furth, & Hoek, 2006a). Although overall incidence may be stable in the female population, rates may be increasing in adolescents who are in the age of peak risk. Incidence rates estimated from review of medical records increased from 56.4 to 109.2 per 100,000 person years in The Netherlands for adolescent females aged 15–19 years (van Son et al., 2006a) and increased linearly from 1935 to 1989 in adolescent and young adult women aged 15–25 years in the United States (Lucas, Crowson, O'Fallon, & Melton, 1999). These increases may reflect earlier detection or an earlier age of onset, as the overall incidence rate has not changed (Smink et al., 2012). Reflecting the rarity of AN, incidence rates estimated from medical records have ranged from 1.2 per 100,000 person years for severe AN (Milos et al., 2004) to 8.3 per 100,000 person years for broadly defined AN (Lucas et al., 1999). Fitting with observed gender differences in prevalence, incidence rates estimated from medical records are greater for women (151.5 per 100,000 people per year) than for men (8.4 per 100,000 people per year) (Larrañaga, Docet, & García-Mayor, 2012).

The *DSM-5* (American Psychiatric Association, 2013) criteria for AN do not include the amenorrhea criterion, with the intention of shifting diagnoses from ED-NOS to AN. Research supports that the number of cases increases by 40% using the broader criteria for diagnosing AN, with most cases shifting from a diagnosis of ED-NOS to a diagnosis of AN (Keel, Brown, Holm-Denoma, & Bodell, 2011). In a sample of female adolescents, the lifetime prevalence by age 20 increased from 0.6% with *DSM-IV-TR* (2000) AN criteria (Stice et al., 2009) to 0.8% with *DSM-5* AN criteria (Stice, Marti, & Rohde, 2013). In a sample of female high-school and university students, the lifetime prevalence of AN increased from 0.59% with *DSM-IV-TR* criteria to 0.69% with *DSM-5* criteria (Machado et al., 2013). Taken together, these findings suggest the prevalence of AN may increase by approximately 30%. However, given the low prevalence of this illness, this will not result in a dramatic change in population-based estimates following the adoption of proposed *DSM-5* criteria.

Bulimia Nervosa

Nearly 100 years after AN was first described, Gerard Russell introduced the term "bulimia nervosa" (BN) to describe women who experienced uncontrollable binge-eating episodes and purged while at a normal weight (Russell, 1979; see also Chapter 3). The presence of a normal weight distinguishes BN from AN, though Russell noted that many of his patients reported a history of partial or full AN (Russell, 1979; see also Chapter 55). Compared to AN, less information exists regarding the prevalence and incidence of BN because less time has passed since the syndrome was first introduced to the literature.

The *DSM-IV-TR* states that 1 to 3% of women will meet criteria for BN in their lifetime. Consistent with this range, epidemiological samples suggest that 0.88% of European women (Preti et al., 2009) and 1.5% of U.S. women will meet criteria for BN in their lifetimes. Other epidemiological samples have found lifetime prevalences ranging from 0.1 to 1.3 in Western countries (Kessler et al., 2013). Studies using broad definitions of BN have found higher lifetime

prevalences (5%) (Baker et al., 2010) than studies that rely on more narrow *DSM-IV-TR* criteria (1.2%) (Trace et al., 2012). Lifetime prevalence in female adolescents is similar to that reported for adults in the United States, with estimates ranging from 1% to 1.6% in adolescents using both questionnaire (Field et al., 2012) and interview methods (Stice et al., 2009; Swanson et al., 2011). In contrast, lifetime prevalence in female adolescents in Finland is approximately 0.4% (Isomaa et al., 2009). Lower prevalence estimates in adolescents than in adults are expected given a mean (SD) age of onset of 19.7 (1.3) years (Hudson et al., 2007). However, it is unclear why expected differences are more apparent in samples drawn from Europe than from the United States.

Contrary to popular media portrayals of EDs as being most common in White individuals, recent data from a nationally representative sample in the United States suggest that BN is more common among Latinos and African-Americans than among non-Latinos (Marques et al., 2011). The *DSM-IV-TR* suggests that women are 10 times more likely to suffer from BN than men. This gender ratio is greater than has been observed in the literature. Epidemiological studies in Europe indicate a greater lifetime prevalence of BN in women (0.88%) than in men (0.12%). Similarly, a study in the United States indicated that 1.5% of women experienced BN in their lifetime compared to 0.5% of men. Thus, women appear to be significantly more likely to suffer from BN compared to men; however, they may not be 10 times more likely to suffer from the illness. Including both genders, 12-month prevalence ranges from 0 to 0.6% across studies of the United States, Canada, and Europe (Gauvin et al., 2009; Hudson et al., 2007; Kessler et al., 2013; Preti et al., 2009).

Given BN's more recent description as a syndrome, less literature exists on changes in its incidence rates. One study of medical records indicated a BN incidence of 4.4 per 100,000 inhabitants per year (Larrañaga et al., 2012). This may be an underestimate as individuals may not disclose symptoms to their physicians. Isomaa and colleagues (2009) found an incidence of 438 per 100,000 person years for broadly defined BN in females aged 15 to 18 years. One meta-analysis indicated a significant increase in BN incidence over the 20th century (Keel & Klump, 2003). Consistent with this, prevalence is higher in younger cohorts than older cohorts (Hudson et al., 2007), suggesting prevalence increased over time.

In contrast to evidence that BN is on the rise, recent research suggests that BN incidence may have decreased over more recent decades (Smink et al., 2012). Currin et al. (2005), using a primary care database, reported a 39.9% decline in BN incidence from 1996 to 2000 in women aged 10–39. Specifically, in women aged 20–39, incidence decreased from 56.7 per 100,000 person years in 1993 to 28.6 per 100,000 person years in 2000, while incidence rates remained stable in women aged 10–19 (Currin et al., 2005). A decrease of 29%, though not statistically significant, was observed in BN incidence from 1985–89 to 1995–99 in The Netherlands (van Son et al., 2006a), and this trend continued when the population was studied again in 2005–2009, resulting in a statistically significant decline in BN incidence from 1985 to 2009 (Hoek, 2012). Finally, a significant decrease in BN point prevalence was observed in a college cohort assessed in 1982, 1992, and 2002 using diagnostic algorithms applied to survey data (Keel et al., 2006).

In contrast to a potential decline in BN incidence and point prevalence, Crowther, Armey, Luce, Dalton, and Leahey (2008) observed stability in BN point prevalence using questionnaires in college cohorts from 1990 to 2004, and a population-based questionnaire study in Australia found that binge eating and purging increased two-fold from 1995 to 2005 (Hay, Mond, Buttner, & Darby, 2008). Another report from this same group found that from 1998 to 2008, the prevalence of objective binge eating and extreme dieting (i.e., 8 or more waking

hours without eating) doubled (2.7% to 4.9%, and 1.5% to 3.3%, respectively), whereas the prevalence of purging behaviors did not change (0.9% and 1.0%, respectively) (Mitchison, Hay, Slewa-Younan, & Mond, 2012). Taken together, these studies suggest BN and bulimic behaviors have demonstrated variability over time.

Across studies with estimates of either BN prevalence or incidence from the 1980s, shortly following the introduction of BN to the medical literature, data suggest that the disorder was alarmingly common, contributing to fears that EDs were an epidemic. Although some estimates from the 1980s may have been inflated by use of less stringent diagnostic criteria, at least three studies, representing data from the United States (Keel et al., 2006), England (Currin et al., 2005), and The Netherlands (Hoek, 2012), have documented a statistically significant decline in either BN point prevalence or incidence from the 1980s to the 21st century while employing a uniform method of case identification. Significant variability in epidemiological patterns may reflect rapid shifts in cultural factors rather than sudden mutations in the genetic make-up of populations, and are broadly consistent with the view of BN as a culture-bound syndrome (Keel & Klump, 2003).

Like AN, the *DSM-5* (2013) criteria for BN are broader than *DSM-IV-TR* (2000) criteria. Within *DSM-IV-TR*, binge eating and compensatory behaviors must occur, on average, twice a week for 3 months for a diagnosis of BN. The *DSM-5* (2013) criteria lowered this threshold to once a week. When combined with the (then proposed) changes for AN diagnostic criteria, one study found minimal impact of the proposed *DSM-5* criteria on number of cases diagnosed with BN compared to cases identified with *DSM-IV* criteria (Keel et al., 2011). A study of Swedish female twins found BN lifetime prevalence increased from 1.2% using *DSM-IV-TR* (2000) criteria to 1.6% using the (then proposed) *DSM-5* criteria (Trace et al., 2012). This increase was even more pronounced in a sample of female adolescents, where lifetime BN prevalence increased from 1.6% (Stice et al., 2009) to 2.6% (Stice et al., 2013). A sample of female Portuguese high-school and university students found a relatively smaller increase from *DSM-IV-TR* to *DSM-5* criteria, from a point prevalence of 0.46% to 0.59% (Machado et al., 2013). Taken together, it appears *DSM-5* (2013) criteria will increase the prevalence of BN; however, the magnitude of this increase remains somewhat unclear given that some cases of *DSM-IV* BN may be diagnosed as *DSM-5* AN, and relatively few individuals may exist in the discrete group of those who binge and purge at least once but not twice per week, on average, over a 3-month period (Keel et al., 2011).

Binge Eating Disorder

Binge eating disorder (BED) was a provisional research diagnosis in *DSM-IV-TR* (2000) and is an ED in *DSM-5* (2013; see also Chapters 4 & 10). First named in 1959 by Albert Stunkard (1959), BED is characterized by recurrent binge-eating episodes in the absence of compensatory behaviors. Although BED was described decades before BN, less is known about the prevalence and incidence of BED due to its more recent recognition in the *DSM*.

Lifetime prevalence estimates for BED using the *DSM-IV-TR* research criteria were approximately 3.5% among U.S. women (Hudson et al., 2007) and 1.9% in European countries (Preti et al., 2009). The prevalence of BED does not differ amongst ethnic groups in the United States (Marques et al., 2011). While gender differences are not as large as those observed in AN or BN, women are more likely to have BED than are men (Hudson et al., 2007). Approximately 2.0% of U.S. men (Hudson et al., 2007) and 0.26% of European men will experience BED in their lifetimes (Preti et al., 2009).

The *DSM-5* (2013) criteria require binge eating once a week, on average, for 3 months in comparison to binge eating 2 days a week, on average, for 6 months proposed in the *DSM-IV-TR*. Adopting these criteria increases the lifetime prevalence from 2.0% to 2.1% in the United States, and from 3.5% to 3.6% in women (Hudson et al., 2012). Likewise, the lifetime prevalence of BED in a Swedish sample of women increased from 0.17% to 0.35% when using *DSM-IV-TR* versus *DSM-5* criteria (Trace et al., 2012). Using *DSM-5* criteria, 2.3% of female and 0.8% of male adolescents will meet criteria for BED in their lifetime (Swanson et al., 2011). Other studies in adolescents have found BED prevalence ranging from 2 to 3% using both questionnaire and interview methods (Field et al., 2012; Stice et al., 2013). While incidence studies have not yet been published for BED, the risk of BED diagnosis increased over birth cohorts in a U.S. sample, suggesting that BED has become more common over time (Hudson et al., 2007).

Eating Disorder Not Otherwise Specified

The most common eating disorder diagnosis in epidemiological studies is ED-NOS (Crowther et al., 2008; Favaro et al., 2003; Hay et al., 2008; Wade et al., 2006; see also Chapters 4 & 11). ED-NOS is a catch-all category that includes any significant disorder of eating that does not meet criteria for AN or BN. Because BED was included as a provisional eating disorder diagnosis in the *DSM-IV-TR* (2000), some studies exclude BED from those diagnosed with ED-NOS while others include it, consistent with formal identification as an ED-NOS in the *DSM-IV-TR*. Lifetime prevalence estimates for ED-NOS range from 5.3% (Favaro et al., 2003) to 10.6% (Wade et al., 2006) in adult populations, and from 3% to 15% in adolescents and young adults using questionnaires (Field et al., 2012). Adopting *DSM-5* (2013) criteria reduced ED-NOS in one population of adolescents and young adults from 2.8% to 1.7% (Machado et al., 2013). However, other studies using the *DSM-5* criteria report a lifetime prevalence in females of 11.5% by age 20, with an incidence of 1,434 per 100,000 person years (Stice et al., 2013).

Purging disorder (PD) is a form of ED-NOS first named by Keel and colleagues in 2005 (Keel, Haedt, & Edler, 2005). Despite its recent introduction to the literature, several studies have examined a syndrome characterized by recurrent purging in the absence of objectively large binge episodes among individuals of minimally normal weight (Keel & Striegel-Moore, 2009). PD is one of five prototypical examples of what is designated as an Other Specified Feeding or Eating Disorder (OSFED) in *DSM-5* (2013).

Estimates of the lifetime prevalence of PD have ranged from 1.1% (Favaro et al., 2003) to 5.3% (Wade et al., 2006) in women. These estimates do not include individuals with a lifetime history of AN, BN, or BED, indicating that PD has a lifetime prevalence comparable to that reported for *DSM-IV-TR* (2000) BN. Lifetime prevalence estimates in adolescents fall within the range reported for adult women, with estimates ranging from 2% using questionnaires (Field et al., 2012) to 3.4% using diagnostic interviews (Stice et al., 2013), and a reported incidence of 447 per 100,000 person years (Stice et al., 2013).

The reported point prevalence of PD is 0.5% in Australian women using questionnaires (Hay et al., 2008) and 0.6% in Canadian women using diagnostic interviews (Gauvin et al., 2008). Comparable estimates of point prevalence have been reported in college samples using questionnaire methodology, ranging from 0.3% to 1.0% (Crowther et al., 2008; Haedt & Keel, 2010). It is not known if prevalence of PD differs by ethnic group, but one study reported that PD is significantly more common in women than in men (Haedt & Keel,

2010). Studies of successive cohorts of college students have found no significant changes in point prevalence over time (Crowther et al., 2008; Haedt & Keel, 2010). No data have been published regarding the prevalence of PD prior to 1982, leaving unclear whether PD became increasingly common during the 20th century, leading up to its first description, or whether PD represented a significant source of psychiatric morbidity in late adolescent and young adult women that was missed due to assessment methods that do not evaluate the presence of purging unless criteria for binge eating are met (Swanson, Brown, Crosby, & Keel, 2013).

Night Eating Syndrome (NES) (Stunkard et al., 2009) is another prototypical example of what is designated as an OSFED in *DSM-5* (2013). NES was first described in 1955 (Stunkard, Grace, & Wolff, 1955), suggesting that there would be ample time to establish the prevalence and incidence of this illness compared to more recently identified EDs. However, in the absence of agreed-upon criteria for this syndrome (Striegel-Moore, Franko, & Garcia, 2009), studies have tended to describe frequency of night eating rather than NES and have been limited to populations affected by other conditions, such as obesity, BED, or other mental illnesses. Even following the publication of recommended research criteria (Allison et al., 2010), limited data are available to evaluate the prevalence or incidence of NES, likely because the features differ qualitatively from those evaluated in diagnosing AN or BN. In a small sample of 68 overweight or obese individuals diagnosed with a serious mental illness and seeking weight loss, 25% met proposed research criteria for NES (Lundgren, Rempfer, Brown, Goetz, & Hamera, 2010). In a sample of 395 families in Canada in which one parent was obese, NES was found to affect 0% of children, 0.5% of mothers, and 0.3% of fathers using questionnaires (Lundgren et al., 2012). In a multisite study of 845 patients diagnosed with type 2 diabetes, 3.8% were diagnosed with NES (Allison et al., 2007). This represents a very large range of prevalence estimates due to differences in sample size and inclusion criteria (e.g., severe mental illness and seeking weight loss, obesity in one parent, type 2 diabetes). To our knowledge, NES has not been examined in any population-based epidemiological study, and this represents an important future direction.

Conclusion and Future Directions

Eating disorders have affected increasing proportions of the population over the course of the 20th century, and current evidence suggests this increase may have stabilized as we reached the end of the 20th century and entered the beginning of the 21st century (Smink et al., 2012). Increases in binge eating and syndromes marked by binge eating may reflect increased urbanization (Hoek et al., 1995; van Son, van Hoeken, Bartelds, van Furth, & Hoek, 2006b) and increased access to large quantities of readily edible food (Keel & Klump, 2003), while increases in the prevalence of AN and EDs characterized by body image disturbance may reflect the increased idealization of thinness in modern Western culture (Keel & Klump, 2003). Little is known about time trends for the incidence or prevalence of EDs in the *DSM-5* (2013) OSFED category that differ in clinical presentation from AN and BN, such as PD and NES. Although purging, as a behavior, demonstrated an increase in one study (Hay et al., 2008), other research indicates no significant change in purging (Mitchison et al., 2012) or PD (Crowther et al., 2008; Haedt & Keel, 2010). No data are available to evaluate time trends for NES; however, the early identification of this syndrome, predating the description of BN by 25 years, suggests that it is not a "new" disorder.

In conclusion, most is known about AN, the least prevalent but longest studied ED. In contrast, less is known about BED and ED-NOS/OSFED, which are the most frequently occurring EDs. More research is needed to understand the epidemiology of these disorders. However, for these studies to be informative, it will be crucial for investigators to separate different forms of ED-NOS in epidemiological reports. This has been hampered by assessment methods in which questions required to make ED-NOS diagnoses are skipped if the individual does not endorse core criteria required for a diagnosis of AN (low weight), BN, or BED (binge eating). *DSM-5*'s (2013) inclusion of five named conditions with descriptions within the OSFED diagnosis (see Chapters 4 & 11) should facilitate this approach to research.

Future research directions for the field include:

1 Determine whether there is a valid threshold for distinguishing AN from atypical AN (i.e., OSFED that resembles AN, except that weight loss has not resulted in medically low weight) based on epidemiological patterns.
2 Determine whether there is a valid threshold for distinguishing BN from partial BN (i.e., OSFED that resembles BN, except not all criteria are fully met) based on epidemiological patterns.
3 Determine whether the incidence of AN has stabilized and whether these patterns vary by age group.
4 Determine whether the incidence of BN has continued to decrease and whether this varies by age group.
5 In both community and clinic-based populations, evaluate whether incidence rates for BED, PD, and NES have changed over time.
6 Adopt alternative approaches to assessment (i.e., do not employ skip rules; see Swanson et al., 2013) to evaluate what other OSFED or Unspecified Feeding or Eating Disorder (USFED; *DSM-5*, 2013) presentations are common and whether the incidence of these presentations has changed over time.

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6

Prevalence and Incidence of Eating Disorders in Asian Societies

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Since the turn of the millennium, our understanding of eating disorders (EDs) has evolved dramatically due, in large part, to the growing body of literature on EDs in countries and among cultures well beyond the boundaries of the Western hemisphere. As reports of EDs in non-Western cultures have increased, earlier conceptions of EDs as "culture-bound syndromes" primarily affecting young Caucasian women in industrialized Western nations, have been replaced with a growing appreciation for the significance of EDs globally (see Chapter 23). This chapter explores the emergence of EDs in Asia and highlights the unique contribution to the study of culture and EDs that Asia provides when examined in juxtaposition to what has become the normative reference provided by the West.

We begin with a brief overview of the timeline of the appearance of EDs, introducing key themes and models that have been used to explain the emergence of EDs in the region. Highlighting and challenging assumptions often made about EDs in non-Western countries, we discuss the evidence for, and limitations of, the model of "Westernization" and examine the link between EDs and "cultures in transition" within the Asian context. Subsequently, we review the existing data on the prevalence and incidence of EDs in various countries within Asia, and conclude with a discussion of trends, projected developments, and future directions for the study of EDs in Asia.

Rise of Eating Disorders in Asia and Cultures in Transition

The geopolitical construct "Asia" designates a large and diverse segment of the globe, encompassing a vast landmass that extends from the Suez Canal in the west, to multiple island chains in the Pacific Ocean. The region is composed of approximately 48 distinct countries; historically and currently, the social, political, and economic conditions of these countries

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vary significantly (S. Lee, 2004). Consequently, the individual histories of these countries over time are instructive in terms of understanding the emergence and expression of EDs.

Gordon (2001) observed that EDs were rarely reported in non-Western countries prior to the early 1990s. This held true for much of Asia, with the exception of Japan, where reports of EDs began to surface in the mid-1970s (Pike & Borovoy, 2004). Similar to other regions in the world, the emergence of EDs in Asia has coincided with times of sociocultural transition characterized by rapid industrialization, urbanization, and modernization (Becker, 2003; Nasser, Katzman, & Gordon, 2001), and accompanied by shifting gender roles, new standards of beauty, and dramatically increased exposure to Western media and foods. Evidence suggests that a culture's population is at particularly high risk for developing EDs during such phases of societal transformation (Nasser et al., 2001). Accordingly, the emergence of EDs within "cultures in transition" has been observed in multiple countries across Asia as discussed below.

Nutrition Transition: From Farms to Fast Food

One of the defining features of economic development that arises in tandem with industrialization is the migration of sizeable portions of a population from rural areas to urban centers. Across Asia, this process of urbanization occurred rapidly, involved unprecedented numbers of people, and has been accompanied by dramatic social changes and increases in per capita income (Marcotullio, 2001). An unfortunate corollary to urbanization, however, is an increase in the overall rate of psychopathology (Galea, Uddin, & Koenen, 2011), including EDs (van Son, Hoeken, Bartelds, van Furth, & Hoek, 2006).

Another consequence of urbanization is a concurrent and profound transformation of nutrition. In Asia, this shift has been characterized by the "Americanization" of diets, wherein the increased availability of processed "Western" foods high in sugar, fat, salt, and calories, replaced traditional Asian diets, which typically consist of more fiber, grains, and vegetables (Madanat, Hawks, Campbell, Fowler, & Hawks, 2010). Integral to this dietary transition was the arrival of American fast food chains in the 1970s, first in Japan and now across all of Asia (Watson, 1997). In turn, rising consumption of these unhealthy foods triggers a cascade of "nutrition-related non-communicable diseases" (NR-NCDs) including obesity, hypertension, diabetes, and other metabolic disorders (Madanat et al., 2010).

"Westernization"

In addition to the role the West has played in Asia's economic development (e.g. foreign investment and the arrival of U.S. and European companies) and changing diet, explanations for the rise of EDs in Asia have focused considerable attention on the implications of Asia's increased sociocultural exposure to, and interaction with, the West—in particular, the Western media. From this perspective, the notion of "Westernization" as an etiological risk factor emerged, positing that exportation of the "thin ideal" and Western notions of "body work" to non-Western cultures generates rising rates of body dissatisfaction, dieting, and EDs.

In support of the "Westernization" thesis, correlations between high levels of Westernization and greater body image disturbance have been reported in Singapore (Soh et al., 2008; Ung, 2003), Hong Kong (S. Lee & Lee, 1999), and Pakistan (Choudry & Mumford, 1992; Mumford, Whitehouse, & Choudry, 1992). However, other research reveals a number of limitations of this thesis. Specifically this perspective conflates "Westernization" with other processes of societal transformation (e.g., modernization) and fails to take into account the fact that the rapid development

in Asia over the last few decades has occurred within the context of "globalization" (Marcotullio, 2001). As a result, the "Westernization" thesis overattributes the changes and developments in Asia, including the emergence of EDs, to the influence of the West rather than to other processes of change and the distinct evolutions of individual Asian cultures. Further, the model of "Westernization" suggests that "Western" culture is exportable while other cultures are immobile and fixed (Lester, 2004). It is highly significant then that variations in the expression of EDs across cultures in Asia are evident, as this is not a development we would expect if it were the case that these cultures were simply absorbing Western beauty and body ideas.

Gender Roles

Among the manifold changes set in motion by the processes of industrialization, urbanization, and modernization underway in Asia, there exists ample evidence of a profound and dramatic shift in gender roles, particularly for women. The "gendered" nature of societal transformation and globalization is especially striking in Asia. This reflects in part the fact that economic development in the region was initially largely driven by the growth in the manufacturing (specifically garment) industry, and later, the service industries, which resulted in a spike in demand for women's labor in particular (Pettman, 2003). Unsurprisingly, with growing numbers of women pursuing employment and educational opportunities, familiar definitions of "femininity" and conceptions about gender roles are called into question, and traditional family structures are subject to change as well. Striegel-Moore, Silberstein, and Rodin (1986) suggest that, during such times of rapidly changing gender roles, girls, in particular, are at increased risk for experiencing stress related to these shifting roles and evolving expectations, which in turn may place them at greater risk for overall psychological distress. Further complicating matters, and perhaps predisposing women in Asia to EDs specifically, are changing images and definitions of female beauty.

Significantly, the growth of market economies and concentrated urban populations in Asia coincided with the globalization of the fashion and beauty industries. In Asia, Western-based companies discovered not only masses of female factory workers but also a new customer base whose spending power was on the upswing. Arguably, one of the most widely consumed "imports" from the West quickly became aesthetic beauty ideals glorifying "thinness" and the notion of "body instrumentality," whereby women strive to reshape their bodies as a means of self-transformation to reflect a transformed society. In an ill-fated twist of irony, increasing numbers of Asian women who entered the workforce seeking greater autonomy and financial leverage in a fast-changing world, found employment in the factories of the very clothing, beauty, and luxury goods brands responsible for manufacturing the "thin ideal" and promoting potentially harmful conceptions of beauty. Constructed as both commodities and consumers (Leitch, 1996), women's bodies became the site of identity construction through body maintenance (Crewe, 2001).

Ultimately, women in Asia and in cultures undergoing transition throughout the world, find themselves inhabiting a tenuous position as they seek to navigate a new and unfamiliar landscape altered by shifting gender roles, changing definitions of "femininity," increasingly abundant and calorie-dense food, and a pervasive "thin" beauty ideal. Inundated with competing and often confusing messages, the burden of navigating a healthy course to adulthood is made ever more treacherous and stressful, placing women in Asia at increasing risk for developing ED symptoms (Becker, 2004). Consequently, while periods of sociocultural transition in Asia and elsewhere have spurred profound gains for women in the form of increased rights and opportunities, one of the costs is increasing rates of EDs.

EDs in Asia Today: Prevalence, Incidence, and Trends Over Time

The following section provides a closer examination of the unique trajectories of EDs in specific countries in Asia, with the intent of further elucidating the distinct contributions, and interactions, of both culture and the various processes of societal transformation to the emergence and rise of EDs across Asia.

Japan

The 1970s marked the beginning of the "modern study" of EDs in Japan. Over the course of the next two decades, reports indicated that the incidence and prevalence of EDs were steadily rising but remained lower than in the West. Several of the earliest studies focused on anorexia nervosa (AN) exclusively, and although community-based public data are not available from this period, clinic-based studies documented a steady rise in the number of individuals presenting for treatment of EDs during the latter half of the 20th century in Japan (Suematsu, Ishikawa, Kuboki, & Ito, 1985). Consistent with the demographic distribution of EDs in the West, the prevalence of AN was estimated to be highest among females ages 13–29 at 25.2–30.7 (per 100,000), as compared to 6.3–9.7 among all Japanese females, and 3.6–4.5 within the general population (Kuboki, Nomura, Ide, Suematsu, & Araki, 1996).

A study of bulimia nervosa (BN) and binge eating in college women conducted in the 1980s found that 8.4% of the women reported bingeing and self-induced vomiting, 2.9% binge-purged more than once a week, and 4.6% used purgatives as a weight-control strategy (Kiriike et al., 1988). Meanwhile, the first formal reports of binge eating disorder (BED) emerged in Japan near the close of the millennium (Nogami, 1997) and the most recent data from Nakai, Nin, and Noma (2014) documents a steady rise in eating disorders from 1982 to 2002. During the first decade of 2000, rates of AN increased four-fold and BN became approximately 4.7 times more prevalent than it was in the 1990s (Yasuhara et al., 2002). As described by Pike and Borovoy (2004), this rise in EDs in Japan transpired against a backdrop of increasing globalization, urbanization, and industrialization, which served to catalyze significant changes in gender roles and the traditional Japanese family structure—a pattern observed repeatedly around the globe.

Hong Kong

Hong Kong (HK), which has long resided at the crossroads of "West" and "East", saw cases of AN emerge in the 1980s and increase thereafter (S. Lee, 1991; S. Lee, Ng, Kwok, & Fung, 2010). An early prevalence estimate for AN of 0.002% was inferred based on the identification of 10 cases over a 5-year period at a hospital serving a catchment area of 500,000 (S. Lee, Chiu, & Chen, 1989), while a study in the 1990s estimated that the prevalence for the full spectrum of EDs was 0.46% (S. Lee, 1993).

The most significant development during this time was the emergence of a new variant of AN termed "non-fat-phobic anorexia nervosa" (NFP-AN). This subgroup encompassed individuals with AN who appeared to lack the explicit "fear of fat" and/or distorted body image characteristic of AN patients in the West. Instead, these patients frequently attributed their symptoms to somatic complaints such as epigastric bloating, abdominal/stomach pain, or an absence of hunger/appetite (S. Lee, Lee, Ngai, Lee & Wing, 2001). In general, individuals with NFP-AN tended to be significantly slimmer premorbidly and were less likely to exhibit bulimic symptoms, as compared to typical AN patients (S. Lee, Chan, & Hsu, 2003; S. Lee et al., 2010). This variant form of AN attracted much attention in the field because it challenged the

core assumptions regarding the essential features of AN. In fact, the number of NFP-AN cases in HK has steadily diminished over time, despite concurrent increases in overall ED rates and, specifically, in cases of BN and fat-phobic AN, such that today the clinical profile of anorexic patients in HK more consistently resembles that seen in the West (S. Lee et al., 2010).

Community-based studies from HK reveal a common theme wherein many women express a desire to lose weight and are inclined to diet despite having BMIs in the "normal" or "underweight" range. By contrast, males typically express the desire to be taller and to gain weight, specifically in relation to developing stronger upper bodies and increasing musculature, which conforms to the male body ideal (S. Lee, 1993; S. Lee, Leung, Lee, Yu, & Leung, 1996). On the whole, studies from the late 1990s through the present show that levels of body dissatisfaction and the "desire for thinness" continue to become increasingly widespread (Fung & Yuen, 2003; Lai et al., 2013; Leung & Mak, 2003), contributing in turn to higher rates of disordered eating and EDs. Furthermore, a 1999 study comparing high school girls in urban HK to similar samples in rural (Hunan) and semi-urban (Shenzhen) locations in mainland China, demonstrated that disordered eating attitudes and behaviors increased in tandem with the level of industrialization, with the girls in HK showing the greatest eating disturbances and body dissatisfaction, followed by girls in Shenzhen and Hunan provinces, respectively (S. Lee & Lee, 1999).

Korea

Powered by one of the fastest growing economies in the world, Korean society underwent rapid and pervasive change from the early 1960s through the late 1990s. This period witnessed a concurrent rise in risk factors and clinical cases of EDs in Korea. In a 1998 epidemiological study of 3,062 Koreans, 8.5% of the sample scored above the cut-off (>20) on the Korean version of the EAT-26 (K-EAT-26) (Y. H. Lee et al., 1998). EDs in Korea present clinically much like those in the West, and recent studies suggest that risk factors like body dissatisfaction and "thin-ideal internalization" are actually higher in Korea than in the West.

In a study comparing 167 second-generation female, young adult Korean-Americans to 37 Korean immigrant women, and 937 native Korean women, Korean-American women scored significantly lower on the EAT-26 than both Korean immigrants and native Koreans. No significant differences were found between Korean immigrants and native Korean women, despite native Korean women reporting significantly lower BMIs than Korean immigrant women. Acculturation levels—measured using the Suinn–Lew Asian Self-Identity Acculturation Scale (SL-ASIA)—were not associated with EAT-26 scores for either group (Jackson, Keel, & Lee, 2006). Another cross-cultural, multicountry study comparing Chinese (n = 109), Korean (n = 137), and U.S. college women (n = 102) indicated that Korean college women exhibited the greatest body dissatisfaction and disordered eating, followed by Chinese women, and, lastly, U.S. women. These results contradict the "Westernization" model of EDs, which would have expected U.S. women to exhibit the greatest ED-related dysfunction (Jung & Forbes, 2007).

Malaysia

An early study of EDs in Asia, conducted in Malaysia, estimated the national prevalence of AN was 0.05% based on a sample of 6,000 psychiatric patients (Buhrich, 1981). Additional studies didn't emerge until almost 20 years later, coincident with a period of increased economic development that saw a simultaneous increase in both population BMIs and rates of body

dissatisfaction and dieting behaviors (Pon, Kandiah, & Mohd Nasir, 2004). In general, the research from Malaysia largely implicates gender and ethnicity as primary factors influencing ED risk.

Significant gender differences indicate that females typically display higher body dissatisfaction and concern related to eating and weight/shape issues, prefer very slim or even underweight ideal body figures, and employ various weight-loss strategies, including dieting, self-induced vomiting, exercise, and laxatives (Kuan, Ho, Shuhaili, Siti, & Gudum, 2011; Mellor et al., 2009; Swami, Tovee, & Harris, 2013). Males, for their part, report engaging in muscle-building activities (Mellor et al., 2009), are more likely to perceive themselves as overweight, select an overweight figure as "ideal," and fail to perceive if they're underweight (Kuan et al., 2011).

Studies examining ethnicity and EDs in Malaysia have compared ethnic Malays (Malaysian Malays) with Malaysian Chinese and/or Malaysian Indians. One such study, which analyzed eating attitudes among 187 ethnic Malays (87 males [mean age = 21.46 years], 100 females [20.42 years]) and 80 Chinese students (33 males [21.03 years], 47 females [20.96 years]) at a Malaysian university, revealed that Malay students were at greater risk for EDs based on significantly higher EAT-26 scores (Edman & Yates, 2004). In another study of 584 male and female university students, approximately one in five students (18.2%) were at elevated risk for EDs. Females were at greater risk than males (21.3% vs. 13.5%) and reported higher stress scores (Gan, Nasir, Zalilah, & Hazizi, 2011).

Swami et al. (2013) examined how ethnicity moderated the discrepancy between actual versus ideal weight in 459 Malay, 307 Chinese, and 150 Indian women. Chinese women showed the smallest weight discrepancy when compared to Malay and Indian women, though the effect size was small. There was also some evidence that the women internalized appearance-related media messages differently, according to ethnicity. Conversely, a 2013 study comparing adolescents of Malaysian Malay ($n = 58$), Malaysian Chinese ($n = 95$), Chinese native ($n = 242$), and non-Asian Australian backgrounds ($n = 81$), reported that Malaysian Chinese adolescents had the greatest degree of body dissatisfaction. In particular, it was found that overall body dissatisfaction was positively correlated with dissatisfaction with facial features among Malaysian Malays and Australians (Mellor et al., 2013)—a finding that is consistent with traditional Asian beauty ideals that focus on facial features.

Fiji

The archipelago of Fiji, although technically located in Oceania, has figured prominently in our understanding of the emergence and rise of EDs in non-Western countries, and so is included here. Like many preindustrialized countries, traditional Fijian notions of beauty favored heavier, more robust female body types, and EDs were quite rare, with only one case of an ED documented prior to the mid-1990s. The islands were largely isolated from Western influences—specifically Western media—until the late 1990s, at which point TV was introduced into Fijian society. In this way, Fiji has provided a real-world laboratory in which the thesis of "Westernization" has been tested; researchers have been able to measure the changes in eating psychopathology and beauty ideals, both before and after the introduction of TV. Over the course of the next decade, in the wake of TV's arrival on the island, a surge in rates of EDs among ethnic Fijian women, as well as a perceptible shift in definitions of female beauty and body ideals to a more "Westernized" "thin ideal" were documented within the broader Fijian society (Becker, 2004; Becker, Burwell, Gilman, Herzog, & Hamburg, 2002; Williams, Ricciardelli, McCabe, Waqa, & Bavadra, 2006; see also Chapter 23).

Studies conducted in Fiji during the late 1990s and the first decade of the new millennium, as the country was beginning to industrialize and population BMIs began rising, document the emergence and proliferation of clinical BN and BED (Becker, Burwell, Navara, & Gilman, 2003; Becker, Gilman, & Burwell, 2005). In tandem, increases in body dissatisfaction, dieting, desire to lose weight, thin ideal internalization, and disordered eating behaviors including binge eating and self-induced vomiting, were also observed (Becker et al., 2002, 2003; McCabe et al., 2011). One study, which revealed a significant association between binge eating and acculturated body attitudes, suggests that acculturation is a key mediating factor (Becker, Fay, Gilman, & Striegel-Moore, 2007). Becker (2004) suggests that Fijian females' explicit modeling of their behavior and appearance on Western TV characters may reflect a desire to position themselves competitively within a rapidly changing culture. The latest research from Fiji indicates EDs are still on the rise and that males increasingly report experiencing sociocultural pressure to achieve a muscular body ideal (McCabe et al., 2011; see also Chapter 37). Additionally, it also appears that contemporary social media may exert a significant adverse influence on vulnerable individuals, increasing risk for developing an ED among Fijians (Becker et al., 2011).

Singapore

About the time that reports of EDs emerged in Malaysia and Hong Kong, cases of ED patients in Singapore also began surfacing (Ong, Tsoi, & Cheah, 1982; Ung, Lee, & Kua, 1997). Over the course of the 1990s, rates of EDs continued to increase significantly, and by 2003, evidence suggested that levels of body dissatisfaction among specific demographics (e.g., university students, Singaporean Chinese schoolgirls) were similar to Western rates (H. Y. Lee, Lee, Pathy, & Chan, 2005; Ung, 2003; Ung et al., 1997), although the prevalence of documented clinical EDs was still relatively low. While epidemiological data are not currently available, surveys of clinical samples suggest that ED rates are continuing on their upward trajectory (Tan, Karim, Lee, Goh, & Lee, 2013).

H. Y. Lee et al. (2005) explored the manifestation of different patterns of EDs according to ethnicity in an 8-year retrospective review of individuals with AN seen at an ED clinic between 1994 and 2002. Results showed that Malays (4.8% of cases) were underrepresented among AN cases in Singapore, as compared to Malaysians of Chinese (84.1%) and Indian (7.9%) descent. In contrast, ethnic Malays appear to account for a significantly larger proportion of bulimic and binge eating disorder cases (Ho, Tai, Lee, Cheng, & Liow, 2006).

A particular social experiment in Singapore during the 1990s is noteworthy. In an attempt to address the rising rates of childhood obesity the government established a compulsory school-based program called "Trim and Fit" (TAF). The scheme had modest success in terms of addressing childhood obesity and furthermore had unintended adverse consequences including increasing the risk of EDs among former participants. Moreover, H. Y. Lee et al. (2005) reported that 11.1% of the AN patients seen during the 1994–2002 period were former members of the program, and participants in the TAF club reported experiencing social stigma and teasing from their peers, both of which may have contributed to ED onset in vulnerable individuals.

Two recent cross-cultural studies comparing Singaporean women to Australian women, both with and without EDs, yielded inconsistent results. While one study revealed a significant association between greater acculturation to Western culture and increased body image disturbance (Soh et al., 2008), the second study found no significant relationship between acculturation and eating pathology (Mond, Chen, & Kumar, 2010).

Taiwan

Data on EDs from Taiwan suggest that the prevalence of clinical EDs is likely lower than in the West; however, rates of body dissatisfaction and dieting are similar (Tsai, 2000). In one survey of 336 female students at two universities in Taiwan, 43% of respondents were identified as at-risk for EDs based on their high bulimia score, high depression index, binge eating, use of laxatives/medicine for weight control, and a loss of 20 or more pounds (approximately 9 kg) in the last 6 months (Yeh et al., 2009).

A separate survey, of 1,605 female high-school students, indicated that 17.11% exhibited disturbed eating pathology based on elevated EAT-26 scores (Y. J. Chang, Lin, & Wong, 2011). Interested in the type of weight-control strategies used by Taiwanese adolescents, Liou et al. (2012) examined 15,716 adolescents aged 10–18 years from 120 representative schools and found that self-induced vomiting was more common among younger individuals. Frequent daily media use (e.g., TV, internet), night-time snacking, and consumption of fried foods were associated with greater self-induced vomiting (Liou et al., 2012). Research from Taiwan also provides support for the correlation between media exposure and thin-ideal internalization, and increased likelihood of body dissatisfaction among adolescents. Moreover, body dissatisfaction and media pressure contributed significantly to restrained eating and unhealthy weight control behaviors, even after controlling for all other variables (F. C. Chang et al., 2013).

India

The picture of EDs in India is complex. Some of the earliest cases of EDs reported in India appeared in the mid-1990s, when Khandelwal, Sharan, and Saxena (1995) described five cases of young, single Hindu females (15–22 years old) who presented with persistent vomiting, significant weight loss, amenorrhea, refusal to eat, and other somatic complaints. Likewise, Srinivasan, Suresh, Vasantha, and Fernandez (1995) published an early study of 210 Indian university students in which 14.8% of the sample presented with Eating Distress Syndrome (EDS). In both of these studies, a significant number of the patients did not show explicit "fat phobia" and appeared to have NFP-AN, much like individuals seen in Hong Kong around the same time.

Despite research indicating that university students report typical eating, shape, and weight concerns, which would predict more typical fat-phobic presentation of EDs (Sjostedt, Schumaker, & Nathawat, 1998), there is no evidence that unhealthy weight management is widespread in India (Basker, Mathai, Korula, & Mammen, 2013; Stigler et al., 2011) or that EDs are increasing in India (Mammen, Russell, & Russell, 2007). Moreover, these data suggest that the most common ED diagnosis in India is psychogenic vomiting (approximately 84%), which was seen mainly in subjects that developed the illness prepubescently, were first-born or only children, and came from middle or lower-socioeconomic status (SES) groups. Individuals diagnosed with AN (14.6% of all ED cases) were more likely to be from an upper-SES background, not be a first-born child, and have had ED onset during adolescence.

A 2012 survey of practicing psychiatrists well illustrates the complexity of clinical presentations of EDs in India. Of the 66 psychiatrists surveyed, 45 reported having seen patients with EDs in the last year, yielding a total of 74 ED cases. Among those patients, 32 had been diagnosed with AN, 12 with BN, and 30 with ED-NOS (Eating Disorders Not Otherwise Specified). When asked whether they believed EDs to be a "serious clinical issue" in India, 23.5% of psychiatrists surveyed felt EDs were increasing in Bangalore; 26.5% felt rates were

stable, and a plurality of 42% were unsure (Chandra, Abbas, & Palmer, 2012). Some data fail to support the notion that acculturation is associated with increased eating pathology (Bhugra, Bhui, & Gupta, 2000), whereas other studies suggest that urbanization and SES (often correlated with acculturation) are associated with increased risk for body weight dissatisfaction and dieting (Mishra & Mukhopadhyay, 2011; Talukdar, 2012). Given the size of the country and diversity of the population, it is likely that different trends characterize distinct segments of the Indian population, and much greater attention to cultural variations within India will be necessary to fully understand EDs within the country.

Pakistan

In the early 1990s, case studies of EDs began to emerge (Yager & Smith, 1993) and studies were conducted with schoolgirls in Pakistan and Lahore, respectively, the first of which identified one girl who met *DSM-III-TR* (*Diagnostic and Statistical Manual of Mental Disorders,* 3rd edition, text revision) criteria for BN (Choudry & Mumford, 1992), and the second of which reported a 1.6% combined prevalence for full- and partial EDs (Mumford et al., 1992). Notably, the latter study also produced some evidence that the students who were the most "Westernized" were at greatest risk of developing an ED. Recent studies from Pakistan indicate that roughly one third (33.73%) of university women report weight dissatisfaction (Sirang et al., 2013), and support the notion that media exposure exerts an overall negative effect on body image regardless of gender (Khan, Khalid, Khan, & Jabeen, 2011).

Thailand

Reports of EDs in Thailand are extremely limited, but include a 2006 cross-cultural investigation of eating-related attitudes and psychopathology among Thai ($n=101$), Caucasian Australian ($n=110$), and Asian Australian ($n=130$) university students at a university in Australia. Results showed that Thai students scored the highest on both the EAT-26 and Eating Disorders Inventory-2 (EDI-2), placing them at greatest risk of ED onset, while Australian Asians' and Caucasian Australians' scores were not significantly different (Jennings, Forbes, McDermott, Hulse, & Juniper, 2006). In addition, a 2012 case study of a 13-year-old Thai female with restrictive AN was notable since the girl hailed from Khon Kaen province, which has the lowest GDP nationally, thus reaffirming that girls from rural, low-SES settings are also at risk for EDs (Areemit & Patjanasoontorn, 2012).

China

Relative to many other Asian countries, mainland China began to industrialize and modernize slightly later, and thus has a comparably shorter history of documented EDs. In the early 1990s, several reports of ED cases emerged, including cases of BN among two females and one male (Schmidt, 1993) and one case of AN in a 4-year-old Chinese boy (K. Y. C. Lai, Pang, & Wong, 1995). Unlike initial cases in Hong Kong, all of these patients exhibited the hallmark "fat-phobia." It is worth noting that evidence from China suggests Asian males may be at greater risk for EDs as compared to males in the West.

Seeking to explain these findings, researchers have posited factors including sociocultural change, such as the transition from rural to urban life and the accompanying economic stress, individual factors such as intense workload and insufficient emotional support, and

Westernization. Additional factors that have been found to be associated with increased ED risk in general, include higher SES background (H. Chen & Jackson, 2008); preference for the thin-ideal and fatness concern (particularly in girls) (T. Jackson & Chen, 2008); concerns related to media idols, nonharmonious parental relationships and history of child abuse, body dissatisfaction and interoceptive awareness factors of the EDI, and anxiety (Liang, Guo, & Liu, 2008). As for environmental factors, T. Jackson and Chen (2010) provide strong evidence that adolescents with BN felt greater appearance pressure from mass media, their close relationships, and on account of fear of negative appearance evaluation. Meanwhile, other study findings indicate that appearance-based pressure from the media and adult relatives is predictive of body change behaviors among males and females (Tao & Zhong, 2010; Xu et al., 2010).

At the start of the new millennium, several larger community-based studies examining body image and eating behaviors among both male and female Chinese adolescents appeared (Huon, Qian, Oliver, & Xiao, 2002; Li, Hu, Ma, Wu, & Ma, 2005). Collectively, these studies suggested that the incidence rate for clinical EDs was somewhat negligible; however, cases of partial EDs were not uncommon and risk factors like body dissatisfaction and maladaptive eating behaviors were found to be widespread. Evidence from the most recent studies suggests that BN is becoming increasingly common and cases of clinical BN are rising among young Chinese females (Fan et al., 2010; Liao et al., 2010). Not only that, a large two-stage study of Chinese female university students in Wuhan (n = 8,444) yielded prevalence rates of 1.05% for AN, 2.98% for BN, and 3.58% for BED, all of which are on a par with those typically seen among similar demographics in the West (Tong et al., 2014).

Conclusions and Future Directions

Despite several challenges that hamper our ability to determine with greater precision and confidence the current incidence and prevalence of EDs in Asia—most notably, the absence of large, epidemiological studies—it is clear that EDs are now a recognized and increasingly prevalent form of psychopathology among young Asian females, and to a lesser extent, their male counterparts. We submit that the cumulative effects of "Westernization," globalization, and cultural transition (including changes in nutrition, gender roles, and urbanization) have contributed to the rise of EDs in Asia. The processes of change that a country typically undergoes in the course of building a robust global economy inevitably have the effect of bringing members from that particular society into progressively greater contact with the West, as well as the global community at large. In Asia, the steady proliferation of Western media images that condone and promote an unhealthy, ultrathin female body ideal, have helped put traditional feminine beauty ideals in flux, while the broader forces of societal transition have profoundly impacted gender roles and expectations. Although there is a common tendency in studies of EDs in non-Western countries to conflate the aforementioned processes of change with the concept of "Westernization," in actuality many of these economic and social developments are not uniquely Western—save of course, the actual exposure to culturally unique values and ideals directly imported (or adopted) from "Western" cultures.

The emergence of clinical EDs in Asia has occurred as part of a broader upsurge in dysregulated eating, which has likewise contributed to rising population BMIs and obesity rates. Current evidence indicates that these trends are likely to continue. As a result, women and men across Asia will be increasingly at risk for EDs. However, the existence of a significant evidence base from which to develop culturally informed prevention, early intervention, and

treatment programs, means that the field is faced with an extraordinary opportunity and challenge to develop and advance a proactive agenda that reduces risk for and effectively treats eating disorders in Asia. Such efforts will be essential to positively impact the future of EDs in Asia.

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7

Prevalence and Incidence of Eating Disorders in Underrepresented Countries

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Knowledge of eating disorders (EDs) has primarily emerged from the West, specifically Europe and North America, and in particular, urban settings. In the main, this has shaped our understanding of these conditions to the point that they have been viewed as culture-bound syndromes (Gordon, 2001). Interestingly, they still are—but not as was originally thought. It is in fact the emergence of EDs in societies and cultures outside of Western, urban settings that has prompted a reconceptualization of this understanding. While "culture bound" was previously understood to mean that sufferers would conform to a certain stereotype with regard to geography, setting, race, age, and gender, that is, Western, urban, Caucasian, adolescent/young adult, and female, such thinking has shifted. While adolescent/young adult females may still predominate among patient populations (Hoek & van Hoeken, 2003) the notion of geography, setting, and race has evolved based on the emergence of EDs in settings not previously associated with the existence of such conditions, such as Africa and Asia (Gordon, 2001).

A critical shift has occurred within the context of a change in how one understands "culture," accepting that any understanding of "culture" within this clinical context is likely to be limited in terms of the social sciences (Hoek & van Hoeken, 2003; Swartz, 2001). This understanding has moved beyond the narrow parameters of the past and is now universal in that the culture in question is that of *modernity* (Lee, 1996), which comprises a number of features that include: a capitalist economy, urbanization, lower fertility rates, increasing average weight, role choice for women, information technology, immigration, and body-oriented advertising. Such an understanding emerged from countries—for example, from Asia (Hong Kong, Japan, Singapore, Republic of Korea, China, India, Malaysia, the Philippines and Indonesia)—that were relatively underrepresented both in terms of the documented existence of EDs and the contribution to the EDs literature generally (Lee, 2001). In this regard, it was the then underrepresented countries that have most likely contributed to one of the major conceptual shifts in our understanding of the etiology of EDs at the level of societal factors. In essence, modernity brings culture change and cultures in transition have been identified as those increasingly vulnerable to the emergence of EDs (Nasser, Katzman, & Gordon, 2001; see also Chapter 6).

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The current chapter deals with countries that remain underrepresented with respect to EDs. Given that the preceding chapters deal with Western and Asian societies, it is prudent to focus on a number of major geographical areas to complete the picture. In this regard Africa and the Middle East as well as Central and South America would appear to do just that. The primary focus is on the incidence and prevalence of EDs. It has been noted that epidemiological studies are time as well as cost intensive (Hoek & van Hoeken, 2003). As will be seen from the content that follows, such data do not generally exist for countries in these underrepresented regions, with a few exceptions. This is not to say that the data that do exist do not provide useful information, but such information is more at the level of documenting the existence of EDs, establishing the potential for risk of developing EDs, or determining cases of EDs from among respondents screened for EDs.

While the need for data on the extent of EDs is not contested, one might ask whether the documentation of existence and risk provides clinicians, researchers, and government with sufficient information to treat, research, and plan. A specific concern has been a tendency for EDs to be relegated—so to speak—to the lower rungs of importance with regard to resource allocation for both clinical services and research funding, which would argue for the need to ascertain incidence and prevalence. In order to gather such data, the regions in question would require the necessary infrastructure and resources both to gather accurate data at source (incidence/clinical sites) and acquire data beyond (prevalence/community). This generally requires an investment in information technology and the provision of funding for skills development, based on an acknowledged need and prioritization by government. In essence, the study of EDs most likely constitutes a niche area within general psychiatry, with psychiatry being a generally stigmatized and underfunded discipline within medicine. In developing world settings, which would characterize the regions under discussion, one needs to be mindful of such limitations. This echoes an earlier commentary on the epidemiology of EDs, specifically related to the issue of funding for such research (Lee, 2003).

Given the preceding content, a pragmatic approach that views the status quo as providing useful information potentially serving as a basis to motivate for more detailed and scientifically valid information is called for. However, in light of the state of flux regarding diagnostic entities and classification of EDs (Walsh, 2009)—over and above the unresolved issue of culturally appropriate diagnostic criteria and research instruments (Fabrega, 2001; Lee, 2003)—this may be a shifting target not worth the investment of limited resources. Notwithstanding deficiencies, one should not lose sight of the value, in terms of insights into EDs locally and generally, that the published literature from countries in underrepresented regions has provided.

Africa

The documentation of EDs in the published literature about Africa dates back to the 1970s and is characterized by the clinical nature of the papers, with specific detail related to clinical characteristics of anorexia nervosa (AN) sufferers (all White females) at sites in both Cape Town and Johannesburg in South Africa (Beumont, George, & Smart, 1976; Norris, 1979; Touyz & Beumont, 1984). None of these papers spoke of either prevalence or incidence. In fact there is very little in the African literature that deals with such issues within the context of clinical cases of EDs. The closest one gets to an attempt at establishing the prevalence of AN is an incorrectly titled South African paper claiming to measure prevalence but in fact using a

weight measurement of more than 20% below desired weight among a sample of White high-school girls as a proxy for AN, with no clinical assessment, and establishing a prevalence of 2.9% (Ballot et al., 1981).

The tendency has been to focus on clinical samples within a hospital setting, either a general hospital or a specialized unit within a specialist hospital setting. While the South African literature involved exclusively hospital-based Caucasian (White) samples at this time, the 1980s and 1990s saw the publication of case reports of African (Black) patients from other African countries, thus documenting the occurrence of these conditions in settings and among females not generally thought to suffer from EDs (Buchan & Gregory, 1984; Fahy, Robinson, Russell, & Sheinman, 1988; Famuyiwa, 1988; Ilechukwu & Nhiwatiwa, 1988; Nwaefuna, 1981). This ultimately included a South African case series of Black female sufferers in the 1990s (Szabo, Berk, Tlou, & Allwood, 1995), emerging at a time of political change with a move to a postapartheid, representative democracy (Szabo & le Grange, 2001). Interestingly, it appears that the earliest mention of EDs in Africa is to be found in an Egyptian study (Okasha, Kamel, Sadek, Lotaif, & Bishry, 1977) where the authors make reference to two cases of AN among a range of "psychiatric morbidity" established among university students in Egypt, but provide no clinical information.

The aforementioned case reports from the 1980s were complemented by a study from Zimbabwe (Hooper & Garner,1986), which used an English language version of the Eating Disorders Inventory (EDI) completed by a racially diverse sample of female, adolescent, urban subjects. Although respondents were assumed to be conversant and comfortable with the content of the instrument this was not explored or accounted for (without any clear attention to issues of reliability or validity—both critical in assessing utility of derived data). A South African study of female university students ($n = 350$; 87.7% White), utilizing the Eating Attitudes Test (40 item), established a prevalence of 11.8% for those scoring above 30, described as being in the "anorexic range." A range of related behaviors was documented with approximately one third reporting binge eating and weight control behaviors (in the total sample), including vomiting (6.3%), laxatives (9.4%), strict diets (15.6%), fasting (15.3%), diet pills (12.2%), and exercise (67.5%); further, 76.8% felt they were overweight, with 91.6% wanting to lose weight. In addition, 1.2% ($n = 4$) reported having been diagnosed anorexic by a mental health professional, and 1.2% ($n = 4$) as bulimic (Shefer, 1987).

The 1990s heralded the emergence of further such papers from Africa, specifically Nigeria (Oyewumi & Kazarian, 1992a,b), Egypt (Nasser, 1986a, 1994a), and South Africa (Szabo & Hollands, 1997; Walker, Walker, Locke, Cassim, & Molefe, 1991) that focused on nonclinical samples in community settings with a focus on presumed vulnerable individuals, that is, adolescent females. The Nigerian study (Oyewumi & Kazarian, 1992b), utilizing the EAT-26, established an overall prevalence rate of 14.1% of a female sample of high-school, college, and university students scoring more than 20 (denotes disordered eating), which was interpreted as a proxy for anorexic behavior. Bulimic behavior in the same sample was assessed using the Binge Eating Questionnaire, and established rates of 21.16% for binge eating and 22.2% for vomiting (Oyewumi & Kazarian, 1992a). The earlier South African study (Walker et al., 1991) did not make use of an established questionnaire but established rates of weight and body shape dissatisfaction, in relation to weight status, among rural and urban Black schoolgirls as well as urban White and Indian (Asian origin) schoolgirls. The subsequent South African study by Szabo and Hollands (1997) established an overall prevalence rate of 21.66% for high-school girls scoring more than 20 on the EAT-26 (with a rate of 37.5% for Black respondents and 20.67% for White respondents). The Nigerian data comprised university students from

Lagos, and the South African study comprised students from a racially integrated, girls-only, private high school in Johannesburg. Both cities in either country would be regarded as the leading urban centers, most exposed to international trends from outside of Africa. The Egyptian studies made use of an Arabic version of the EAT-40, establishing varying rates of those scoring more than 30 (denoting disordered eating and described as EAT +ve). Among Arab students in London the rate was 22%, while among those in Cairo it was 12% (Nasser, 1986a). A subsequent study undertaken in Egypt among secondary-school girls established a prevalence of 11.4% ($n = 40$) scoring more than 30 (EAT +ve), with subsequent clinical interviews of the 40 EAT +ve respondents establishing that three met criteria for bulimia nervosa (BN) and a further 12 had features of a partial syndrome of BN.

Regarding the EAT-40, researchers while initially noting the instrument as having utility when utilized in a non-Western setting (Nasser, 1986b) subsequently concluded that instruments other than this one would be needed to assess bulimic behavior (Nasser, 1994b), specifically with translated versions, which in this instance was to Arabic. It should be noted that the application of an instrument in settings other than where it was derived may require cautious interpretation of findings without having subjected the instrument to a process of validation. However, Nasser (1994b, p. 94) argued that the EAT-40 should be retained as a screening instrument for both "dieting behavior and concerns about weight and shape in non-Western populations." Notwithstanding the apparent limitations, these studies gave an indication of the prevalence (given their cross-sectional nature) of aberrant eating attitudes and behaviors that might be viewed as suggesting risk and by implication gave a proxy for prevalence.

The aforementioned Zimbabwean study conducted among Black, White, and mixed race urban high-school girls in Harare by Hooper and Garner (1986) found that Black respondents demonstrated not only a drive for thinness but also bulimic tendencies. This suggested that Black adolescent females in an urban setting with Western exposure were potentially vulnerable to the development of EDs—but less so than their White counterparts. There was a somewhat different message emerging from the subsequent South African study (Szabo & Hollands, 1997), which was conducted some 10 years later, and demonstrated that Black adolescent females appeared to have a greater vulnerability. Further South African studies established prevalence rates of high scores on the Bulimic Investigatory Test, Edinburgh and the Eating Attitudes Test, with 5% for the former and 9% for the latter among university students in Cape Town, with Black students scoring consistently higher than White or mixed race students (le Grange, Telch, & Tibbs, 1998). A study of South African adolescent girls by Caradas, Lambert, and Charlton (2001) established an overall prevalence of 18.8% of abnormal eating attitudes using the EAT-26 (score >20) with no difference between the racial groups (Black, White, mixed race) but with 19.6% of Black respondents, 25.9% of mixed race respondents, and 32.9% of White respondents demonstrating significant body image concerns using the Body Shape Questionnaire (score >129). A study by Wassenaar, le Grange, Winship, and Lachenicht (2000, p. 225) whose purpose was "to establish the prevalence of eating disorder pathology in a cross-ethnic population of female students in South Africa" did not in fact do that; the research used the EDI and compared scores of the various subscales between the studied racial groups—demonstrating that eating pathology as measured by the EDI was present among Black university students. In essence, all of the aforementioned studies suggested that Black female populations (adolescent and young adult), by virtue of responses to Western-derived self-report questionnaires—some validated locally and others not—were either suffering from or at risk for EDs.

The aforementioned South African study by Szabo and Hollands (1997) led to a study that sought to extend the population being sampled, and did so by involving several multiracial

public sector girls-only high schools in Johannesburg (urban sample) as well as employing a translated version of the EAT-26 (Szabo & Allwood, 2004a). Specifically, the EAT-26 was translated into Zulu and administered to a rural sample; this constituted the first such translation and usage, with subsequent data provided related to validity (Szabo & Allwood, 2004b). The urban data demonstrated that 18.7% (Black equivalent to White) scored more than 20, with a figure of 3% among rural respondents. While such data appear intuitively correct, that is, with a continuum between urban and rural, the issue of validity of the EAT-26 was a consideration. Specifically, whilst validity for the translated version was demonstrated amongst rural respondents, they performed somewhat differently than urban White respondents, who were closest in performance to the original study by Garner, Olmsted, Bohr, and Garfinkel (1982). Concerns regarding validity in South African samples have been borne out in studies by Edwards and Moldan (2004) and le Grange et al. (2004).

Two further studies of African samples, from Ghana (Bennett, Sharpe, Freeman, & Carson, 2004) and Tanzania (Eddy, Hennessey, & Thompson-Brenner, 2007), used both questionnaires and structured clinical interviews. Bennett et al. (2004, p. 315) studied a sample of rural secondary-school girls and established that 10 of the sample of 668 were diagnosable with AN, that is, 1.5%, but without "a morbid fear of fatness nor a pervasive need to be slim." The authors proposed that "morbid self starvation" is the core feature of AN, with attribution varying according to culture (Bennett et al., 2004, p. 316). They further noted that the EAT (40-item version) was not a valid screening instrument in the population studied. The Tanzanian study, among a sample of young females ($n = 214$), established that 1.9% met modified criteria for AN, 4.7% had features consistent with an eating disorder not otherwise specified (EDNOS), with one participant meeting criteria for BN as given in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV*; American Psychiatric Association, 1994). The authors further noted that both media exposure and travel to Western countries were associated with ED symptoms.

There appear to be limited data on binge eating disorder (BED), with only one study from Tunisia formally assessing the presence of this disorder in an obese population compared to nonobese controls, using the Binge Eating Scale. In this study, the prevalence of BED was 40% among obese versus 8.3% among nonobese participants, with BED among obese participants being associated with an earlier onset of the obesity as well as a higher prevalence of anxiety, depression, and poorer quality of life (Ketata, Aloulou, Charfi, Abid, & Amami, 2009).

The Middle East

Using the "Middle East" as a geographical region poses some interesting challenges, specifically because this region encompasses religions and cultures that are congruent with countries located in the northern region of Africa, namely, Egypt, Libya, Morocco, Algeria, and Tunisia. The question is whether these countries should be considered within an African or Middle Eastern context. If one thinks in purely political terms, these countries have been in the forefront of change described as the "Arab spring," which would suggest a closer link with the Middle East. Interestingly, they are all Mediterranean countries, with each having links with Europe, and again from a political perspective there are those who would have them viewed within such a context. Of specific interest was the inclusion of Turkish samples—which were not considered—and Egyptian samples, which were considered but could have readily been included with the African content. On a personal note, when undertaking research among

Black female South Africans, the Egyptian data of Nasser (1986a,b, 1994a,b) were considered to be African for purely geographical reasons and her research has been considered as such for the purposes of this chapter, as has the paper by Okasha et al. (1977). Yet, as the studies involve Arab females, the citations are grouped, in PubMed, with studies from the Middle East.

The cited content emanates from a range of Middle Eastern countries including: Iran, Israel, the United Arab Emirates (UAE), Jordan, Egypt, Oman, Lebanon, and Saudi Arabia, with the majority involving female samples from Israel. As with the preceding section one sees that the published material predominantly includes prevalence rates established through Western-derived screening instruments, with some studies employing follow-up interviews to establish cases. There are also case reports.

The cited literature commences with the papers by Nasser (1986a,b, 1994a,b) involving use of the EAT-40 among Arab females, signaling the utilization of this instrument in this population and providing prevalence data that has been discussed in the previous section on Africa. Subsequent to Nasser's work (1986a,b, 1994a,b), a study in Saudi Arabia sought to establish the validity of an Arabic version of the EAT-26 (Al-Subaie et al., 1996). Of 129 grade 7–12 female students (in Riyadh), 25 were identified with abnormal eating attitudes giving a prevalence of 19%. Of these students, one was identified with AN by follow-up interview. A paper by Abou-Saleh, Younis, and Karim (1998) reported on five cases of AN in the UAE, of whom three were female and two male with two being UAE nationals, two Omanis, and one Sudanese. The authors contended that the emergence of AN in this setting was linked to Westernization.

This theme emerged in a study of Saudi schoolgirls (grades 7–11) in Riyadh (Al-Subaie, 2000). Using the EDI, 15.9% scored positively on the EDI-Drive for Thinness subscale, with this finding being associated with both individual (speaking a Western language, having lived in a Western country) and family factors (small family size, higher parental education, and better occupation). Iranian data on the prevalence of EDs were established using a Persian version of the EAT-26 with follow-up interviews, both clinical and structured, using the Eating Disorder Diagnostic Inventory (Nobakht & Dezhkam, 2000). It was established that from a sample of 15–18-year-old girls (n = 3,100) there was a lifetime prevalence of 0.9% for AN, 3.2% for BN, and 6.6% for partial syndromes. These findings were very similar to a later Iranian study that reported the presence of eating disorders in 11.5% of a sample of participants aged 14–55 (n = 1,204) with specific figures as follows: 0.8% for AN, 6.2% for BN, 1.4% for subthreshold AN, and 3% for subthreshold BED (Garrusi & Baneshi, 2012).

Comparing Omani teenagers and adults, varying rates of anorexic-like behavior (as measured by the EAT-26) and bulimic behavior (as measured by the Bulimic Investigatory Test Edinburgh) were established, with 33% of teenagers demonstrating anorexic-like behavior and 12.3% showing a propensity for BN (Al-Adawi et al., 2002a). Of interest was that males were almost as likely to demonstrate either tendency as females, with fewer than 2% of adults demonstrating any possible propensity towards an ED. A further study among Omani adolescents by Al-Adawi, Dorvio, Burke, Moosa, & Al-Bahlani (2002b) reported a prevalence of 29% scoring positively on the EAT-26, with 9.5% identified as anorexic cases with follow-up interview. Whilst this demonstrates the low positive predictive value of the EAT-26, the figure of 9.5% would appear to be significantly higher than would be anticipated in an adolescent sample. Once again, the utility of the EAT-26 in non-Western settings was raised, but more interesting was the finding that one third of these identified cases were male. Further to the case reports of AN in the UAE (Abou-Saleh et al., 1998), a study by Eapen, Mabrouk, and Bin-Othman (2006) among adolescent girls in the UAE (n=495) using the EAT-40,

established a prevalence of 23.4% of those scoring more than 30. Follow-up interviews of 50 of these respondents led to the diagnosis of AN in 2%. This once again demonstrated the low predictive value of the EAT. A later study by Thomas, Khan, and Abdulrahman (2010) established a prevalence of 24% of a sample (n = 228) of female university students in the UAE scoring more than 20 on the EAT-26.

Israeli data on rates of EAT-26 positive scores (>20) among university students who were Israeli-born, veteran immigrants (4–15 years since immigration), or new immigrants (3 years or less since immigration), from the former Soviet Union, demonstrated rates of 19.6%, 18.8%, and 7.9% respectively. The impact of acculturation was postulated to account for the similarities between Israeli-born and veteran immigrant students compared to new immigrant students (Greenberg, Cwikel, & Mirsky, 2007). Arab schoolgirls (grades 7–12) in Israel were the focus of an Israeli epidemiological study using the EDI—and comprised Muslim, Christian, and Druze religious subgroups (Latzer, Tzischinsky, & Azaiza, 2007). It was noted that 13% of the sample scored higher than the cut-off point of 14 on the Drive for Thinness subscale. In a similar study comparing Israeli-Arab and Jewish schoolgirls, Latzer, Tzischinsky, and Geraisy (2007) noted that the drive for thinness found in Arab-Israeli schoolgirls had not led to equivalent referrals to EDs clinics. It appears that the sample used in the study by Latzer, Tzischinsky, and Azaiza (2007) also completed the EAT-26, which was reported on separately (Latzer, Tzischinsky, & Azaiza, 2009) and found that 25% of the sample scored more than 20. The one and only incidence study emerges from Israel where Mitrany, Lubin, Chetrit, & Modan (1995) obtained nationwide data from all community and hospital-based adolescent psychiatric services for a 5-year period (1989–93), using *DSM-III-R* criteria (American Psychiatric Association, 1987) for EDs. Notwithstanding incomplete data, the mean annual incidence for Jewish Israeli females aged 12–18 was established to be 48.8 per 100,000 for all eating disorders, 29.0 for AN, and 8.6 for BN. The distribution for the 632 new cases identified over the period of study was as follows: AN 60%, BN 17%, AN and BN 4%, and EDNOS 20%.

Jordanian data were reported in two published studies by the same group of researchers (Mousa, Mashal, Al-Domi, & Jibril, 2010a,b), with 21.2% of a sample (n = 326) of adolescent girls aged 10–16 in Amman displaying body image dissatisfaction (Mousa et al., 2010a) and one third of a sample (n = 432) of adolescent girls aged 10–16 in Amman reportedly having EDs (Mousa et al., 2010b). No clinical assessment was undertaken in determining the presence of an ED, but 0.6% were reported as having BN, 1.8% as having BED, and 31% as having an EDNOS. These figures were based on participant responses to the Eating Habits Questionnaire (EHQ), an instrument used to detect EDs (Greenfield, Quinlan, Harding, Glass, & Bliss, 1987), with further evaluation using *DSM-IV-TR* criteria (American Psychiatric Association, 2000). For completeness, it needs to be noted that a case of AN in Kuwait has been described in the literature (Qadan, 2009).

Central and South America

This geographical region is predominantly Spanish speaking, with the exception of Brazil where Portuguese is the official language. Brazilian studies have dominated the research literature, with a number of publications from various regions within the country. Samples of surveyed adolescents have demonstrated various prevalence rates using screening questionnaires. A study by Hulsmeyer, Marcon, Santana, and Kallas. (2011) established that 15.97% of an adolescent sample of 407 had symptoms of AN (using the EAT) with those reporting body

image dissatisfaction having a 2.56 times increased risk. Using the Bulimic Investigatory Test, Edinburgh (BITE) in a sample of 652 female adolescents between the ages of 14 and 20, do Vale, Kerr, and Bosi (2011) reported that 25% displayed a "risk eating pattern" with 1.2% demonstrating signs of an "installed" eating disorder. The Body-Shape Questionnaire (BSQ-34) together with the EAT-26 were used in a study of university students in southern Brazil, finding that 8.3% demonstrated scores greater than 20 on the EAT-26, with 20% demonstrating body image dissatisfaction (Costa, Vasconcelos, & Peres, 2010). Somewhat different rates were established from a study using the EAT and BSQ, in a different Brazilian site in a younger population of school-going adolescents, with prevalence rates of 15.6% for "EAT +" (scores >20) and 18.8% being dissatisfied with their body image (Alves, Vasconcelos, Calvo, & Neves, 2008). An important cautionary emerged from a 4-year follow-up study of women with abnormal eating behavior—as determined by the EAT-26 and BITE—noting that while such behavior persisted, there was no association with the development of subsequent EDs and that these women were more at risk for other psychiatric diagnoses (Nunes, Olinto, Camey, Morgan, & de Jesus Mari, 2006).

Diet composition was the focus of a study among female adolescents, whereby using the EAT, 21% of 279 girls showed symptoms of AN associated with consumption of fewer calories and less iron but more protein than those without symptoms of AN (Dunker & Philippi, 2005). A study from southeast Brazil reported on risk for EDs whereby 1.4% of a sample of 1,251 boys and girls aged 7–14, surveyed using interviews of both children and parents to establish the possible presence of EDs as per *DSM-IV* criteria (American Psychiatric Association, 1994), were deemed to be at risk of having EDs, with females and older subjects being at higher risk among those at risk (Moya, Fleitlich-Bilyk, & Goodman, 2006). Among a sample of 7–19-year-old school children, 13.3% were determined to have "inappropriate eating behaviors" as measured by EAT scores, with 1.1% having possible BN, as measured by the BITE (Vilela et al., 2004). A population-based study (Nunes, Barros, Anselmo Olinto, Carney, & Mari, 2003), which involved researchers visiting 1,524 randomly selected homes and administering the EAT-26 and BITE to females between the ages of 12 and 29 (n = 513), established that 16.5% of the sample had disturbed eating behavior (score on the EAT-26 >20), with 2.9% having BITE symptom scores exceeding 21 (evidence of BN).

In one of the few studies of BED and body image perception among university students, results showed a prevalence of 12.9% using the Binge Eating Scale (Nicoli & Junior, 2011). However, binge eating was also the focus of a study using the Questionnaire on Eating and Weight Patterns among teenagers aged 14 to 19 (Pivetta & Goncalves-Silva, 2010) ,finding that the prevalence of such was episodes was 24.6%. A study of 12–19-year-olds established a prevalence of binge eating among 37.3% of a sample of 561, with older students being more "susceptible" to binge eating and younger students being more "susceptible" to weekly strict dieting—24.7%—with females more frequently engaged in either behavior (de Souza Ferreira & da Veiga, 2008). The study of binge eating has also extended to older female samples: a sample of 1,298 women aged 35 and older established the prevalence of binge eating (two or more episodes per week) to be 11.5% (de Freitas, Appolinario, Souza Ade, & Sichieri, 2008). A review of Latin American studies related to BED ultimately included 30, with 27 from Brazil, and one each from Argentina, Colombia, and Venezuela (underscoring this author's impression of Brazil being the leading contributor to the EDs literature in this region); reported prevalence rates were between 16 and 51.6% among obese people attending weight-loss programs (Palavras, Kaio, Mari, & Claudino, 2011).

Brazilian researchers have also focused on specific groups, for example, dancers. One study established that 31% of a sample of classical ballet dancers demonstrated risk behaviors for EDs, using Portuguese-language versions of the EAT and BITE (Ribeiro & da Veiga, 2010). A later study of elite professional female ballet dancers (n = 19) using a range of instruments (Mini International Neuropsychiatric Interview, somatoform and eating disorders module of the Structured Clinical Interview for *DSM-IV* disorders, the BITE and the Beck Depression Inventory) established that 15.78% had a lifetime diagnosis of AN (restrictive subtype) and 10.52% a current diagnosis of body dysmorphic disorder (Nascimento, Luna, & Fontenelle, 2012).

A number of publications from other South and Central American countries exist and cover a range of clinical and research areas. By country these include the following:

- *Chile:* the documentation of males with EDs (Salas, Hodgson, Figueroa, & Urrejola, 2011) and establishing the prevalence of muscle dysmorphia (13.6%) among male weight-lifters (Behar & Molinari, 2010).
- *Ecuador:* the administration of the EAT to students, with 14% demonstrating abnormal eating attitudes (Power, Power, & Canadas, 2008).
- *Colombia*: documenting the impact of traumatic experiences on women with eating disorders (Rodriguez, Perez, & Garcia, 2005).
- *Curacao*: documenting the existence of AN among a subculture of women of mixed race, high education, and high income (Katzman, Hermans, Van Hoeken, & Hoek, 2004).
- *Argentina:* a survey of eating behaviors among high-school students of whom 50% were dissatisfied with their weight, 43% had ever dieted, with 23.5% having bulimic behaviors (Leiderman & Triskier, 2004).
- *Venezuela:* a prevalence study of EDs in a random selection of 1,363 students from a population of 23,831 demonstrating rates of 0% for AN, 1.58% for BN, and 0.66% for BED (Quintero-Parraga et al., 2003).
- *Guatemala:* an exploration of ED development among a sample of 347 grade 5 and 6 girls in Guatemala City (Vander Wal, Gibbons, & Grazioso, 2008).

These publications demonstrate that whilst the literature from Central and South America is dominated by Brazil, the existence of EDs and related problems pervades this region.

Conclusions and Future Directions

In considering the three regions, each seems dominated by a specific country in terms of relative contribution to the published literature, namely South Africa in Africa, Israel in the Middle East, and Brazil in Central and South America. This may of course be an artifact related to publications cited in PubMed. However, intuitively each of these countries is indeed a regional leader with Brazil and South Africa being part of the BRICS (Brazil, Russia, India, China, and South Africa) grouping of countries (http://en.wikipedia.org/wiki/BRICS). Prior to South Africa becoming a member of BRIC (to form BRICS) collectively these countries—Brazil, Russia, India, and China—contributed 18% of global gross domestic product and were home to 40% of the world's population. The constellation of countries thus constitutes a powerful grouping both in terms of economics and representation of peoples. Most recently, there is discussion regarding the formation of a development bank to pool resources and thus increase global competitiveness and ultimately, power (Rosenberg, n.d.).

In terms of either prevalence or incidence of EDs, there is a general tendency across the regions to document cases of EDs or study samples of eating-disordered patients but limited formal data of either prevalence or incidence through epidemiological studies. Instead, selected samples are either subject to questionnaire-based screening, which establishes prevalence of ED-related behavior and, at best, potential for developing EDs, with some studies following such questionnaire-based screening with follow-up interviews of "high scorers" to ascertain the existence of cases.

What is clear is that while these regions may be relatively underrepresented in terms of available literature, one should not underestimate the relevance of EDs in these regions. EDs clearly exist. Moreover, it appears that the risk for the development of EDs, while variable based on the use of screening instruments, is present. In this sense the phenomenon of EDs is truly universal—with a global spread and emergence—which attests to the notion of "modernization" as a common context.

With the emergence of interventions such as deep brain stimulation with the potential to treat AN (Lipsman et al., 2013), there will no doubt be an increasing emphasis on biological aspects of causation and treatment of EDs. However, biology exists within a social context and in crystallizing the literature on EDs in underrepresented regions the "social" component is emphasized.

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Section III
Diagnosis

8

Diagnosing Anorexia Nervosa

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Anorexia nervosa (AN) is a complex syndrome characterized by physical, behavioral, and cognitive criteria and is associated with substantial medical morbidity and psychiatric comorbidity (*Diagnostic and Statistical Manual of Mental Disorders*, 5th ed. [*DSM-5*]; American Psychiatric Association, 2013). The illness commonly onsets during adolescence (see Chapter 55) and historically has affected predominantly females (see Chapters 5 & 27). Epidemiological studies (see Chapters 5 to 7) indicate lifetime prevalence rates of 0.3% among male and female adolescents (Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011), and 0.3% and 0.9% among adult males and females, respectively (Hudson, Hiripi, Pope, & Kessler, 2007). The etiology of AN is thought to involve a confluence of biological, psychological, and social factors (see Chapters 22 to 34 & 67). Although AN was first described in the medical literature in the 17th century (see Chapter 2), much about how to recognize, understand, and in turn, treat this illness remains unknown.

A core objective of the *DSM-5* (2013) is to provide a common language through which clinicians and investigators may diagnose, communicate, study, and treat people with mental disorders. As a clinical tool, the *DSM-5* is a practical guide wherein criteria for psychiatric illnesses are carefully outlined in order to facilitate diagnosis, case formulation, and treatment planning. This chapter explores the diagnostic criteria for AN and considers implications for clinical practice and future research.

DSM-5 Diagnostic Criteria for Anorexia Nervosa

According to *DSM-5*, AN is defined by three specific criteria: low body weight (criterion A); fat phobia or persistent engagement in behaviors that subvert weight gain (criterion B); and a disturbance in body image (criterion C).

The Wiley Handbook of Eating Disorders, First Edition. Edited by Linda Smolak and Michael P. Levine.

Criterion A

Low body weight is the hallmark feature of AN. Criterion A is defined by "restriction of energy intake…leading to significantly low body weight" (*DSM-5*, 2013, p. 338) for age, gender, and height. Weight loss in AN is often achieved through severely restrictive dieting or adherence to strict rules governing food choices. In addition, excessive physical activity can raise energy requirements and thus contribute to low body weight maintenance. Low body weight is considered to be one that is less than minimally normal, or less than what would be minimally expected for youth who should be growing. To define low weight, *DSM-IV-TR* (American Psychiatric Association, 2000) suggested a threshold of 85% or less of expected weight. Rather than provide a specific threshold to guide judgment of what constitutes low weight, the *DSM-5* allows this low weight criterion to be evaluated on the basis of clinical judgment. While an adult weight below a body mass index (BMI) of 17.0 kg/m^2 (e.g., 99 lb [~45 kg] at 5′4″ [1.63 m]) is definitely significantly low, depending on weight history and other physical signs, an adult BMI of between 17.0 and 18.5 kg/m^2 (e.g., 108 lb [49 kg] at 5′4″ [1.63 m]) or greater may also be considered low weight.

For younger patients, this criterion need not be met through weight loss but may instead be achieved by failure to make gains expected during a given developmental window. For children and adolescents, a BMI-for-age that is below the fifth percentile is clearly underweight. In addition, a BMI-for-age that is above the fifth percentile may also be considered low weight for a child who has fallen off his or her prior growth trajectory, as seen in the example of a 14-year-old girl with a BMI-for-age in the 7th percentile (ht.: 5′4″/1.63 m = 63rd percentile; wt.: 94 lb/42.7 kg = 17th percentile) who was historically above the 50th percentile growth curve for height, weight, and BMI-for-age. In addition, by leaving this category loosely defined, there is greater room to include clinical judgment of the severity of physical complications related to malnutrition (e.g., bradycardia, hypothermia, lanugo, alopecia, reproductive hormone abnormalities; Katzman, 2005; Rosen, 2010; see also Chapters 14 & 52).

Criterion B

Historically, fat phobia has been considered the sine qua non of AN (Becker, Thomas, & Pike, 2009; see also Chapter 2). Indeed, individuals with AN prototypically express an intense fear of weight gain and a terror that they will become fat. They often worry that once they begin to gain weight, gain will be interminable and they will lose control over their weight and shape. Yet, clinical data suggest that approximately one fifth of adults with AN deny or significantly minimize fat phobia and that these rates are likely higher among adolescents (Couturier & Lock, 2006; Eddy, Doyle, Hoste, Herzog, & Le Grange, 2008; Eddy et al., 2010; Thomas, Hartmann, & Killgore, 2013). Although the reasons behind denial or minimization are likely heterogeneous, data indicate that individuals with AN score higher on measures of impression management, raising the possibility that they may be concealing symptoms intentionally (Thomas et al., 2013).

In the *DSM-IV-TR* (2000) fat phobia was solely based on a patient's subjective report of intense fear of gaining weight or becoming fat. The *DSM-5* (2013) allows criterion B to be met on the basis of the presence of persistent behaviors that maintain low weight and/or subvert weight gain, based on the clinician's objective assessment. Inclusion of clinical observation of behaviors aimed at achieving/sustaining a low weight, in addition to what an individual may endorse, allows for recognition of a broader clinical presentation. For example, even those

patients who deny fat phobia due to limited insight, self-presentation biases, or poor treatment alliance/reluctance to self-disclose symptoms, may be characterized as meeting one of the key criteria of AN on the basis of consistent and pervasive engagement in a set of behaviors that contribute to low weight. Outside of restricting and exercising, such behaviors include compensatory actions intended to eliminate ingested calories by purging, through self-induced vomiting or misuse of diuretics, diet pills, or laxatives.

Criterion C

Individuals with AN also present with body image disturbance. Body image can be conceptualized as comprising attitude toward body and body size perception (see Chapter 22). Thus, for some, body image disturbance is manifested in a distorted internal representation of the size of one's body and an inability to accurately perceive or experience one's underweight shape. Others may acknowledge weight loss or small size but lack insight into or fail to appreciate the medical gravity of their low body weight. Assessment of body image disturbance through clinical interview or self-report questionnaires, such as the Eating Disorder Examination interview (EDE; Fairburn, 2008), can elicit the extent of body dissatisfaction (e.g., "How dissatisfied have you felt about your weight?") as well as distorted perception of body shape or weight (e.g., "Have you felt fat?"). Altered perception of one's body weight or shape—namely, seeing oneself as fat in spite of low weight—is thought to drive motivated weight loss behaviors. Measures such as the EDE questionnaire version (EDE-Q; Fairburn & Beglin, 1994) or the Body Checking Questionnaire (Reas, Whisenhunt, Netemeyer, & Williamson, 2002) can also be useful by indexing behavioral avoidance (e.g., "I avoid wearing form-fitting clothing") and body checking (e.g., "I pinch the flesh on my stomach"), which in concert with engagement in weight loss behaviors reinforce an overvaluation of weight and shape, contributing to a cyclical nature of the disorder (Fairburn, 2008). This criterion remains the same in *DSM-5* as in *DSM-IV-TR*.

Subtypes and Specifiers

After the AN diagnosis is conferred, subtype (i.e., restricting type or binge-eating/purge type) is assigned, and one or more diagnostic specifier(s) may also be noted. Diagnostic specifiers for AN represent an addition to *DSM-5*.

Subtypes

DSM-5 distinguishes between two mutually exclusive subtypes: restricting type AN (AN-R) and binge-eating/purge type (AN-BP). Individuals with AN-R engage in predominant restriction of dietary intake, while for those with AN-BP restrictive eating is punctuated by episodes of binge eating and/or purging. Presence of binge eating and purging among those with AN can increase medical risk (e.g., when low weight is compounded by purging behaviors; see Chapters 14 & 52). Further, binge/purge symptoms may be indicative of a protracted course (see Chapter 55) and be harbingers of increased impulsivity, substance use problems, and elevated suicide risk.

Yet, subtypes are recognized in order to convey information about predominant *current* symptom presentations. Prospective studies of adolescents suggest dieting tends to precede development of binge eating and/or purging (Field, Camargo, Taylor, Berkey, & Colditz,

1999; Stice, 2002; Stice, Agras, & Hammer, 1999), and longitudinal follow-up of clinical samples (see Chapter 55) indicates that the course of AN frequently involves migration over time from restrictive to binge and/or purge behaviors (Eddy et al., 2008; Milos, Spindler, Schnyder, & Fairburn, 2005; Tozzi et al., 2005). In fact, more than half of those with AN-R will likely develop AN-BP or BN over time (e.g., Eddy et al., 2008). Therefore, given the likelihood of diagnostic migration, the AN subtype captures current symptom presentation but is of limited predictive validity.

Partial and Full Remission Specifiers

For patients who previously met full criteria for AN, specifiers of partial or full remission should be assigned if symptomatic improvement is observed for a sustained period of time. Partial remission is noted when weight recovery is achieved but the cognitive or behavioral symptoms (criteria B and/or C) persist, and full remission is indicated when all symptoms are absent (see Chapter 64). The longitudinal course of AN can be variable; some patients will achieve full remission, while others may be chronically ill, or achieve partial remission before relapsing or recovering, and even relapsing again (Steinhausen, 2002; see also Chapters 12 & 55). Recognition of partial remission allows for acknowledgment of a significantly improved state without requiring a new diagnosis (Eddy, Swanson, et al., 2010). Further, notation of full remission also allows clinicians to be aware of the patient's history (e.g., in the event of a current diagnosis of Major Depressive Disorder) and may indicate that she or he is still vulnerable to relapse (Eddy et al., 2007; Eddy, Dorer, et al., 2008). Given that the longitudinal course of AN involves waxing and waning of symptoms, recognition of course specifiers conveys clinically meaningful information.

Severity Specifiers

Although AN is universally associated with increased medical and psychiatric risks, *DSM-5* recommends four levels of severity on the basis of current low BMI (or BMI percentile for children and adolescents) using World Health Organization (1995) guidelines. *Mild* denotes a BMI of 17 kg/m^2 or more; *moderate*, a BMI of 16–16.99 kg/m^2; *severe*, a BMI of 15–15.99 kg/m^2; and *extreme*, a BMI of less than 15 kg/m^2. These levels may be flexibly adapted and integrated with collateral clinical data such as clinical symptoms, functional disability, and the need for supervision. Like subtype and remission specifiers, severity reflects current presentation and can be particularly useful in clinical communication and treatment planning, for example, informing decisions about appropriate level of care (see Chapter 50).

Notable Revisions in AN Criteria from *DSM-IV-TR* to *DSM-5*

While the hallmark features of AN, including low body weight, aberrant eating behaviors that maintain low weight, and a disturbance in body image, have persisted across diagnostic manuals over the past 33 years, a number of specific changes in the *DSM-5* AN criteria reflect research findings and clinical consensus amassed over the nearly 15 years since the publication of *DSM-IV-TR* (Keel, Brown, Holland, & Bodell, 2012). The revision of criterion A to allow low weight to be determined by clinical judgment and consideration of the range of physiological sequelae of low weight may promote detection of individuals with AN who formerly would

have narrowly missed the low weight threshold. Furthermore, the slight change in the wording from *DSM-IV-TR*, which described an individual's "refusal" to maintain a healthy body weight, avoids the possibly blaming connotation of the term "refusal," and allows for diagnosis in the context of patients who are low weight but actively engaged in treatment (e.g., and therefore no longer "refusing"; Becker, Eddy & Perloe, 2009). The rewording of criterion B now allows for recognition of individuals engaging in motivated weight loss/low weight maintenance behaviors, even in the absence of voiced fat phobia.

In addition to the modifications inherent in the descriptions of criteria A and B, the most notable change from *DSM-IV-TR* (2000) to *DSM-5* (2013) is the omission of criterion D, which required amenorrhea presence for three consecutive months. This change was made because amenorrhea is understood to represent a physiological sequela of malnutrition that presents for many but not all with AN (Roberto, Steinglass, Mayer, Attia, & Walsh, 2008). A further notable change with *DSM-5* is the addition of diagnostic specifiers, which function as a useful descriptive characteristic for clinical purposes. The intention of all of the revisions is to better reflect the clinical reality of the patient presenting with this syndrome, and in turn to promote early detection and improve treatment outcomes.

Considerations for Diagnostic Presentation

While the *DSM-5* aims to provide comprehensive and otherwise valid criteria to frame diagnosis, individuals with AN may have a complicated presentation. It is imperative that clinicians consider the variability in presentation to help guide an informed decision about assessment and treatment. In order to provide a context to understanding application of a diagnosis, the roles of detection, development, gender, and comorbidities are detailed in this section as particular considerations.

Detection

Recognizing and diagnosing AN can be surprisingly complex. Physically, the prototypic patient presents with extreme weight loss, emaciation, and signs of malnutrition (e.g., lanugo, bradycardia, hypotension; see Chapters 14 & 52). For some patients, primary care physicians and pediatricians are often on the front line in terms of early recognition and diagnosis of AN. For example, low weight is often detected by physicians who notice a change—weight loss, weight-for-height statistics that fall off the growth chart, or a change in weight/growth trajectory—during a routine visit. In other instances, family members, partners, or friends observe changes in their loved one's weight, eating or activity patterns, or mood. However, AN often goes unnoticed because patients and family members minimize, or are in denial about, symptoms. Physicians and others in the individual's environment can play a crucial role in recognizing symptoms of AN.

As the broadened AN criteria in *DSM-5* are incorporated into standard practice, it is likely that more affected individuals will be identified and referred for appropriate treatment (Ornstein et al., 2013). Ideally, physicians will track their patients' height and weight over time in addition to implementing brief eating disorder screenings at annual physicals (Rosen, 2010). With the revision of criterion A, a specific expected weight threshold to categorize a low weight no longer exists. Thus, physicians, as well as community partners (e.g., teachers, school nurses, and school counselors), hold more power in using clinical judgment to make sure that patient symptoms do not go unnoticed. They may choose to utilize screening tools (see Chapter 38), such as the SCOFF

(Morgan, Reid, & Lacey, 1999) or EDE-Q (Fairburn & Beglin, 1994), or simply ask a short set of questions that would provide enough information to suggest whether or not the patient should be referred for an evaluation with a mental health professional.

Developmental Considerations

Anorexia nervosa presentation may vary across childhood and adolescence (WCEDCA, 2007; see also Chapter 13). Under the *DSM-IV-TR* (2000), children and adolescents often failed to meet full criteria for AN because pediatric AN often presents with characteristics such as a failure to gain weight rather than marked weight loss, and a minimization or denial of body image dissatisfaction or weight/shape overvaluation. Rather than considering only weight loss or relying solely on comparisons to population norms, clinicians working with children and adolescents should review growth charts to develop a growth trajectory to assist in determining whether weight or height has failed to progress as anticipated (Rosen, 2010). In addition, children and adolescents often lack the capacity to self-report symptoms because of developmental, cognitive, and emotional factors (Weaver & Liebman, 2011). These factors may also contribute to an inability to fully comprehend clinician questioning, to limited insight into the severity of their symptoms, and an inability to explicitly endorse psychological symptoms (e.g., fear of weight gain). Consequently, behavioral markers (e.g., failure to gain weight) can be more salient indicators of AN than endorsement of cognitive criteria (e.g., excessive influence of body and weight on self-evaluation) in pediatric patients (see Chapter 13).

Gender Considerations

Historically, males with eating disorders have been underrepresented in clinical populations and research has predominantly focused on females; males may be less frequently screened for EDs, and, further, they may be less likely to seek out treatment than females (see Chapter 37). Recent epidemiological research using *DSM-IV-TR* (2000) criteria suggests that males comprise approximately 25% of those with lifetime AN (Hudson et al., 2007), and these relative rates may be even higher among adolescents (see Chapter 5). AN features are generally similar in males and females. Physically, males show lowered testosterone levels (Goldstein, Herzog, Misra, & Sagar, 2008) and often have a premorbid weight history (Andersen, 1999; Gueguen et al., 2012). Psychologically, males with AN may be more likely to focus on certain parts of the body or on muscle mass rather than weight (Weltzin et al., 2005). No longer starkly gender-specific (i.e., through removal of the amenorrhea criterion), the *DSM-5* may increase access to services and direct research for males with AN. The Eating Pathology Symptom Inventory (Forbush et al., 2013) and future evaluation instruments may additionally aid in these processes.

Differential Diagnosis

Both physical and psychiatric conditions can be associated with symptoms that mimic or resemble those of AN. Among the physical disorders to be evaluated and ruled out are endocrine disorders such as diabetes mellitus and thyroid disease; gastrointestinal conditions such as inflammatory bowel disease, malabsorption, peptic acid diseases, and motility disorders; and neurological conditions including brain tumors. In addition, conversion disorders, mood disorders, anxiety disorders (e.g., obsessive compulsive disorder), and schizophrenia are among the psychiatric disorders that may present with manifest weight loss and binge eating or purging.

Case Study

In determining diagnosis, there are often many factors to consider. A composite of cases is presented as a case study with the aim of providing a clinical picture of how to apply a careful and comprehensive diagnosis.

Presenting Information

Eleanor was a prepubertal 12-year-old female who was entering the 7th grade. She was referred for evaluation by her pediatrician who, like her parents, was concerned about maintaining her growth trajectory and about Eleanor's compulsive exercising. She had steadily maintained a height and weight around the 40th percentile, but at her yearly physical it was determined that she had dropped to the 11th percentile. At 73 pounds (33.1 kg) and 4 feet 9½ inches (1.46 m), she was referred for outpatient care. Her pediatrician reported that Eleanor's school had noted that she was frequently throwing away her lunch, and, per mother, Eleanor was just a "picky eater," sometimes skipping dinner because she said she did not like what was available.

With no history of prior psychiatric or therapeutic treatment, Eleanor, accompanied by her biological mother, was evaluated at an eating disorder outpatient center. She presented as outgoing, actively engaging in conversation and making direct eye contact. Physically, her appearance was thin and by this point she was in the sixth BMI percentile for her age (now 71 lb/32.2 kg at 4 feet 9½ inches tall). Her vital signs were also notable for a low resting heart rate as well as orthostatic hypotension. Eleanor described that she was a newly "health motivated" person but was not worried about her behavior. In addition to attending 2-hour soccer practices 5 days a week, she had started running 4 miles a day every morning. Eleanor's mother reported that Eleanor had also started reading fitness magazines during the ride home from school. When questioned, Eleanor reported that her coach said that she and her teammates needed to stay in shape outside of practice so that they would be ready for their summer tournaments.

Mother also reported that, during the prior 2 weeks, Eleanor had begun to eat snacks late at night; mother found 10 empty cracker boxes and a jar of peanut butter under Eleanor's bed when cleaning her room. Upon further conversation with Eleanor, she described her nightly eating as something she hated but felt she could not stop. She would look in her mirror for up to an hour afterwards, thinking the entire time that all she wanted to do was to "get a new body."

Clinical Discussion

Eleanor's evaluation involved both medical and psychological assessments, and the integration of patient self-reports with information from collateral informants. It is the integration of these data that determined that a diagnosis of AN was appropriate. Although there was evidence of binge eating in the past 2 weeks, her presentation was primarily restricting, resulting in a restricting type. A severity specifier of "moderate" was assigned to best capture the seriousness of her physical complications (e.g., bradycardia and orthostatic hypotension), in spite of her BMI percentile being greater than the fifth.

In Eleanor's case there are various developmental factors to consider. There are also other questions for clinicians to think about as they see clients and consider application

of a diagnosis. How would Eleanor's case have presented differently if she had been younger or older, such as an 8-year-old versus an 18-year-old or young adult? If Eleanor were male, would large amounts of exercise be ignored when seen through the lens of a gender norm? Would comorbid pathologies such as depression or obsessive compulsive disorder (OCD) have either clouded her clinical presentation or made her symptoms appear more severe? Also, a diagnosis of AN-R reflected Eleanor's *initial* presentation. Her symptoms may change over time, and establishing a longitudinal understanding of a client's history can greatly aid in guiding the treatment process, particularly in providing information about risk and vulnerabilities for relapse over time (see Chapter 55).

Conclusions and Future Directions

Diagnosis is a tool that informs clinical work and guides patient-oriented research. *DSM-5* criteria for AN capture a complex syndrome. Revisions to the diagnostic criteria reflected in *DSM-5* are supported by more than a decade's worth of nosological research and result in a more nuanced clinical picture that better reflects the reality of individuals across the age spectrum with this heterogeneous illness (Attia & Roberto, 2009; Thomas, Vartanian, & Brownwell, 2009; Wonderlich, Joiner, Keel, Williamson, & Crosby, 2007). Preliminary studies suggest that the *DSM-5* diagnostic criteria for AN can be reliably applied (e.g., kappa = .81–.97 for AN using proposed criteria prior to *DSM-5* publication; Sysko et al., 2012). The next wave of nosological research will build on extant work that has carefully characterized the AN phenotypes in order to identify the neurobiological and genetic underpinnings of this likely heterogeneous illness. Ongoing research investigating biomarkers (e.g., neural correlates, genetics; Kaye et al., 2009; Root et al., 2011) of AN are promising, yet inconclusive (see Chapters 17, 28, & 30). Further, longitudinal studies to examine the predictive validity of the diagnostic categories (and AN specifiers) can inform our understanding of what interventions work best, for whom, and in what time course. In concert, clinical observations and strategic research approaches such as the National Institute of Mental Health's Research Domain Criteria (RDoC) initiative (NIMH, 2011) will continue to refine criteria and the overarching classification scheme. In turn, this work will facilitate detection, treatment, and prevention of eating disorders.

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9

Diagnosing Bulimia Nervosa

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Since its initial description by Russell (1979), just prior to its inclusion in the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.: *DSM-III*; American Psychiatric Association, 1980), bulimia nervosa (BN) has represented a particular diagnostic challenge (see Chapter 3). There are several reasons for this. First, there remains substantial stigma and shame associated with BN symptoms and, at least partly for this reason, most individuals are quite secretive about their symptoms early on (Hepworth & Paxton, 2007). It is well recognized that there is typically a significant lag (usually several years) between the onset of BN symptoms and the point at which the person with BN first reveals the symptoms to someone else. A second factor that makes the diagnosis more difficult on average than, say, the diagnosis of AN, relates to the weight pattern observed in BN. When an individual develops anorexia nervosa (AN), substantial, usually rapid weight loss occurs, which is readily observable (see Chapters 2 & 8). Such a pattern of marked weight loss typically does not occur in BN. Furthermore, while specific signs of BN are observed in some individuals, these are not universally present and thus not always useful. The signs tend to be relatively subtle and are not encountered in many (perhaps most) people with BN. Therefore, physical signs are not the aid to diagnosis that they are in AN (see Chapters 8, 13, 14, & 52).

This chapter explores the diagnosis of BN through a historical review of changes in the *DSM*'s diagnostic criteria from 1980 to the present, and through consideration of the differential diagnosis of BN, anorexia nervosa-purging type (AN-P), and Binge Eating Disorder (BED). The use of interview, physical, physiological, and questionnaire-based assessments will then be discussed, followed by a case history that demonstrates the diagnosis of BN. The chapter concludes with a summary and a list of important questions meriting further research.

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History of the Diagnosis of Bulimia Nervosa

DSM-III and *DSM-III-R*

As noted, BN first appeared in *DSM-III* (1980). In this initial criteria set, binge eating was required, but purging was only one of a number of potentially associated symptoms and was not required. Also, binge eating was described as rapidly eating a large amount of food in a short period of time; loss of control eating was not specifically required.

In *DSM-III-R* (American Psychiatric Association, 1987), both binge eating and purging/compensatory behavior were required for a BN diagnosis. Examples of purging or compensatory behaviors included self-induced vomiting, use of laxatives or diuretics, strict dieting/fasting, or vigorous exercise to prevent weight gain. "Lack of control over eating" during binges was also included as part of the definition. Overconcern with shape and weight was added to capture cognitive aspects of BN, although no guidance as to the definition or assessment of these was provided. In recognition of the fact that experimentation with bulimic behaviors for brief periods at the beginning of the college-age years is fairly common (Drewnowski, Yee, & Krahn, 1988), a frequency criterion (a minimum of two binges per week for 3 months) was added.

DSM-IV

The criteria for BN in *DSM-IV* (American Psychiatric Association, 1994) changed modestly from those seen in *DSM-III-R*. *DSM-IV* criteria require recurrent episodes of binge eating characterized by "eating, in a discrete period of time (e.g., within a 2-hour period) an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances" (p. 549), as well as "a sense of lack of control over eating in the episode (e.g., feeling that one cannot stop eating or control what or how much one is eating)" (p. 549). In addition, "recurrent inappropriate compensatory behavior in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting; or excessive exercise" (p. 549) is also required. The frequency criterion of at least 3-month duration of symptoms, with symptoms occurring at least twice per week was maintained. The criteria also require self-evaluation to be "unduly influenced by shape and weight" (p. 550). *DSM-IV* recognizes that AN "trumps" BN; that is, AN would be the diagnosis assigned to someone who met criteria for both AN and BN. In *DSM-IV* both purging- and nonpurging subtypes are identified, with the use of the compensatory behaviors of fasting or excessive exercise being assigned to the nonpurging subtype.

How Did Criteria Change in *DSM-5*?

Limited changes were made to *DSM-5* (American Psychiatric Association, 2013) criteria for BN. The criteria were liberalized to allow for binge eating and purging that occurs once per week rather than twice per week during the 3-month period. The goal of this change is to capture individuals under the BN diagnosis who come close to, and appear to meet the spirit of, the BN criteria but do not quite fall into that group. The rationale for this change rested on studies suggesting little difference between people meeting full or subthreshold criteria for BN (Crow, Agras, Halmi, Mitchell, & Kraemer, 2002). It remains to be seen whether this change will substantially broaden the number of people diagnosed with BN, although initial indications from small field trials suggest it may not change the rate of BN very much (Keel, Brown, Holm-Denoma, & Bodell, 2011).

Criterion D ("Self evaluation is unduly influenced by body shape and weight" (American Psychiatric Association, 2013, p. 345) was unchanged in *DSM-5*. This critically important cognitive criterion all too often receives little attention, perhaps because it is not as readily quantified as the behavioral (binge and purge) criteria. An additional change in *DSM-5* is the provision of current severity ratings based on frequency of purging: mild (1–3 episodes/week), moderate (4–7 episodes/week), severe (8–13 episodes/week), and extreme (14+ episodes/week).

Differential Diagnosis of BN

Several alternative diagnoses might be considered when the presenting picture suggests BN. One possibility is AN, purging subtype (AN-P; see Chapter 8). Individuals with AN-P engage in purging behavior, and may have some degree of binge eating (although, in my clinical experience, subjective binges may predominate). The critical diagnostic difference would be weight status. Those failing to maintain a minimally adequate weight-for-height (BMI; see Chapter 8) would be diagnosed with AN-P, not BN. However, application of these criteria can be complicated. For example, say a young woman whom you are assessing is 5'4" tall (1.63 m) and 16 months ago weighed 155 pounds (70.3 kg; BMI = 26.6). Now, she meets all the criteria for BN and weighs 120 pounds (54.4 kg; BMI = 20.6). Thus, her BMI is normal, yet she has lost 22.3% of her body weight in the past 16 months. This common clinical situation might appear to meet the spirit of the criteria for AN-P. Another conundrum relates to the application of a weight cut-off for diagnosis of AN versus BN. While *DSM-5* (and, actually, *DSM-IV* before it) does not specify a weight cut-off, such cut-offs are nonetheless often employed (see Chapter 8). But in doing so, someone might move between the diagnoses of AN and BN based on improving hydration, or resolving ankle edema, or even relief of constipation. Clearly, such rapid diagnostic changes cannot reflect a meaningful reality.

A second consideration is Purging Disorder (Keel & Striegel-Moore, 2009). Purging disorder, one component of what was called Eating Disorder Not Otherwise Specified (ED-NOS) in the last four iterations of *DSM*—and now an example of Other Specified Feeding or Eating Disorder in *DSM-5* (see Chapters 4 & 11)—describes a clinical pattern very similar to that seen in BN, but without any binge eating. Careful history-taking will clarify this distinction. Binge Eating Disorder (BED; see Chapters 4 & 10) is a third consideration. Again, careful history-taking will help to make the correct diagnosis. Some care is warranted, especially for clinicians and patients contemplating application of pharmacotherapy (see Chapter 59). Individuals with purging symptoms are typically counseled to avoid bupropion due to elevated seizure risk, which is thought to be due to electrolyte disturbances from purging, not binge eating. As bupropion gains more attention as a potential weight loss strategy (Gadde et al., 2001; Greenway et al., 2010), individuals with binge eating will have greater and greater exposure to it. Thus, accurate delineation of purging becomes important.

Last, one must recall that BN symptoms can sometimes occur as a part of another psychiatric diagnosis, such as borderline personality disorder. A BN diagnosis would typically be made only if frequency criteria are met.

A critical issue to recall with regard to differential diagnosis is that the frequency of diagnostic crossover between ED diagnoses is high, especially early in the illness course (Agras, Crow, Mitchell, Halmi, & Bryson, 2009; Tozzi et al., 2005; see also Chapter 55). Thus, a given individual may meet several of these diagnoses over time. This has both clinical and

theoretical implications. From the clinical perspective, the high rate of crossover means the clinician must remain alert to the possibility of fluctuation in symptom patterns. Theoretically, frequent crossover could be interpreted as supporting the view of a broader underlying eating disorder (ED) construct with varying presentations (e.g., BN at one point, BED at another).

Interview Approaches to Diagnosis of BN

The diagnosis of BN during clinical interviews can be a substantial challenge. Several factors contribute to this. The first is secrecy. As noted above, for many individuals there is substantial shame and embarrassment attached to having BN symptoms, which tends to lead to secrecy about, or minimization of, symptoms. A second issue is ambivalence. There are clearly individuals with BN who are willing and perhaps even eager for treatment, yet ambivalent about making clinical change. Such individuals may talk about their symptoms if asked, but may not be likely to raise the symptoms of their own accord.

The third challenge is one of case finding. In my experience, among many clinicians, knowledge about EDs is not high. The relative lack of effective ED treatments in some geographical areas probably further contributes to this; clinicians may be unlikely to ask extensively about problems for which they feel they have no effective treatment resources to offer. Furthermore, like many educated people (Gordon, Perez, & Joiner, 2002), clinicians often have ideas about the kinds of individuals who are most likely to exhibit ED symptoms, for example, that they are young, relatively lean, White, relatively affluent females (see Chapter 25). Although there may be some useful truth in these perceptions (see Chapter 5), this assumption inevitably leads to diminished case finding among those who do not fit this stereotypical view (e.g., males, individuals who are overweight or obese, or non-Caucasians).

Several strategies can be used at the time of interview review to try to avoid such pitfalls. Most importantly, making the diagnosis of BN is an area where fairly directed questioning is useful. Most clinicians are taught to use very open-ended approaches to interviewing. This works well in many instances but for the diagnosis of BN it may fall short. A reasonable strategy would be to start with the broad question, "Is eating an issue for you?" and then move on to ask very specific follow-up questions about self-induced vomiting, loss of control eating, compulsive overeating, binge eating, and so forth. This runs counter to what most clinicians are trained to do, but in the diagnosis of EDs (as in some other areas), such approaches may be critical.

In order to do this effectively, the clinician must be comfortable and practiced in asking the questions. Questions such as those on the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 2002) certainly would work in this regard. The SCID is a semistructured diagnostic interview covering a wide range of psychiatric diagnoses. The SCID provides specific questions to be asked for each diagnosis (e.g., "Have you often had times where your eating was out of control?"). It is not critical that questions from the SCID be used; what is critical is that the clinician is prepared to ask questions about purging, binge eating, dietary restriction, and so forth, that feel and sound natural when asked by her or him.

Physical Assessment and Diagnosis of BN

There are several identified physical signs of BN that are classic and probably indicative of the disorder (i.e., relatively pathognomonic), though these signs may not be displayed in most people (see Chapters 14 & 52). The first of these is Russell's sign, an excoriation or

raw area on the back of the hand (Daluiski, Rahbar, & Meals, 1997). This is classically thought to be caused by using the finger to self-induce vomiting, resulting in a combination of abrasion and stomach acid exposure, thus causing the lesion on the back of the hand. Obviously, for individuals who do not self-induce vomiting using a finger, this sign would not be found.

A second common finding is parotid or submandibular salivary gland hypertrophy, resulting in swelling of the cheeks (Mandel & Kaynar, 1992). This usually occurs equally on both sides and is typically painless, distinguishing it from some other parotid gland problems. The swelling typically resolves over the course of successful treatment.

Third, there are classic findings on dental examination, including a shortening of overall teeth height and loss of the white enamel on the surfaces of the lingual (or tongue-side) surfaces in the teeth (Romanos, Javed, Romanos, & Williams, 2012). While most physicians do not receive extensive training in this kind of dental assessment, the findings are readily apparent to dentists in many cases, and thus dental visits provide an opportunity for BN case-finding (Hague, 2010).

Biochemical Assessment and Diagnosis of BN

There are many medical complications of BN and quite a number of those are detectable through testing of blood samples (see Chapters 14 & 52). As such, a substantial amount of work has been conducted trying to identify biochemical markers that could be useful in the diagnosis of BN. Serum electrolyte abnormalities, most notably low potassium, have received substantial attention. Serum electrolyte changes are common (Greenfeld, Mickley, Quinlan, & Roloff, 1995), and one study examined the sensitivity and specificity of low potassium in females with BN and healthy controls (Crow, Salisbury, Crosby, & Mitchell, 1997). Hypokalemia (abnormally low potassium) was found in about one fifth of individuals with BN and in none of the matched young, healthy female controls. As such, hypokalemia appears to be a highly specific but not particularly sensitive marker for BN in young, healthy individuals (see Chapter 38).

A second area of interest has been salivary amylase, an enzyme that is produced by salivary glands. It is thought that increased salivary amylase production would accompany the swollen parotid glands occurring in response to recurring vomiting behavior. A number of studies have documented that such a relationship exists (Gwirtsman, Guze, Yager, & Gainsley, 1990; Mitchell, Pyle, Eckert, Hatsukami, & Lentz, 1983; Walsh, Wong, Pesce, Hadigan, & Bodourian, 1990). However, elevated salivary amylase may not be sensitive or specific enough to be clinically useful, as it occurs in connection with increased stress in general (Takaia et al., 2004).

A third area of interest has been the examination of changes in urine electrolyte patterns as a particularly sensitive marker of purging behavior (Crow, Rosenberg, Mitchell, & Thuras, 2001). Crow et al. (2001) found that a urine sodium to urine chloride rate greater than 1.16 identified about half of those with BN (51.5%) with only a 5% false positive rate. While determination of this ratio has received limited usage clinically, existing evidence suggests that it may be the most sensitive and specific marker of binge eating and purging developed to date. One significant limitation, however, is that while urine electrolytes measures are widely available, they are rarely obtained.

Psychometric Assessments and Diagnosis of BN

A final approach to diagnosis of BN involves the use of questionnaires and structured diagnostic assessments (see Chapters 38 & 40). In research settings these represent the gold standard for making diagnoses—they support consistent, reliable, and valid diagnosis, in that psychometric studies generally support their reliability and validity. Although they are rarely used clinically for diagnostic purposes, for individuals who are clearly recognized to have disordered eating, structured diagnostic assessment such as the Eating Disorder Examination (Berg, Peterson, Frazier, & Crow, 2011; Fairburn, Cooper, & O'Connor, 2008) provide a particularly rigorous assessment and may aid in distinguishing BN from ED-NOS (or in *DSM-5*, OSFED), and they are useful in gauging severity (see Chapter 11).

Questionnaire-based methods such as the EDE-Q (see Chapter 10), a questionnaire version of the Eating Disorder Examination (EDE), could also be used to diagnose BN (Fairburn & Beglin, 2008). The EDE-Q follows the form and wording of the EDE closely. Diagnostic algorithms can be used to generate categorical diagnoses, and subscale scores allow measurement of eating disorder cognitions. The psychometric properties of the EDE-Q are generally good (for a review, see Berg et al., 2011). There are also other questionnaires that ask about purging behavior. An example is the Eating Disorders Diagnostic Scale (EDDS; Stice, Fisher, & Martinez, 2004). One caveat with currently available instruments is that some (such as the EDDS) are calibrated to *DSM-IV*. Consequently, they capture frequency data that support the old BN criterion of two episodes per week, but reveal little below that now-outdated threshold.

Questionnaire-based methods may be particularly useful in screening for disordered eating in broader populations, for example, in a two-stage (screening, followed by clinical interview) clinic-based or epidemiological study (Allison et al., 2007; Lundgren et al., 2006). One particular reason to use reliable and valid questionnaires for screening is that some evidence suggests that questionnaires may yield higher rates of disordered eating reports than interviews. For example, in a study examining questionnaire-based and structured interview-based reports of binge eating and purging behavior, higher rates of these features of disordered eating were identified with the questionnaire method than with interviewing (Keel, Crow, Davis, & Mitchell, 2002).

Case History

Ms A was a 17-year-old woman who presented at her primary care physician's office because her mother thought she had an ED.

At the age of 15 she began running for her high-school cross-country team and decided that she wanted to lose weight (although she was 5′4″ [1.63 m] tall, and weighed 125 lb [56.7 kg]; BMI = 21.5). She gradually began restricting the types of foods that she was eating and then, after losing some weight, began to diminish the amount of food she was eating. Her training volume went up as well when she joined the track team. Her weight and shape became more and more important to her, in terms of her self-evaluation. After 4 months she had lost about 15 pounds, to a weight of 110 pounds (49.9 kg; BMI = 18.9). At about that time she began to binge eat in the late afternoon before her parents got home. No one knew about these binge-eating spells, during which she would eat large amounts of food, usually carbohydrates; at these times

she felt out of control of her eating. Two months later she first made herself vomit after an eating binge, because she felt so upset about the episode. By the time she was 16 her binge eating and purging were happening in tandem, on average, about four times per week, and they continued at that rate until she was 17.

When she was 17, her mother thought she heard her vomiting in the bathroom and asked her about it when she came out of the bathroom. Ms A seemed upset but vehemently denied it. Nevertheless, her parents took her to their family physician for evaluation for an ED. The physician asked about dieting, binge eating, and purging, but seemed uncomfortable asking those questions. The patient repeatedly denied all of the ED symptoms that her family doctor asked about. At that time, everyone concluded that she did not have an ED, and she received no treatment. She had a brief decrease in her symptoms before and just after that clinic visit but they soon returned to their previous frequency. By the time she left for college a year later, she was binge eating and purging six times per week.

The first semester of college went okay, although she didn't do as well in school as she might have hoped; she noticed that she was somewhat more anxious and down and distracted by her thoughts about eating, weight, and shape. Early in the second semester the resident assistant in her dormitory became worried about Ms A's continued weight loss. Additionally, another student met privately with the RA, saying she heard Ms A vomiting in the bathroom.

At that time Ms A was referred to the student health service for evaluation, where she saw both a primary care physician and a psychologist. This time, the primary care physician who asked about ED symptoms seemed well versed in them, and Ms A did admit to binge eating and purging. Additionally, the physician checked her potassium level, which was low, and noted on physical exam that she had bilateral parotid salivary gland swelling. The psychology evaluation included both a clinical interview and completion of the Eating Disorder Examination—Questionnaire version; in both she endorsed binge eating and purging. Ms A was referred for specialized ED treatment at the school.

Conclusions and Future Directions

In summary, although determining the presence of extreme behaviors such as binge eating and purging would seem to be a straightforward matter, the diagnosis of BN is actually quite challenging. Making this diagnosis is complicated by secrecy and shame surrounding symptoms, ambivalence about seeking treatment, and issues with clinical case finding, including stereotypes and stigma. There are specific approaches to clinical interviewing that can help to overcome these challenges. In addition, physical examination signs, biochemical markers, and structured diagnosis assessments in questionnaires can aid in effective diagnosis of BN.

The diagnosis of BN is widely recognized and has been studied fairly extensively. Nevertheless, several areas of uncertainty persist. Future research will likely need to address the following important questions. For example, *DSM-5* (2013) has revised the cut-off for purging and binge eating frequency to once per week, but is this a meaningful cut-off? Similarly, what frequency of purging (if any) can occur in BED and still have BED make the most sense as a diagnosis? Conversely, in those for whom purging predominates, what frequency of binge eating separates BN from purging disorder? Finally, for individuals who have recently lost weight, or who are at the lower end of the "normal" weight range, how does one differentiate AN from BN?

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10

Binge Eating Disorder: Diagnosis and Assessment

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With the publication of the 5th edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*; American Psychiatric Association, 2013), binge eating disorder (BED) was moved from "the back of the book" (i.e., the Appendix) to "the front of the book" (i.e., the main body of the text) where it is now recognized, along with anorexia nervosa (AN) and bulimia nervosa (BN), as a named eating disorder (ED). This official recognition of BED as a distinct ED represents one of only two major changes that were made in the *DSM* eating disorders chapter. The other major change was the reorganization of all eating and feeding disorders into one section rather than separating the feeding disorders most commonly diagnosed in childhood (see Chapter 13). This reorganization was undertaken to reflect clinical experience and emerging scientific evidence that age of onset and course of eating and feeding disorders may span a wider developmental period than once was assumed.

Indeed, BED is a case in point for including all EDs into one chapter (i.e., a single nosological category) rather than segregating them by presumed developmental period of highest risk. Although the modal age of onset of full syndrome BED falls into the 20s (Kessler et al., 2013), loss of control eating has been documented even among grade-school children (Marcus & Kalarchian, 2003; Tanofsky-Kraff, Marcus, Yanovski, & Yanovski, 2008; Tanofsky-Kraff et al., 2011). This finding highlights that in some individuals BED symptoms may emerge even before adolescence. Furthermore, in contrast to AN or BN (see Chapter 5), for which risk of onset decreases in middle adulthood (Favaro, Caregaro, Tenconi, Bosello, & Santonastaso, 2009; Hudson, Hiripi, Pope, & Kessler, 2007), risk of new onset for BED remains stable into adulthood (Hudson et al., 2007). The purpose of this chapter is to describe the key diagnostic features of BED and how best to assess these features using evidence-based procedures.

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Definition and *DSM-5* Diagnostic Criteria

The core clinical feature of BED is recurrent binge eating in the *absence* of recurring inappropriate compensatory behaviors such as self-induced vomiting, laxative use, excessive exercising, or fasting. Binge eating is defined as the consumption of a very large amount of food in a discrete period of time (within 2 hours) accompanied by a feeling of loss of control while eating. Furthermore, a BED diagnosis requires the presence of at least three additional symptoms representing behavioral indicators of lack of control over eating or feeling ashamed about one's eating. These symptoms include eating more rapidly than normal, eating until uncomfortably full, eating large amounts of food when not physically hungry, eating alone because of feeling embarrassed by overeating, and feeling disgusted, depressed, or guilty after eating. The person also must report feeling markedly distressed about the binge eating behavior, and this behavior must occur at a minimum average frequency of once per week for at least 3 months.

The diagnostic criteria instituted in *DSM-5* (2013) differ from the "provisional" diagnostic criteria used in *DSM-IV-TR* (American Psychiatric Association, 2000) in the following three ways:

1 the frequency criterion is now based on the number of episodes of binge eating rather than on the number of days that such episodes occurred;
2 the frequency criterion is reduced from a minimum average of two episodes to one episode per week; and
3 the duration criterion is shortened from 6 to 3 months.

The switch to counting episodes rather than days makes the metric for measuring binge eating behavior comparable to BN, and the reductions in frequency and duration of binge eating mirror *DSM-5* revisions for BN (see Chapter 9). In essence, it is now "easier" to receive a diagnosis of BN or BED (vs. ED-NOS/OSFED; see Chapters 4, 9, & 11). Perhaps surprisingly, however, initial studies (Hudson, Coit, Lalonde, & Pope, 2012) suggest that this change does not result in a large increase in the prevalence of BED, possibly because few people whose symptom profile meets the other BED criteria binge eat only once per week.

Differential Diagnosis of BED

In the *DSM-5*, AN, BN, and BED cannot be diagnosed concurrently. As such, a diagnosis of AN or BN "trumps" a diagnosis of BED. The diagnostic criteria for BED differ from the criteria for BN in two ways. Whereas the presence of compensatory behaviors is necessary to receive a diagnosis of BN, the regular use of such behaviors is an exclusion criterion for BED. Although individuals with BED may occasionally engage in inappropriate compensatory behaviors such as fasting (eating little or nothing all day), a diagnosis of BN may be indicated if such behaviors reach a frequency suggestive of a regular pattern. Additionally, unlike BN, body image disturbance (i.e., overvaluation of weight or shape on one's self-worth; see Chapter 9) is not required for a diagnosis of BED. However, many individuals with BED report that their weight or shape plays a central role in how they judge their own self-worth (Fairburn, 2008; see also Chapter 18). To this end, some experts recommended inclusion of overvaluation of weight or shape as a diagnostic criterion of BED in the *DSM-5* (Grilo, Masheb, & White, 2010).

Binge eating disorder also shares similarities with Night Eating Syndrome (NES), an ED listed in the *DSM-5* under the category of "other specified feeding or eating disorder" (see Chapters 4 & 11). NES involves recurrent episodes of night eating as demonstrated by consumption of most of one's caloric intake late in the day (after the evening meal) or eating after awakening from sleep (i.e., nocturnal eating). Although binge eating episodes are more likely to occur later in the day or at night in individuals with BED (Schreiber-Gregory et al., 2013; Stein et al., 2007), these episodes can occur at other times during the day as well. A diagnosis of NES should not be made if the individual meets criteria for BED or BN.

Additionally, obesity is a very common comorbid medical condition of BED; however, obesity is neither a defining symptom of BED nor a mental disorder (Marcus & Wildes, 2009). Indeed, the vast majority of obese individuals do not have BED (Allison, et al., 2006; Decaluwé & Braet, 2003). Although BED may increase risk for obesity, it can occur in normal weight individuals. Findings suggest that there are few behavioral or psychological differences between obese and nonobese individuals with BED (Barry, Grilo, & Masheb, 2003). Overall, a diagnosis of BED should be made only if the individual reports the defining features of the disorder.

Finally, increased appetite and weight gain are included in the diagnostic criteria for a major depressive episode (American Psychiatric Association, 2013); however, this increase in eating may or may not be associated with loss of control or occur in discrete periods of time. Major depression is a common comorbidity of BED (Hudson et al., 2007), and if full diagnostic criteria for both BED and a mood disorder are met, both diagnoses should be given (American Psychiatric Association, 2013).

Procedures and Instruments Recommended for Making a BED Diagnosis

Like most other mental disorders, BED is not defined by objectively measurable signs, such as increased blood pressure, excessive sweating, or indicators of aberrant kidney functioning. As such, making a diagnosis requires eliciting information about behaviors, contexts, emotional experiences, and patterns from the person being evaluated. In turn, diagnosing BED necessitates that the "assessor" and the individual being assessed share a common understanding of, and a common language in regard to, core features of the disorder. This requirement applies whether the assessment is based on an interpersonal interaction, such as an interview (see Chapter 39), or whether it is based on an impersonal procedure such as filling out a self-report questionnaire that is then scored by a clinician or researcher (see Chapters 38 & 40). Furthermore, this requirement applies whether the decision about the outcome of the assessment (i.e., the diagnosis) is in the hands of the person with the eating problem (e.g., self-screening) or in the hands of someone else (e.g., a clinician or researcher).

Assessment Challenges

In both clinical practice and research settings, a key challenge in making a BED diagnosis arises from the difficulty in determining whether the individual is actually "binge" eating. The defining elements of binge eating (i.e., overeating *and* experiencing an eating episode as "out of control"; see Chapter 9) require a judgment for which no objective criteria have been specified (Wolfe, Baker, Smith, & Kelly-Weeder, 2009). Some researchers have recommended

or used a minimum 1000-calorie threshold (Fairburn, 1987; Kaye et al., 1992; Mitchell, Crow, Peterson, Wonderlich, & Crosby, 1998; Timmerman, 1999); however, this cut-off is not included in the *DSM-5* definition of a binge and has not been strictly followed by all researchers examining binge eating episodes.

Although the *DSM-5* (2013) defines binge eating as eating "an amount of food that is definitely larger than what most people would eat in a similar period of time under similar circumstances" (p. 350), the definition of what constitutes objectively "large(r)" varies significantly based on an individual's culture, geographic location, and perception. As such, the context in which the episode occurred represents an important factor in determining whether the amount of food consumed was clearly larger than expected. For example, a 1500 kcal meal at a fast-food restaurant (e.g., the approximate calorie count of a cheeseburger, a medium-sized order of french fries, a nondiet drink, and a dessert) may be within normal limits in the community. However, a 1500 kcal "meal" consisting of snacklike foods at home (e.g., the approximate calorie count of an entire 10-oz bag of potato chips) may not be. In research settings, whether an episode meets criteria for an objectively large amount of food is often determined through consensus within the research team, but this procedure might not be possible or practical in a clinical setting (e.g., private practice). Furthermore, assessing the size of binge episodes may be difficult if the individual does not remember specifically or accurately what he or she ate. This may be especially true if an individual experiences significant dissociation related to his or her eating behaviors. Similarly, patients may be ashamed by how much they are eating, and in turn, find it difficult to discuss their behavior with someone else. Given the advantages of conducting interviews for diagnostic purposes, building rapport and developing a strong therapeutic alliance represent important aspects of the assessment process and may improve the accuracy of the information being provided (see Chapter 39).

In addition to challenges in assessing whether food consumption is abnormally large and thus problematic, there may be difficulties in assessing lack of control during the episode. Although patients may understand that feeling loss of control is subjective, the answer to whether an individual has experienced this is not always clear and unambiguous. Acknowledging loss of control may be, in part, a function of demographic characteristics (e.g., women more readily endorse loss of control than men; children may have difficulty understanding what is meant by losing control; Arikian et al., 2012). Moreover, the very notion of loss of control implies that control is attempted, desired, or present at some point in time. Indeed, some individuals with longstanding overeating problems may perceive their efforts at controlling their eating as futile and may deny loss of control by explaining that they have given up trying to control their overeating. Additionally, some individuals may have planned to binge, and therefore may not endorse feeling a loss of control.

Interview Assessments

Given the complexities of measuring binge eating, experts recommend that assessment for determining diagnosis be based on an interview rather than questionnaires (Tanofsky-Kraff et al., 2013; Wildes & Marcus, 2010). An interview format allows for clarification of whether a given episode reflects eating "an amount of food that is definitely larger than what most people would eat…under similar circumstances" (*DSM-5*, 2013, p. 350), a task that involves finding out what the person actually ate and under what circumstances. As previously mentioned, eating very large quantities of food in one sitting under certain circumstances (e.g., dining in an all-you-can-eat facility or consuming value-sized meals) is culturally

normative in the United States and other industrialized nations. Thus, making reliable and valid judgments about a given person's eating episodes can be difficult. As such, during an interview, it is important to gather detailed information about the specific types of food, amount of food consumed, duration of eating episode (including time started eating, time stopped eating, and whether there were any breaks during the episode), and the setting in which the food was consumed (e.g., alone or with others) (Peterson, 2005). The latter information is particularly important for determining whether the amount of food consumed was excessively large, given the situation.

During the interview, it is also necessary to ask whether or not the individual experienced a sense of loss of control while eating. If individuals initially deny experiencing loss of control, it is often helpful to rephrase the question in terms of whether they felt they could stop eating once they started or whether the episode could have been prevented from occurring (Peterson, 2005). Negative endorsement of either question also would imply experiencing loss of control.

Columbia Eating Disorder Assessment (CEDA) The decision about which interview to use depends in part on the specific goals of the interview. If only a categorical decision is desired ("does the person meet diagnostic criteria for BED: yes/no?") and the diagnosis is being made in a clinical context, a short unstructured interview focused on the core symptoms may suffice. Researchers at the Columbia Center for Eating Disorders are in the process of creating a new semistructured interview for diagnosing *DSM-5* eating disorders (Glasofer et al., 2012). Preliminary analyses comparing the Columbia Eating Disorder Assessment (CEDA) to the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993; see also Chapter 9) suggest that the CEDA interview is brief, reliable, and valid (Glasofer et al., 2012) and, consequently, may be a useful guide in the diagnosis of BED and other EDs in clinical settings.

Eating Disorder Examination (EDE) In research studies the EDE is considered the "gold standard" assessment interview for ED symptoms, including binge eating episodes (see Chapter 38). The EDE is a semistructured interview that assesses ED psychopathology over the 28 days prior to the assessment (or 3 months prior for diagnostic items). Items are rated on a seven-point scale, with higher scores indicating greater symptom severity. The EDE is useful for generating BED and other ED diagnoses in part because it provides detailed information on the frequency of specific forms of overeating, loss of control, and compensatory behaviors. Additionally, it yields a global ED severity rating along with four subscale scores related to cognitive features of EDs: dietary restraint, eating concerns, shape concerns, and weight concerns.

The EDE has become and remained the gold standard for assessment because research has established its good psychometric properties and utility (Berg, Peterson, Frazier, & Crow, 2012), it has been adapted for children and adolescents (Bryant-Waugh, Cooper, Taylor, & Lask, 1996; Watkins, Frampton, Lask, & Bryant-Waugh, 2005), and it is available in several languages. The EDE has demonstrated good interrater reliability, short-term test-retest reliability, and convergent validity in individuals with BED (Grilo, Masheb, & Wilson, 2001; Grilo, Masheb, Lozano-Blanco, & Barry, 2004; Wilfley, Schwartz, Spurrell, & Fairburn, 2000). Nevertheless, although the EDE may be informative for both clinicians and researchers, it requires extensive training and is often time-consuming to administer. As such, it may not be feasible to administer in all clinical settings.

Self-Report Questionnaire Assessments

Given the challenges involved in arranging to conduct semistructured interviews in clinical settings, it may be more useful for clinicians to supplement unstructured interviews with psychometrically sound self-report questionnaire assessments. Although assessment of binge eating episodes may be more reliable and/or valid when done via interviews (Wilfley, Schwartz, Spurrell, & Fairburn, 1997), self-report measures can be useful in terms of verifying the diagnosis, examining other associated clinical features (e.g., depressed mood), and examining symptom change over time. Indeed, several self-report measures exist that can be used to assess ED diagnoses and determine the presence and severity of eating pathology or other psychological symptoms. Although a review of all these measures is beyond the scope of this chapter, there follows a description of a few widely used questionnaire assessments that may be useful for assessment of BED in clinical settings. For a more thorough review of psychological assessments, including assessments of body image disturbances and of quality of life in EDs, see Túry, Gülec, and Kohls (2010).

EDE: Questionnaire Version (EDE-Q) The EDE-questionnaire (EDE-Q; Fairburn & Beglin, 1994) is a 33-item self-report measure that was adapted from the EDE interview. It focuses on ED symptoms during the 28 days prior to the assessment and yields four subscales identical to those of the EDE. This measure also includes frequency of binge eating and compensatory behaviors. Overall, findings from several studies indicate that the EDE-Q may be a psychometrically sound alternative to the EDE for examination of ED symptoms in individuals with BED (Reas, Grilo, & Masheb, 2006; Wilfley et al., 2000; Wilson, Nonas, & Rosenblum, 1993).

A recent meta-analysis comparing scores on the EDE and EDE-Q indicated that scores on the EDE-Q were generally higher than those on the EDE, but concluded that both can be used as valid assessments of cognitive and behavioral symptoms of eating disorders (Berg, Peterson, Frazier, & Crow, 2011). However, there were some inconsistencies between the measures in the frequency of binge eating (Berg et al., 2011). There is some evidence that reliability and validity of the EDE-Q may be improved by providing, as part of the client's introduction to the measure, more detailed information about the definition of binge eating (Celio, Wilfley, Crow, Mitchell, & Walsh, 2004; Goldfein, Devlin, & Kamenetz, 2005; Wilfley et al., 1997). For example, explaining to the patient what constitutes a binge episode may improve accuracy of frequency ratings, and in turn, aid in diagnosis. Given that the EDE-Q is fairly easy to use and administer, it can be given to patients (older adolescents and adults) on a monthly basis to monitor treatment response.

Eating Disorder Diagnostic Scale (EDDS) Similar to the EDE-Q, the Eating Disorder Diagnostic Scale (EDDS; Stice, Telch, & Rizvi, 2000) also may be a useful tool to verify diagnosis. The EDDS is a 22-item questionnaire designed to assess *DSM-IV* eating disorder diagnoses. This measure assesses average weekly frequency of binge eating and compensatory behaviors over the previous 3 months, as well as cognitive symptoms of eating disorders, including influence of shape and weight on self-evaluation (see Chapter 9). Rather than using the term "binge," which may be misinterpreted, this measure provides an example of a large amount of food (e.g., a quart of ice cream) to help clarify what is meant by overeating. The EDDS has demonstrated good psychometric properties in individuals with BED, including high test-retest reliability and content, criterion, and convergent validity, which provide support for its use in the assessment of ED diagnoses (Stice, Fisher, & Martinez, 2004; Stice et al., 2000). Given that it is slightly shorter than the EDE-Q, the EDDS may be a good alternative to assist in and verify diagnosis of BED if adapted for *DSM-5* criteria.

Three-Factor Eating Questionnaire (TFEQ) Although the EDE-Q and EDDS may be used to verify diagnosis, additional self-report measures such as the Three-Factor Eating Questionnaire (TFEQ; Stunkard & Messick, 1985) and the Binge Eating Scale (Gormally, Black, Daston, & Rardin, 1982) are used in research settings to assess more specific ED symptoms and symptom severity. These measures cannot be used to make a diagnosis or measure binge frequency, but they may provide more information on specific symptoms or be useful as a screening tool for BED.

The TFEQ is a 51-item questionnaire designed to evaluate three dimensions of eating behavior: cognitive restraint, hunger, and disinhibition. This questionnaire is divided into two parts, with the first 36 items consisting of true/false questions and the remaining 15 items rated on Likert-type scales. This measure has demonstrated good reliability across studies (Túry et al., 2010); however, the original factor structure has not been supported (Karlsson, Persson, Sjöström, & Sullivan, 2000; Mazzeo, Aggen, Anderson, Tozzi, & Bulik, 2003) and studies examining the validity of the measure have not been consistent (de Lauzon et al., 2004; Karlsson, et al., 2000; Laessle, Tuschl, Kotthaus, & Prike, 1989). As such, the TFEQ would be most useful for identifying potential individuals at risk for BED or tracking changes in symptoms over the course of treatment (Túry et al., 2010).

Binge Eating Scale (BES) The Binge Eating Scale (BES; Gormally et al., 1982) is a 16-item continuous measure developed to assess binge eating severity. Responses to all items are added together to form a total score (range 0–46), with scores greater than or equal to 27 indicating clinical binge eating. Similar to the TFEQ, this measure does not assess frequency of binge eating, but higher scores have been associated with other measures of binge eating (Celio et al., 2004; Timmerman, 1999). Thus, this measure may be used as a screening tool for two-stage studies (e.g., questionnaire followed by an interview) of binge eating or BED, as well as to track severity of BED-related symptoms across treatment.

Assessing psychiatric comorbidities Given that BED is often associated with other psychiatric comorbidities, including mood, anxiety, substance use, and personality disorders (Cassin & von Ranson, 2005; Hudson et al., 2007; Wonderlich, Gordon, Mitchell, Crosby, & Engel, 2009), a thorough assessment also should include measures of these symptoms (see Chapter 15). For example, the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) is a 21-item continuous measure of the extent and severity of depressive symptoms over a 2-week period. It is widely used in both clinical and research settings, and extensive research has demonstrated its very good psychometric properties (Beck, Steer, & Garbin, 1988). The BDI can be given biweekly to assess change in depressive symptoms. Note that, although self-report measures of associated psychological problems may be informative for treatment planning and monitoring treatment response, more thorough interviews are required to establish diagnoses of any comorbid disorders.

Use of Assessments in Clinical Settings

As previously mentioned, there are several different measures available for assessing BED criteria and symptoms. Although interviews are best for establishing an accurate diagnosis, self-report questionnaires may be useful in verifying the diagnosis, examining other associated clinical features, and measuring change in symptoms over time. The clinician

should combine information from all methods of assessment to establish a clear picture of the patient's presenting problem. It also may be helpful for the clinician to provide diagnostic feedback and communicate findings or general conclusions from the assessment to the patient. Findings suggest that providing diagnostic feedback empathetically can increase a patient's positive emotions and potentially help build good rapport (Holm-Denoma et al., 2008). Once a diagnosis is established, additional information gathered during the assessment via interviews and questionnaires should be used to generate a hypothesis about factors maintaining the disorder, and in turn, assist in treatment planning.

Monitoring Symptoms during Treatment

In addition to aiding in diagnosis and treatment planning, many interview questions and self-report measures can be used to monitor symptoms throughout treatment and therefore to track patients' progress. For example, the EDE-Q assesses symptoms such as binge eating frequency over a 28-day period, so administration of this measure on a monthly basis can provide tangible evidence of whether or not the treatment is working in terms of decreasing frequency of specific symptoms. Self-monitoring of symptoms may be another method for tracking treatment response (Grilo et al., 2001). For example, the patient may keep a daily log of how many binge eating episodes he or she experiences per week, as well as the emotional and situational factors surrounding each of the episodes. In addition to minimizing recall biases inherent in other self-report methods, this momentary monitoring may increase awareness of these types of episodes, elucidate specific factors leading up to or maintaining the problem, potentially emphasize the patient's control over the behaviors, and/or have reactive effects that decrease the frequency of the problem behavior (Korotitsch & Nelson-Gray, 1999).

Other Clinical Considerations when Making a Diagnosis

Compared to individuals with AN or BN, BED is the most underdetected ED. Although treatment-seeking is low among all EDs (Cachelin & Striegel-Moore, 2006; Mond, Hay, Rodgers, & Owen, 2007), epidemiological studies have found that individuals with BED are especially likely to not be diagnosed with the disorder (Kessler et al., 2013). The reasons for this difference in detection rate are unclear and likely not primarily a function of lesser impairment (Kessler et al., 2013). Stigma and shame about binge eating or obesity may explain, in part, why individuals with BED do not seek help for their eating problem (Hepworth & Paxton, 2007; Puhl & Heuer, 2009). It is also possible that individuals may be more familiar with AN or BN and do not yet recognize BED as a clinical problem or at least one that has viable treatment options. Regardless, findings of low treatment rates in BED highlight the importance of inquiring about such symptoms (e.g., overeating episodes, subjective distress regarding eating habits) in clinical or medical settings, even if these symptoms are not the chief complaint.

Conclusions and Future Directions

In summary, although BED was included in the Appendix B of *DSM-IV* ("Criteria Sets and Axes Provided for Further Study"; see Chapter 4), it is now recognized as an official eating disorder diagnosis in *DSM-5*. The core clinical feature of BED is binge eating in the absence

of compensatory behaviors, which can often be challenging to assess. Consequently, interviews are necessary to establish an accurate diagnosis. However, self-report questionnaires addressing the features of the ED (e.g., the EDE-Q) and associated psychological symptoms (e.g., BDI for severity of depression) may provide essential information for treatment planning and monitoring treatment response. Importantly, although binge eating represents the core clinical feature necessary for a BED diagnosis, other associated features are required.

Few studies have specifically examined these associated features (e.g., eating more rapidly than normal) and how important they are for the diagnosis. Moreover, it is important to note there are currently no continuous self-report measures of loss of control. It would be useful for future research to focus on this construct and how best to assess it in both clinical and research settings. Similarly, future research should be conducted to better understand the importance (and necessity) of both loss of control and binge size for the validity of the syndrome, including predictive validity and utility in treatment planning. Given that there are various forms of assessments for BED, each with their own limitations, future research should continue to compare the different methods of assessments used to examine the same construct (i.e., cross-method validation; Tanofsky-Kraff et al., 2013). Finally, future research should focus on how best to define and measure binge eating in children.

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11

Diagnosing Other Specified and Unspecified Feeding and Eating Disorders in *DSM-5* (Formerly Eating Disorders Not Otherwise Specified in *DSM-IV*)

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Collectively, other specified feeding and eating disorders (OSFED) and unspecified feeding and eating disorders (UFED) were formerly known as eating disorders not otherwise specified (ED-NOS). These diagnoses are assigned in the presence of clinically significant eating disorder (ED) symptoms that do not satisfy criteria for another formal ED diagnosis (American Psychiatric Association, 2000, 2013; see also Chapter 4). ED-NOS and now, presumably, OSFED and UFED are associated with levels of symptomatology, psychosocial impairment, and mortality risk comparable to other types of EDs, including anorexia nervosa (AN) and bulimia nervosa (BN) (Crow, Agras, Halmi, Mitchell, & Kraemer, 2002; Crow et al., 2009; Ricca et al., 2001; Turner & Bryant-Waugh, 2004). Previous research suggests that the predecessor to OSFED and UFED, ED-NOS, was the most common ED diagnosis in clinical settings (Button, Benson, Nollett, & Palmer, 2005; Ricca et al., 2001; Turner & Bryant-Waugh, 2004; see also Chapter 5), with one review citing a weighted average of 60% of outpatient ED clinic patients who met criteria for ED-NOS (Fairburn & Bohn, 2005). Recent prevalence estimates in a community sample have suggested rates of ED-NOS between 4% and 5% (Le Grange, Swanson, Crow, & Merikangas, 2012). Fairburn et al. (2007) noted the ironic contrast between the relatively high rates of ED-NOS in clinical settings and the fact that the "not otherwise specified" diagnosis is technically a residual diagnostic category.

Diagnosing OSFED and UFED can be challenging for several reasons. First, other types of EDs, including AN and BN, must be ruled out; however, ED symptoms still need to be of sufficient severity to warrant an ED diagnosis (Fairburn & Bohn, 2005). Second, the parameters of this diagnostic category have changed with the release of the *DSM-5* criteria (American Psychiatric Association, 2013). Binge eating disorder (BED), formerly included in the *DSM-IV-TR* (American Psychiatric Association, 2000) as an example of ED-NOS, is now included with

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AN and BN as a recognized ED diagnosis—and needs to be ruled out before diagnosing OSFED or UFED. Finally, several ED criteria can be particularly challenging to diagnose reliably, including binge eating (especially whether the amount of food consumed during the episode was objectively large) and the overvaluation of shape and weight, which requires both self-awareness and metacognitive ability on the part of the patient for accurate reporting (Peterson, 2005; Wilson, 1993; see also Chapters 9 & 10). In this chapter, we briefly review the historical roots of currently conceptualized OSFED and UFED (see Chapter 4), provide specific guidelines in regard to their assessment and diagnosis within clinical and research settings, and present empirical data supporting the validity of the OSFED, UFED, and ED-NOS constructs.

Historical Definitions

ED-NOS in *DSM-III* and *DSM-III-R*

The *DSM-III* (American Psychiatric Association, 1980) first defined individuals who did not meet the full criteria for AN or BN but who did have clinically significant ED pathology as "atypical," an adjective still in use to describe ED-NOS (e.g., Simpson & Slowey, 2011), along with "partial" and "subthreshold" (Fairburn & Bohn, 2005). In *DSM-III-R* (American Psychiatric Association, 1987), the term "Eating Disorder Not Otherwise Specified" was introduced and defined as "disorders of eating that do not meet criteria for a specific Eating Disorder" (p. 71). Three examples of ED-NOS were included in *DSM-III-R*: individuals of average weight without binge eating who engage in self-induced vomiting because of fear of weight gain; individuals who meet all AN criteria except amenorrhea; and individuals with all symptoms of BN who do not meet the binge eating frequency criterion of twice per week. Interestingly (and, perhaps, prophetically in a field in which prevalence rates of NOS diagnoses are indeed high), the *DSM-III-R* (1987) specified that the NOS label "is used to indicate a category within a class of disorders that is residual to the specific categories in that class, although it is recognized in some settings the category may actually be more common than any of the specific disorders in that particular class" (p. 23).

ED-NOS in *DSM-IV* and *DSM-IV-TR*

The *DSM-IV* (American Psychiatric Association, 1994) and *DSM-IV-TR* (2000) continued to define ED-NOS as a category "…for disorders of eating that do not meet the criteria for any specific Eating Disorder" (pp. 550 & 594, respectively). The *DSM-IV* and *DSM-IV-TR* also provided specific examples of ED-NOS, such as meeting full criteria for AN but still menstruating or with weight in the normal range; compensatory behavior in the absence of objectively large binge eating episodes; chewing and spitting behavior; and BED.

DSM-IV and *DSM-IV-TR* criteria for ED-NOS have been criticized on several grounds (Sysko et al., 2012). The most common criticism, as noted above, is the problem of having a residual category account for the majority of ED cases in treatment settings (Fairburn & Bohn, 2005; Hebebrand & Bulik, 2011). Despite the prevalence of ED-NOS, providers treating ED-NOS were not consistently reimbursed by health insurance in certain countries, notably the United States (Fairburn & Bohn, 2005). Similarly, evidence-based treatment (EBT) studies typically require participants to meet full diagnostic criteria and therefore have often excluded participants with ED-NOS diagnoses (Bisaga & Walsh, 2005; Fairburn & Bohn,

2005). Consistent with other types of mental health NOS conditions that have been neglected in research (Pincus, Davis, & McQueen, 1999), ED-NOS received less attention than AN, BN, and BED in research and in treatment studies in particular. More recent treatment studies appear to be broadening inclusion criteria such that individuals with ED-NOS have been allowed to participate (Schmidt et al., 2012); nonetheless, EBT data for ED-NOS remain limited (NICE, 2004). Finally, the validity of the *DSM-IV* and *DSM-IV-TR* eating disorder criteria, including the designation of NOS, has been challenged due to low levels of diagnostic stability over time (Hebebrand & Bulik, 2011; Peterson et al., 2011) and to inconsistent empirical support for current practices in differentiating eating disorder types (Wonderlich, Crosby, Mitchell, & Engel, 2007; Wonderlich, Gordon, Mitchell, Crosby, & Engel, 2009). Thus, the NOS diagnostic classification as conceptualized in *DSM-IV* and *DSM-IV-TR* had both practical and theoretical limitations, which probably limited both scientific advancement and access to healthcare resources.

The Shift from ED-NOS to OSFED and UFED in *DSM-5*

Given these criticisms, the Eating Disorders Workgroup for *DSM-5* aimed to reduce the prevalence of ED-NOS by broadening the AN and BN criteria (e.g., reducing the required frequency of binge eating and compensatory symptoms to once per week in BN, eliminating the amenorrhea criterion in AN) and by including BED as a formal ED diagnosis rather than an example of ED-NOS (Walsh, 2009). In addition, the ED-NOS category was renamed and divided into two categories, Other Specified Feeding or Eating Disorder (OSFED), which includes five specific clinical examples (described below), and Unspecified Feeding or Eating Disorder (UFED), a category for clinically significant eating or feeding disorders other than AN, BN, BED, Pica, Rumination Disorder, Avoidant/Restrictive Food Intake Disorder (ARFID), or OSFED. Because the OSFED diagnosis includes specific examples of clinically significant eating disorders, UFED is used only to diagnose conditions not specified in the OSFED category. The Not Otherwise Specified (NOS) label has been dropped from the *DSM-5* in general and replaced by the "Other Specified" and "Unspecified" categories throughout the new classification system.

The OSFED and UFED diagnoses are intentionally designed to be useful for clinicians and, consistent with previous versions of the *DSM*, are to be used only with individuals who have a clinically significant eating or feeding disorder but who do not meet criteria for another diagnosis. Given the priority of clinical utility in determining the *DSM-5* criteria (Sysko & Walsh, 2011), modifications of previous criteria were guided by empirical findings as well as ease of use in clinical settings. For example, the AN diagnosis no longer indicates a specific weight threshold for determining whether an individual is underweight, in contrast to the *DSM-IV* and *DSM-IV-TR,* which provided the example of less than 85% ideal body weight. As a result, patients formerly diagnosed with ED-NOS because their weight was slightly higher than 85% of ideal body weight (e.g., an 18-year-old female who is 5′3″ [1.60 m] and weighs 100 pounds [~45.4 kg], which is 86.7% of IBW) will now be eligible for the formal AN diagnosis. The text of *DSM-5* also recommends use of body mass index (BMI; kg/m^2) rather than IBW, with a specific threshold of 18.5 (e.g., 5′3″, 104.5 lb [47.4 kg]) as an example of the lower limit of "normal" body weight, but allows for considerably more flexibility in the definition of underweight based on clinical history and medical status.

This diagnostic revision is especially important given research suggesting that individuals with full and subthreshold AN (by *DSM-IV* standards) are indistinguishable in regard to ED

symptoms, general psychopathology, and level of impairment (Crow et al., 2002; Le Grange et al., 2013). This modification allows clinicians more flexibility with less rigid weight parameters and, as a result, may reduce the prevalence of ED-NOS (or OSFED and UFED as they are currently conceptualized). Indeed, initial data from the following samples suggest that application of *DSM-5* criteria reduces the relative numbers of those diagnosed with ED-NOS compared to older *DSM-IV* criteria: a Japanese outpatient sample (Nakai, Fukushima, Taniguchi, Nin, & Teramukai, 2013); two Portuguese epidemiological samples (Machado, Goncalves, & Hoek, 2013); a Scandinavian clinical sample (Birgegard, Norring, & Clinton, 2012); a longitudinal sample of U.S. college students (Keel, Brown, Holm-Denoma, & Bodell, 2011); and a U.S. clinical sample (Berg, Stiles-Shields, et al., 2012).

The *DSM-5* OSFED criteria specify five specific examples. **Atypical AN** is defined as meeting all AN criteria except that the patient's weight is within or above the normal range (e.g., an obese individual who develops symptoms of AN in the context of a significant weight loss). **BN of low frequency and/or limited duration** is given to individuals meeting all criteria for BN except for the frequency and/or duration criteria (e.g., binge eating and vomiting at a frequency of three times per month rather than four; see Chapter 9). **BED of low frequency and/or limited duration** is defined as meeting all criteria for BED except for the frequency and/or duration criteria (e.g., binge eating weekly for 2 months instead of 3). **Purging disorder** (PD; Keel & Striegel-Moore, 2009), included in the *DSM-IV* as an example of ED-NOS, is characterized by recurrent purging behavior (e.g., self-induced vomiting, misuse of laxatives or diuretics) to influence shape or weight. Individuals diagnosed with PD do not engage in episodes of binge eating that meet *DSM-5* criteria but may experience "subjective" binge episodes in which the amount of food consumed is not objectively large but is accompanied by a sense of loss of control (Fairburn, 2008). The final example, **night eating syndrome** (NES; Striegel-Moore, Franko, & Garcia, 2009), is characterized by excessive evening food consumption prior to sleeping or eating that occurs at night after the onset of sleeping.

The UFED diagnosis is assigned when eating and feeding disorder symptoms are clinically significant (i.e., are associated with occupational, academic, or social impairment or significant distress) but are not adequately captured by any of the other Feeding and Eating Disorders described in *DSM-5* or the syndromes described by OSFED (e.g., atypical AN). For example, the UFED diagnosis would be assigned to an individual who is intensely concerned with body weight and shape, who denies binge eating, night eating, and purging behaviors, and who, despite frequent exercise and regular diet pill use, is not underweight and has not lost a significant amount of weight. The UFED diagnosis can also be applied when there is insufficient information to determine a diagnosis, for example, when an individual is evaluated in an emergency room or other type of crisis setting (*DSM-5*, 2013).

Assessment Strategies

Several approaches can be used to assess and diagnose OSFED and UFED in research and clinical settings, as well as to differentiate between these residual categories and other eating and feeding disorders, other mental health or physical conditions (e.g., anxiety disorders), or the absence of mental illness. Unstructured or semistructured interviews, self-report instruments, or a combination can be utilized to obtain reliable and accurate diagnoses (Berg, Peterson, & Frazier, 2012; Mitchell & Peterson, 2005; Peterson, 2005). Assessment instruments are typically used to determine whether specific ED symptoms are present and,

if so, the severity of those symptoms. These data are then used to determine whether criteria are met for a formal ED diagnosis. The presence of clinically significant (in terms of behavioral severity, distress, and/or impairment) ED symptoms on an ED assessment instrument combined with the absence of meeting full criteria for another ED would indicate a diagnosis of OSFED or UFED. Assessment instruments can also be useful in determining whether the individual meets criteria for a specific subset of OSFED (e.g., Night Eating Syndrome).

Anthropometry

Obtaining an accurate measurement of height and weight is essential for determining whether AN or OSFED (Atypical AN) should be diagnosed. Because accuracy of this measurement is so critical, height and weight should not be based on self-report, given extensive data suggesting that self-reported weight is often inaccurate (Connor Gorber, Tremblay, Moher, & Gorber, 2007). Nonetheless, asking self-reported weight can provide clinically useful information (e.g., self-awareness, minimization of symptoms) when it is combined with objective measurement of height and weight. Because many individuals with EDs experience distress about having their weight measured, offering blind weights at assessment can often be helpful in building and maintaining rapport (Peterson, 2005).

Unstructured Interviews

Clinical interviews are often used to assign diagnoses, including OSFED and UFED, to individuals with ED symptoms (Berg, Peterson, & Frazier, 2012). In both research and clinical settings, interpersonal rapport is essential to ensure that the client or patient feels sufficiently comfortable to provide accurate information, and it is vital to ask diagnostic questions without criticism or negative judgment (Peterson, 2005; Vitousek, Daly & Heiser, 1991). Asking for detailed examples (e.g., contents of a typical binge eating episode and/or number of repetitions, and type and duration of exercise activities) and obtaining collateral information from other healthcare providers and family members can also increase the accuracy of self-reported information used to determine ED diagnoses (Peterson, 2005). To diagnose OSFED or UFED, it is particularly important to ask detailed questions about eating patterns including binge eating (especially to determine if the binge eating episodes meet *DSM-5* criteria in terms of size and frequency), cognitive symptoms including self-evaluation, and compensatory behaviors including self-induced vomiting, misuse of laxatives/diuretics, fasting, and excessive exercise. In addition, discussing clinical impairment (i.e., the extent to which the ED symptoms interfere with work, school, interpersonal relationships, and psychosocial functioning) and distress are important to determine whether the severity of ED symptoms warrants an OSFED or UFED diagnosis.

Structured and Semi-Structured Interviews

Eating Disorder Examination (EDE) Several formal instruments can be utilized in clinical and research settings to diagnose OSFED or UFED. The widely used Eating Disorder Examination (EDE; Fairburn, 2008), described in detail in Chapters 9 & 10, provides structure to the interview process while simultaneously allowing the clinician the flexibility to ask follow-up questions based on individual responses.

The EDE is helpful in diagnosing OSFED and UFED because it can be used both to establish that the individual has symptoms that are clinically significant to a degree that warrants a diagnosis and to exclude the presence of AN, BN, and BED. Considerable psychometric data support the reliability and the validity of the EDE (Berg, Peterson, Frazier, & Crow, 2011, 2012; Berg, Stiles-Shields, et al., 2012; Fairburn, 2008). Administration and scoring of the instrument does require training and expertise in both the EDE and in ED psychopathology more generally (Fairburn, 2008). In addition, the EDE often requires over an hour to complete with symptomatic individuals. However, trained clinicians who use the EDE solely for diagnostic purposes may selectively use diagnostic items from the interview to shorten the duration of administration and to focus unstructured interviews through empirically developed questions that assess ED symptoms.

Structured Clinical Interview for DSM-IV (SCID) Another limitation of the EDE is that it provides only current diagnostic information. The Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1995; currently being updated for *DSM-5*) can be administered to diagnose the presence of both current and lifetime history of ED-NOS, OSFED, and UFED. Notably, past diagnoses of AN, BN, and/or BED do not preclude a current diagnosis of ED-NOS, OSFED, or UFED and, in fact, longitudinal changes in ED diagnoses are quite common, with individuals moving between different eating disorder categories over time (Peterson et al., 2011; see also Chapter 55). However, when establishing a diagnosis of OSFED or UFED for a particular time period, the formal ED must be ruled out for that specific time period. To diagnose OSFED or UFED accurately, the SCID must be administered without the "skip out" procedures in order to establish exactly which symptoms are present along with the severity of each symptom. Note that the Structured Interview for Anorexic and Bulimic Disorders (SIAB; Fichter & Quadflieg, 2001) is an empirically supported semistructured interview designed for *DSM-IV* (Fichter & Quadflieg, 2001) that can be used to establish the presence of both current and lifetime ED diagnoses.

Questionnaires

Eating Disorder Diagnostic Scale (EDDS) The Eating Disorder Diagnostic Scale (EDDS; Stice, Telch, & Rizvi, 2000) was developed to determine the presence of *DSM-IV* eating disorder diagnoses (see Chapter 10). This measure, which can be modified for *DSM-5*, is a brief self-report questionnaire that provides information about the presence and frequency of symptoms that correspond to the diagnostic criteria. Psychometric data support the reliability and validity of the EDDS (Krabbenborg et al., 2012; Stice, Fisher, & Martinez, 2004). Although the measure was designed for use in nonclinical samples, more recent data include cut-off points that can be used in clinical samples as well (Krabbenborg et al., 2012).

Eating Disorder Examination - Questionnaire (EDE-Q) The Eating Disorder Examination-Questionnaire (EDE-Q; Fairburn, 2008) is a self-report measure based on the EDE interview. Similar in content to the interview version, the EDE-Q yields information across the same four subscales (restraint, eating concern, weight concern, shape concern), as well as a global score and estimates of symptom frequency, which can be used to help establish ED diagnoses. Studies support the reliability and validity of the EDE-Q (Berg, Peterson, Frazier, & Crow, 2012; Fairburn, 2008; Rizvi, Peterson, Crow, & Agras, 2000), and the EDE and EDE-Q show some convergence in ED diagnoses, including ED-NOS (Berg, Stiles-Shields, et al., 2012). However,

recent research suggests important differences between the two instruments, including higher scores on the subscales of the questionnaire (Berg et al., 2011), inconsistent reporting of binge eating frequency between the interview and questionnaire (Berg et al., 2011), and discrepancies between overall response patterns on the two measures (Berg et al., 2013). For these reasons, the EDE-Q should not be used interchangeably with the interview version.

Night Eating Questionnaire (NEQ) The Night Eating Questionnaire (NEQ; Allison et al., 2008) is a 14-item measure that can be used to assess the psychological and behavioral symptoms associated with night eating syndrome, a subcategory of OSFED. Research has supported the reliability and validity of the NEQ in community and clinical samples (Allison et al., 2008). The NEQ can be used in conjunction with a diagnostic interview to establish that an individual has symptoms of night eating syndrome.

Clinical Impairment Assessment (CIA) The Clinical Impairment Assessment (CIA; Bohn & Fairburn, 2008) is a 16-item self-report questionnaire that can be used to establish the extent to which ED symptoms are associated with sufficient distress and psychosocial and cognitive impairment (e.g., work performance, decision making, interpersonal relationships, self-esteem) to constitute a mental disorder. Although the CIA cannot be used to establish an ED diagnosis, it is particularly helpful as an accompaniment to a diagnostic interview.

Questionnaires: Summary In summary, questionnaires can be helpful in determining whether an individual should be diagnosed with OSFED or UFED and may be especially useful as screening tools (see Chapter 38). Similar to interviews, ED diagnostic questionnaires can be used to establish whether an individual reports clinically significant ED symptoms but does not meet criteria for AN, BN, or BED. Questionnaires can also be helpful in assessing clinical impairment to make a distinction between OSFED or UFED and the absence of an ED. Given the discrepancies between interview and questionnaire-based methods in assessing ED symptoms (Berg et al., 2011, 2013), including binge eating (Wilson, 1993), it is generally recommended that a clinical diagnosis of OSFED or UFED be assigned based on interview data or a combination of interview and questionnaire data.

Differential Diagnosis: Distinguishing OSFED and UFED from Eating Disorders, Feeding Disorders, Other Disorders, and No Diagnosis

Distinguishing OSFED and UFED from other types of EDs typically involves three steps. The first step is to establish whether diagnostic criteria are met for AN, BN, BED, or a feeding disorder. If not, the second step is to determine whether symptoms are of sufficient severity, distress, and/or impairment to warrant diagnosis of a disorder or mental illness. The third step is to confirm that the most appropriate diagnosis is OSFED or UFED and not another *DSM-5* disorder (e.g., mood) or medical condition (e.g., cancer, celiac disease).

Anorexia Nervosa

As shown in Figure 11.1, AN (in *DSM-5*, 2013) requires that the individual is underweight, is fearful of weight gain or acts in a way to prevent weight gain, and endorses one of several psychological criteria (see Chapter 8; e.g., body image disturbance, in that

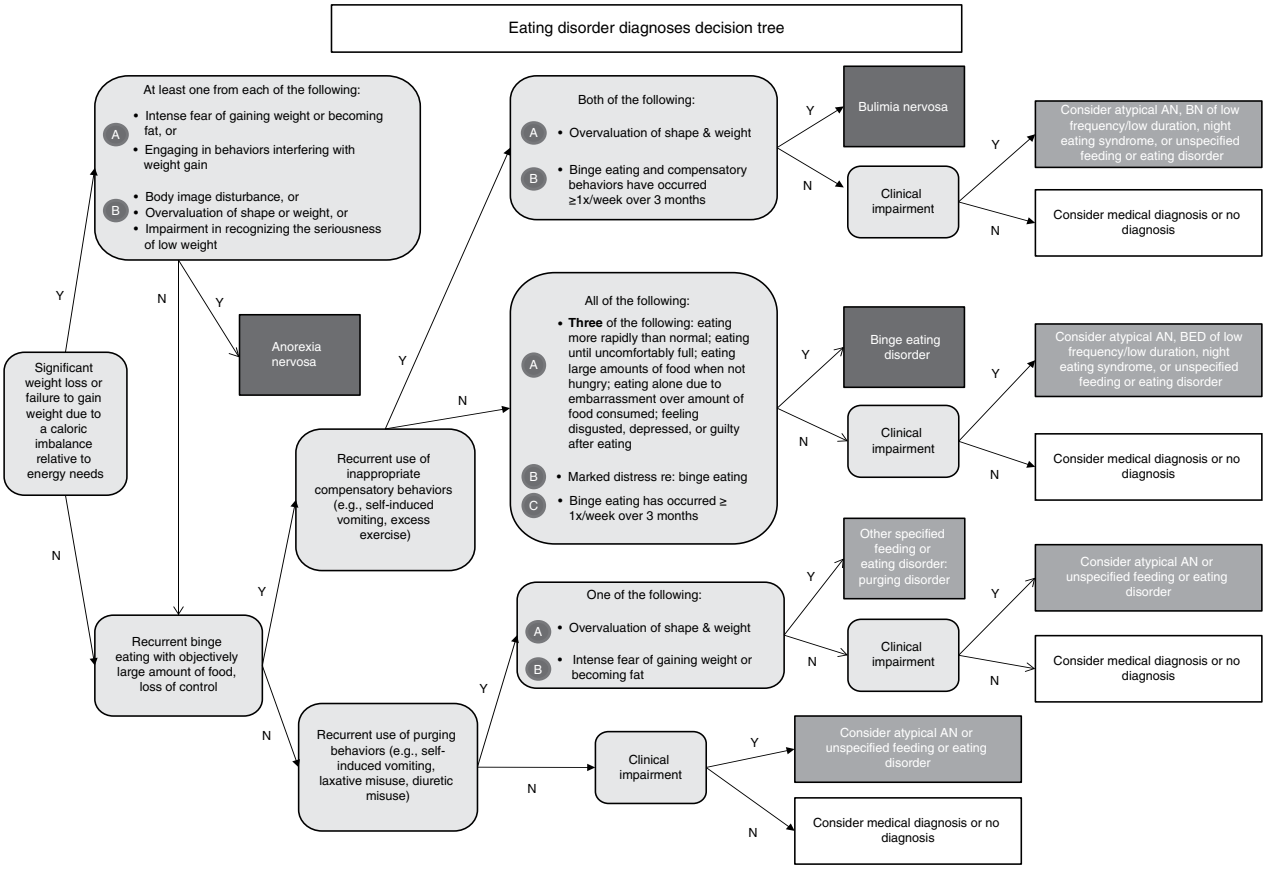

Figure 11.1 Eating disorder diagnoses decision tree.

individuals may report that they are overweight despite their low weight status and/or base their self-evaluation primarily on their shape and weight, as described below). A diagnosis of OSFED or UFED should be considered for individuals who meet some but not all symptoms of AN. As noted previously, *DSM-5* specifies that atypical AN can be diagnosed within OSFED for individuals who have lost weight and meet all criteria for AN but whose weight is still in the normal range. Others with some combination of AN symptoms can be diagnosed with UFED. For example, patients who are underweight and fear weight gain but who do not acknowledge the seriousness of their underweight status and deny both body image disturbance and overvaluation of shape/weight might be most appropriately diagnosed with UFED.

Bulimia Nervosa

According to *DSM-5* (2013), BN requires the presence of binge eating and compensatory behaviors and that these behaviors occur a minimum of once per week for 3 months (Figure 11.1 and Chapter 9). The BN criteria also require that individuals endorse the undue influence of shape and/or weight on self-evaluation. This symptom can be assessed using various structured assessments (e.g., EDE, EDE-Q) as well as unstructured interviews. Using the EDE to determine if self-evaluation is unduly influenced by weight and/or shape, the interviewer asks the individual to construct a list of domains that influence her or his self-worth (e.g., relationships, work performance, appearance). This list is then ranked and transcribed into a pie chart to clarify the relative importance of each self-domain. The criterion for undue influence is met if weight and/or shape are among the main aspects of determining one's self-evaluation.

The *DSM-5* specifies that individuals engaging in binge eating and compensatory behaviors for less than once per week and/or less than 3 months who overvalue shape and/or weight can be diagnosed as bulimia nervosa of low frequency and/or limited duration within OSFED. Similarly, individuals with at least weekly binge eating and purging episodes, accompanied by overvaluation of shape and weight, who have been symptomatic for 2 instead of 3 months can also be diagnosed with this form of OSFED. In contrast, individuals with binge eating and compensatory behaviors at least once per week over 3 months who do *not* exhibit undue influence of shape and/or weight on self-evaluation would be diagnosed with UFED instead of OSFED because OSFED diagnostic criteria require the presence of overvaluation of weight and/or shape.

Binge Eating Disorder

For a diagnosis of BED, the *DSM-5* (2013) requires binge eating at least weekly for the past 3 months accompanied by associated symptoms (e.g., eating until uncomfortably full) and endorsement of significant distress (e.g., on a scale of 1 to 10, rating distress about binge eating as an 8) and/or role impairment (e.g., leaving work early in order to binge eat, being unable to concentrate at school because of thoughts about food) (see Figure 11.1 and Chapter 10). BED of low frequency and/or limited duration within OSFED can be assigned as a diagnosis for individuals who engage in binge eating less than weekly and/or for less than 3 months. Individuals who engage in binge eating and exhibit distress but who do not report at least three associated features can be diagnosed with UFED.

Rumination

Rumination is categorized by regurgitation of ingested food, which may then be rechewed, reswallowed, or spat out (*DSM-5*, 2013). Thus, the primary symptom of rumination (i.e., regurgitation of food) is similar to self-induced vomiting, which can be present in many other EDs. However, regurgitation can be distinguished from self-induced vomiting by the primary motivation for the behavior. Whereas self-induced vomiting is most often motivated by attempts to influence body shape or weight, regurgitation of food in rumination may be motivated by attempts to derive pleasure from the behavior or as a way to seek attention. Rumination and self-induced vomiting can be further differentiated by age of onset, with rumination often occurring in infancy or childhood in contrast to self-induced vomiting, which typically occurs in adolescence or adulthood (*DSM-5*, 2013). Detailed questions about precipitants of regurgitation (e.g., concerns about weight gain) and about emotional consequences (e.g., pleasure, relief) can clarify the appropriateness of this diagnosis. Because rumination can also occur in the context of EDs, including AN and BN, it should not be given a separate diagnosis when it occurs exclusively in the context of another formal ED. Generally, individuals who regurgitate food will be diagnosed with rumination rather than OSFED or UFED.

Avoidant/Restrictive Food Intake Disorder (ARFID)

Introduced in *DSM-5* (2013), ARFID is characterized by inadequate nutritional intake (e.g., overall intake or a willingness to ingest only a narrow category of foods of a certain texture) and can occur in children and adults (Kreipe & Palomaki, 2012). In contrast to other EDs, including AN, the criteria for ARFID do not include weight and shape disturbance or a motivation to restrict in order to influence weight or shape. Because the diagnosis of ARFID requires the presence of psychosocial or nutritional impairment, individuals with ARFID are often underweight. Consequently, differential diagnoses between ARFID and EDs, including OSFED and UFED, require careful evaluation of eating patterns, motivations for restrictive eating behavior, body image, and overvaluation of shape and weight. Collaborating with medical personnel who can provide a detailed examination of physical and nutritional status is also important (see Chapters 14 & 52).

No Eating Disorder Diagnosis

The diagnosis of OSFED or UFED requires the presence of clinically significant ED symptoms. For this reason, individuals with unusual eating patterns that are not accompanied by weight or shape concerns, medical or nutritional problems, underweight status, and/or psychosocial impairment or distress should not be diagnosed with OSFED or UFED. Similarly, the fact that an individual is underweight should not necessitate diagnosis of an eating or feeding disorder, given that some individuals have a naturally low body weight. However, individuals with EDs may deny—sometimes fiercely—psychological symptoms for various reasons (Vitousek et al., 1991; see also Chapter 18). In some instances further information may be required from family members and medical records in order to establish whether an underweight individual who denies ED symptoms should be diagnosed with OSFED, UFED, AN, or no eating disorder. In general, individuals who overeat without a sense of loss of control, distress, or impairment and have no other symptoms are not usually diagnosed with OSFED, UFED, or BED.

A Different Type of Psychiatric Disorder or a Medical Condition

Because significant changes in weight and eating are associated with various types of psychiatric disturbances, the presence of these changes does not necessarily indicate an ED diagnosis. Examples include changes in appetite and weight in major depression, fear of eating based on paranoia in psychotic disorders, fear of choking or vomiting in anxiety disorders, and weight loss associated with stimulant or cocaine use in substance use disorders. Therefore, accurate diagnosis of an ED, including OSFED or UFED, requires that anxiety, mood, psychotic, and substance use disorders all be evaluated.

Similarly, a feeding or eating disorder, including OSFED and UFED, should not be diagnosed if the behavior or symptom is caused by a medical condition. Examples include weight loss and/or vomiting due to hyperthyroidism, cancer, diabetes, celiac disease, and other gastrointestinal disturbances. For this reason, a thorough medical evaluation is an important component in establishing an accurate ED diagnosis.

In summary, differential diagnosis to establish OSFED or UFED requires accurate assessment of height and weight, eating patterns, ED symptoms, motivation for weight loss, undue influence of shape and weight on self-evaluation, food aversions, and body image disturbance. In addition, careful assessment of other types of psychopathology, including mood and anxiety disorders, as well as a thorough medical examination, are needed to establish an accurate differential diagnosis between OSFED or UFED and EDs, feeding disorders, psychiatric disorders, and medical conditions.

Empirical Support for ED-NOS, OSFED, and UFED Diagnosis

The majority of research on the ED-NOS diagnosis has focused on determining whether ED-NOS, and, by extension, OSFED and UFED, are clinically valid and can be meaningfully distinguished from the other types of ED diagnoses described in *DSM-IV* (1994) and *DSM-IV-TR* (2000). Such an empirical distinction is critical for demonstrating the validity of ED-NOS (now OSFED and UFED) as distinct from other EDs. Overall, research data suggest that individuals with ED-NOS diagnoses are similar to individuals with *DSM-IV/DSM-IV-TR* eating disorder diagnoses (e.g., AN and BN) with regard to eating pathology, general psychopathology, and mortality (Crow et al., 2002, 2009, 2012; Thomas & Vartanian, 2011).

These findings supported the expansion of the diagnostic criteria for the *DSM-5* (2013) EDs, given that individuals with conditions formerly considered "subthreshold" appear to have psychiatric, medical, and psychosocial comorbidity comparable to AN and BN. However, when ED-NOS is parsed into more specific subtypes, the results have been more complex. Some syndrome comparisons, including high- versus low-frequency BN and AN with menses versus AN without menses, have yielded nonsignificant findings (Attia & Roberto, 2009; Thomas & Vartanian, 2011; Wilson & Sysko, 2009). These data have been used to inform changes to the ED criteria in *DSM-5*, such as lowering the frequency threshold for binge eating and compensatory behaviors from twice per week to once per week in BN and eliminating the amenorrhea criteria in AN (see Chapters 8 & 9). Yet, other comparisons such as AN versus non-fat phobic AN (i.e., all criteria for AN are met except fear of weight gain) suggest significant differences in some areas (e.g., less severe eating and general psychopathology in non-fat-phobic AN than in AN) but not others (e.g., similar treatment responses and medical complications in non-fat-phobic AN and AN; Thomas & Vartanian, 2011). These findings

suggest that there may be meaningful differences between the formal EDs and some syndromes included in ED-NOS, OSFED, and UFED. Nevertheless, in general, research examining differences between the formal EDs and ED-NOS has served to underscore the clinical significance of these "subthreshold" symptom presentations. Although some research (Peterson et al., 2011) has suggested that combining some ED-NOS conditions with formal EDs may improve the longitudinal stability of these diagnoses, the distinction between ED-NOS (now renamed OSFED and UFED) and the other EDs has been retained in the interest of clinical utility.

Other research on the validity of the ED-NOS diagnosis has focused on establishing the validity and clinical utility of specific syndromes described in ED-NOS. The majority of this research has been conducted on BED, and the findings suggest that BED is characterized by levels of eating pathology, general psychopathology, clinical distress, and functional impairment that are similar to those observed in AN and BN. These data also suggest that BED can be meaningfully distinguished from AN and BN with regard to symptom presentation, course of illness, and recovery rates (Wonderlich et al., 2009; see also Chapters 4 & 10). As such, the *DSM-5* workgroup recommended that BED become a formal ED diagnosis in *DSM-5* (2013). Similar research has been conducted on PD (Keel & Striegel-Moore, 2009) and NES (Striegel-Moore et al., 2009), both of which are now included in OSFED.

Finally, recent research using advanced statistical methodologies such as latent structure analysis (LSA) and taxometric analysis has attempted to identify empirically defined categories or classes of EDs. These studies have commonly found classes of EDs resembling AN, BN, and BED (Crow et al., 2012), but two "alternate" syndromes have also emerged across multiple studies. One of these classes is characterized by frequent purging or other compensatory behaviors and infrequent or no binge eating. Individuals assigned to this class, which resembles current conceptualizations of PD, appear to have levels of eating pathology and general psychopathology similar to those in other classes of EDs (Pinheiro, Bulik, Sullivan, & Machado, 2008; Striegel-Moore et al., 2005).

The second "alternate" class of EDs that has repeatedly appeared in LSA studies is characterized by low-weight individuals who deny ED cognitions and behaviors and may be similar to descriptions of non-fat-phobic AN. Individuals assigned to this class tend to have lower levels of eating pathology but have similar medical problems, poorer treatment response, and higher mortality than individuals in other classes of EDs (Crow et al., 2012; Eddy et al., 2009, 2010; Keel et al., 2004; Mitchell et al., 2007). Although these data do not provide enough evidence to support PD and non-fat-phobic AN as distinct ED diagnoses, they do indicate that further research is needed to evaluate the clinical utility and validity of these syndromes. In general, these empirical classification studies also provide some support for the *DSM-5* (2013) diagnostic criteria, including differentiation of OSFED and UFED from AN, BN, and BED.

Conclusions and Future Directions

In summary, ED-NOS, a diagnosis reconceptualized as OSFED and UFED in *DSM-5* (2013), has been found to be the most common ED in clinical settings as well as in community epidemiology samples (Fairburn et al., 2007). Although individuals with ED-NOS and, presumably, OSFED and UFED, do not meet full criteria for a formal ED diagnosis such as AN, BN, or BED (see Chapters 8 to 10), they nonetheless show comparable levels of associated psychopathology, psychosocial impairment, and mortality risk (Crow et al., 2002, 2009,

2012). To reduce the prevalence of ED-NOS as a residual category, diagnostic criteria for AN and BN in *DSM-5* (2013) have been broadened (see Chapters 8 & 9) and BED has been added as a formal eating disorder (see Chapters 4 & 10). Preliminary data suggest that ED-NOS and, as a result, OSFED and UFED, may indeed be less prevalent with the adoption of *DSM-5* criteria (Keel et al., 2011). The proper diagnosis of OSFED or UFED requires knowledge of ED pathology and can be accomplished with the aid of empirically supported diagnostic interviews and self-report questionnaires. Further studies are needed to better understand OSFED and UFED in many respects, including clinical features, heterogeneity and potential subtypes, etiological and maintenance mechanisms, and longitudinal course. In addition, with the adoption of new criteria in *DSM-5* (2013), future work is needed to help guide the differentiation of OSFED and UFED from other types of eating and feeding disorders, including outcome research to establish effective prevention and treatment strategies.

Thus, the recent introduction of *DSM-5* (2013) has brought to the fore the need for new research in a variety of areas to better understand, assess, and treat individuals who are diagnosed with OSFED and UFED. Data are needed to determine the prevalence and incidence of both OSFED and UFED, as well as to establish empirical validation of these criteria in community and clinical samples. Extensive research will be needed to determine the reliability and validity of existing psychometric instruments to assess and diagnose OSFED and UFED. Longitudinal data are also needed to clarify the short- and long-term course of OSFED and UFED, including diagnostic crossover with other ED diagnoses as well as the absence of an eating disorder (see Chapter 55). Future investigations are also essential to develop more accurate assessment and diagnostic strategies for OSFED and UFED, and to differentiate OSFED and UFED from other eating and feeding disorders through the use of interviews, questionnaires, and innovative techniques that incorporate the use of technology (e.g., ecological momentary assessment).

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12

Severe and Enduring Anorexia Nervosa: Diagnosis, Features, and Radical New Treatment Approaches

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Gull (1874) and Lasègue (1873), in their pioneering accounts of anorexia nervosa (AN) published in the late 19th century, expressed optimism about the illness's eventual outcome. This view was supported by clinical descriptions of cohorts, often collected by charismatic psychiatrists in the latter part of the last century, which seemed to indicate there were few nonresponders and generally an excellent outcome with treatment. This view persisted until the first scientifically based prognostic studies showed the hidden reality. For instance, in a review of AN outcome in the 20th century, Steinhausen (2002) identified 119 studies of 5,590 participants with length of follow-up of 1–29 years. Of these participants, a mean of 47% recovered (range 0–92%), 34% improved (range 0–75%), 5% died, while 21% developed a chronic or severe and enduring eating disorder (ED; range 0–79%). Even these figures give a gloss to reality (Von Holle et al., 2008). Using conservative criteria of recovery, Von Holle et al. reported that after 10 years of illness one tenth of AN and BN patients had recovered, with only 16% of AN and 25% of BN meeting recovery criteria after 15 years.

This chapter discusses chronicity as it pertains to AN. It sets out diagnostic criteria of severe and enduring anorexia and separates the syndrome from acute AN. It describes the clinical features that reflect chronicity: social and psychological as well as behavioral, and includes common comorbid symptom clusters. It addresses the particular clinical needs of the patients and their prognosis. The chapter outlines proven and putative treatment approaches, including coerced and compulsory treatment.

Chronicity

There is no generally accepted definition of what constitutes "chronicity" in AN (Tierney & Fox, 2009). Steinhausen (2002) suggests that after 6–7 years of illness, the likelihood of people recovering reaches a plateau but fails to reach zero, although most clinical evidence

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suggests that such a plateau does not appear until 10–20 years after the onset of the disorder. Basing the concept of chronicity on the passage of time alone has led to sterile debate. Some argue, on little evidence, that chronic AN should only be considered after 9 years or after 11 years of illness (Touyz et al., 2013). Others say that it is the psychological presentation, not time, that is the essence and the term should be restricted to those who score high on standardized measures—yet there is little evidence that marked abnormality on questionnaires is indicative of prognosis. Others claim that meaningful diagnosis can only be made in the clinical interview, for the benchmark in longstanding AN is the severity of the psychopathology and particularly that the anorectic beliefs become "hard-wired," pushing the patient into denial. "Chronic" also implies that the anorectic will not recover, though many clinicians have shown that recovery is always possible (Steinhausen, 2002; Strober, 2009) and it is best to consider patients as always having the potential to recover. So the concept of chronic anorexia as a diagnostic term has or, more accurately, is being dropped and replaced with a more inclusive and hopeful term: severe and enduring anorexia.

Severe and Enduring Anorexia Nervosa (SE-AN)

To make a diagnosis of SE-AN, six main features need to be present. The thinking of people with AN is archetypal and diagnostic. Indeed it is pathognomonic. The **psychopathology** of SE-AN is dominated by a pursuit of thinness combined with disparagement and hatred of the body (see Chapters 2 & 8). The core element—one that doesn't occur in any other condition—is an irrational fear, or phobia, of normal body weight. People with AN have an intense fear of putting on weight, as opposed to a mere dislike. Furthermore, it is not obesity of which they are fearful, but normal body weight. There is a pursuit of thinness, a hatred and fear of a normal weight, and a profound disparagement of body shape.

These beliefs lead to **behavioral changes** aimed at weight loss. It is important to remember the obvious point when considering treatment: it is a phobia, which leads to weight loss behavior and not vice versa. The behavior, such as dietary restriction, vomiting, or excessive exercise, leads to weight loss, and body weight falls below BMI 17.5 (e.g., at 65 in. [1.65 m], 105 lb [47.6 kg]). This weight loss in turn leads to an **endocrine disturbance** sufficient, if a woman, to stop menstruation, and in both sexes leads to low sexual drive.

The illness must have been **persistent** and periods of remission are not common, though they do not exclude the diagnosis. The data referred to above suggest the diagnosis should not be used unless the illness has been present for a period of 7 years at least and consistently so at that. If the patient has had a number of remissions the length of illness should be correspondingly increased, and most experts wouldn't feel comfortable making the diagnosis in a patient with a fluctuating course unless there had been 11 or more years of ill health.

SE-AN sufferers are highly **resistant to treatment**, having repeated "treatment failures." In other words, a patient with longstanding but untreated AN should not be diagnosed with SE-AN. Further, they should have failed to respond to a broad spectrum of therapies specifically designed to address the ED and delivered by different therapists. For example, someone who has had years of psychodynamic therapy without weight recovery has not been adequately treated for the diagnosis of SE-AN.

Severity is measured not by questionnaire but by the effect of the illness on quality of life and health. Sufferers tend to be socially isolated, unemployed, or not to have fulfilled their potential. They are intensive users of general practice or family doctors and community health

services as well as the welfare system. Comorbid features such as depression, obsessional features, and addictive and self-damaging behavior are frequent (see Chapters 15 & 54).

These six diagnostic concepts will guide the clinician away from the obvious pitfalls. SE-AN should not be used when a patient has a lengthy history of being symptom free but a tendency to relapse, or with someone who is recovering despite the length of illness, or with someone who has been inadequately treated.

Characteristics of SE-AN

For almost all, the length and severity of the illness changes the psychological, social, and physical aspects of the anorectic illness. The sum seems like a personality change, though the component parts are all part of acute AN, writ large and rigid.

Psychological Features of SE-AN

SE-AN patients often have a strong sense of self-determination, characterized by perfectionism and rigid inflexibility. In the clinic, you sense the patient's vulnerability; she is cut off from obvious reality. She denies emaciation even if asked to calculate her own BMI; it seems as if her understanding of her body and the real world of biology and physics become parallel tracts. Even a well-educated woman will claim her metabolism is different than everyone else's and that her intake of food or her overexercising are unrelated to her weight, seemingly a denial of the laws of thermodynamics. Her value judgments are skewed and her outside reality is not that of the observer's. Yet there is no question of psychosis: it is an overvalued idea. Reasoned discussion relating her statements to common knowledge of metabolism, or even Newton's laws, will bring her briefly to reality. The latter is particularly successful with the more scientifically trained patient! For some, the removal of denial is too much and they will "run away" or "fail" the next appointment. Fully established SE-AN does not allow the window of truth to open and reality to enter the mind. Patients rationalize that they do not need help and can deal with their own difficulties.

It is sometimes said that SE-AN patients are mistrustful of interpersonal relationships. In reality this is fear, usually from the pressure to gain weight, and they just wish to avoid their doctor. The terror of weight gain makes them extremely apprehensive. Any food intake is perceived as loss of control. Cognitive therapists will describe this as distorted thinking, and dynamic therapists as ineffectiveness due to earlier trauma. Whichever, there is an adverse impact on conceptual, perceptual, and decision-making abilities.

SE-AN patients are distressed by their mood. They will describe being depressed but the clinician should avoid antidepressant medication. The mood change is usually understandable in the context of the patient's lifestyle and problems. Depression and anxiety can be best seen as stemming from underlying issues and trauma, though undoubtedly they are exacerbated by the physiological and metabolic disturbances that accompany starvation and malnutrition (see Chapters 14 & 52). It is interesting, however, that they always become worse, not better, with weight gain. Patients find it easy to talk about sadness and anxiety, but the dominant emotion in SE-AN is anger. Anger is not an easy thing to talk about to a GP and is often missed. However, it is probably the most crippling emotion of the condition.

Lastly, it should not be forgotten that SE-AN patients quickly pick up if their professional advisors give up hope. They will be adversely affected by a less than optimistic view regarding the long-term outcome of the disorder.

Social Features of SE-AN

SE-AN patients retreat into their own world, becoming perfectionists and working long hours. They lose concentration, as their thoughts become increasingly limited to food and weight. They give up their friends and distance themselves from their families. The boundaries of their world become very narrow. These patients pose a significant burden to parents and the community as well as their professional carers (Striegel-Moore et al., 2008; Strober, 2004a). Longstanding AN has the highest mortality rate of all mental illnesses, with one fifth dying as a result of suicide, emaciation, or intercurrent disease (Steinhausen, Seidel, & Winkler, 2000). It has been estimated that direct inpatient costs exceed that of schizophrenia (Rieger et al., 2000). The AN is often denied and the patient avoids coming to doctors or therapists until there is an emergency.

Physical Complications of SE-AN

Many of the physical complications of acute AN (see Chapters 14 & 52) are progressive. The persistent emaciation in SE-AN patients leads therefore to significant medical comorbidity (Arkell & Robinson, 2008; Birmingham & Treasure, 2010; Robinson, 2009; Treasure et al., 2001) that results in repeated presentations to general and specialist medical facilities.

Osteoporosis and osteopenia are particular problems. Bones become demineralized of calcium and liable to fracture. Chronic exercising can lead to early stress or frank fractures after only slight overactivity. It is indicative of the power of the weight phobia that patients will continue to run even when they know they have a stress fracture and are in pain. The psychological pain of stopping exercise is more powerful that the physical pain of activity.

People with SE-AN at low weight describe being intermittently dizzy: a combination of electrolyte abnormalities and intermittent phobic anxiety. Tachycardia and low blood pressure are the norm when a SE-AN patient is nasogastrically fed, whether under restraint or not. Again, the panic of weight phobia preconditions the body to sympathetic discharge. Chronic hepatic and cardiac complications occur but are rare.

Like patients with acute AN, SE-AN patients are sensitive to cold and, interestingly, wake early in the morning like patients with melancholic depression. They are restless, and the authors have wondered if these phenomena follow starvation and emaciation and have evolutionary survival value; put simply it is the early bird that catches the worm.

The emaciation also leads to physical changes such as dry skin and weak muscles. SE-AN patients who purge develop a swelling in their salivary glands and their teeth become eroded, particularly on the palatal surfaces. They also develop Russell's sign, which is abrasions on the knuckles of the fore- and middle fingers, usually of the left hand (see Chapters 3 & 9). The abrasions, which can become infected, are caused by friction when using the fingers to initiate vomiting or by vomitus or by acidic drinks, such as a cola, used to help vomiting. The difference from acute AN is that these features are persistent: the salivary glands remain permanently swollen (producing what the patient describes as a hamsterlike appearance), the knuckles ulcerate, and teeth need to be extracted.

The majority of SE-AN patients are infertile because of their low weight, though male patients may have reduced fertility but lack potency. Most women have major concerns about their potential to have children. They can be reassured that even though they may have had three decades of amenorrhea, their fertility on weight restoration is unaffected. Further, at persistent low weight, it cannot be assumed that ovulation will not take place. Occasionally it does; put another way, any SE-AN patient may be fertile and be unaware of the need for contraception. Counseling is required around this difficult issue.

Multi-Impulsive Behavior

Increasingly, SE-AN is associated with addictive and self-damaging behavior, known as multi-impulsive behavior (see Chapters 15, 54, & 57). This behavior may include abusing alcohol, other drugs, and other substances, unwise sexual activity, and overspending. The sufferer perceives these behaviors as being "out-of-control," and all may be described as having the same function, which is to quash feelings (Lacey & Evans, 1986).

Multi-impulsive behavior is reciprocal; in other words, one behavior dominates whilst the others are recessive. For example, a bulimic may abuse alcohol, during which time the ED becomes quiescent. In addition to feelings of being out-of-control, multi-impulsive behavior is associated with low self-esteem, marked depression, and, interestingly, anger.

Alcohol and Other Drug Abuse

Eating disorders and substance abuse disorders (SUDs, including abuse of alcohol and other drugs) often exist side by side, but the mechanism is not understood (Lacey, 1993; see Chapters 15 & 54). Some believe that both disorders are manifestations of a common shared underlying cause or set of causes; others believe that the two disorders may share the same risk factors. In addition, it is theorized that EDs and SUDs are manifestations of a predisposition toward being impulsive. This is thought to relate to the opioid compounds that occur naturally in the body and act like opiates in specific circumstances.

The relationship works in both directions, that is, just as alcohol and other substance abuse may occur in AN, so EDs are common in women with alcoholism. In fact, figures as high as 30% have been quoted. Of these, one third was diagnosed with AN and two thirds with BN. The ED is usually primary, preceding the alcohol abuse (Beary, Lacey, & Merry, 1986; Lacey & Mourelli, 1986).

Self-Harm

Cutting, scratching, or burning occurs in about half of SE-AN patients (Kent et al., 1997; Lacey, 1993; Morgan & Lacey, 1999; see also Chapters 15 & 54). Cutting is the most common form of self-mutilation and is generally carried out on the arms, legs, or abdomen with a pin, knife, or razor blade. Some cuts are no more than scratches, but the individual may pick at them so they do not heal easily. Other cuts are so severe that they require several stitches, but the person may remove these on returning home as part of the self-damaging behavior. Self-harmers may burn their limbs or breasts with a lit cigarette, or bang their heads or fists against a wall, causing cuts and bruises that are explained away in credible tales. In women, the most pernicious forms of self-damage are cuts and stabbings to the vagina, usually internally. Although light cuts in the area of the vagina are sometimes used as a means of fabricating a "period," more usually it is an indication of profound psychological illness. Fortunately, this behavior is rare.

Self-mutilation may seem like a cry for attention. However, individuals invariably try to cover their scars or burns with long sleeves and trousers. Some self-harmers feel numb or dead inside and report that the action of cutting (or scratching or burning) is triggered by a desperate need to feel something—even physical pain. Other self-harmers are aware of a build-up of tension so great that they feel they would explode if they did not do something. On carrying out the cut, scratch, or burn, the self-harmer experiences a rush of endorphins (the hormones that generate a feelgood factor), which effectively releases all tension and stress—at least for a moment or two.

Overdosing

Most overdoses are impulsive and not thought through (Kent et al., 1997). SE-AN patients are not usually actively suicidal, though death may occur by omission. An overdose is more a means of indicating to others how desperate and depressed they are feeling. Most commonly, compounds containing aspirin or paracetamol (acetaminophen) are used, which in an emaciated patient are particularly dangerous, leading to liver and kidney damage and death.

Sexual Behavior

People with SE-AN lack libido or sexual drive (Morgan, Lacey, & Reid, 1999). This is because their weight is below the threshold necessary for sex hormone activity and hence sexual awareness. Some purging SE-AN patients, however, are at a higher weight and so are sexually aware, though they lack the maturity to handle it. Sometimes pedophiles or men who are inadequate emotionally are drawn to the immature body of a chronic anorectic. Such men may be particularly interested because the girl or woman is of a legal age to consent and hence they avoid prosecution.

A multi-impulsive SE-AN patient does not value her body, hating and despising it, and, often under the influence of alcohol, becomes sexually disinhibited. This is not promiscuity. It stems from low self-esteem and the inability to value herself or her body. The sexual activity is described as punitive and pleasureless.

Anorexic, addictive, and self-damaging behavior are all visible symptoms of underlying problems that serve to protect the individual from real difficulties that he or she cannot handle. Giving up this pernicious form of AN or BN is particularly difficult, for in so doing, the individual feels defenseless against troubles of which he or she is not fully aware.

Prognosis

The prognosis of EDs in adults is not good (see Chapter 55). Progressively, publications over the last 30 years have indicated a deteriorating response. By the start of the 21st century, only a quarter of bulimics and 16% of anorectics (Von Holle et al., 2008) were making a full recovery when measured on strict criteria. Some believe the deteriorating response is because of pathoplasticity, that is, the tendency for these disorders to become associated with multi-impulsive behavior (Lacey & Evans, 1986). Others suggest that adolescents with AN are not treated with sufficient vigor. Significant numbers of children with eating disorders are being missed (BBC News, 2013; see also Chapter 13). The current fashion is to restrict the use of inpatient treatment with a brief admission with only partial weight recovery before discharge to outpatients. The policy is financially driven but rationalized by emphasizing the advantage of keeping the adolescent in the home and school environment. The result is that patients spend many years at a low but medically safe weight, during which time the psychopathology becomes fused and chronic.

Some wonder if cognitive-behavioral therapy (CBT; see Chapter 56), which is experimentally validated in the treatment of EDs, is not being followed exactly as the manuals describe. Others, on the contrary, believe that "pure culture" CBT is too narrow and a more eclectic therapy should be used, where cognitive and behavior methods are mixed with dynamic insight-directed therapy or family therapy. The jury is still out, but the message is that AN must be treated early in adolescence or it is likely to become severe and enduring (see Chapter 55).

Treatment

Despite the problems in treating AN (see Chapters 50–63)—or perhaps because of them—there have been only 10 randomized controlled trials (RCTs), and these have involved fewer than 400 patients. These studies have been criticized for having high dropout rates (approximately 30%) and nonconclusive results (Strober, 2004a; Touyz et al., 2013). There is, therefore, no clear treatment despite the seriousness of AN. There has been only one RCT for SE-AN (Touyz et al., 2013). Further there is no evidence that any one model of psychotherapy is superior to others. Whilst current clinical opinion regards CBT as the "best hope" for EDs, when applied to AN the results have been inconsistent and, indeed, disappointing. As a result, in clinical practice there is a tendency to implement therapy changes in ways that are impressionistic and not scientific. Wonderlich et al. (2012) write that "Clinicians often modify treatment, target co-morbid complicating disorders, switch to intermittent supportive treatments, or intensify treatments with higher levels of care, all of which are based on clinical decision making with a minimal of scientific guidance" (p. 467).

Expert consensus, rather than rigorous research, is the foundation of the recommended treatment of adults with AN. Because so little is known, the general response is to recommend the least restrictive treatment compatible with safety. Most national guidelines (American Psychiatric Association, 2000; Beumont, Hay, & Beumont, 2003; NICE, 2004) agree that there should be a continuum of care, with outpatient individual therapy being favored initially if a patient is medically stable. There is no agreed "best" outpatient therapy, and significant dropout is often reported in published treatments and is even more common than reported in inpatients populations (Bulik, Berkman, Brownley, Sedway, & Lohr, 2007; Dare, Eisler, Russell, Treasure, & Dodge, 2001; Halmi et al., 2005). CBT has been shown to be effective in relapse prevention in two independent trials for adults, including (but not exclusively) many individuals with SE-AN (Carter, Woodside, & Kaplan, 2000; Pike, Walsh, Vitousek, Wilson, & Bauer, 2003).

Thus, in the absence of scientific guidelines, clinicians who have the responsibility to treat patients with SE-AN resort to modifying existing treatment protocols, seeking out alternative strategies that make accommodations for patients' chronic status, paying close attention to comorbidities and blending supportive, harm-reduction, and recovery-based strategies. Wonderlich et al. (2012) state that, in the attempt to meet this therapeutic challenge, "treatments may devolve into relatively unfocused, intermittent, supportive interventions, where goals become unclear and monitoring of clinical status becomes impressionistic and imprecise" (p. 476).

Longer duration of illness is a predictor of treatment resistance and mortality in the eating disorders (Keel et al., 2003; Reas, Williamson, Martin, & Zucker, 2000). Treatment resistance has traditionally been defined as a lack of improvement in ED pathology following intervention. AN patients typically develop a history of negative treatment experience and repeated treatment failures (Woodside, Carter, & Blackmore, 2004). Both patients and clinicians often experience a sense of hopelessness about the possibility of change (George, Thornton, Touyz, Waller, & Beumont, 2004).

Touyz and his collaborators, which include the first author (J. H. L.), discuss the challenges in tailoring treatment for individuals with SE-AN (Touyz et al., 2013). They point out that treatments that focus on both physical and psychological recovery run the risk of misalignment with patient aims and readiness for recovery, which results in high dropout. As weight phobia is pathognomonic to the disorder, any treatment that prioritizes weight gain in a patient who has suffered repeated "treatment failures" will lead to avoidance of treatment

or dropout at the first opportunity. Insurance companies often refuse to treat on the basis that these individuals do not respond to known treatments, and in the United Kingdom, for instance, some National Health Service (NHS) funders insist that specialist ED services discharge patients with SE-AN to generic psychiatric services or nothing on the same grounds. Globally, treatment programs are limited in their capacity to treat these patients, and it is not uncommon for nonspecific medical palliation to become the default care (Lopez, Yager, & Feinstein, 2010; Strober, 2009).

Given the choice of palliation, especially for relatively young individuals with SE-AN, and taking the challenges and complexities of treatment into account, a different paradigm is needed (Goldner, 1989; Robinson, 2009; Strober, 2004b; Vitousek, Watson, & Wilson, 1998; Williams, Dobney, & Geller, 2010; Yager, 1992). Such a paradigm must reflect the severe and enduring nature of this debilitating disorder, the weight phobia at its core, and the understandable avoidance that is manifest as dropout. Rather than recovery being the basic premise, treatment should focus more upon retention, improved quality of life with harm minimization, and avoidance of further treatment failure (Strober, 2009; Williams et al., 2010). Such an approach needs to take into account the challenges in treating patients with longstanding low levels of motivation for change, neurocognitive deficits, and a self-view and lifestyle dominated by the illness (Hatch et al., 2010; Schmidt & Treasure, 2006; Strober, 2004a; Treasure & Russell, 2011).

Any treatment for SE-AN must intrigue patients by offering something other than, or something more than, weight gain. Most importantly SE-AN patients wish to address their quality of life and mood. The treatment would need to retain the patient by being clinically meaningful. Two approaches, if adapted, have promise. A modified cognitive-behavioral therapy for AN (CBT-AN; Pike et al., 2003) has documented efficacy for relapse prevention for adult AN, and a modified Specialist Supportive Clinical Management (SSCM; McIntosh, Jordan, & Bulik, 2010; McIntosh et al., 2006) is a treatment that has demonstrated utility in adult AN. Although individuals with SE-AN participated in the initial studies evaluating both CBT-AN and SSCM for adult AN, no study had focused exclusively on SE-AN until the Touyz et al. (2013) study.

Specialist Supportive Clinical Management

SSCM mimics usual outpatient clinical practice by combining features of clinical management and supportive psychotherapy. It is flexible and readily tailored to the individual patient's specific needs and goals. Clinical management includes education, advice, care, and support, and fostering a therapeutic alliance that promotes adherence to treatment. Supportive psychotherapy aims to assist the patient through use of praise, reassurance, and advice, without mandating change. There is also collaborative goal setting and symptom monitoring. Active strategies are suggested but not enforced. The abnormal dietary patterns typical of AN (see Chapters 2 & 6) are addressed in SSCM through education about normal eating and increase in weight, strategies for weight maintenance, information about energy requirements, and relearning to eat normally (see Chapter 61). Information is provided via discussions and in handouts.

Manualized SSCM for SE-AN was modified such that weight gain was not prioritized. Instead, SSCM encouraged patients to make changes to improve quality of life. The rationale for this emphasis (Touyz et al., 2013) is that research suggests that treatments that target psychosocial functioning are especially appropriate when there has been repeated relapse or a

long duration of illness. By helping individuals improve quality of life, they will be motivated to make progress on their core ED symptoms.

Cognitive-Behavioral Therapy

CBT-AN follows four clearly defined phases of treatment, each of which is manualized and adapted for SE-AN. Unlike SSCM, CBT-AN is an active treatment aimed at restoring normal eating habits by challenging underlying beliefs and through cognitive restructuring and change in behavior (see Chapter 56). It involves planned "homework" and it sets clear aims.

The CBT-AN for SE-AN is based on the CBT-AN protocol developed by Pike et al. (2003), which focuses on the cognitive and behavioral disturbances linked to the core features of AN and also on more global issues associated with AN, including motivational (see Chapter 63) and schema-based work. Phase I provides specific strategies for initiating treatment, orienting patients to CBT, and addressing issues of motivation. Phase II focuses on strategies for addressing weight gain and addresses cognitive distortions and behavioral disturbances associated with eating and weight. Phase III expands the focus of treatment to schema-based work, which addresses relevant issues extending beyond the specific domains of eating and weight. Phase IV focuses on reviewing the course of therapy, consolidating gains, and preparing continuation work for after therapy ends.

Although the four phases of treatment are described sequentially, depending on the course of therapy for each individual, the treatment is flexible in terms of applying modules of the protocol as needed throughout the course of treatment. CBT-AN for SE-AN is modified in the way SSCM is modified to reflect the shift in treatment goals. Specifically, weight gain and recovery from core features of the ED were not assumed to be treatment priorities. Instead, treatment goals were set collaboratively and weight gain, though encouraged, was not mandatory.

Touyz et al. (2013) tested these two treatments on a severely ill AN population with significant emotional, social, and medical problems. The sample had an average age of 34 years and had been ill for a mean of 17 years. Most were unemployed, despite having had higher education. Most had never had an emotional relationship, let alone a meaningful one. The majority were having medical treatment and three quarters were causing medical concern. The study yielded a number of surprises that have changed the way we view the treatment of chronic AN. Unlike any other AN treatment study, only a few (12%) patients dropped out of treatment. Further, the completion rates were the same in both treatments. This must be a reflection of something that is common in both treatments and different from treatment in other studies. The most likely is that weight restoration was not central to treatment and therapists worked on areas that the patient herself deemed important, in particular, quality of life.

The relative efficacy of CBT and SSCM was not marked. The patients responded to the treatments irrespective of orientation. Both treatment groups reported significant improvement in quality of life measures, mood disorder symptoms, and social adjustment. Those who received CBT showed a greater improvement in ED symptoms and a greater readiness to change their illness, whilst those who had SSCM had greater improvement in health-related quality of life, with improved mood and social adjustment. This might give an indication of which patients should try which of the two approaches first.

Although weight gain was not emphasized in either treatment, not one patient lost weight and almost all gained weight. Three patients have since the end of the study had a baby! This confirms the authors' belief that improvement in domains outside the core

pathology can significantly affect patient well-being and disease burden and improve their quality of life, which will further motivate and enable them to make progress on their core ED symptoms.

These findings challenge the view that individuals with an enduring course of AN have little or no motivation to change, are unlikely to respond to conventional psychosocial treatments, and have a high treatment dropout. The Touyz et al. (2013) study shows that SE-AN patients can be retained in treatment and can make significant and meaningful improvements with therapy. The authors argue the findings have public health implications, in particular that specialist clinics should work with chronic anorectics and not pass them to family practitioners or generic services, or give up on them in any other way.

Coerced Treatment of SE-AN

Patients with a diagnosis of SE-AN are likely to have experienced various forms of coercion from health professionals during the course of their illness. This ranges from strong persuasion by clinicians to engage with and remain in treatment to formally compelled treatment under a legal restriction.

Those with SE-AN have, by definition, received treatment on numerous occasions across different treatment modalities. As a result, they are likely to have experienced levels of **perceived coercion** stemming from clinical persuasion, which verges on *de facto* compelled treatment. In treatment for AN, it is common for "*strong persuasion, brow beating, or even outright moral blackmail* [emphasis added]" to be deployed (Carney, Tait, & Touyz, 2007, p. 390). Clinicians use this form of leverage to try to obtain treatment compliance, with patients being directly or indirectly notified that a lack of adherence to treatment would likely result in the enforcement of legally compelled treatment.

For many patients with SE-AN, such experiences are likely to magnify the power imbalance between patient and clinician, and have the effect of actually delaying, or preventing, the development of therapeutic alliance. Perceived coercion is also likely to influence their future engagement with treatment and their opinions of clinicians in general. Guarda et al. (2007) studied perceived coercion in 139 ED patients at admission and again 2 weeks into inpatient treatment. Those with a diagnosis of AN reported higher levels of perceived coercion and pressure and a lower sense of procedural justice compared to those with BN. The assertion that perceived coercion is viewed entirely negatively by patients is challenged by these findings, in which nearly half of patients who originally objected to their admission changed their minds within 2 weeks of treatment.

Compulsory Treatment

Compulsory treatment in the management of seriously ill patients is used when their AN is "life threatening and associated with core features of denial of illness or thinness to a degree that the use of involuntary legal commitment may be appropriate" (Watson, Bowers, & Andersen, 2000, p. 1809). Carney et al. (2007) identify three main indicators associated with the use of compulsory treatment: the number of previous treatment episodes; the complexity of the patient's condition; and the current health risk (typically measured by BMI). These indicators can be seen to closely link in with the clinical factors associated with patients diagnosed with SE-AN. Again, the use of compulsory treatment highlights both a power imbalance and the lack of therapeutic alliance. This may be

magnified still further due to the fact that the majority of patients placed under a legal restraining order have entered treatment as an informal (noncompelled) patient (Ramsay, Ward, Treasure, & Russell, 1999). So, those with SE-AN are often requested to attend residential treatment informally, and once within the program they become "trapped" there if and when clinical staff deem it necessary for treatment to be compulsory. It is perhaps no wonder then that many such patients "down tools" and cease cooperating with the treatment they are prescribed, often leading to a protracted battle of wills between patients and staff. Such experiences are also likely to influence their engagement—or lack thereof—with clinicians during future treatment episodes both in the community and in residential settings.

However, the decision to enforce compulsory treatment is not taken lightly, and, as noted above, is used primarily as a last resort. The aims of such a treatment approach are typically to achieve medical stability, and what limited data there are tend to suggest that these aims are broadly met. The short-term outcomes of compulsory treatment have been found to be largely comparable to those of voluntary patients (Watson, Bowers, & Andersen, 2001; Yager & Andersen, 2005). With these outcomes in mind, it is also worth remembering that the patients receiving compelled treatment would have been otherwise lost to treatment. The long-term benefits of compelled treatment are at best marginal, but at least the patients are kept alive, with the chance of recovery still there, somewhere. This approach, which essentially delivers patients from risk of harm to a low, if medically stable weight, maintains the constancy of living with the illness that is the hallmark of SE-AN.

Compulsory Recovery

The use of compulsory treatment is almost overwhelmingly used to restore medical stability to SE-AN patients. The previous section has highlighted that, although this approach does achieve these aims, it also furthers the experience of those with SE-AN that the perpetuation of low weight is all that is achievable—and all that is expected—from treatment. This in some way could be said to reinforce the core facets of the SE-AN diagnostic criteria. One recent intervention developed by the first author (J. H. L.) is "compulsory recovery." The central tenet of compulsory recovery is to provide a "last chance" for patients at high risk of being categorised SE-AN. This intervention was designed for patients at a dangerously low weight who have already experienced many failed treatment episodes, including treatment under a Mental Health Act restriction order. All patients had an illness duration of less than 7 years, and clinicians held the view that unless they responded to treatment on this occasion, these patients would become readily classified as SE-AN. In essence, this was a calculated clinical attempt to prevent the transition from AN to SE-AN.

Standard compulsory treatment for AN is primarily focused on weight gain and provides little psychotherapeutic input for the patient. Weight targets are typically low, for example around a BMI of 15. Where compulsory recovery differs is that target weights for patients on this treatment are around the normal weight range (BMI = 20), and patients receive intensive psychotherapeutic input throughout their treatment. *Any* posttreatment weight loss would be cause for rapid readmission to treatment. These characteristics of compulsory recovery ensure that this is not a pure refeeding exercise, but rather an intensive therapeutic program with disciplined compulsory weight gain structured into it.

Conclusions and Future Directions

Treating those with SE-AN is undoubtedly a challenge for clinicians, and future research would do well to concentrate on means to enhance the outcome for those patients diagnosed as such. The work by Touyz et al. (2013) has provided a foundation on which to build. Further examination of the efficacy of SSCM and CBT-AN would enable the building of an evidence base from which we could more confidently respond to the needs of SE-AN patients. The Touyz et al. (2013) study had low dropout rates, which intimates that a focus on quality of life rather than target weight was more acceptable to SE-AN patients. Future research may wish to consider other means by which to "fast-track" the development of therapeutic alliance with this population, to enhance engagement, and maximise treatment outcome (see Chapter 63). Long-term follow-up work is also required to chart the course of illness of these patients receiving such treatment. The use of compelled treatment may be at times contentious and controversial, but for some patients this may be the best option, as highlighted by the compulsory recovery section above. Again, the long-term course of illness and health service usage of patients receiving such treatment is an important area on which to focus.

The recalcitrant psychopathology of AN ensures that many patients, over the course of time, are further classified as SE-AN (see Chapter 55). In the eyes of many, those with SE-AN are beyond any reasonable chance of recovery, but this chapter has elucidated that this is not always the case. The chance of recovery from SE-AN is in fact ever present, although it may take many treatment episodes and new treatment approaches to fully "tap into" these chances with any hope of success. The emerging treatment approaches identified in this chapter shift the primary emphasis away from weight targets and towards the identification of aspects of quality of life that the patients themselves want to identify. This appears to be a crucial factor in the engagement with treatment, from which meaningful change can be made. A flip side of this approach is the stance of "compulsory recovery." As a last chance, designed to prevent the descent into SE-AN, this approach aims to proactively "take on" AN during a treatment episode that is designed to get the patient to a physical and psychological place of safety. This is in direct contrast to the inadvertent maintenance of AN that many compelled treatments achieve. As a preventative approach to SE-AN, this is a tool at the disposal of the proactive clinician. For those that are already diagnosed as SE-AN, a collaborative treatment involving CBT-AN or SSCM would seem the best option at this stage.

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13

Diagnosing Eating Disorders in Children and Adolescents

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Childhood and adolescence represent crucial time periods for growth, development, and establishing lifetime healthy habits and behaviors. Lack of appropriate nutrition or development of unhealthy eating attitudes and behaviors can result in delays in puberty, compromised bone development due to osteopenia (reduced bone mineral density), and other medical challenges. Such disturbances in nutrition may result in significant harm during vulnerable periods of brain development and physical growth (Bravender et al., 2007; DeSouza, 2006). Eating disorders (EDs) in children and adolescents can go unrecognized by the unsuspecting clinician or parent, magnifying this risk further. With appropriate vigilance and knowing how to ask the right questions, children with disordered eating can be more easily recognized, with earlier detection potentially leading to improved outcomes (Rome & Committee on Adolescence, 2003; Rosen & Committee on Adolescence, 2010).

The *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., *DSM-IV*; American Psychiatric Association, 1994) did not capture some of the nuances of the presentation of EDs in youth, leading to later diagnosis in some cases, with a negative impact on outcomes (Bravender et al., 2007; Peebles, Wilson, & Lock, 2006). The challenge for *DSM-5* (American Psychiatric Association, 2013) was to improve diagnostic criteria for various ED subgroups (see Chapters 8–11), allowing for improved recognition and earlier and more appropriate treatment by disease category, all toward a goal of improved outcomes. This chapter will review *DSM-IV* versus *DSM-5* criteria for diagnosing EDs in children and adolescents, including newer categories such as Avoidant Restrictive Food Intake Disorder or Other Specified Feeding or Eating Disorder (OSFED), which includes "atypical Anorexia Nervosa" (*DSM-5*, 2013, p. 353), and "Bulimia Nervosa (of low frequency and/or limited duration)" (*DSM-5*, 2013, p. 353). Strategies for recognizing EDs in children and adolescents will be highlighted.

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What's New in *DSM-5*?

The changes in the fifth edition of the *DSM* (2013) were designed to produce an evidence-based document that could help clinicians consistently and accurately diagnose mental disorders, including those related to disordered eating (see Chapters 8–11). This more precise categorization would provide a basis for research criteria, as well as a common language to communicate about mental disorders. *DSM-5* represents cumulative expert opinion from over 700 distinguished mental health and medical experts, with draft criteria opened for public review over three separate time periods during the development process to ensure representation of a broad sampling of perspectives.

Of the 13 working groups for the various mental disorders, Dr B. Timothy Walsh led the Working Group on Eating Disorders. This group confirmed that over 50% of children and adolescents diagnosed as having an ED do not meet full *DSM-IV* (1994) and *DSM-4-TR* (American Psychiatric Association, 2000) criteria for anorexia nervosa (AN) or bulimia nervosa (BN). Consequently, application of the criteria from both *DSM-IV* iterations has resulted in diagnoses that have lumped a majority of children and adolescents with an ED into the category, eating disorder not otherwise specified (ED-NOS) (see Chapters 4 & 11). For instance, children with body image distortion and fear of weight gain who have not lost 15% of ideal body weight (per *DSM-IV* and *DSM-IV-TR* criteria) would not be diagnosed as having AN. Instead, according to *DSM-5*, they would be assigned the diagnosis of atypical AN, which, as noted above, is a subcategory of OSFED (see Chapters 4 & 11). Similarly, children or adolescents who have been binge eating and purging for less than 3 months or less than twice a week over 3 or more months would not meet criteria for BN. Using the *DSM-5* (2013), this subthreshold form of BN would now be diagnosed with a different subcategory of OSFED: BN of low frequency and/or limited duration. The remaining three OSFED categories in *DSM-5*—purging disorder, night eating syndrome, and binge-eating disorder of low frequency and limited duration—could also be applied to children or adolescents. In the two versions of *DSM-IV* these syndromes would have been considered forms of ED-NOS. Conversely, the form of ED-NOS emphasized in *DSM-IV*, binge eating disorder (BED), is now an eating disorder with its own category in *DSM-5*.

Another shortcoming of *DSM-IV* (1994) and *DSM-IV-TR* (2000) involved inattention to growth. Children and adolescents should be in a period of growth, including gaining in height, weight, and bone density at various ages and stages. Thus, most healthy children in growth phases should not merely maintain a weight, let alone lose weight during these growth periods. *DSM-5* (2013) acknowledges the importance of "developmental trajectory" (p. 338) and therefore the initial *DSM-5* criterion for diagnosis of AN in children and adolescents is "significantly low weight…that is…less than minimally expected" (p. 338). Another aspect of this inattention to growth is that girls who are prepubertal or who have not experienced menarche will not be captured accurately with the old criteria for AN, making amenorrhea a limiting criterion, as well as being completely irrelevant for boys with AN. In *DSM-5,* amenorrhea is no longer a criterion for the diagnosis of AN in girls or women.

Another difference unique to younger cohorts is the observation that children and adolescents may not experience their body weight, shape, and size in the same way adults do (Bravender et al., 2007). Younger children may lack a vocabulary for their illness, so they may not state that they are afraid of getting fat, dislike their bodies, or are afraid of certain foods. However, in certain children these words begin to emerge after the refeeding process has begun, and some children will voice fears of getting bigger, taller, or having the changes of

puberty occur. Even before the vocabulary "catches up," their actions still are consistent with the classic ED behavior of food refusal, compensatory behaviors, and resultant failure to grow along expected parameters (Bravender et al., 2007).

Avoidant/Restrictive Food Intake Disorder (ARFID)

A separate subgroup does not merely lack the vocabulary of weight concerns or distorted body image, because, for this subgroup, "avoidant/restrictive food intake" (*DSM-5*, 2013, p. 334) is not about weight or body image at all. Adolescent medicine physicians specializing in the medical care of children and adolescents with disordered eating have recently recognized this subgroup as distinct from the overly inclusive ED-NOS diagnostic (Fisher et al., 2014). Children and adolescents in this cohort tend to be younger than those with AN, BN, or other subgroups of ED-NOS, and they lack the distorted body image and/or fear of weight gain. Approximately 10% of patients presenting to pediatric and adolescent medicine ED programs fit this category. Some of these children or adolescents, after having gagged or choked, develop aversive eating patterns to avoid vomiting again. Some parents remember these children and adolescents as having been picky eaters as babies or toddlers, or as having had sensory issues with feeding from a young age. Another cluster of these patients had chronic or intermittent abdominal pain that interfered with intake.

Previously, experts used the terms functional dysphagia, selective eating, and food avoidance emotional disorder to describe aversive eaters in childhood (Bryant-Waugh, 2000; Fisher et al., 2014; Watkins & Lask, 2000). Instead of the *DSM-IV* definition of Feeding Disorder of Infancy or Early Childhood (FDIEC), *DSM-5* (2013) now has a category called Avoidant/Restrictive Food Intake Disorder (ARFID). To determine the utility of this new category and delineate differences between this group and both AN and BN patients, seven divisions of adolescent medicine across North America retrospectively performed chart reviews of patients aged 8 to 18 years presenting to their programs in 2010, recording *DSM-IV* and *DSM-5* diagnoses (Fisher et al., 2014). Those patients (n = 98) meeting newly proposed *DSM-5* criteria were compared to a random sample of patients with AN (n = 98) and BN (n = 66).

The results helped establish the usefulness of ARFID as a distinct entity worthy of a new category in *DSM-5* (Fisher et al., 2014). The ARFID patients were younger (ARFID = 12.9 vs. AN = 15.6 vs. BN = 16.5 years), were ill longer prior to initial intake (33.3 vs. 14.5 vs. 23.5 months), and had a median percent BMI intermediate between the AN and BN groups (86.5% vs. 81.0% vs. 107.5%). The ARFID group represented 13.6% of the total number of new ED patients presenting to the seven centers in 2010. Fisher et al. (2014) also looked at subcategories within ARFID: 28.7% were restrictive eaters, 21.4% had generalized anxiety, 19.4% had gastrointestinal symptoms leading to food aversion, 13.2% had difficulty with vomiting or choking, 4.1% had significant food allergies, and 13.2% were categorized as "other." Patients with ARFID were more likely to have a medical condition (51% vs. 10% vs. 11%). The prevalence of comorbid mental health disorder was high in all groups (see Chapters 15 & 54), with anxiety highest in the ARFID group (58% vs. 35% vs. 33%) and mood disorder lowest (19% vs. 31% vs. 58%). The percentage of boys was higher in all groups than in older literature, with the highest percentage of boys in the ARFID group (29% vs. 15% vs. 6%). This finding supports prior findings leading to increased recognition of boys with disordered eating in childhood and adolescence (Peebles et al., 2006; see also Chapter 37).

In summary, data from the multisite study by Fisher et al. (2014) support inclusion of ARFID as a distinct ED in the *DSM-5*, as ARFID patients demographically and clinically were

distinct from patients with AN and BN. Further research is needed to determine the prevalence of ARFID in other clinical settings and to better define the clinical presentation to allow for earlier recognition and intervention.

Atypical Anorexia Nervosa and Atypical Bulimia Nervosa

The *DSM-IV* category of ED-NOS encompassed a medically heterogeneous category with serious physiological sequelae in children and adolescents. In a study of 1,310 girls aged 8 to 19 years presenting to an ED program, Peebles, Hardy, Wilson, and Lock (2010) found that 25.2% of girls had AN, 12.4% had BN, and 62.4% had ED-NOS. When the ED-NOS patients were subcategorized into "partial AN" and "partial BN," with less severity of illness but symptoms paralleling the AN and BN groups, only 14.3% remained in the ED-NOS group. This percentage of patients who do not meet criteria for AN, atypical AN, BN, or atypical BN is remarkably similar to the 14.6% found in the seven-site study of ARFID by Fisher et al. (2014).

Peebles et al.'s (2010) category of partial AN (within *DSM-IV* ED-NOS) has evolved in *DSM-5* (2013) to "Atypical Anorexia Nervosa," defined as meeting all the criteria for AN except that body weight is not significantly low (see Chapters 4 & 11). Atypical AN can be seen with children, adolescents, and young adults who have been obese and lost weight in an unhealthy way, and who now have a distorted body image and undue influence of body image or shape on their self-esteem but who are not significantly underweight. Atypical or subthreshold BN is the diagnosis now given to those who would meet the criteria for BN but have had low frequency of vomiting or have not yet binged and purged at least once weekly for at least 3 months.

Other *DSM-5* Disorders

Purging disorder (PD) in *DSM-5* (2013) is the diagnosis for those who predominantly purge without binges or restricting, and subthreshold binge eating disorder (BED) refers to those who meet criteria for BED but have had low frequency of binges or limited duration of symptoms. Night Eating Syndrome is another diagnostic category in *DSM-5* (see Chapter 4). These *DSM-5* changes allow for more accurate diagnosis in children and adolescents and help increase specificity when looking at interventions and outcomes. These diagnoses are less frequently made in youth, with more frequent findings of ARFID than Night Eating Syndrome, PD, or subthreshold BED. Obscuring diagnosis is the perception that it may be "normal" in college to be too busy to eat in the day, then go out for pizza and beer with friends at night; this pattern is not healthy, but becomes pathologic when accompanied by distorted body image or abnormal eating attitudes.

Recognizing and Assessing Disordered Eating in Children and Adolescents

A key principle of ED recognition in children and adolescents is to trust parental and other caregivers' instincts. In other words, when a parent or other caring adult voices concern about an ED, the astute clinician should evaluate closely (Rome & Committee on Adolescence, 2003; Rosen & Committee on Adolescence, 2010); if an ED is not already present, it may be in its initial, developing stages and not yet entrenched. Early intervention at this stage can improve outcomes (Rome & Committee on Adolescence, 2003).

Working Collaboratively with Parents, Children, and Adolescents

With both children and adolescents, the astute clinician can learn much from working with parents and the designated patient, both together as well as each individually. Most physicians tend to start with the parent(s) and child together, learning much from not just the words spoken but from the body language of each participant. Ask the child or adolescent first why they think they are there today. Does the parent automatically answer for the child, interrupt, or otherwise take over ("hijack") the conversation? Does the child look to the parent for "permission" to answer or for the parent to speak for him or her? Is the adolescent avoiding eye contact and trying to erase themselves from the room? How the clinician assesses and responds to parent–child interactions can set a tone for building trust and empowering both parent and child to work together toward this child's health. Questions that are useful to ask with both parent and child in the room are presented in Table 13.1.

Skillful interviewing can tease out the hidden agenda and clarify any discrepancies in perspective between parent(s) and child (Rome & Gillespie, 2011). If the parent tries to "dominate all airspace," that is, control the conversation for whatever reasons, simple redirection may be necessary, such as, "Hold on, Mom, I would like to hear your daughter's words here, and we will have time to hear from you in a bit." If intrusive behaviors continue, it may be useful to make confidential time for each party early on, and to ask the child if they want "first dibs or second dibs." In adolescent medicine it used to be assumed that the child would want to speak second so that they did not feel "reported on." However, many adolescents want to speak first to make sure you have the accurate picture from their perspective without the parents' bias. In either case, giving the child or adolescent the choice empowers them in a useful way to partner with you and their parents toward improved health, while also helping to build trust between patient and clinician as well as parent and child.

Table 13.1 Useful questions with both parent and child in the room.

What is the most you ever weighed? When was that? At what height?
What is the least you have ever weighed? When was that? At what height?
What do you think you ought to weigh? [ask for a range if they give you an exact number] *How much time and/or energy do you put into that?*
What numbers feel too high, and too low?
How often do you weigh yourself?
Do you feel guilty or badly after eating certain foods?
How much of your day is spent on food/body thoughts?
Are there areas of your body that truly distress you? Which areas? Do you check these areas at all? Regularly?
What, if anything, do you do for exercise? How much, how often?
How stressed are you if you miss a workout or miss exercising?
What did you eat yesterday, start to finish? Today?
Do you count calories? If yes, how much do you allow? Do you count fat grams? If yes, how much do you allow?
Any taboo foods that you avoid?
For postmenarchal girls:
When was your last period? What did you weigh then?
How old were you at your first period?
Have they ever been regular? Any cramps?
If they have ceased, when did that happen?

Adolescents tend to reveal more confidential information if the definition of confidentiality *and its limits* are clearly articulated (Ford, Millstein, Halpern-Felsher, & Irwin, 1997). The clinician can say, "Everything we talk about without your parent (or your child, depending on which party you are addressing) in the room will be confidential, unless you tell me something life threatening or dangerous, in which case I will tell you that I need to tell your parent (or your child, depending on which party you are addressing)." If the parent remains reluctant to allow the child time alone with the clinician, the clinician can also share that such confidential time allows the child to become a better healthcare consumer, speaking up for themselves and working toward making healthy choices even when the parent is not literally or metaphorically looking over their shoulder. Parents may want to address certain concerns without the child, also. As noted previously, in obtaining a confidential history, asking each party about specific disordered eating behaviors such as purging, compulsive exercising, and other habits can be quite revealing.

Taking a Good History: Specific Assessment Targets

Feelings About Weight, Food, and Body Shape Does the child or adolescent feel overweight, underweight, or just right? Has he or she tried to change weight or shape through exercise or change of eating habits? If so, what did he or she do and for how long? How much is food or body on her or his mind? Are there specific body areas that make him or her most uncomfortable? Asking how much effort she or he must put in to maintain, gain, or lose weight can assess both magnitude of any abnormal eating attitudes and behaviors while simultaneously acknowledging the stress she or he may feel over weight and body issues (Rome & Gillespie, 2011).

Dietary History and Recent Changes in Eating The dietary history is best obtained from both parent and child, with the former serving as a reality check for the latter. For instance, if the child states that she had cereal for breakfast, the mother might add, "Only three bites!" if she is restricting, or "Half the box!" for someone who is binge eating. How often do family meals occur? Are they stressful? Can the child or adolescent eat at friends' houses comfortably? Does she avoid social situations where food is involved? Who prepares the food? Does she eat only what she has made herself, or only what Mom or another family member has prepared? Has she recently shifted to "healthy eating," and what does that mean? Does anyone else in the home have dietary restrictions? For instance, if Dad has had a heart attack and is following an Ornish ("nothing with a face, nothing with a mom") or Esselstyn diet ("only foods grown from the ground"), has the child picked up on that and are they trying to eat the same things for their own "health"?

Evidence of changes in eating is especially deserving of attention. A shift to vegetarian or vegan eating may follow a health education class, particularly if the child or adolescent was shown videos of inhumane animal treatment. Parents may not be aware that the child is now eating smaller portions of, or avoiding entirely, specific food groups, especially if the rest of the family are carnivorous (i.e., are enthusiastically part of the "meat and potatoes" crowd). Such shifts may initially be seen as a positive health move by the family, without recognizing over time how little the child actually consumes (American Dietetic Association, 1988). Some children start to avoid a specific food group out of ignorance of their bodies' needs, while others use seemingly healthy changes to mask progressive restriction. In athletes, an initial drive for improved performance can be usurped by a progressively more starved brain's drive to be thinner, with the latter goal overtaking the former as AN ensues.

Determining What is "Taboo" Given the importance of seemingly healthy changes in the context of an ED, the clinician should ask what food the patient will not eat, or what is "taboo." In this regard, it is important to determine if the child or adolescent is conscientiously restricting fat grams or reading labels to keep a running tally of calories, fat grams, or other items. Many parents and youth do not realize that the adolescent brain requires a minimum of 30–50 grams of fat daily to myelinate or "hard wire" neural processes, and this need continues through age 26 years (Rome & Gillespie, 2011).

Magical Thinking and Food-Related Rituals Thinking about what foods to avoid, what foods are "good," and what foods are "fattening" are not the only types of cognitions and emotions to assess. Magical thinking can also occur, especially in the chronically nutritionally depleted brain. One adolescent girl I treated believed that a specific mirror added weight to her hips; we had her cover that mirror with a drape to avoid the phenomenon until her mindset was clearer. Another boy needed constant reassurance that the act of watching television would not make him gain weight; that the calories did not magically flow through the TV to his abdomen to make him fat. He had heard repeatedly that watching TV "makes kids obese," and his starved brain and obsessive-compulsive personality combined to have him fixate on the topic. This kind of delusional thinking usually resolves with refeeding (Beaumont, Russell, & Touyz, 1993), but occasionally judicious use of psychopharmacology can facilitate the process.

Assessment of magical behavior or rituals in children and adolescents is also important. Some children with obsessive-compulsive features may not allow different foods to touch on the plate, or they have unique ways to utilize utensils. Other children will chew food multiple times, or chew each bite multiple times, or cut food into minute pieces. Some of these rituals are obsessive; others are a means of avoiding ingestion of food. A child may also engage in restless or repetitive movements as a means of purging calories and/or as a manifestation of generalized anxiety.

Binge Eating and Weight Management Confidential questioning, using simple and age-appropriate terms, can be employed to assess binge eating, dieting, and weight management practices, including behaviors that may be embarrassing to the child or that they may not yet wish parents to know. What makes up a binge? It may actually be a normal or undersized portion, mislabeled inappropriately as a binge (see Chapter 10). Has the child or adolescent tried vomiting to control weight? If so, via what methods—spoon, finger, or spontaneously without initiating a gag reflex mechanically? After vomiting, do they immediately brush their teeth, or gargle/rinse? With mouthwash, water, or water/baking soda? This line of questioning helps clarify the disordered eating and it also can be used to educate a purging patient on not brushing after vomiting, as it gets acid to far areas of the mouth that the acid may otherwise not have reached, in contrast to mouthwash (best) or water/baking soda (second best), or even plain water (acceptable). Even if the child or adolescent presents with self-induced vomiting, it is important to ask about other methods of purging used, including diet pills, laxatives, and diuretics, and including questions about which brands are used, how often, and over what time period. A thorough and useful assessment also inquires about daily consumption of caffeine and energy drinks.

Psychosocial Functioning and Psychosocial Risk Status In a survey of 50 adolescents with EDs and 57 age-matched healthy controls aged 13 to 25 years, no significant differences in sexual activity (52% vs. 39%, $p = .16$) or type of birth control method used between the two groups

(Hicks et al., 2013). However, ED patients incorrectly associated more health risks with condoms, spermicides/gels/foams, and the rhythm method (all values of $p < .05$); identified fewer health benefits of oral contraceptives ($p = .05$); and were less well informed about the HIV protection afforded by various methods of birth control ($p < .05$). ED patients also underestimated the overall pregnancy risks associated with various methods of birth control ($p = .005$). Thus, it cannot be presumed that adolescents and young adults with EDs are asexual beings without risks on that front. The clinician needs to ascertain level of risk in order to help keep patients safe, disease free, and pregnancy free, while addressing ED issues.

Even if a child or adolescent comes in specifically for an ED evaluation, confidential use of a significant portion of the HEADSSS questions (Goldenring & Rosen, 2004; see Table 13.2) remains a useful assessment of the child's psychosocial functioning and psychosocial risk. How you ask the questions matters, in that open-ended, nonjudgmental questions tend to get more honest responses. It is important to ask questions about home and school, as they may reveal narrowing of interests, avoidance of situations where food is central, such as family dinners or social gatherings, and other troubling behaviors.

Psychiatric Symptoms and Physical Symptoms Symptoms of anxiety, depression, mania, suicidal ideation, and obsessive-compulsive behaviors should of course also be assessed. Does the child or adolescent have panic attacks? In what situations and how often? Do obsessive-compulsive behaviors center around food and body, or are there other compulsive rituals or obsessions

Table 13.2 The HEADSSS assessment (adapted from Goldenring & Rosen, 2004).

Home: Who lives at home? What happens when there is an argument in the home? If parents do not live together, how often does the child see each parent? Any food/meal patterns from each house? Number of family dinners at each?
Education: What grade are you in school? How are your grades this year, and last year? [A sudden drop in grades can be a sign of increasing brain starvation, a new drug habit, depression, or other stressor]
Activities: How do you fill your time? In school and out of school activities? Overscheduled? Religious activities? Protective factors? Gangs, guns, other risks?
Drugs: Do your friends smoke cigarettes? Do you smoke cigarettes? How much, how often? Do your friends use drugs? Do you use drugs? Over the counter, prescription, marijuana, other? How much, how often? Alcohol use? How much, how often? Do your friends drink alcohol? Do you drink alcohol? What do you drink? How much, how often?
Sex: Are you attracted to guys, girls, or both? (assure them you ask ALL patients this question). Have you had sex? With guys, girls, or both? (If heterosexual and has had sex) What do you use for protection? If condoms, do you use them sometimes, most of the time, or all the time? What do you use for a second method? [This line of questioning provides an easy means of motivational interviewing, or asking questions while helping them shape/change behaviors to make healthier choices]
Has anyone done anything sexually to you that made you uncomfortable? If yes, how old were you? Who was the perpetrator? Where is that person now? How did you get it to stop? [And if ongoing, that is a child protective services referral]
Suicide/depression? Ever to the point of wishing they didn't exist? If so, how often? Today? Ever thought, "I'm going to kill myself?" If so, how often? Ever had a plan? What specific plans?

Note. The final S in the acronym HEADSSS stands for **S**afety from injury and violence.

about homework, house cleaning, or violent thoughts? There should also be a review of physical systems that includes questions about early satiety (i.e., whether the patient gets full early on in the meal), bloating, and reflux; stool frequency, diarrhea and constipation, and treatments tried; dizziness, syncope (fainting; transient loss of consciousness) and near syncope ("gray-outs"); weakness and fatigue; signs of chronic disease, such as thyroid, infection, or malignancy; and easy bruising or bleeding, pallor, fevers, and urinary frequency.

Physical Examination

Height and weight should be obtained in a gown, standing backwards on the scale, after voiding. Having nursing assistants trained not to share weights or even make potentially misconstrued comments is useful, because even a well-intentioned "Oh, that is much better!" can imply to the child or adolescent struggling with AN that significant weight gain has occurred, causing immense distress in the moment and a missed opportunity. Whether to share information about body weight and weight gain or loss should be a therapeutic decision by the care provider, with some patients clearly showing enhanced speed of recovery when numbers are not revealed, just trends; this is similar to use of the Maudsley method of parent-driven refeeding to help a child face a buffet without terror (see Chapters 53 & 60). Other patients can make effective use of a therapy session to process the weight-related numbers or challenge the cognitive distortions that cause the numbers to feel "weighty" and the client to feel "fat."

It is important that taking blinded or unblinded weights be a clinical decision, not a misinterpreted moment that sabotages therapy. Sophisticated patients may try to manipulate the measurements, with weights hidden in bras or underwear, or even with extreme measures, such as a roll of coins intravaginally. Hence, the patient should be weighed wearing only a gown, with urinalysis helping to assess degree of hydration (a specific gravity of 1.000 indicates water-loading or consumption of noncaloric beverages to falsely elevate the weight). Certain patients may need a "pat down" to look for hidden weights.

Vital signs. Measurements of pulse and blood pressure should also be taken (see Chapters 14 & 52). As simple as these sound, do not assume that all clinicians do these measurements accurately. The patient should be lying down for initial blood pressure (BP) and pulse readings, followed by standing (i.e., orthostatic) measurements, which are most accurate after the patient has been standing for 2–3 minutes. In an outpatient setting, sitting measurements can be skipped. Similarly, a typical patient normalizes their BP and pulse readings fairly quickly (within seconds), so that to save time in a busy practice the patient can stand and have their measurements taken immediately. However, obtaining vital signs in both positions is preferable because if the pulse jumps up 20 points from lying to standing or diastolic BP drops 10 points, that change means the patient is "orthostatic." Systolic changes tend not to be clinically significant. Pulse tends to jump up excessively in the face of nutritional depletion with dehydration, so orthostatic changes should be treated with an increase in caloric beverages consumed daily, even if the patient is asymptomatic. In this regard, note that the majority of patients who are orthostatic by pulse and/or blood pressure may not experience dizziness with standing, let alone syncope or near syncope. In addition to orthostatic hypotension, other signs of nutritional depletion are bradycardia (abnormally slow heart rate) and hypothermia (abnormally, sometimes dangerously, low body temperature).

A thorough head to toe examination can reveal other findings common in patients who restrict or purge (see Chapters 9, 14, & 52). The astute clinician can evaluate for Russell's sign (callus of the knuckle from the teeth hitting that area during finger-induced purging) and

parotitis (from acid backwash through the parotid duct, inflaming the cheeks; patients see this sign as evidence that their face is getting "fat"). Tanner staging can help determine stage of puberty, such that atrophic breasts emerge as a sign of malnutrition. Often the untrained eye will note Tanner 4 or 5 pubic hair (too many to count for stage 4, hair out to the thighs for 5), while breasts are Tanner 2. The astute clinician will notice the presence of atrophic breasts consistent with undernutrition rather than a discrepancy in puberty. A difference of 2 or more Tanner stages in an individual patient is a cause for further evaluation to ascertain the origin of this pubertal delay or abnormality. The starved patient may also have a scaphoid abdomen, meaning that it is concave from ribs to hipbones. Palpable loops of stool from chronic constipation can appear as abdominal masses that may be found on abdominal exam. Acrocyanosis (bluing of the hands and feet) and peripheral edema (swelling) may be found, with the latter occurring in reaction to both starvation and, in particular, misuse of laxatives and diuretics. Lanugo, or fine, downy hair covering the trunk and elsewhere, can reflect prolonged starvation. During the physical examination, the patient should also be evaluated for psychomotor retardation, or slowing of thought, speech, and/or movement, which can occur in starved patients.

Challenges in Diagnosis

Prior to *DSM-5* (2013), the issue of "organic" versus "behavioral" disordered eating led to inconsistencies in working definitions that are useful for research across sites. As many as 16–30% of feeding problems have been described as "organic" (Williams, Riegel, & Kerwin, 2009), and up to 80% of referrals to pediatric specialists for feeding disorders have been found to have a significant behavioral component (Benoit, 2000; Burklow, Phelps, Schulz, McConnell, & Rudolf, 1998; Sanders, Patel, Le Grice, & Sheppard, 2003). Budd et al. (1992) found that nearly two thirds of their cohort of children with feeding problems presenting for outpatient treatment had a combination of behavioral disturbances and sensorimotor problems in connection with the lips, tongue, and/or jaw. In other words, certain patients manifest a biological predisposition to feeding challenges, as might occur with the sensory-neural challenges found in autism or with a genetic predisposition to AN or other disordered eating (see Chapter 28).

These findings also clearly demonstrate that EDs in children and adolescents, as with other biopsychosocial illnesses (e.g. type 2 diabetes), represent a complex interplay of nature and nurture (see Chapter 67). In this regard, some patients have environmental risks that predispose them to disordered eating in childhood, as can occur with prolonged intubation in the neonatal period leading to difficulties with eating in general and/or with specific textures. Add to these dynamics the challenges of parenting in the context(s) of a biological predisposition and environments shared by parent(s) and child, and EDs in childhood can flourish. For example, a mother with generalized anxiety disorder is more likely to have challenges with learning to feed a child who by nature and/or neonatal experience is a picky eater. For a variety of reasons, including their own mental health challenges, parents may manifest overly rigid behavior with regard to their child's eating habits, they may fail to recognize hunger and satiety, and the parents' own disorganized or chaotic eating patterns may lead to lack of proper food access or to development of healthy eating attitudes and behaviors (Fisher, Birch, Smiciklas-Wright, & Picciano, 2000; Fomon, 1994; Patrick, Nicklas, & Hughes, 2005; see also Chapter 26).

How a parent responds to food refusal can be quite revealing. Parents who dissolve into tears or move quickly to anger may have more challenges learning to appropriately feed their

child than parents who by nature are calmer and less autocratic in parenting style. Assessing the parents' relative strengths and weaknesses can provide clues as to how the child developed disordered eating, as well as illuminate strategies that will aid a particular family in learning to feed their child appropriately and successfully. Scales have been developed and standardized to assess parental feeding skills. For example, Crist and Napier-Philips (2001) used the Behavioral Pediatrics Feeding Assessment Scale (BPFAS) to identify behavioral features of children with feeding problems. The main difference between clinically referred children with disordered eating (n = 249) and the controls (n = 96) was that the parents of the children with feeding problems reported significantly more problem behaviors.

Validated symptom checklists can be useful for consistent comparisons of patients before, during, and after treatment and across programs. The Eating Disorders Examination-Questionnaire (EDE-Q; see Chapters 9–11) is currently recommended by the Academy for Eating Disorders Inpatient Standards Task Force in its Clinical Practice Recommendations for Residential and Inpatient Eating Disorder Programs (preliminary communication, July 8, 2013). The EDE-Q is available at no charge under assessment measures at http://www.rcpsych.ac.uk/pdf/EDE-Q.pdf, with age-adjusted versions available for evaluation of younger patients (Bryant-Waugh, Cooper, Taylor, & Lask, 1996; Fairburn & Beglin, 1994). Another psychometrically sound scale is the 26-item modified Eating Attitudes Test (EAT), or the child version, the chEAT (Garner, Olmsted, Bohr, & Garfinkel, 1982; Maloney, McGuire, & Daniels, 1988; see also Chapter 38). For evaluating quality of life, the Eating Disorders Quality of Life Scale (EDQOL; Bryant-Waugh et al., 1996) can be a useful measure. Keep in mind that many other measures are available for adults but they have not been validated for use in children. None of these measures is perfect; the savvy adolescent can "game" the system or underreport symptoms on a questionnaire. These assessments are tools, not end-all or be-all measures. Clinical judgment, a good history, and a thorough physical examination (see Chapter 14) remain core aspects of diagnosis of EDs in children and adolescents.

Conclusions and Future Directions

Children and adolescents suffering from EDs may not walk into the clinician's office with ready packaging, a diagnosis readable on their foreheads or in an emaciated body. Concerns raised by a parent, teacher, caring adult, or peer should be taken seriously. Tools for assessment and diagnosis such as the EAT, chEAT, or EDE-Q can augment diagnosis based on careful, multifaceted clinical interviewing and behavioral observations and can be used for tracking patients' symptomology over time, as well as research tools across programs. Such tools supplement but do not replace what can be learned from a careful history and physical examination.

New categories such as Avoidant/Restrictive Food Intake Disorder, partial Anorexia Nervosa, and partial Bulimia Nervosa describe newly recognized subgroups relevant for diagnosis in children and adolescents. Future research should help clarify aspects of presentation and valid assessment in these newer subgroups, determine prevalence in different clinical settings and countries, and perhaps further refine and validate particular eating disorder phenotypes in these younger age groups. The ultimate goal is to enhance earlier detection to improve outcomes, aid in treatment planning, and augment efforts at prevention.

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14

Medical Complications and Diagnosing Eating Disorders

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Students and clinicians may be fascinated by the complexity of eating disorders (EDs) and their treatment (see Chapters 12, 50–64, & 67). However, they often refuse to treat patients with EDs for the same reason, as well as the likelihood of medical morbidity and mortality (see Chapters 52 & 55). A basic knowledge of the medical complications of EDs will help you decide when medical help is needed and thereby allow you to comfortably treat patients with EDs. This chapter will help you answer the following questions: Do my patient's symptoms mean they need to see a physician? Does my patient need an immediate medical assessment? What does the physician's assessment mean?

Making or Supplementing the Diagnosis of an Eating Disorder

The diagnosis of an eating disorder (ED) is based on history, not on physical signs (Uyeda, Tyler, Pinzon, & Birmingham, 2002; see also Chapters 8–11 & 13). The diagnosis of bulimia nervosa (BN) is usually straightforward because the patient provides a clear history. In anorexia nervosa (AN) the opposite is usually true because the patient fears "recovery." Physical signs of an ED may suggest the diagnosis of an ED to the examiner, reassure the patient that the examiner has ED expertise, spontaneously open discussion about the ED because patients are interested in their physical complications, and help to estimate the risk of morbidity and mortality (Birmingham, Su, Hlynsky, Goldner, & Gao, 2005; Harbottle, Birmingham, & Sayani, 2008). A medical assessment should take place at the outset of treatment for all patients with AN and moderate or severe BN and eating disorder not otherwise specified (ED-NOS; now categorized as either Binge Eating Disorder [BED], Other Specified Feeding or Eating Disorder [OSFED], or Unspecified Feeding or Eating Disorder [USFED]; see Chapters 4, 10, & 11).

The interval between medical follow-up appointments depends on the severity of medical illness, the rate of weight change, the rate of increase in caloric intake, the seriousness of other medical conditions or disorders from which the patient suffers, and whether medical complications

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have developed. The most common medical conditions and disorders that increase the need for frequent medical follow-up are (whether or not they are related to the ED): pregnancy, diabetes mellitus, arrhythmias, congestive heart failure, stroke, substance use (see Chapters 15 & 54), seizures, hypoglycemia, renal stones, and pancreatitis. Medical assessment should be conducted at least weekly if weight is increasing at 1 kg (2.2 lb) per week or more, but can usually be reduced in frequency after the first 6 weeks of weight gain. Medical assessments should continue every 6 months, long term, because medical complications can occur years after recovery (Marks, Beumont, & Birmingham, 2003). For example, recovery of the diaphragmatic muscle can take 1 or 2 years, and symptomatic deficiencies of vitamin A and selenium can take more than a decade to manifest (Birmingham & Tan, 2003).

The Medical Differential Diagnosis of Eating Disorders

Physicians may fail to consider an ED as a possible diagnosis, even when suggestive physical signs and symptoms are present (Su & Birmingham, 2003). Patients with an ED who abuse laxatives often develop a deformity of their nails called clubbing. But they are usually investigated for rare causes of clubbing (e.g., bacterial infection of the heart valves, Crohn's disease, and cancer) without asking questions that might lead to the diagnosis of an ED. Similarly, extensive medical investigations usually precede the consideration of AN when physicians examine a patient with marked weight loss.

Conversely, the diagnosis of an ED may be made in error, or the established diagnosis of an ED may lead the clinician to overlook a medical illness. "False positive" diagnoses can occur when the actual diagnosis is hyperthyroidism, Crohn's disease (chronic inflammation of the intestines), gastrointestinal malabsorption, Addison's disease (chronic adrenal insufficiency), or cancer. False positive diagnoses are most common when the real diagnosis is one of the following psychiatric conditions: schizophrenia, obsessive-compulsive disorder, major depressive disorder, and somatoform disorder (Birmingham & Sidhu, 2007).

Differential Diagnosis of AN

What are the physical signs and laboratory tests that help distinguish AN from the medical conditions? Hyperthyroidism can cause weight loss, increased energy, hair loss, anxiety, clubbing of the fingers, and occasionally vomiting (Birmingham, Gritzner, & Gutierrez, 2006). An elevated resting heart rate and temporal balding (loss of hair where it parts on either side of the scalp) suggest hyperthyroidism, and a low level of thyroid-stimulating hormone (TSH) confirms it.

Celiac disease is caused when antibodies against gluten from grain act against the body (Kneepkens & von Blomberg, 2012). Celiac disease may cause symptoms such as weight loss, abdominal discomfort, vomiting, deficiencies (e.g., iron-deficiency anemia), and generalized aches and pains. When the patient is eating gluten, the diagnosis is confirmed, by an elevated antigliadin antibody blood test or a small bowel biopsy.

Superior mesenteric artery syndrome (SMAS) mimics certain features of AN, because it can cause significant weight loss, epigastric distress (including feeling bloated), and vomiting (Welsch, Büchler, & Kienle, 2007). SMAS is a partial blockage of the upper bowel caused by its compression between the superior mesenteric artery and the aorta. Compression occurs when the fatty tissue that normally provides padding between the two arteries disappears with

marked weight loss. SMAS can be caused by marked weight loss from any cause, including AN. When AN and SMAS are comorbid, the latter is a complicating factor because it prevents weight gain or causes weight loss. Ultrasound or CT scanning confirms the diagnosis of SMAS.

Substance use can complicate or confuse the presentation of EDs (see Chapters 15 & 54). Use of ephedra and cocaine, for example, can cause a syndrome of weight loss, anxiety, denial of problems, and resistance to change that suggests AN. Patients with concurrent ED and substance use may seek admission to an ED program hoping for a goal other than abstinence.

Unexpected medication side effects can occur because of marked variation between individuals in absorption, distribution, receptors, medication effects, and medication interactions. Increased vomiting, depression, anxiety, weakness, and weight loss are all possible medication side effects. ED patients often use over-the-counter medications, antihistamines, weight loss medications, medications from friends and family, and herbal remedies, as well as recreational substances. A thorough history of pills, liquids, and remedies, drug testing, and pill counts, as well as a review of databases of patient drug prescriptions from third party payers, can help identify a drug as a possible cause of an apparent change in ED symptoms.

Food allergies are rarely confused with EDs, but they can be. These allergies, which usually result from an allergy to a protein component of a food, can cause weight loss, vomiting, tiredness, and diarrhea. Most food allergies are associated with an immediate onset of symptoms, but there may be a slight delay between ingestion and symptoms. Compared to patients with an ED, patients with food allergies are more likely to have a history of eczema, asthma, hay fever, or anaphylaxis themselves or in their blood relatives; more often have dark circles under their eyes; are extremely keen to try introducing foods; and prefer frequent follow-up or hospitalization.

Brain tumors can cause rapid weight gain or loss. The cognitive aspects of EDs (see Chapters 8–11 & 18) are usually absent. If there is drowsiness or intermittent drowsiness, apparent change in personality such as loss of inhibition, or an unexplained muscular or sensory change, a brain tumor should be considered and neurological assessment obtained (Davidson & Birmingham, 2001).

Differential Diagnosis of BN

What signs and tests can help distinguish BN from medical conditions or suggest medical complications? Involuntary emesis, in a patient with BN, is usually due to esophageal reflux. A 3-week trial of a prokinetic agent (domperidone) and proton-pump inhibitor (e.g., esomeprazole) usually stops nausea and vomiting due to esophageal reflux. Less common medical causes of frequent vomiting are: air swallowing, ulceration or inflammation of the stomach, overactive thyroid, medication side effect, covert alcohol ingestion, superior mesenteric artery syndrome, and gastric bezoar (like the hair balls "coughed" up by cats) (Birmingham, Cardew, & Gritzner, 2007).

About 5 to 10% of patients who have BN for longer than a few years repeatedly chew, swallow, and regurgitate food from the stomach back into the mouth—they ruminate (Birmingham & Firoz, 2006). Untreated rumination prevents recovery from BN. All patients who ruminate say it has a calming effect on them. This is likely caused by stimulation of the vagus nerve (which supplies the upper intestine), and thereby the parasympathetic nervous system. Patients avoid reporting rumination because they believe the therapist will regard it as repugnant. Ask every patient, in a nonjudgmental way, whether she or he repeatedly regurgitates, chews, and then swallows food.

The symptoms of severe and even life-threatening diseases of the abdomen in young patients with ED may be erroneously and even tragically attributed to the ED. The overlapping symptoms include abdominal pain, constipation, diarrhea, mucus or small amounts of blood in the stool, vomiting, bloating, gas, heartburn, chest pain, back pain, increased or decreased frequency of urination, sweating, and feeling cold or dizzy. As a result, patients with an ED who develop a bowel obstruction, pancreatitis, or kidney stone may not be diagnosed (Birmingham & Boone, 2004; Jonat & Birmingham, 2003).

Hyperthyroidism or vitamin A toxicity can mimic BN or a worsening of BN. About 1% of cases of hyperthyroidism present with repeated vomiting and abdominal pain. High doses of vitamin A can cause headaches and vomiting. Similarly, the diagnoses of hyperemesis gravidarum and BN can be confused. Most patients with BN stop binge eating and purging when pregnant, while one third have an increase in bulimic symptoms. If the delivery team is not aware the patient has an ED they may presume the patient has hyperemesis gravidarum.

Conducting a Thorough Medical Assessment: The Physical Examination

Patients experience less anxiety if they are given information about the physical examination beforehand and advised that they can ask questions during it. Who will perform the physical examination, will anyone else be present, can a family member or friend be present, and what parts of a prototypical "complete" examination will not be done (e.g., breast, rectal, pelvic)? Examining the patient with a staff member present may be preferable to the presence of a parent (for children or adolescents) or partner or friend (for adults) because it enhances confidentiality, reduces embarrassment, and allows discussion. It may be helpful to have a female staff member present when the patient is female, as is often the case, and the examiner is male. Breast, pelvic, and rectal examinations should be done by the family doctor before referral, or the ED physician should refer the patient back to the family doctor to have those systems examined.

Keep the examination room well lit. In addition, have an adjustable lamp that can be used to focus on skin lesions such as those due to self-injurious behavior, or to a zinc or vitamin C deficiency (Tyler, Wiseman, Crawford, & Birmingham, 2002). Ensure the patient is comfortably and modestly draped at all times. Keep the examination room warm to prevent patients from becoming cold and uncomfortable. Remember, AN patients are somewhat poikilothermic (i.e., they have an internal temperature that varies significantly with the surroundings) and therefore their temperature will rapidly drop if they are wet or the ambient temperature is low. Tell the patient the results of the examination in a matter of fact way during the course of the examination. This demonstrates your openness with them about your findings.

An efficient examination helps keep it brief and demonstrates competency. The examination is often anxiety provoking, and the patient is often weak and unsteady when first seen. In the case of AN, this is related to malnutrition, while in BN and other EDs this is often due to dehydration caused by a worsening of purging due to anxiety related to the examination. Start by observing the person walk towards the office. For all patients, observe (a) their gait for muscle control problems (ataxia) or an antalgic limp due to injury from overexercising or bone fracture; (b) clothing that may be excessive for the outside temperature; (c) skin color that may be yellow all over their body and blue in their extremities; (d) their level of consciousness and the quality of their speech; and (e) whether they have excessive micromovements indicating increased nonexercise activity. Next measure their weight, height, and anthropometrics

(skinfold measurements should be done only by those who have training and experience). Ask patients whether "it is OK" to weigh them, whether they want to know their weight, whether they are usually "told their weight," and how often they weigh themselves. Most patients want to know their weight at the first assessment. They may comment on the measurement. The examiner should not comment on their weight or show any interest in it. If they ask why I am not interested I tell them, "You are more than a number!"

Next, have the patient lie down. Measure the patient's heart rate and blood pressure (BP; see Chapter 13). To test for postural change have the patient stand up and then measure the heart rate and BP every 15 seconds until the results stabilize. In young people who are dehydrated it is common for the BP to remain constant while heart rate increases by more than 10 beats per minute. Then have the patient sit. Measure the patient's temperature, oxygen saturation, and examine the head, neck, arms, and back. Next, ask the patient to lie down once again. Measure the jugular venous pressure, and then examine heart, lungs from the front, abdomen, and lower extremities. While the patient remains supine, perform the beginning of a nervous system examination. The patient should then stand up to complete the nervous system examination.

Encourage questions but be concise with your answers. Remember, like every one of us, these young people are frightened of what might be found wrong with them and of how, from their perspective, the information may used to "control" them and/or make them abandon valued coping mechanisms. However, if they ask, "Why are you doing all these assessments? It's not like I am really sick or brain damaged or something!?" you can tell them:

> You are young. You should have the body of a Ferrari! If you reduce the horsepower of a Ferrari by 50% you still have a fine car, but it isn't what it could be!
>
> [*and/or*]
>
> Do you remember how you feel the first day after a cold or flu? You feel great! But you don't feel great the next day. That is because our mind doesn't carry around all of our experiences; likely, because we need to be able to forget in order to carry on. So, even though you may believe you feel well today, it is quite possible that you felt even better before your eating disorder.

The cognitive function in patients with ED, especially with mood symptoms, is abnormal on specialized testing. But this can be reversed, along with all other physical complications, with weeks of treatment.

Take time to summarize your conclusions at the end of the history and physical examinations, continue to answer questions briefly and in a matter of fact way, and discuss treatment options briefly. Your conclusions should include their ED diagnosis (see Chapters 8–13), concurrent medical and psychiatric disorders (see Chapters 15 & 54), and the complications of the eating disorder (see Chapter 52). Clearly indicate whether a diagnosis is firm, possible, or will require another opinion or further testing.

Physical Signs Commonly Seen in ED

Physical signs alone are not sufficient to make the diagnosis of an ED. However, certain physical signs are suggestive and supportive of the diagnosis. Note that the interobserver reliability of the diagnosis of physical signs (between medical practitioners) is low. So, if a

physical sign is diagnosed for the first time, it may or may not have been there before. In general, the more experienced the physician, the more reliable they are in diagnosis (Chen, Lear, Gao, Frohlich, & Birmingham, 2001; Mazloum, Johnston, Lundrigan, & Birmingham, 2008; Tyler & Birmingham, 2001).

Glands

The parotids are the oyster-shaped glands immediately in front of each ear. The submandibular glands feel like small meatballs and are located just under the jawbone half way along the jaw bilaterally (Clare, Gritzner, Hlynsky, & Birmingham, 2005). Enlargement (hypertrophy) of the parotids and submandibular glands occurs with malnutrition alone, likely due to autonomic dysfunction, but is greater with purging. Thus, with restricting AN (i.e., AN-R) there is often mild parotid and submandibular gland hypertrophy, whereas in both the binge-purging form of AN (i.e., AN-BP) and BN there is usually marked hypertrophy (see Chapter 9). The glands decrease in size once purging stops over a couple of months, and warming speeds the decrease in size (Birmingham, Gutierrez, Jonat, & Beumont, 2004).

Hair and Skin

Hair The hair loss that often occurs with AN is generalized and not associated with any rash or itch of the scalp. Lanugo hair is the new growth of fine hair, especially noticeable on the back and abdomen. Lanugo hair is the same color as the patient's normal hair and disappears at a healthy weight.

Skin in General Erythema ab igne is the hyperpigmentation of skin that occurs when part of the body (usually the back or abdomen) is exposed to heat (Vahasoini, Vazquez, Birmingham, & Gutierrez, 2004). This occurs when patients with AN, who feel cold, repeatedly warm themselves using water bottles and warming pads, or by standing close to hot air vents.

Hypercarotenemia presents as yellow skin, but without yellowing of the whites of the eyes. Jaundice from liver disease causes yellowing of the whites of the eyes and yellow skin (Birmingham, 2002). Linear or perfectly shaped excoriations, bruises, burn marks, and nail or skin-picking excoriations are signs of self-injurious behavior.

Mouth Thinning of the teeth caused by regurgitated acid can be appreciated by shining a light through the teeth. The gums may appear swollen. Bleeding from swollen gums suggests scurvy. A rash at the side of the mouth, when not caused by licking, may be caused by riboflavin deficiency.

Hands Professor Gerald Russell described scarring of the dorsum of the hands caused by pressure from the teeth when fingers are used to induce purging (see Chapters 3 & 9). Dry hands with flaky skin may be due to zinc deficiency, especially if associated with poor healing of cuts and a change in preferences to tastes (Bakan, Birmingham, Aeberhardt, & Goldner, 1993; Birmingham, Goldner, & Bakan, 1994; Kopala, Good, Goldner, & Birmingham, 1995; Su & Birmingham, 2002).

Cardiovascular

Shortness of breath on exertion that is made worse by lying down and associated with an increased heart rate, may indicate congestive heart failure (Birmingham & Gritzner, 2007; Coxson et al., 2004). Congestive heart failure is most likely to occur during the first month of feeding as a complication of the treatment of AN and is usually caused by hypophosphatemia (Birmingham, Alothman, & Goldner, 1996; Birmingham, Lear, et al., 2003; see also Chapter 52). Refer a patient with these symptoms to the emergency department of a medical facility for assessment that may include a chest x-ray, electrocardiogram, serum electrolytes, creatinine, phosphorus, and magnesium (Birmingham, Puddicombe, & Hlynsky, 2004; Gambling, Birmingham, & Jenkins, 1988). Congestive heart failure in AN, which is often confused with comorbid asthma, is confirmed if the chest x-ray shows a "bat wing" distribution.

Mitral valve prolapse is common in females but can develop or worsen in AN. High-pitched clicks or a soft midsystolic murmur from the mitral valve prolapse are best heard with the patient standing and straining, as if they were trying to have a bowel movement.

Muscles and Tendons

Weakness of the muscles can be caused by deficiency of protein and calories, as seen in all patients with severe weight loss, but is more common with deficiencies of potassium, magnesium, calcium, selenium, or phosphorus (Birmingham, 1999). Carpopedal spasm, or latent tetany, is a spontaneous contraction of the muscles of the hands and feet that happens to all of us when we hyperventilate long enough. This often occurs in ED due to a total body deficiency of magnesium. It can also occur if the blood pH becomes alkaline due to loss of acid from vomiting. Signs that carpopedal spasm may occur are very brisk tendon reflexes, spasm of the hands when the blood pressure cuff is left inflated (Trousseau's sign), and contraction of the facial muscles when the side of the face is tapped (Chvostek's sign).

When the body is malnourished the metabolic rate falls as a protective mechanism against further weight loss. This is partly accomplished by the "sick euthyroid syndrome," which means there is less thyroid hormone secreted. An indication of the presence of the sick euthyroid syndrome is a delay in the relaxation phase of the tendon jerks.

Importance of Medical Assessment to the Patient, Family, and Treatment Team

Building rapport is essential to successful treatment in ED. In this regard ED patients often have greater confidence in medical doctors than psychological healthcare professionals. For this reason, the physician may help establish rapport that is generalized to the ED team (Birmingham, Muller, & Goldner, 1998; see also Chapter 50).

Understanding the ED as a Disease

The patient's acceptance of his or her ED as a disease is often intermittent and weak, but acceptance and concern about physical complications, particularly objective ones, tends to be constant and strong. It is therefore paramount that the physician performing the medical

examination and then interpreting and communicating its results be honest, forthright, and knowledgeable (see Chapter 52). A discussion of the abnormal findings on physical examination, as well as laboratory abnormalities, should be clear, brief, and unbiased. The patient and family will ask questions about the seriousness of identified physical findings. A calm and clear response should be used to convince them of the importance of the findings and their relationship to the ED. These goals reduce noncompliance and allow patients to focus on objective data as a measure of their recovery. Overstating the importance of physical complications, on the other hand, may result in a loss of trust and rapport.

Medical complications provide a particularly strong and simple incentive for the family and friends to provide support. This tends to focus them away from issues of self-blame, anger, and control that may otherwise limit their ability to aid in the recovery of the patient (see Chapters 53, 60, & 63). Family and friends can drive the patient to their medical appointment or help purchase food or medications. This is much better accepted than helping with meal support or supervision of exercise.

Other Important Issues in Medical Assessment of EDs

Signs and Symptoms Indicate Different Things in Different Populations

Signs and symptoms may have different meanings or normal values in males versus females, in different ethnicities, and for different age groups. For example, a normal body fat in Japanese women is usually about 4% lower than in Caucasian women (Lear, Humphries, Kohli, & Birmingham, 2007). Chronic weight loss in the elderly is more often due to cancer or depression than an ED. Weight loss in a young gay male could indicate HIV/AIDS rather than AN (Smith & Birmingham, 1990).

What is a Dangerous Level of BMI or Body Fat?

Body mass index (BMI) is typically calculated by dividing the weight in kilograms by the square of the height in meters; this is equal to dividing the person's weight in pounds by the square of his or her height in inches, and then multiplying the result by 703. In other words, it standardizes weight by height. But it does not take into account what the weight is made up of. People with different frame sizes will have different BMIs even if they have the same body fat and nutritional status. The BMI does not tell you whether a gain or loss in weight is due to fluid, stool, fetus, or muscle. Nutritional status also depends on the patient's diet before she or he became ill, whether the patient binge eats and purges or restricts, whether the patient has co-occurring conditions, or whether the patient has been given vitamins and minerals and an improved diet for the last month (see Chapter 61). For all of these reasons the BMI itself is not a good indicator of risk (McCargar, Taunton, Birmingham, Pare, & Simmons, 1993).

Anthropometrics (skinfolds), BIA (bioelectrical impedance analysis), and DEXA (dual x-ray absorptiometry) are common methods of measuring total body fat. BIA is not reliable when there are fluid shifts in the body, as there are in EDs, and DEXA requires x-rays. Physicians should purchase stainless steel Harpenden calipers, carefully review the accompanying pamphlet and video, and practice so they can measure, via skinfolds, changes in total body fat (Birmingham, Jones, et al., 1996; Orphanidou, McCargar, Birmingham, & Belzberg, 1997; Orphanidou, McCargar, Birmingham, Mathieson, & Goldner, 1994).

What Physical Signs Can Predict an Increased Risk for Death?

Death or severe illness from physical causes in EDs usually occurs in AN, not BN or ED-NOS, and often occurs without warning (Table 14.1). This is because the body adapts well to disease, humans are often unaware of illness if it is slow in onset, and many physical complications are either present or absent—unlike measures such as temperature or heart rate. Congestive heart failure (Birmingham, Stigant, & Goldner, 1999), rapid weight loss, onset of marked muscle fatigue on exercise, postprandial hypoglycemia, seizures, arrhythmia, and loss of consciousness (Puddicombe & Birmingham, 2006) all predict an increase in the risk of death in patients with ED.

What Complications are Underdiagnosed?

If a person with an ED experiences dizziness or confusion 30 to 90 minutes after meals, it is usually due to hypoglycemia. The hypoglycemia is due to inadequate stores of glycogen in the liver.

Examine for signs of self-injurious behavior (SIB; see Chapters 15 & 54). SIB may appear in unusual shapes due to injury from unusual objects such as cups or cigarettes. Search for needle marks as a sign of self-induced bleeding (i.e., self-phlebotomy). When self-phlebotomy, which can cause life-threatening anemia, occurs, it is almost always in patients who are healthcare professionals (Grewal & Birmingham, 2003).

Infection is usually diagnosed late because fever and increased white cell count are delayed in AN. The most common sites of infection are urinary tract, dental, lungs (pneumonia/empyema), and bone or bone marrow (osteomyelitis) (Birmingham, Hodgson, et al., 2003; Brown, Bartrop, Beumont, & Birmingham, 2005).

Table 14.1 ED complications that cause mortality.

Signs	*Possible Significance*	*Test*	*Treatment*
Rapid onset of shortness of breath that is less when sitting, fast heart rate	Congestive heart failure, usually due to hypophosphatemia	Stat chest x-ray, electrocardiogram, serum electrolytes, creatinine, phosphorus, magnesium	Transfer to emergency department or medical facility by ambulance
Confusion after meals	Hypoglycemia	Blood sugar	Glucose or another simple sugar that is quickly absorbed
Seizure or loss of consciousness	Hypoglycemia, hypomagnesemia, alkalosis, arrhythmia, Wernicke's encephalopathy, drug or substance side effect, withdrawal, or interaction, medication side effect of withdrawal of substance	The main significance is the underlying cause	Treat as for hypoglycemia, and give thiamine, examine for signs of Wernicke's, and take history of use of barbiturates or alcohol outside treatment

Laboratory Assessment

The interpretation of laboratory values is complex and must be done with knowledge of medicine, nutrition, and the patient's clinical status (see Chapters 50 & 52). We usually use blood tests to determine whether there are deficiencies of minerals or nutrients. However, serum tests do not tell us the whole story.

The level of nutrients in the body is analogous to the financial situation of a student at college or university. The money in the student's wallet is like the serum level of a nutrient, the money is the bank is like the stores in the body, and their apartment and car are like the organs that need the nutrient. At first, there is money (and nutrients) everywhere. So, if a student remains in a stable situation without need to use their money, their bank account remains the same. However, when at college or university they begin to use their money and the money in the bank account (body stores) gradually diminishes; thus, later on the student has to give up their car and later their apartment (organ dysfunction). Thus, the serum level (money in the wallet) doesn't tell us much about the total body stores of a nutrient. With refeeding (going to college and using the money) the stores will definitely drop. Depending on how low the total body nutrient levels fall, parts of the body may fail. So, it is only during the phase of refeeding or renourishment that deficiencies commonly occur.

Serum levels can also be misleading for a variety of other reasons (Birmingham, & Treasure, 2010). In some instances the level of the transporting protein confounds some nutrient levels. For example, serum calcium and vitamin A are both carried by proteins and consequently a low protein level will give the appearance of low levels of these nutrients. An important type of misleading finding concerns serum creatinine, which is proportional to lean body mass. Thus, the serum creatinine should be markedly lower than normal in AN, so the patient may have renal dysfunction with a normal serum creatinine. Usually this can be appreciated by using the low end of the serum creatinine range as the patient's normal level. In sick euthyroid syndrome the patient may have symptoms of hypothyroid functioning (e.g., fatigue, anxiety, and mild cognitive impairment) that coincide with the symptom picture in EDs, but levels of TSH and triiodothyronine (T_3) and thyroxine (T_4) are within the normal range. However, this syndrome is associated with delayed relaxation phase of the tendon jerks and a low free thyroxine. Note that all patients with marked malnutrition will have a slightly low white cell count, hemoglobin, and creatinine, all of which have no clinical significance.

Drug Treatment Can Change the Medical Assessment

A patient's physiology is changed by the drugs they ingest (see Chapter 59), the interactions between those drugs, the food they eat, and their genetic, psychological, and physical makeup. Serotonin reuptake inhibitors can cause anything from a tremor to the serotoninergic syndrome with body aches, confusion, and fever. Major tranquilizers can cause dystonia (uncontrollable muscle contractions, leading to repetitive, odd movements and postures), malignant neuroleptic syndrome, and other extrapyramidal complications. Some drugs have overlapping side effects based on their pharmacology, even though we use them for completely different purposes. For example, domperidone, commonly used to reduce stomach fullness in ED, is a major tranquilizer. It commonly causes an increase in prolactin with decreased libido and galactorrhea (spontaneous emission of milk from the breast). Any physical complaints the patient has could be due to the drugs the patient is prescribed, getting over the counter, or taking covertly. Always consider drugs as a possible cause of physical and psychological complaints.

Conclusion and Future Directions

The physician's assessment (see Chapter 52) is underutilized in the treatment of ED. The physician can help suggest the diagnosis of a covert ED, exclude diseases that can mimic an ED, uncover and treat medical complications of an ED, help determine the level of care needed (see Chapter 50), indicate pharmacological treatments that are useful while monitoring and treating complications (see Chapter 59), and estimate the urgency of treatment. Recognition of the medical complications helps increase the motivation of the patient, the family, and even the treatment team as a whole (see Chapter 50). Ongoing medical assessment decreases morbidity, addresses comorbidities (see Chapter 54), and helps guide treatment goals. Remember, the medical professional is a trusted source of information and problem solving, and can help establish trust and rapport with the patient and family.

A number of exciting questions remain to be answered in medical ED research. Three of the most important are:

1 How can the brain-body link be measured, quantified, and modified? We are starting research with neurofeedback using three-dimensional electroencephalography (neuroimaging via low-resolution brain electromagnetic tomography, i.e., LORETA) to work in this area.
2 How can we best monitor, understand, and treat abnormal heart rate variability (see Chapter 52)? Rapid changes in heart rate variability are, in my opinion, the most frequent cause of cardiac death in AN.
3 What is the most effective way to use external warming to treat EDs (Birmingham, Gutierrez, et al., 2004)?

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15

Psychiatric Comorbidity in Diagnosis

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The assessment of individuals with an eating disorder (ED) requires a comprehensive evaluation of not only eating-related symptoms (see Chapters 8–11 & 13), but also of co-occurring psychiatric symptoms that may influence eating-symptom expression, clinical care (see Chapter 54), course (see Chapter 55), and outcome (see Chapter 64). Research has demonstrated extremely high rates of psychiatric comorbidity in individuals with EDs, with one study reporting that 97% of female inpatients with a primary diagnosis of an ED had at least one additional mental disorder (Blinder, Cumella, & Sanathara, 2006; see also Chapter 54). However, merely assessing for presence of comorbidity is inadequate. To improve clinical care, it is also important to understand the timeline and sequencing of onset of co-occurring conditions, and to consider the potential impact of co-occurring psychiatric diagnoses on the diagnosis, treatment, and prognosis of the primary condition for which an individual is seeking treatment—namely, the ED (Valderas, Starfield, Sibbald, Salisbury, & Roland, 2009).

In other words, an assessment for comorbidity is an essential component of the overall evaluation of any individual presenting with an ED. Important as it is, diagnosing concurrent disorders in an ED context presents many challenges. In this chapter we highlight some of the common complications that occur when EDs coincide with such comorbid problems as mood disorders, anxiety disorders, substance use disorders, sexual dysfunction, and personality disorders. We will also recommend tools that are useful for assessing concurrent symptoms in clinical practice.

When considering the impact of psychiatric comorbidity in this context, we remind readers of three things. First, with the recent publication of *The Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*; American Psychiatric Association, 2013), there are likely to be some changes in the prevalence of co-occurring psychiatric disorders relative to the research presented in this chapter, which is based on *DSM-IV-TR* diagnoses (American Psychiatric Association, 2000). Second, in considering data on the prevalence of any comorbid psychiatric diagnoses, both the specific definition of the term "comorbidity" and criteria for establishing its presence are critical. In the discussion of the assessment of psychiatric comorbidity in

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individuals with EDs, we will be referring to diagnostic co-occurrence, that is, the concurrent diagnosis of two or more psychiatric conditions (Lilenfeld, Waldman, & Israel, 2006).

Third, it can be very important and challenging to tease apart whether co-occurring conditions may be complications of the ED (due to malnutrition or the dietary restriction that inevitably occurs in individuals with an ED) or, rather, represent predisposing factors or independent diagnoses. The pioneering study of human starvation conducted by Ancel Keys and colleagues demonstrated that malnutrition and weight loss profoundly affect personality and behavior, with weight loss reportedly leading to symptoms of depression, irritability, and social withdrawal (Keys, Brožek, Henschel, Mickelsen, & Taylor, 1950). In other words, some "comorbid" symptoms may be secondary effects of malnutrition, and will improve as eating behavior is normalized. A comprehensive assessment of the sequencing of comorbid-symptom onset in relation to the development of eating pathology can be invaluable in treatment planning.

Mood Disorders

Mood disorders, including major depressive disorder (MDD), dysthymia, and bipolar I or II disorder, are common across all ED variants. A large nationally representative sample from the National Comorbidity Replication study demonstrated that approximately 40% of individuals with anorexia nervosa (AN) reported a lifetime diagnosis of a mood disorder, as did 70% of those with bulimia nervosa (BN) and 45% of those with binge eating disorder (BED) (Hudson, Hiripi, Pope, & Kessler, 2007). Across each of the ED subtypes, MDD was the most common mood disorder diagnosis. In a separate sample of adults in treatment for an ED, no significant differences in the prevalence of a MDD were found across restrictive or binge-eating/purging subtypes of AN, and BN (Godart et al., 2006). Although a similar pattern exists in adolescents with binge-eating ED variants—with 50% of adolescents with BN and 45% of those with BED showing a lifetime mood-disorder diagnosis (Swanson, Crow, Le Grange, Swendson, & Merikangas, 2011)—adolescents with AN had a relatively low prevalence of lifetime mood disorder, at approximately 10%. An apparent implication is that mood disorders may be less common in adolescents with AN than they are in adults with AN.

Researchers have studied the time course of depressive symptoms in relation to eating symptomatology, to tease apart whether depression typically precedes or follows the onset of an ED. A large prospective study of adolescents demonstrated that increases in depressive symptomatology were associated with subsequent increases in bulimic symptoms, and vice versa (Presnell, Stice, Seidel, & Madeley, 2009). In other words, the development of eating symptoms exhibits no clear-cut temporal precedence over development of mood symptoms, and vice versa. Presence of unipolar depression has, however, been associated with diagnostic instability ("crossover," i.e., a change in diagnosis from AN to BN, or vice versa) over a 6-year period in individuals with an ED (Castellini et al., 2011). Clinicians who are providing long-term treatment for clients with an ED may therefore wish to consider the impact of concurrent diagnoses on the diagnosis and evolution of a person's symptoms over time. Reassessment of symptoms at regular intervals using multifaceted techniques (e.g., interview and self-report questionnaires) can be helpful in longer-term treatments.

A variety of brief self-report measures that have been demonstrated to have good reliability and validity can be used to assess mood disorders in an ED context (see Table 15.1). Two such measures, both available in the public domain, are the Mood Disorder Questionnaire (Hirschfeld et al., 2000), which can be used to screen for bipolar spectrum disorders, and the

Table 15.1 Self-report measures to screen for concurrent psychiatric conditions.

Comorbidity	*Instrument*	*Length*	*Reliability/Validity*
Mood disorders:			
• Depressive symptoms	Centre for Epidemiological Studies – Depression Scale (Radloff, 1977)	20 items	Good internal reliability (Cronbach's alpha of .89) in female adolescents/young adults (Lewinsohn, Striegel-Moore & Seeley, 2000)
• Bipolar spectrum	Mood Disorder Questionnaire (Hirschfield et al., 2000)	15 items	Good internal reliability (Cronbach's alpha of .90) in a sample of patients with mood disorders. A cut-off score of 7 has been suggested for use as a screening tool (Hirschfield et al., 2000)
Suicidality	Suicidal Behaviors Questionnaire-Revised (Osman et al., 2001)	4 items	Good internal reliability in adolescent and adult samples, both clinical and nonclinical (Cronbach's alpha ranged from .76 to .88, Osman et al., 2001)
Self-harm	Inventory of Statements about Self-Injury (Behaviors) (Klonsky & Glenn, 2009)	7 items assessing 12 types of self-harm behavior	Excellent internal reliability in a nonclinical sample (Cronbach's alpha of .84; Klonsky & Olino, 2008)
Anxiety disorders:			
• OCD	OCI-R (Foa et al., 2002)	18 items	Good internal reliability (Cronbach's alpha for total score >.9) in a sample of individuals with AN and healthy controls (Roberts, Lavender, & Tchanturia, 2011)
• Social Anxiety Disorder	Mini-SPIN (Connor, Koback, Churchill, Katzelnick, & Davidson, 2001)	3 items	Good internal reliability (Cronbach's alpha of .85 in a treatment-seeking sample with anxiety disorders), with good construct validity. A cut-off score of 6 has been suggested to have reasonable sensitivity and diagnostic efficiency (Weeks, Spokas, & Heimberg, 2007)
• Panic Disorder	Beck Anxiety Inventory (BAI) (Beck & Steer, 1990)	21 items	The BAI has good sensitivity and specificity for detection of panic disorder (Leyfer, Ruberg, & Woodruff-Borden, 2006)
• Generalized Anxiety Disorder	GAD-7 (Spitzer, Kroenke, Williams, & Löwe, 2006)	7 items	Excellent internal reliability (Cronbach's alpha of .92 in a patient sample), with a recommended cut-off score of 10 for identifying cases with generalized anxiety disorder (Spitzer et al., 2006)

(*Continued*)

Table 15.1 (*Continued*)

Comorbidity	*Instrument*	*Length*	*Reliability/Validity*
PTSD/trauma	Traumatic Life Events Questionnaire (TLEQ; Kubany et al., 2000)	Items assessing 16 types of traumatic events	Good temporal stability in a variety of samples (i.e., college students, battered women) and good convergent validity with structured interview (Kubany et al., 2000)
	Posttraumatic Stress Diagnostic Scale (PTDS; Foa et al., 1997)	33 items	Good internal consistency (Cronbach's alpha for total and subscales ranged from .78 to .92) in a sample of individuals with high risk for trauma and with PTSD (Foa et al., 1997)
	Short Post-Traumatic Stress Disorder Rating Interview (SPRINT; Connor & Davidson, 2001)	8 items	Good internal consistency (Cronbach's alpha of .77) and good construct validity (Connor & Davidson, 2001)
Substance use disorders	Shorter PROMIS Questionnaire (Christo et al., 2003)	160 items assessing substance use and impulsivity	Good internal reliability (mean Cronbach's alpha of .89 across subscales) and good convergent validity with existing validated measures of addictive behaviors (Christo et al., 2003)
Sexual dysfunction	Female Sexual Function Index (Rosen et al., 2000)	19 items	Good internal consistency (Cronbach's alpha of .82 and higher for subscales and total score) and good discriminant validity (Rosen et al., 2000)
Personality disorders	Schedule for Nonadaptive and Adaptive Personality Functioning (SNAP; Clark, 1993)	375 items	Good internal consistency and adequate content validity (Melley, Oltmanns, & Turkheimer, 2002)
	Personality Diagnostic Questionnaire-4 (PDQ-4; (Hyler, 1994)	99 items	Good content validity and clinical utility (Widiger, 2008)

Centre for Epidemiological Studies – Depression Scale (Radloff, 1977). For clinicians who would like to do a more comprehensive interview for mood disorders, semistructured interviews are available. The most commonly used interview for *DSM-IV* Axis I disorders, including mood disorders, is the Structured Clinical Interview for DSM-IV-TR (SCID; First, Spitzer, Gibbon, & Williams, 1997). The SCID helps differentiate between unipolar and bipolar depression and has high interrater agreement for MDD diagnoses (weighted *k* values of .80 to .91; Ventura, Liberman, Green, Shaner, & Mintz, 1998).

Suicidality and Self-Harm

Suicide attempts are disquietingly common in individuals with AN and BN, making it vital for clinicians to conduct a careful suicide risk assessment in clients with EDs. In a prospective study of a large sample of women with an ED, a total of 15% of the sample made a suicide attempt with a serious intent to die during the 8.6-year course of the study (Franko et al., 2004). The same study suggested, not surprisingly, that co-occurring psychiatric problems, such as depression and substance use, increase suicide risk. Personality features also appear important to consider, as individuals with an ED and co-occurring borderline personality disorder report higher likelihood of self-harm behaviors and suicide attempts than do individuals with an ED who have another personality disorder, or no personality disorder (Wonderlich & Swift, 1990).

In a study involving a large sample of inpatients with EDs, the lifetime prevalence of self-harm behavior was estimated to be approximately 35%, with no apparent differences in rates obtained across people with AN, BN, or eating disorder not otherwise specified (ED-NOS) diagnoses (Paul, Schroeter, Dahme, & Nutzinger, 2002). The preceding suggests that screening for self-harm behaviors, and distinguishing between self-harming behaviors that do, or do not, bear suicidal intent, are both important components of good ED assessment and treatment planning, including with inpatients. Although we believe that no self-report tool can replace the skilled clinical assessment of suicidal or self-harm urges, screening tools that assess self-harm and suicidality are available (see Table 15.1); these have been shown to be reliable and have some preliminary support for their validity and sensitivity. Furthermore, research has demonstrated good agreement between information about suicidal behaviors disclosed in clinical interviews and self-report (Kaplan et al., 1994). However, a study by Kaplan and colleagues (1994) found that patients may be more likely to disclose current suicidal ideation in self-report format questionnaires than in a clinical interview. This finding suggests that self-report measures are a good complement to information provided by clients in clinical interviews.

Anxiety Disorders

Anxiety disorders are also highly prevalent in individuals with EDs. In a large sample with heterogeneous ED diagnoses, nearly two thirds had a lifetime anxiety-disorder diagnosis (Kaye et al., 2004). In this sample, obsessive-compulsive disorder (OCD) and social anxiety disorder were the most common diagnoses, with no significant differences emerging between individuals with AN (restrictive or binge/purge subtype) or BN as to the prevalence of diverse anxiety disorders (including OCD, panic disorder, social phobia, specific phobia, agoraphobia, or generalized anxiety disorder).

A shared transmission model (Pallister & Waller, 2008) has been proposed, which accounts for high rates of comorbidity between eating and anxiety disorders. According to this model, cognitive avoidance strategies develop based on cognitive and environmental experiences. These

avoidance strategies, in turn, can take the form of disordered eating behaviors or anxiety-related behaviors (or both). Susceptibility to anxiety disorders is increased once avoidance strategies consistent with EDs develop (and vice versa). As with mood disorders, researchers have shown that the time course of the anxiety disorder diagnosis is an important feature. Findings suggest that some anxiety disorders (such as social phobia) typically precede the development of an ED, whereas others (such as panic disorder and generalized anxiety disorder) develop in concurrence with, or after, the development of an ED (Godart et al., 2003; Pallister & Waller, 2008).

Some presentations of anxiety disorders may include food-related fears, which need to be carefully differentiated from anxieties that are consistent with an ED. For example, according to *DSM-5* (2013), in individuals with an ED, an additional diagnosis of social phobia should be applied only if fear of negative evaluation about ED symptoms is not the sole source of social anxiety. Similarly, a differential diagnosis is important for OCD, as clients may present with food-related obsessions and compulsions (e.g., fear of contamination of food) in the absence of concerns about weight or shape. Adding a new diagnostic challenge, Avoidant/Restrictive Food Intake Disorder (ARFID), introduced in *DSM-5* (2013), includes conditions in which people become severely blocked in the ability to nourish themselves or be nourished by others due to such things as intense aversion to certain food tastes or textures, preoccupation with the nutritional value of certain foods, or disproportionate fears that eating will cause indigestion or vomiting (see Chapter 13). The ARFID diagnosis (Bryant-Waugh & Kreipe, 2012) classifies a range of symptoms as an eating/feeding disorder that, because they are characterized by food-related anxiety, need to be carefully differentiated from anxiety symptoms. Certain food-related fears, such as the fear of choking or vomiting, can also be considered to be a specific phobia, and may be difficult to distinguish from ARFID. According to the *DSM-5*, an ARFID diagnosis should be given if the eating-related problems require a primary clinical focus.

Research has indicated that anxiety disorders (i.e., generalized anxiety disorder, OCD, panic disorder with agoraphobia, social anxiety disorder, and specific phobia) are significantly less likely to be diagnosed after an unstructured clinical evaluation than a semistructured interview (Zimmerman & Mattia, 1999). To conduct a comprehensive assessment of anxiety disorders, clinicians should use a validated semistructured interview, such as the Anxiety Disorders Interview Schedule for DSM-IV (DiNardo, Brown, & Barlow, 1994). A summary of the psychometric properties of self-report measures that tap into generalized anxiety disorder, OCD, social anxiety disorder, and panic disorder is presented in Table 15.1.

Posttraumatic Stress Disorder, Childhood Abuse, and Trauma

There are well-established links among EDs, exposure to abuse or trauma, and Posttraumatic Stress Disorder (PTSD; see Chapter 34). About 30% of adults with BN report unwanted sexual experiences during childhood, and more than half report childhood physical maltreatment (Wonderlich, Brewerton, Jocic, Dansky, & Abbott, 1997). However, a history of trauma is more common in ED variants characterized by binge eating and purging (i.e., BN, AN-BP, ED-NOS with binge eating or purging, and BED) than in those characterized mainly by restriction (e.g., AN-R) (Carter, Bewell, Blackmore, & Woodside, 2006; Wonderlich et al., 1997). It has been suggested that trauma contributes to ED etiology. Although the specific mechanisms that could underlie a link between trauma and EDs remain to be ascertained, it is possible that exposure to traumatic stressors may lead to a psychobiological dysregulation that predisposes an individual to develop a variety of psychological disorders. Alternatively, traumas that directly affect the body (e.g., sexual abuse) may have direct consequences for body image, and in turn for eating and weight-control behaviors.

Given the general association between EDs characterized by binge eating and purging and exposure to traumatic experiences, it is not surprising to note that there exists a marked co-occurrence between BN and PTSD. The National Comorbidity Survey indicated that lifetime PTSD occurs in approximately 45% of individuals with BN, versus in only 10–15% of those with AN (including both restrictive and binge eating/purging types) (Hudson et al., 2007). Some theorists have attributed the association between PTSD and EDs to shared genetic vulnerabilities or shared symptoms (e.g., dissociation and emotion dysregulation) across the two disorders (Mitchell, Mazzeo, Schlesinger, Brewerton, & Smith, 2012).

Abuse and victimization experiences are common enough in individuals with an ED that a thorough assessment of prior traumatic experiences should be standard practice when assessing people affected by any ED diagnosis. Patients may not be comfortable in revealing major traumatic events during an initial assessment, so the evaluation of such experiences should be an ongoing process that merges into treatment.

To assess for a history of traumatic experiences, we recommend the Traumatic Life Events Questionnaire (TLEQ; Kubany et al., 2000), which assesses exposure to 16 types of potentially traumatic events and breaks down general traumatic experiences into more specific items that include contextual factors (see Table 15.1). In general, brief scales and self-report measures can complement structured interviews in determining whether a PTSD diagnosis is warranted. The "gold standard" interview for PTSD is the Clinician Administered PTSD Scale (CAPS; Blake et al., 1995), which contains a checklist of traumatic events and items that correspond to *DSM-IV* criteria for PTSD. The Posttraumatic Stress Diagnostic Scale (PTDS; Foa, Cashman, Jaycox, & Perry, 1997) is a solid self- report measure that yields both a *DSM-IV-TR* (2000) PTSD diagnosis and a measure of PTSD symptom severity. The scale contains a checklist of 12 traumatic events and questions aimed to ascertain a PTSD diagnosis and provide a measure of symptom severity. The PTDS has demonstrated high internal consistency and test-retest reliability, high diagnostic agreement with SCID, and good sensitivity and specificity (see Table 15.1).

Substance Use Disorders

High rates of alcohol and/or other drug (i.e., substance) use disorders have been reported in individuals with an ED, with approximately 25% of those with AN, 35% of those with BN, and 25% of those with BED reporting a lifetime diagnosis of a substance use disorder (Hudson et al., 2007). Individuals who report binge-eating symptomatology have a stronger likelihood of having a substance use disorder than do those with restrictive or purging symptoms in the absence of binge eating (Krug et al., 2004). That being said, approximately 25% of individuals with AN-restrictive subtype reported current use of illegal substances (including cannabis and opioids), suggesting that clinicians should not disregard substance use in individuals who do not have binge-eating symptomatology. Individuals with an ED were also significantly more likely than were healthy controls to report using some substances (e.g., cigarettes, illegal drugs) as an appetite suppressant or weight control technique (Krug et al., 2004). Clinical interviews should therefore include a comprehensive assessment of substance use, including substances that may be taken to control weight or as a compensatory measure to counteract binge eating (e.g., amphetamine, diet pills, diuretics and laxatives, and over-the-counter emetics such as ipecac).

Clinicians who are seeking a self-report questionnaire to assess substance use could employ the Shorter PROMIS Questionnaire (Christo et al., 2003), which assesses multiple addictive behaviors, including nicotine use, and drug and alcohol use.

Sexual Dysfunctions

In *DSM-IV-TR* (2000) sexual dysfunctions are characterized by a disturbance in the processes that characterize the sexual response cycle (i.e., desire, arousal, orgasm) or by pain associated with sexual intercourse. The female sexual dysfunctions include Hypoactive Sexual Desire Disorder, Sexual Aversion Disorder, Female Sexual Arousal Disorder, Female Orgasmic Disorder, Dyspareunia (painful intercourse, in general), and Vaginismus (painful vaginal sex, and especially intercourse, due to uncontrollable, spasmodic contractions of the vagina). Sexual dysfunctions seem to be common in women suffering from an ED, with about 75% of women with AN, 40% of women with BN, and 45% of women with ED-NOS reporting a loss of sexual desire (Pinheiro et al., 2010). As compared to women without an ED, women with AN and BN have higher levels of sexual anxiety, relationships without sexual intercourse, problems with arousal and orgasm, as well as lower levels of sexual satisfaction (Castellini, Lelli, Lo Sauro, Fioravanti, et al., 2012; Morgan, Wiederman, & Pryor, 1995). Prevalence estimates for sexual dysfunctions in men with EDs are currently lacking.

EDs and sexual dysfunctions may co-occur for various reasons. First, decreases in sexual desire seen in women with AN could be due to secondary effects of emaciation, given that weight restoration often seems to produce increases in, or a return to, baseline levels of sexual desire (Castellini, Lelli, Lo Sauro, Fioravanti, et al., 2012). Second, other ED features, such as depression and/or discomfort with body image can affect sexual functioning directly, in that negative body image and preoccupation with the body during sexual activity predict greater sexual dissatisfaction and higher levels of sexual dysfunction (Ackard, Kearney-Cooke, & Peterson, 2000; Morgan et al., 1995). Third, since a history of childhood abuse is a well-documented risk factor for the development of EDs and for sexual dysfunction in adulthood, this aspect constitutes a possible common factor linking sexual dysfunction and EDs in some individuals (Castellini, Lelli, Lo Sauro, Vignozzi, et al., 2012).

In the assessment and treatment of someone with an ED, it is important to determine whether sexual dysfunctions preceded or followed onset of the ED. If sexual difficulties predate the onset of the ED, then a diagnosis of a specific sexual dysfunction could be made. However, if sexual problems seem to be a direct consequence of the ED, then diagnosis of a sexual dysfunction is not yet warranted. As no standardized diagnostic interview exists for sexual dysfunction, it is advisable to conduct a thorough clinical interview to assess for the presence of sexual difficulties.

As is the case for trauma, during an initial assessment patients may be uncomfortable discussing details pertaining to their sexual behaviors. Therefore, evaluation of sexual function should be an ongoing process. Furthermore, the clinician can model comfort in discussing matters of a sexual nature to help the patient become more at ease with disclosing personal sexual information. Helpful approaches include asking patients' permission to ask a few questions relating to their sexual life, and using a neutral tone while asking direct, nonjudgmental screening questions relating to sexual history (Althof, Rosen, Perelman, & Rubio-Aurioles, 2013). During the initial and subsequent assessments or sessions, the clinician should inquire about the extent of the problem, conditions under which it occurs, duration, frequency, and impact of the problem. Additionally, obtaining information about relevant biological, psychological, and social/interpersonal factors affecting sexuality will help elucidate whether an individual is suffering from a co-occurring sexual dysfunction. As a possible complement, the Female Sexual Function Index (Rosen et al., 2000) is a brief and well-validated self-report measure of sexual dysfunction for women (see Table 15.1). It contains 19 items assessing six domains of sexual functioning: desire, arousal, lubrication, orgasm, satisfaction, and pain.

Personality Disorders

Co-occurrence of EDs with personality disorders (PDs) constitutes an important form of comorbidity in an ED context (see Chapter 32). PDs often engender conflict, distress, and dysphoria, each of which can have negative effects on interpersonal functioning. Consequently, assessment of concurrent PDs is needed, because they may directly affect the therapeutic relationship that is at the core of effective treatment. Notably, individuals with an ED are much more likely to display PDs than are people in the general population (Lilenfeld, Wonderlich, Riso, Crosby, & Mitchell, 2006). The estimated prevalence of PDs in the general population is between 6 and 13% (Paris, 2010), whereas a majority of individuals with an ED are found to display a co-occurring PD (Rosenvinge, Martinussen, & Ostensen, 2000). According to Cassin and von Ranson (2005), the most common PD in individuals with an ED is avoidant PD. Obsessive-compulsive PD is one of the most common PD forms in those with AN-R, but is also seen in BN. Dependent PD is also common to both AN-R and BN. Borderline PD is one of the most common PDs in those who engage in binge eating (Cassin & von Ranson, 2005). While there are fewer data on PDs in BED, co-occurrence of PDs in BED seems to be comparable to that seen in BN, such that those with BED display high rates of co-occurrence with avoidant, obsessive-compulsive, and borderline PDs (Becker, Masheb, White, & Grilo, 2010; Cassin & von Ranson, 2005).

It is possible that having a PD may predispose someone to develop an ED, or a PD could be a simple consequence of the ED. Another possibility is that both EDs and PDs are caused by some third factor such as shared developmental, familial, neurobiological, or hereditary pathways (Steiger & Bruce, 2004). A particular difficulty in diagnosing a PD in someone with an ED (as is the case with other disorders) is that the sequelae of malnutrition, hypomania, or substance use may influence behavioral and affective regulation, thereby confounding the clinical profile and making it hard to ascertain the presence of a PD. For example, malnutrition can have adverse effects on personality functioning and can alter personality presentation. Extended periods of starvation can increase levels of anxiety, depression, rigidity, obsessiveness, and irritability (Keys et al., 1950). Thus, a critical research-related issue pertains to the persistence of PD symptoms after recovery from an ED. Certain personality features seem to be quite stable, regardless of state of activity of the ED. For instance, PD diagnoses often persist after ED remission in individuals with AN-BP (Cassin & von Ranson, 2005). However, certain personality traits, such as impulsivity and affective instability, appear to improve with nutritional stabilization, suggesting that there is a degree of state-dependency. Therefore, clinicians need to exercise caution when diagnosing PDs in someone with an active ED.

The preferred method for diagnosing PDs is the semistructured interview. Such interviews ensure that a systematic and comprehensive assessment of the diagnostic criteria of each personality disorder has been made. Two valid and widely used semistructured interviews are the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II; First, Gibbon, & Spitzer, 1996) and the International Personality Disorder Examination (IPDE; Loranger, 1999). Due to their depth and comprehensiveness, semistructured interviews often take quite a long time to administer. Therefore, it is practical for clinicians to first administer a self-report inventory to identify the principal areas of dysfunction that warrant additional attention in a subsequent interview. The Schedule for Nonadaptive and Adaptive Personality Functioning (SNAP; Clark, 1993) and the Personality Diagnostic Questionnaire-4 (PDQ-4; Hyler, 1994) are two validated and potentially useful self-report measures (see Table 15.1).

Conclusions and Future Directions

Individuals with EDs commonly display one or more concurrent psychiatric disorders, and diagnosing comorbid conditions in those with EDs presents numerous challenges. We suggest that a comprehensive clinical evaluation, complemented by self-report measures, can optimize the screening for concurrent psychiatric disorders in an ED context (see Chapter 39). This comprehensive evaluation often takes place over the course of one or two extended appointments prior to beginning treatment. However, if a clinician chooses to use semi-structured interviews in an assessment for specific concurrent conditions (e.g., mood or anxiety disorders), the length of the evaluation period can be considerably extended. There are numerous clinical implications of the presence of psychiatric comorbidity in an individual seeking treatment for an ED. Concurrent diagnoses may affect global functioning, treatment outcome, and prognosis. A thorough assessment of concurrent conditions can therefore aid the process of clinical formulation and treatment planning.

A comprehensive assessment can also be useful given that comorbid psychiatric conditions may sometimes be associated with complaints that mimic ED symptoms. For instance, depression can present with loss of interest in eating and weight loss, or OCD can impede eating due to concerns about being contaminated in the process of touching or eating certain foods. A comprehensive evaluation also allows for determination of the time course of symptoms in relation to the ED. Concurrent symptoms that develop only after the onset of the ED may not represent a comorbid disorder but, more likely, the sequelae of the ED on psychological functioning. For instance, a period of marked behavioral dysregulation (characterized by self-mutilation or recklessness in other areas) need not imply an underlying personality pathology—especially if present only transiently, during a period of marked dietary restriction, or during a hypomanic episode. Likewise, decreased interest in sex, if present only subsequent to ED onset, would not justify a diagnosis of sexual dysfunction. Finally, as malnutrition may underlie many symptoms seen in concurrent disorders, care must be taken to tease out the role of malnutrition in the clinical profile of an individual with an ED. Ongoing assessment of concurrent psychiatric symptoms throughout the course of treatment for an ED is suggested, as some symptoms may ameliorate as a client's nutritional status improves.

Further insight into the sequencing of concurrent conditions represents one of the key areas for future research, as knowledge of the precursors to an ED can aid in the development of existing biopsychosocial models of EDs. With the recent changes to some of the diagnostic criteria for EDs in the *DSM-5* (2013), future research will also need to examine whether there are shifts in the patterns of diagnosable concurrent disorders across the ED subtypes. As highlighted throughout the chapter, there are some differences in the prevalence of concurrent conditions depending on whether binge-eating/purging symptomatology is present. This, in part, has led some researchers to examine more critically the utility of the diagnostic system, given the heterogeneity of symptoms present in individuals with EDs, and given the relatively high prevalence of diagnostic crossover (Peat, Mitchell, Hoek, & Wonderlich, 2009). Future research that includes a dimensional approach to the classification of eating and concurrent symptomatology may also help elucidate what specific phenomenology is associated with comorbid conditions, which in turn can also contribute to models of EDs (see Chapter 1).

The role of comorbidity in the treatment of individuals with EDs is addressed further elsewhere (Steiger & Israel, 2009; see also Chapter 54). Here, we simply note that there can be "spinoff" benefits of work in the general psychopathological domain (e.g., affective

or interpersonal disturbances) for positive developments in eating-specific domains, and vice versa. For instance, it has been well demonstrated that ED-focused therapies yield demonstrable benefits in reducing general psychopathology and depression, and improving social adjustment (Fairburn, Kirk, O'Connor & Cooper, 1986). In contrast, attention to comorbidity, when it is present, optimizes outcomes on eating-specific dimensions (Fairburn et al., 2009).

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16

Cross-Cultural and Ethnicity Issues in Diagnosis

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Countries such as the United States, Canada, and the United Kingdom have steadily become more ethnically diverse (Office for National Statistics, 2012; Statistics Canada, 2011; U.S. Census Bureau, 2010), and with technological advances in communication and transportation, the world as a whole has moved towards global integration. Along with increased multicultural contact within and between countries has come an agreement in the mental health field regarding the importance of taking into consideration race, ethnicity, and culture in the diagnosis, treatment, and research of psychiatric distress (Alarcon et al., 2002; Lewis-Fernandez & Diaz, 2002; Lu, Lim, & Mezzich, 1995; see also Chapter 51).

This chapter will focus on cultural and ethnicity issues that are important to consider in the diagnosis of eating disturbances. Because race, ethnicity, and culture are related factors that can be complicated for even experienced clinicians to differentiate and incorporate into assessment and treatment, this chapter will begin with a definition of each. Then the cross-cultural applicability of current diagnostic classifications of eating disorders (EDs) and of diagnostic instruments will be explored. Lastly, cultural issues that affect the therapeutic interaction and recommendations regarding future research will be presented. The focus of this chapter will be on the diagnoses of anorexia nervosa (AN) and bulimia nervosa (BN) in women due to limited cross-cultural research on the diagnosis of EDs in men and children and on binge eating disorder (BED).

Race, Ethnicity, and Culture

Scientific studies have debunked previous notions of race as a genetic construct, and *race* has come to be defined as a culturally constructed categorization where descriptive, physiognomic qualities have been given meaning by social, economic, and political forces (Alarcon et al., 2002). *Ethnicity* (see Chapters 23, 25, & 51) is also a cultural construction, though variables such as shared ancestry, language, religion, culture, or nationality are more central in defining ethnic groups as compared to superficial racial categorizations. In the United States, ethnicity is often linked to race, though in other countries ethnicity may be determined more by shared religion or language.

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European Americans, African Americans, Hispanics, Asian Americans, and American Indians are considered the main ethnic groups in the United States (Levine & Smolak, 2010). Each ethnic group consists of subgroups that may differ considerably with regard to language, history, and culture (see Chapter 23). For example, Asian American subgroups include descendants of people from China, the Philippines, and India; the term African American might refer to third-generation Africans or more recent immigrants from Africa and the Caribbean. Similarly, the term American Indian encompasses over 500 diverse tribes, while Hispanic ethnic subgroups include individuals from countries as diverse as Mexico, Puerto Rico, and Cuba and do not include Portuguese-speaking Brazilians who are considered Latino but not Hispanic (U.S. Census Bureau, 2010).

Culture refers to socially transmitted values, beliefs, traditions, and behavioral norms shared by a group of people (see Chapters 7 & 23). Cultural groups are not limited to ethnic groups but can also define the military, people of similar sexual orientation, or religious or political organizations (Lewis-Fernanez & Diaz, 2002; see also Chapter 35).

As part of a culturally sensitive assessment process, clinicians need to be aware of the great diversity that exists within racial, ethnic, and cultural groups and should avoid broad generalizations that may or may not apply to the individual. Clinicians need to be attentive to the specific ethnic or cultural groups their clients identify with and should take into consideration the effects that race, ethnicity, and culture may have on their clients' experience and expression of psychiatric distress (see Chapters 23, 25, & 51). It is important to note that culture does not exist only for individuals from minority groups or from foreign or developing countries; culture influences the social, physical, and emotional experience of all individuals (Alarcon et al., 2002; see also Chapter 21).

Cultural Perspectives on Diagnostic Classification Systems

The most widely used diagnostic classification systems are the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*; American Psychiatric Association, 2013) and the *International Classification of Mental and Behavioural Disorders* (10th ed; *ICD-10*; World Health Organization, 2010). Established nosological systems such as these are essential in guiding clinical interventions because they provide information regarding operational criteria, prognosis, and the preferred treatment of mental disorders (Kirmayer, 2005). In addition, the *DSM* and *ICD* codify labels and explanations of distress in ways that may help patients and their families feel that an illness is understood and thus potentially manageable. These diagnostic systems also facilitate communication between helping agencies, assist in the training of health professionals, and guide policies regarding resource allocation for healthcare and research (Kirmayer, 2005; Smart & Smart, 1997). Because of the wide and varied use of both the *DSM* and the *ICD*, it is imperative that these classification systems be examined for cross-cultural applicability. Otherwise, misdiagnoses can lead to negative consequences such as inadequate or inappropriate treatment, misleading prevalence estimates, and ethnic disparities in healthcare (Becker, 2007).

A Universal Perspective

Both the *DSM* and the *ICD* reflect a universal perspective in which core symptoms and syndromal patterns of psychiatric disorders are assumed to be applicable to individuals from various cultural backgrounds. The universal stance of the *DSM* and the *ICD* has been criticized

because in both instances diagnostic categories were developed based on the expert opinions of mostly Western professionals and on research conducted with mostly Western populations (Becker, 2007; Canino & Alegría, 2008; Good & Good, 1986). According to critics, applying these culturally constructed categories can lead to a "category fallacy" (Kleinman, 1988) in which a narrow, homogeneous diagnostic classification developed in one culture is assumed without conceptual and psychometric validation (and thus often mistakenly) to be meaningful and relevant to individuals from all cultures. In comparison to a universal perspective, a relativist perspective emphasizes the importance of establishing a diagnostic system based on an understanding of disorders that is grounded in the cultural contexts of their manifestations. Relativists view psychiatric disorders as being shaped to a substantial degree by culture's impact on not only the likelihood of developing a disorder but also on the symptoms and the organization of symptoms into disorders (Canino & Alegría, 2008).

The *ICD*'s own advisory group states that the assumed universality of the *ICD* has not been proven and that "culture has largely been viewed as a distraction or source of error in classification," though "steps are being taken to improve the cultural applicability of the next edition" (International Advisory Group for the Revision of ICD-10, 2011, p. 88). Likewise, in *DSM-5* (2013) the American Psychiatric Association issues a cautionary statement that the illness categories described in its manual do not encompass all mental disorders seen throughout the world. Both the *ICD-10* and the *DSM-5* contain appendices that catalogue "Culture-Specific Disorders" and a "Glossary of Cultural Concepts of Distress," respectively, but these features have been criticized as giving the impression that their contents are exotic and foreign, in contrast to the disorders in the main text, which are presented as culture-free or universal (Smart & Smart, 1997). The *DSM-5* also includes text descriptions of "Culture Related Diagnostic Issues" for specific disorders. However, culture is often described as affecting risk and protective factors, not core symptoms, which implies that the nature of a disorder is still consistent across cultures (Canino & Alegría, 2008). Despite these limitations, the *DSM-5* (2013) does improve upon previous editions by expanding its discussions of the ways in which culture can impact the diagnosis of mental illness. Both in the introduction and in a section on "Emerging Measures and Models," the *DSM-5* describes in detail the various ways in which culture can affect the experience, expression, and explanation of psychological distress. The *DSM-5* also offers a semistructured interview, the Cultural Formulation Interview (CFI) described later in the chapter, to address these cultural factors during a diagnostic assessment.

Evaluating the Universality of AN and BN

There has been much debate regarding whether the *DSM* and *ICD* diagnoses for EDs can be applied universally across diverse cultural settings (Habermas, 1996; Keel & Klump, 2003; see also Chapters 2, 6, 8, & 23). Some argue that the diagnoses are relative to Western culture and may not apply to non-Western cultures that have different ideals, rituals, and beliefs regarding food and the body (Becker, 2007). According to Lewis-Fernandez and Kleinman (1995), the Coordinating Group for the Task Force on Culture and DSM-IV had even recommended that AN be moved to the appendix of Culture Bound Syndromes, stating the illness was bound to Western culture and its emphasis on thinness and dieting. Others argue that eating disorders should remain in the main text because of their presence and prevalence in multiple cultures (Keel & Klump, 2003). Western influence is seen as increasing the risk of eating disorders, but not as a necessary causal agent because many other factors, including biopsychiatric, psychological, and interpersonal issues, contribute to the

development of EDs (Levine & Smolak, 2010; see also Chapters 6 & 23). Though AN has remained in the main text, the *DSM* does acknowledge that there may be cultural variations in the presentation and occurrence of AN. *DSM-5* (2013) also points out that both AN and BN are more prevalent in industrialized societies and that research is lacking regarding the prevalence of AN and BN in other cultures, particularly those that are not economically advantaged.

There are also criticisms regarding not the universality of EDs per se, but the universality of the specific criteria used in the diagnosis of EDs (Keel & Klump, 2003). In particular, many theorists, led by Hong Kong psychiatrist Sing Lee (Lee, Ho, & Hsu, 1993; Lee, Lee, Ngai, Lee & Wing, 2001; Lee, Ng, Kwok, & Fung, 2010), have questioned whether the fear of fatness (or weight gain) should be included as an essential criterion for the diagnosis of AN. Research in locations such as Hong Kong, India, west Malaysia, and Ghana has found that some individuals with AN or BN provide explanations other than fat phobia for their food restriction (Becker, Thomas, & Pike, 2009; see also Chapter 23). Historical investigations of AN have also revealed that early reports of food refusal leading to emaciation lacked weight and shape concerns (Keel & Klump, 2003; see also Chapter 2). In these cross-cultural and historical accounts of AN, alternate rationales for food restriction have included abdominal bloating or pain, loss of appetite, religious motives, eccentric nutritional ideas, or simply "don't know" (Becker, 2007; Lee et al., 1993). The predominant rationale for food restriction still appears to be a fear of fatness, but it is clear that it is not the only expressed motivation, especially in children (see Chapter 13), adolescents (see Chapter 8), and in cultures that do not overvalue thinness or dieting (Lee et al., 2001).

DSM-5 (2013) acknowledges cultural variations in AN, noting that the expressed motivation for food restriction may vary across cultural contexts and that an absence of "fat phobia" is found more frequently in Asia "where the rationale for dietary restriction is commonly related to a more culturally sanctioned complaint such as gastrointestinal discomfort" (p. 342). This statement is in the text on Culture-Related Diagnostic issues, which may be overlooked or dismissed as nonessential. Critics have argued that without a more flexible definition in the diagnosis itself that allows for a plurality of rationales for food restriction, there is the risk that clinicians and researchers may miss or dismiss cases of AN where the fear of fatness is absent (Becker, Thomas, & Pike, 2009; Lee et al., 2010). AN without fat phobia might be misdiagnosed as Other Specified Feeding or Eating Disorder (OSFED; previously Eating Disorder Not Otherwise Specified [ED-NOS]; see Chapters 4 & 11) when in fact it shares more clinical and treatment similarities with traditional AN (Becker, 2007). Misdiagnosis of AN may partially explain why in Asian populations, the prevalence rates for diagnosable AN and BN remain low, despite studies showing high rates of ED symptoms (Cummins, Simmons, & Zane, 2005).

To address some of the limitations in using fear of fatness as a core diagnostic criterion for AN, some theorists have suggested avoiding inferences regarding motivation and focusing instead on descriptions of behaviors that interfere with maintaining a minimum standard of weight (Becker, Eddy, & Perloe, 2009). This would capture the ego-syntonic nature of the disorder (see Chapter 18) and would allow for inclusion of individuals who may have difficulties expressing a rationale for food restriction, for example, children with limited insight into their symptoms or individuals from cultures that discourage the sharing of private information (Keel & Klump, 2003). The *DSM-5* (2013) reflects this suggestion to focus on overt behaviors. Criterion A was reworded from a "refusal to maintain body weight" to

describe a behavioral "restriction of energy intake" (p. 338), and Criterion B, which previously described just a fear of fatness, added a clause to include the possibility of "persistent behavior that interferes with weight gain, even though at a significantly low weight" (p. 338).

With these new criteria, behaviors leading to significantly low body weight are now the core of AN, and cultural variations in the forms of the disease or disorder are more likely to be captured. There is concern that without a specific rationale for weight loss, AN might be confused with conversion disorder (e.g., food intake is reduced due to the experience of distressing abdominal bloating or pain), depressive conditions that include loss of appetite (literal "anorexia"), or restriction of calories due to less pathological reasons such as the belief that calorie restriction leads to longevity (Becker, Thomas, & Pike, 2009; Rieger, Touyz, Swain, & Beumont, 2001). Criterion C, which still describes disturbance in body image or a lack of recognition regarding the seriousness of the low body weight, could assist in the differentiation between AN and these other conditions.

There appears to be less controversy regarding cross-cultural variations in the manifestation of BN (Keel & Klump, 2003). Some studies have shown ethnic differences in the prevalence of specific compensatory behaviors (Hood, Vander Wal, & Gibbons, 2009) but descriptions in both the *DSM* and *ICD* regarding compensatory behaviors are sufficiently broad enough to capture a wide range of behaviors. There may be cultural differences, though, regarding what the *DSM-5* (2013) describes as "an amount of food that is definitely larger than what most people would eat during a similar period of time under similar circumstances" (p. 345) or "recurrent inappropriate compensatory behaviors" (p. 345). These statements require the clinician to consider what is deemed normal within the individual's cultural milieu. For instance, in Fiji, the use of herbal purgatives following a feast is culturally sanctioned and therefore may not be distressing to the individual or abnormal according to Fijian cultural norms (Becker, 2007).

There have been concerns regarding the number of criteria required for a diagnosis of BN. Tsai and Gray (2000) found that in a sample of 257 Asian American women ranging in age from 18 to 30, an "unrealistically" low number of women were diagnosed with BN when the four primary criteria in the *DSM-IV* (2000) were used, but the prevalence jumped from approximately 0.8% to 5.1% when a threshold of only three of the four criteria was used. The researchers suggest that the diagnostic criteria be adjusted to account for subsyndromal levels of EDs that still evidence a high level of dysfunction or distress (see Chapters 4 & 11). Another diagnostic criterion that has been criticized is the frequency of binge eating. Both the *DSM-IV* and the *ICD-10* require at least two binge eating and purging episodes per week in the last 3 months. These requirements for frequency and duration have been criticized as being too stringent for use in some cultures, which may result in missed cases of individuals who evidence symptoms of BN but may have been diagnosed as ED-NOS because they did not meet the criteria for the frequency of binge eating and purging episodes (Alegría et al., 2007). The *DSM-5* (2013) reflects these concerns in changes made to the frequency requirement to one binge and purge episode per week in the past 3 months.

Culture and Diagnostic Instruments

Researchers who accept the universality of the *DSM* and *ICD* often utilize what is referred to as an "etic" approach (Morris, Leung, Ames, & Lickel, 1999). In an etic approach, diagnostic instruments based on what are presumed to be universal classifications are used to measure

psychological constructs in individuals from different cultures (Becker, 2007). Although an etic approach is useful for making ethnic and national group comparisons and for describing general cross-cultural phenomena, it has been criticized for being insensitive to cultural nuances (Morris et al., 1999). Clinicians and researchers who adopt a relativistic perspective often utilize an "emic" approach that emphasizes local representations of illness, their localized meanings, and their distribution and correlates within the particular culture of interest (Alarcon et al., 2002). An emic approach provides an in-depth understanding of a particular culture but its narrow, culture-specific focus will probably make it difficult to compare data across cultures. A "derived etic" approach combines both etic and emic approaches in that "standard" procedures and instruments are adapted based on information from the local culture (Morris et al., 1999).

Many clinicians and researchers are utilizing an etic approach to the diagnosis of EDs when using instruments such as the Eating Attitudes Test (EAT; Garfinkel & Newman, 2005), Eating Disorder Inventory (EDI; Garner, Olmstead, & Polivy, 1983), and the Eating Disorder Examination (EDE; Cooper, Cooper, & Fairburn, 1989) in culturally diverse settings (see Chapters 38 & 40). The problem is that these "standardized" and widely used (and thus extremely familiar) instruments and interviews (see Chapters 8–11) were developed based on Western diagnostic classification systems and were then validated and normed on primarily Western populations (Wildes, Emery, & Simons, 2001). The reliability and validity of ED instruments for use in cultures other than those they were developed in needs to be ascertained, otherwise the instruments risk being culturally insensitive and ineffective in detecting EDs (Canino & Alegría, 2008; Good & Good, 1986; see also Chapter 7). The process of adapting a diagnostic tool used in one culture for use in another culture requires testing the two versions for semantic, content, conceptual, technical, and criterion equivalence, and few cross-cultural studies undertake this comprehensive validation process (Canino & Alegría, 2008). Cultural observations, consultation with local experts, qualitative interviews, and pilot testing can assist in the modification and validation of a "standardized" instrument or interview before use in the culture of interest (Becker, 2007). Efforts to gather emic understandings before making etic comparisons are necessary in order to gather meaningful information regarding EDs in non-Western cultures.

To enhance the use of diagnostic instruments across cultures, several theorists have recommended a two-stage process in which standardized questionnaires are first administered to screen for symptomatic individuals who are then followed up with a semistructured interview (Cummins et al., 2005; Garfinkel & Newman, 2001; Le Grange, Louw, Breen, & Katzman, 2004). The interview allows for an explanation of the meaning behind symptom endorsement on a survey, thus facilitating exploration of localized thoughts, feelings, and behaviors related to eating pathology that may not be captured with standardized instruments. For example, in their study of South African adolescents, Le Grange and colleagues (2004) followed up administration of the EAT with interviews and found that symptoms of self-starvation endorsed on a self-report questionnaire were related to poverty and cultural beliefs rather than a desire to lose weight (see Chapter 23). One young girl described eating pig's meat because it was the only food available but then vomiting that food because digesting it went against her cultural beliefs (Le Grange et al., 2004). Careful and patient exploration of the meanings behind symptoms may not only prevent misdiagnosis and missed diagnoses but may also serve to strengthen the therapeutic relationship.

Culture and the Diagnostic Process

Diagnostic classification systems and diagnostic instruments both contain discrete items that can be examined for meanings and relevance across cultures, but perhaps more complicated is ascertaining the impact of culture on the complex set of social interactions that constitute the diagnostic process. Both the clinician and the client are individuals influenced by their own cultural backgrounds while engaging in a complex interaction in a clinical setting that also occurs in a cultural context (see Chapter 39). In the Western culture it is often the "licensed or certified" clinician who dominates the diagnostic process, determining when to probe for more information and then deciding who is legitimately distressed or not (Kirmayer, 2005). Being in this position of power, clinicians need to be aware of the possibility of their own cultural biases, which may occur outside of their awareness and can negatively impact the diagnostic process.

Patient factors that are framed by cultural influences may have a significant effect on the diagnostic process, beginning with help seeking. In some cultural groups, there may be great shame related to seeking help for psychological disorders, including EDs (Hood et al., 2009). Feelings of shame may contribute to a focus on somatic symptoms rather than on emotional distress, and thus clinicians need to be attuned to the possibility that psychological distress may underlie somatic complaints. In addition, in some cultures such as Japan, expressing distress may be viewed as a sign of weakness while enduring hardships is characterized as a virtue, which may further limit self-disclosure regarding psychiatric distress (Pike & Borovoy, 2004). Some cultural groups may tolerate greater levels of distress before seeking help. For example, as compared to European American women, African American women have been found to experience higher levels of distress related to eating or their bodies before seeking help (Grilo, Lozano, & Masheb, 2005). Additional cultural variables that may be barriers to help seeking include mistrust of mental health professionals or lack of knowledge regarding mental health treatment (Cachelin, Rebeck, Veisel, & Striegel-Moore, 2001). These impediments to help seeking may likewise deter disclosure of personal information to friends and family or to medical or mental health professionals who may assist in the detection of EDs.

There is evidence of clinician biases that appear to be based on the stereotype that EDs occur only in Caucasian populations (Becker, Franko, Speck,& Herzog, 2003; Gordon, Brattole, Wingate, & Joiner, 2006). For example, Gordon et al. (2006) found that when clinicians were provided with nearly identical fictional descriptions of an individual with ED symptoms, the discrete symptoms were recognized similarly whether the individual was Caucasian, Hispanic, or African American. However, when the individual was African American, those specific symptoms were not conceptualized as a group into ED syndromes, making it more likely for clinicians to suggest the individual did not need to receive any form of help, whether from friends and family or from a mental health professional. In a separate study, clinicians were given detailed instructions for detecting ED symptoms, as well as flow sheet algorithms for making referral recommendations (Becker et al., 2003). Even with comparable symptoms, ethnic minority individuals were less likely than nonethnic minority individuals to be referred for any further treatment. In fact, Latina participants had the most severe symptoms and were still less likely to be referred for further evaluation. When individuals from this same study were later contacted, the researchers learned that ethnic minority individuals were also less likely to have been queried by a physician about ED symptoms than were nonethnic minority individuals.

Clinician beliefs may be based on research that has shown lower levels of body dissatisfaction and EDs in ethnic minority groups, though there are certainly studies that suggest otherwise (Levine & Smolak, 2010; Wildes et al., 2001; see also Chapters 23 & 25). African American and Hispanic women may also be viewed as protected from EDs because their cultural groups do not value an overly thin physique, and the petite body frame of Asian Americans may contribute to a lack of recognition of EDs in this ethnic group. Asian Americans are also subjected to the "model minority" stereotype of being successful and capable and thus suffering from few psychological problems. The reality is much more complicated (see Chapters 23 & 25). At the very least, pathological eating behaviors have not been found to be completely absent from any cultural group, and because of the potentially severe consequences of EDs, the goal of ED assessment should be to identify EDs in all individuals who may be suffering regardless of trends within their ethnic group (Levine & Smolak, 2010). In addition, because individuals with EDs may present in a variety of clinical and medical settings, all practitioners who may come into contact with EDs should be knowledgeable about both the extremely diverse range of people suffering from EDs and the diverse range of presentations in ED symptoms.

Language differences may also impact the diagnostic process. In the process of translation, not just words but meanings and idiomatic expressions also need to be understood from the perspectives of both cultures, as difficulties can occur when two cultures do not share equivalent meanings. For example, the frequently used concepts of "binge eating" ("binge") and "dieting behavior" do not have corresponding words in the Pakistani Urdu language (Choudry & Mumford, 1992), and "calories" is a difficult word to translate for those cultures that do not emphasize counting calories (Becker et al., 2010). Even with the same language, there may still be nuances in the meanings of words that differ between cultures. For example, in a study using an Arabic version of the Eating Attitudes Test, Al-Adawi, Dorvio, Burke, Moosa, and Al-Bahlani (2002) found that a Saudi Arabic translation created linguistic and conceptual misunderstandings in their Omani sample, necessitating the development of an Omani Arabic version.

To minimize barriers to effective diagnosis and treatment, clinicians need to be trained in general cultural competence (*DSM-5*, 2013, pp. 749–759; Lu et al., 1995). This requires a genuine respect for cultural differences, knowledge of how issues such as racism, discrimination, and poverty can negatively affect individuals from minority groups, and an awareness of ways in which social and cultural contexts can impact psychiatric distress. To be culturally competent, clinicians also need to be aware of the effects that their own cultural identity and biases and their style of interviewing have on the diagnostic process. Cultural competence also includes an awareness of one's own limitations and biases and knowing when to seek additional assistance through cultural consultants, specially trained mental health translators, or family members. Without cultural competence, clinicians may unintentionally speak or act in a way that a client finds incomprehensible, judgmental, or offensive and thus be vulnerable to making diagnostic errors and providing inappropriate treatment (*DSM-5*, 2013; Lu et al., 1995).

To facilitate the incorporation of cultural factors in the diagnostic process, Cultural Formulation guidelines for a "mini clinical ethnography" were created for the *DSM-IV* (American Psychiatric Association, 2000; Lewis-Fernandez & Diaz, 2002) and retained for *DSM-5* (2013, pp. 749–759). The Outline for Cultural Formulation suggests exploring the following aspects: cultural identity of the client; cultural conceptualizations of distress; psychosocial stressors and supports that can impact vulnerability and resilience, respectively; elements of the relationship between the client and the clinician; and an overall cultural assessment for diagnosis and care. These guidelines encourage clinicians and clients to collaborate in developing a narrative—a story that has meaning to the client in the context of his or her own

cultural perspective—regarding the client's illness experience. With this information, a more accurate diagnosis and a treatment plan acceptable to the client can be rendered. A client who feels understood might be more likely to engage in and remain in treatment (see Chapter 63). In fact, "being understood" has been cited as one of the most important factors for those recovering from AN (Hsu, Crisp, & Callender, 1992).

A criticism of the Cultural Formulation Outline in *DSM-IV* (2000) was that it failed to provide a specific guideline or any case illustrations to facilitate its use, which may have contributed to its limited use (Mezzich, Caracci, Fabrega, & Kirmayer, 2009). To address these concerns, the *DSM-5* (2013, pp. 750–759) has updated the outline format with the Cultural Formulation Interview (CFI), which consists of 16 questions to easily assess the various areas outlined in the Cultural Formulation guidelines. Most of the questions are general in nature and focus on the client's own view as opposed to cultural or ethnic issues. Two examples are "People often understand their problems in their own way, which may be similar or different from how doctors describe the problem. How would *you* describe your problem to someone else?" (*DSM-5*, p. 752; italics in the original); and "Why do you think this is happening to you? What do you think are the causes of your [PROBLEM]?" (p. 752; bracketed insert is in the original). The *DSM-5* describes the CFI as being relevant for use "in the initial assessment of individuals in all clinical settings, regardless of the cultural background of the individual or of the clinician" (p. 751), though the CFI is seen as particularly useful when there are cultural differences between clinician and patient and/or a need to explore culturally expressed symptoms. Despite attempts to make the CFI relevant to all individuals, it remains (pp. 750–759) in the 74-page Section III (Emerging Measures and Models) of the *DSM-5*, which was placed right before the 108-page set of Appendices, including the "Glossary of Cultural Concepts of Distress" (pp. 833–837) containing information about what were previously called "Culture Bound Syndromes." Thus, the CFI is located approximately 400 pages after the conclusion of the Feeding and Eating Disorders section, which may limit its use with ethnic minority patients.

Conclusions and Future Directions

Though culture shapes the lives of all individuals, this chapter focused on diagnostic issues most relevant to those from non-Western cultures. The universality of mental illness categories developed by Western professionals has been questioned, and there exist challenges in applying the Western-based diagnostic categories of the *DSM-5* and *ICD-10* across multiple cultural contexts. With regard to EDs, there is sufficient evidence that illness presentations may differ across cultures, especially regarding the emphasis on the fear of fatness. Thus, when working with individuals from non-Western cultures, caution must be used in applying diagnostic categories based on Western ED presentations. There are similar concerns regarding the use of diagnostic measurements developed and normed on Western populations with individuals from other cultures. Ideally, etic approaches to diagnostic measures should be infused with emic understandings of localized presentations of EDs. The diagnostic process may also be impacted by cultural factors. For example, clinician biases based on racial stereotypes, language differences, and a clinician's limited cultural competence may interfere with obtaining an accurate diagnosis. The *DSM-5*'s Cultural Formulation Interview provides a way in which cultural factors can be incorporated into the diagnostic process. Patient factors, such as feelings of shame, a focus on physical complaints, higher distress tolerance, and mistrust of mental health professions, may also limit self-disclosure or impede seeking help from a mental health professional.

Accompanying the introduction and dissemination of the *DSM-5* (2013) will be important questions regarding whether the updated diagnostic criteria adequately capture cultural variations in EDs. It will be important for researchers to examine whether changes in the diagnostic criteria (see Chapters 8, 9, 11, & 13) and the addition of BED (see Chapter 10) lead to increased and more accurate identification of eating disorders in culturally diverse populations (see Chapters 23 & 25) and whether this in turn leads to positive changes in referrals and treatment outcome for those who have typically been underdiagnosed or misdiagnosed. Similarly, it is very likely that new diagnostic instruments that mirror changes made to the *DSM-5* will be necessary to capture the shift in focus to observable behaviors. These measures will need to be carefully validated for use with diverse populations within and across cultures and ideally would be developed utilizing a derived etic approach with information from multiple cultures so as to limit the narrow focus on Western ED presentations. Existing ED instruments also require more rigorous studies regarding their validity for use across cultures before the data obtained by using these measures can be meaningfully interpreted (Chapter 25 presents a parallel argument for careful validation of measures to be used with various ethnic groups).

To decrease ethnic disparities in the treatment of EDs, future studies should also examine additional factors that might reduce misdiagnosis. Here are three important hypotheses to be tested:

1 Increasing clinician awareness of ethnic variations in ED presentation and of potential therapist and patient biases will facilitate the identification of EDs in individuals from non-Western cultures (see Chapter 6).
2 Training programs that increase general cultural competence or that encourage the use of the Cultural Formulation Interview will improve the diagnosis and treatment of EDs.
3 Community-based, psychoeducational outreach programs, especially for vulnerable populations, will facilitate the disclosure of ED symptoms.

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Section IV
Theories

17

Biopsychiatric Theories of Eating Disorders

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Descriptions of self-starvation and disordered eating have appeared throughout history, often related to religious beliefs and asceticism (see Chapters 2 & 3). One of the earliest descriptions was from Rome in 383 AD and described a group of women, disciples of Saint Jerome, who starved themselves to achieve spiritual purity, with one of the group dying as a result. Similar descriptions are seen during the European Renaissance when young women such as Saint Catherine of Sienna and Saint Theresa of Avila fasted and purged as a route to God (Pearce, 2004).

It wasn't, however, until 1873 and 1874 when two physicians, Charles Lasègue and William Gull, independently described a condition that would be recognizable to modern readers as anorexia nervosa (AN; see Chapter 2). While it was Gull who coined the term "anorexia nervosa," both authors described a condition in which young women lost weight through deliberate dietary restriction. Both described many of the characteristic physical findings of the disorder including emaciation, bradycardia, hypothermia, psychomotor agitation, and amenorrhea (see Chapters 14 & 52) but also stressed the underlying psychological etiology (Gull, 1874; Lasègue, 1873). In the early 20th century, confusion regarding the nature and etiology of AN arose when in 1914 and 1916 Simmonds published accounts of cases of cachexia (emaciation) seen in women who were found on postmortem to have atrophy of the anterior lobe of the pituitary. For the subsequent three decades, AN was regarded as a disturbance of pituitary function, and it was not until Sheehan's work was published in 1949 that this association was dispelled (Bemporad, 1996; Parry-Jones, 1985; Pearce, 2004).

Throughout the late 1940s and the 1950s the etiology of AN was postulated in orthodox psychodynamic formulations (see Chapter 20) that focused on possible unconscious motives behind the expressed symptoms. A recurring theme in these formulations saw AN as a defense against sexuality. In the 1940s for instance, Waller, Kaufman, and Deutsch proposed that AN represented a defense against unconscious fantasies of oral insemination (Bemporad, 1996; Russell & Treasure, 1989).

In 1962 Hilde Bruch first highlighted the issues of abnormal body image and fear of fatness, linking these symptoms to distorted attempts at mastery in individuals who felt helpless in their environments (Bruch, 1962; see also Chapter 2). Since the 1970s, diagnostic formulations for

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AN have focused on symptomatic descriptions and etiology seen in the context of complex interactions between genes, biology, and the environment (see Chapter 67).

The last three decades have seen an increasing focus on understanding the biological basis of eating disorders. Central to this has been a focus on the role of genetics and gene–environment interactions. Family and twin studies have shown high levels of heritability of both AN and bulimia nervosa (BN) indicating a substantial genetic influence in both disorders (see Chapter 28). Heritability estimates for BN have been reported to be between 28% and 83%, and for AN from 48% to 88% (Hinney, Scherag, & Hebebrand, 2010). More recent research in eating disorders has used techniques including genome-wide association studies (GWAS) to look for specific genes that play a role in eating disorders, though there are no positive findings to date.

In addition to genetic studies there has been a growing focus on the use of neuroimaging and neuropsychological studies to elucidate the underlying changes in brain function that make an individual vulnerable to develop an eating disorder (see Chapter 30). The majority of these studies have been in individuals with AN and the focus of this chapter will be on AN. While taken individually no one study can provide a comprehensive explanation of AN, the growing number of studies and the overlap in findings have allowed for the development of a number of theoretical neurobiological models of AN. This chapter will look in particular at the neuroimaging and neuropsychological findings in the areas of body image and food processing, abnormal reward processing, emotional process, and the role of the insula. Each of these models offers us insights into the etiology of AN and options for the direction of future treatments.

Food and Body Image Processing

Fear of food and abnormal body image are central to the clinical presentation of anorexia nervosa (see Chapters 2 & 8). Not surprisingly many of the neuroimaging studies carried out in eating disorders and AN in particular have focused on brain responses to illness-salient stimuli, in particular food and body image. Interestingly, regardless of the methodology, there has been considerable overlap in the findings from responses to food images and body image.

Food-related challenges have involved both the viewing of food images as well as the ingestion of sweetened fluids. While no one area of the brain has been universally identified in food-related imaging studies, a number of specific brain regions important in attention, food-related pleasure, anticipatory reward, and threat perception have been identified.

Perhaps the most robust finding has been increased activation of the right amygdala in response to both food images and food ingestion (Joos et al., 2011; Santel, Baving, Krauel, Munte, & Rotte, 2006; Uher et al., 2004; Vocks, Herpertz, Rosenberger, Senf, & Gizewski, 2011). The amygdala is central to the body's threat perception network and appears to be central to the response of individuals with AN to food.

Another common finding in response to food-related stimuli has been decreased activity in the inferior parietal lobule, which is thought to be important in food-related pleasure (Santel et al., 2006; Uher et al., 2004), coupled with decreased activity in the anterior cingulate cortex, thought to play a role in anticipatory reward (Joos et al., 2011; Vocks et al., 2011). Related to these findings of decreased food-related pleasure and decreased anticipatory reward has been the finding in a single study of decreased responses in the insula and antero-ventral striatum, areas important in both reward processing and taste (Wagner et al., 2008).

In eye tracking studies, individuals with AN have demonstrated less attention to food-related pictures than controls (Giel et al., 2011). Functional magnetic resonance imaging (fMRI) studies have demonstrated decreased activity in the brain's visual cortex or occipital lobes in response to food-related images, suggesting in individuals with AN a basis for the capacity to ignore food (Santel et al., 2006). In all of these studies the differences between controls and those with AN were most marked in the fasting state. The implications of this finding underline not only the ability of individuals with AN to ignore food but to do so during periods of fasting, overriding the body's normal response to hunger. Taken together these findings suggest that in individuals with AN the brain responds to food as threatening rather than pleasurable or rewarding and is more able to ignore food-related stimuli, particularly in a fasting state, compared to controls.

The findings in response to body image processing and AN are less clear. Findings to date indicate that, in general, individuals with AN recruit similar regions of the brain to nonaffected individuals when viewing nonself images (Sachdev, Mondraty, Wen, & Gulliford, 2008; Uher et al., 2005). Studies have suggested that when looking at nonself images individuals with AN focus more on the detail of specific body parts rather than the body as a whole and whole body movements, which would be consistent with the tendency for individuals with AN to focus on specific body parts as overweight (Urgesi et al., 2012).

Significant differences in brain activation are, however, seen in individuals with AN looking at self images. In particular, studies have shown decreased activity in the occipital cortex (visual processing), the insula and precuneus (important in the integration of both internal and external body-related stimuli and interoceptive awareness), and the prefrontal cortex (attention). These findings suggest that when viewing images of themselves, individuals with AN experience a suppression of the normal cognitive, perceptual, and emotional processing of body image. This breakdown of normal body image processing has been suggested as underlying the abnormal body image seen in AN, in which individuals respond to fears of being overweight rather than the reality of what they see when looking at themselves (Sachdev et al., 2008). Supporting this hypothesis are findings of increased activation of the amygdala in individuals with AN when compared with controls in response to words related to body image (Miyake et al., 2010).

Both in the processing of food-related stimuli and body images, individuals with AN demonstrate increased activity of the amygdala (important in fear, threat, and anxiety), an ability to focus less attention on actual food and self images, and decreased activation of the brain's visual processing regions. These findings are consistent with the clinical presentation of AN characterized by intense fear of food and weight gain and abnormal body image such that an individual's response to his or her own shape and weight is based on a fear of being overweight rather than what is seen in the mirror.

Reward Processing

The body's reward processing network is complex and involves multiple brain regions, including the insula, the basal ganglia (antero-ventral and dorsal striatum, nucleus accumbens), the anterior cingulate cortex, the orbitofrontal cortex, the mesial temporal cortex, and the ventral tegmental area. These regions function to maximize positive outcome in response to external stimuli (Frank, 2011).

Clinical descriptions of AN frequently describe high levels of anhedonia and abnormal responses to normally rewarding stimuli, in particular food. Studies using both psychometric tests and fMRI have demonstrated differences in reward processing responses to both generic reward tasks and to food in individuals with AN. It is postulated that abnormal reward processing in eating disorders is mediated by alterations in dopamine metabolism and receptor binding. Dopamine (see Chapter 30) is considered the key neurotransmitter in mediation of reward, with studies identifying altered frequency of functional polymorphisms of dopamine D_2 receptor genes (different genetic sequences coding for variations of the D_2 receptor), abnormal dopamine metabolism, and abnormal dopamine receptor binding in the striatal regions (brain's reward centers) in particular (Bergen et al., 2005; Frank et al., 2005; Kaye, Frank, & McConaha, 1999).

Studies have identified two differences in generic reward processing in individuals with AN. First, in response to gambling related tasks, underweight individuals with AN have been shown to perform more poorly than controls due to difficulty in distinguishing positive and negative outcomes (Tchanturia et al., 2007). This same study did not see these differences between healthy controls and individuals recovered from AN, though in a second study persisting fMRI abnormalities were seen in a recovered AN group compared with controls when undertaking a similar reward-based task (Wagner et al., 2007). In the recovered AN group, similar responses to wins and losses were seen in the anterior ventral striatum, highlighting the failure of the brain's reward system to respond differentially to positive and negative outcomes, as compared with the control women, who showed greater activation of the brain's reward network in response to wins compared with losses. In the recovered AN group, however, there was greater activation in the caudate, a region of the brain associated with the modulation of reward response based on assessment of the potential of strategies to elicit a reward. Put together the findings of these studies suggest that individuals with AN have an underlying deficit in the body's normal reward processing system, characterized by an inability to distinguish positive and negative outcomes, relying on a secondary and less efficient cognitively focused strategy. This deficit appears to be exacerbated by starvation, though it remains even after refeeding.

The second suggested difference in generic reward processing in individuals with AN compared with nonaffected individuals is a greater ability to delay immediate reward over the potential for future reward (Steinglass et al., 2012). This finding is not surprising, if as previously suggested, individuals with AN have difficulty in distinguishing between positive and negative outcomes and rely on a more detail-focused cognitive strategy to predict the outcome of decisions and behaviors. Applying these findings to food, coupled with the impact of starvation on exaggerating these difficulties, it is not surprising that individuals with AN have a greater capacity to restrict food intake than controls and that this increases with starvation.

As detailed previously, there have been a large number of studies of AN that have used food-related challenges (both food images and ingestion of liquid food), with abnormal reward processing a key finding. Two studies, one in response to food images (Joos et al., 2011) and one in response to chocolate milk (Vocks et al., 2011), demonstrated decreased activity in the anterior cingulate cortex in individuals with AN, which was thought to represent a decrease in anticipatory reward. This difference was most marked in the fasting state. Only one study has noted differential activity in the striatal system (the brain's key reward processing region) in response to a sucrose solution, with both decreased activity in the insula (thought to be critical in taste) and decreased activity in the antero-ventral striatum (Wagner et al., 2008), though two studies have not found decreased activity in the inferior parietal lobule, a region of the brain outside of the striatal system, thought important in food-related pleasure (Santel et al., 2006; Uher et al., 2004).

Reward processing is the key to individuals achieving maximum positive outcome in response to external stimuli. Individuals with AN appear to have a less efficient system of processing reward, relying on more complex cognitive strategies. This may explain higher levels of anxiety in response to normal environmental stimuli, less capacity for efficient decision making, and the higher degrees of perfectionism seen in AN. In addition, individuals with AN appear to have a breakdown in reward processing for food, making them more susceptible to and more able to persist with dietary restriction. Malnutrition associated with dietary restriction has been shown to further disrupt reward processing, providing a biologically based positive feedback mechanism for dietary restriction.

Emotional Processing

While current diagnostic classifications of AN focus on cognitive distortions and the physical complications of AN (see Chapter 8), there has been a growing interest in the role of deficits in emotional processing in the etiology of AN. Difficulty in identifying and describing emotions has been noted in individuals with AN since 1973 (Bruch, 1973). This construct, defined as alexithymia, has been consistently reported by clinicians and demonstrated by researchers with rates as high as 77.1% in individuals with AN compared to 6.7% in healthy matched controls (Hatch, Madden, Kohn, Clarke, Touyz, & Williams, 2010). In addition, rates of comorbid depression and anxiety in AN are high (see Chapters 15 & 54). The lifetime prevalence of anxiety disorders in AN has been reported to range between 23% and 75%, with most commonly reported figures in the 50% range. Anxiety disorders are the most commonly identified comorbid psychiatric disorders in individuals with AN and are significantly higher in this group than age-matched controls (Milos, Spindler, & Schnyder, 2004; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011; Swinbourne & Touyz, 2007; Wentz, Gillberg, Anckarsater, Gillberg, & Rastam, 2009). Comorbid mood disorders, in particular major depressive disorder, are commonly seen in AN. Average lifetime prevalence of major depressive disorder in individuals with AN has been reported as 40%, or approximately double the risk of that in age-matched controls (Godart et al., 2007).

Researchers have investigated abnormal emotional processing using a variety of tools, including psychometric tests, measures of physiological arousal in response to threatening and illness salient stimuli, and neurophysiological investigations such as event-related potentials (ERP) and functional neuroimaging. The most commonly used measure for studying emotional processing in AN has been the use of emotional faces, typically depicting six basic emotions: anger, disgust, fear, sadness, surprise, and happiness. Studies have shown that relative to controls both adolescents and adults show deficits in recognizing facial expressions of emotions (Hatch, Madden, Kohn, Clarke, Touyz, & Williams, 2010). Similar deficits have also been demonstrated in women at high risk for developing AN, suggesting deficits in recognizing emotions may be a risk factor for developing AN rather than occurring as a result of the disorder and associated malnutrition (Jones, Harmer, Cowen, & Cooper, 2008). Further, some studies have shown that there are deficits in facial emotion recognition, predominantly difficulty in accurately recognizing happy or neutral faces rather than faces depicting negative emotions (Hatch, Madden, Kohn, Clarke, Touyz, Gordon, & Williams, 2010; Jones et al., 2008). The tendency to misinterpret positive emotional feedback, coupled with a sensitivity to negative emotions, is consistent with many clinical descriptions of AN.

Investigators have looked at three different types of evidence to understand the mechanisms, timing of cognitive responses to emotional stimuli (unconscious or conscious), and brain

regions involved in the disturbance in emotional processing seen in AN. Studies have demonstrated arousal dysfunction in AN, with aversive responses to positive stimuli (Friederich et al., 2006) and decreased responses to negative stimuli (Tchanturia et al., 2007). Even in cases where individuals with AN have responded with increased physiological arousal to stress-inducing tasks such as public speaking, unlike controls, they have not reported changes in their feeling state (Hatch, Madden, Kohn, Clarke, Touyz, & Williams, 2010). These abnormal responses to positive and aversive stimuli and the disjunction between physiological changes and subjectively reported emotion further underline the evidence of abnormal emotional processing in AN that is independent of illness-salient stimuli such as food and body image.

While the above evidence supports the presence of a global emotional processing deficit in AN, it is important to recognize that emotional processing is not a single process but involves a number of steps including both unconscious and conscious processing of emotional cues. One such model of emotional processing is the Integrate Model proposed by Hatch, Madden, Kohn, Clarke, Touyz, and Williams (2010). It proposes that the first response to emotional cues involves an early automatic response, occurring outside of conscious awareness within 200 ms of presentation. This response is seen as a highly conditioned response designed to distinguish danger from reward. This early automatic response they propose is followed by a conscious process of identifying and attending to these emotions in order to make a voluntary response to the emotional cue. In a final step they propose that individuals with AN regulate their behavior over the short to medium term in response to their early emotional processing to minimize danger and maximize reward, with eating disorder behavior seen as a response to earlier breakdowns in this system (Hatch, Madden, Kohn, Clarke, Touyz, & Williams, 2010).

In order to look at both unconscious and early conscious responses to emotional cues, Hatch, Madden, Kohn, Clarke, Touyz, Gordon, et al. (2010) measured event-related potentials (ERP) (a summation of electrical activity in neuronal pathways) in response to emotional faces. They found reduced electrical activity in patients with AN in response to emotional faces at 120 ms. This finding was present with all emotional faces both before and after refeeding, suggesting an early unconscious deficit in emotional processing occurring regardless of emotional valence and nutritional state. They proposed that the reduction in response in AN represented a loss of ability to form an early unconscious, visual gestalt of emotion that could influence subsequent conscious emotional responses and behavioral regulation. They further argued that the presence of this difference independent of nutritional state indicated that this is a trait marker of AN. These differences were noted to be most marked in the temporo-occipital regions, that is, the regions of the brain most commonly associated with memory and emotion (Hatch, Madden, Kohn, Clarke, Touyz, & Williams, 2010).

The neuroimaging literature in this area is less conclusive. fMRI studies have clearly demonstrated differences in metabolism in individuals with AN in brain regions associated with emotional processing (amygdala, insula, and anterior cingulate gyrus) in response to both food and body image stimuli (Friederich et al., 2010; Joos et al., 2011; Miyake et al., 2010; Vocks et al., 2011). Despite this, in the one study to use fMRI to look at brain response to emotional faces, no difference was noted in patterns of brain activation between healthy controls and individuals who had recovered from AN (Cowdrey, Harmer, Park, & McCabe, 2012).

In summary, there is a growing body of evidence suggesting abnormalities in emotional processing in AN. Evidence suggests that this is not only related to disease-salient stimuli such as food and body image but is more global in nature. While there is some contradictory evidence, it appears that, though emotional processing is impacted by malnutrition, deficits in emotional processing persist even with nutritional recovery. In the only study to look at the timing of

emotional response, it has been suggested that there is a failure of unconscious emotional processing, predominantly in the temporal lobes, that impacts downstream responses and behavior in individuals with AN. If this is the case it may explain why psychological therapies that presuppose conscious awareness of feelings have been less successful in this group of patients.

Anorexia Nervosa and the Insula Hypothesis

The insula hypothesis, first proposed by Nunn and colleagues in 2008 (Nunn, Frampton, Gordon, & Lask, 2008), proposes AN as a disconnection model with failure of the different regions of the brain processing sensory stimuli, emotional responses, and memory to appropriately connect with each other, similar to models put forward in schizophrenia and dyslexia (Nunn, Frampton, Fuglset, Torzsok-Sonnevend, & Lask, 2011). Central to this model is the role of the insula in connecting those parts of the brain that deal with adaptation to the external environment to those parts that are responsible for internal homeostasis.

The insula is located deep in the sylvian fissure between the junction of the anterior and posterior cerebral hemispheres. It lies below the frontal lobes, medial to the temporal lobes, and in front of the parietal lobes. Thus, the insula is covered by these three regions of the brain. The insula is divided into a larger anterior segment and a posterior segment. It is connected to nearly all major regions of the brain, including the frontal (short-term memory, planning, motivation and attention), temporal (memory and language), and parietal lobes (sensory integration), the limbic system (emotion processing), the basal ganglia (movement and mood), the striatum (reward), and the thalamus. The only parts of the brain that the insula does not have direct contact with are the occipital lobe and the cerebellum. It is this connectivity that is central to the insula hypothesis of AN. Namely, while nearly all of the aforementioned regions of the brain have been individually identified in different neuroimaging studies in AN, the idea that there are underlying abnormalities in each of these areas seems less likely than dysfunction in the single region of the brain (the insula) that links these various regions (Nunn et al., 2008, 2011).

Of the many functions of the insula, a number of those associated with the anterior insula are particularly relevant to eating disorders and especially AN. These include the regulation of taste and appetite, including food reward and disgust, integration of thoughts and feelings, interoceptive awareness, monitoring of the body state, and regulation of the autonomic nervous system (Nunn et al., 2011; Wagner et al., 2008). It is possible to group these functions into three broad areas: response to food, awareness of internal and external body state, and emotional processing. Dysfunction in these three areas serves to explain many of the key symptoms and behaviors in AN such as food refusal and food-related anxiety, particularly to high-energy foods, abnormal body image processing, hyperarousal and comorbid anxiety, depression, and alexithymia. While each of these three areas has been previously dealt with in this chapter, it is worth looking at the evidence of insular dysfunction as a possible way to integrate these previous findings.

The insula is critical in regulating appetite through its role in connecting the frontal lobes with the hypothalamus (Nunn et al., 2011). Additionally the insula acts as a primary gustatory cortex, responding to taste and the physical quality of food activated by both the intensity and valence of pleasant and unpleasant tastes (Wagner et al., 2008). Animal studies have shown that lesions in the insula result in loss of fine tuning of feeding responses to salient food items, including lack of appropriate response to highly palatable foods, foods associated with sickening agents, and satiety.

Functional MRI studies in humans have demonstrated differences in insula activation in individuals with AN compared with controls. Wagner and colleagues (2008) showed that,

compared to healthy controls, women with AN had a reduced insular response to the blind administration of sucrose or water. Downstream areas of the brain, including the ventral striatum, thought to play a critical role in the consumption of highly palatable, high-energy foods, also showed decreased activation in response to administration of sucrose solutions in women with AN. This suggests a breakdown of the normal reward response to these foods in AN. In addition, unlike healthy controls for whom self-reported ratings of pleasantness of taste to the sucrose solution were directly related to insular activation, there was no connection between ratings of pleasantness of taste and insular activity in women with AN. This indicates not only a lack of reward value for highly palatable food but also an inability to differentiate more palatable from less palatable food. Coupled with high levels of self-reported disgust to images of high-energy foods (Uher et al., 2004), the lack of reward in response to food may explain both food restriction and avoidance of high-energy foods.

In addition to its role in the processing of taste, the anterior insula plays a central role in the regulation of the autonomic nervous system (ANS). ANS regulation includes the inhibition of the amygdala and its output to the central nervous system, as well as a central stimulator to the parasympathetic nervous system. Dysfunction in this system would account for the hyperarousal seen in AN, particularly in response to food stimuli (Nunn et al., 2011).

Abnormalities in insular function have also been noted in fMRI studies of patients with AN in response to images of self and others. Study findings have demonstrated that patterns of brain region activation are similar in participants with AN and healthy controls when looking at nonself images. However, when individuals with AN look at images of themselves they fail to activate regions important in body image processing, including the insula and the occipital lobe (Sachdev et al., 2008). Given the insula's role in monitoring the "body state"(Nunn et al., 2011) by linking internal and external feeling states, failure of insular function would provide a plausible explanation of both the abnormal body image and misperception of visceral feedback such as feelings of fullness.

An increasing number of studies have identified differences in insular function between individuals with AN in response to food and body image related challenges. In addition, dysfunction of known insular functions overlaps with core psychopathology in anorexia nervosa. While far from being proven, the "insula hypothesis" provides a parsimonious and testable neurobiological explanation of AN.

Conclusions and Future Directions

While it is generally accepted that eating disorders occur in the context of complex gene–environment interactions (see Chapters 28 & 67), neuroimaging studies over the past two decades are providing increasing support for dysfunction in key brain processes in the genesis of eating disorders. While no single finding or study can clearly explain the etiology of or pathology underlying particular eating disorders, findings can be increasingly grouped into a number of key theoretical models including:

- abnormalities in the reward processing to both generic and illness-salient stimuli, in particular food;
- abnormalities in emotional processing, again both generic emotional processing and threat-related processing of food and body image;

- failure of the brain's normal mechanisms of body image processing, particularly self-image processing;
- an insula disconnection model.

Grouping findings into theoretical frameworks is important in that it allows future research to be more systematically targeted to underlying brain networks rather than becoming a "New Age" phrenology focusing on individual brain regions as being responsible for specific eating disorder symptoms. It is also important as it allows theories to be disproved so resources can be devoted into more fruitful areas of research. Finally, the ability to link neuropsychiatric findings into coherent narratives is critical in guiding future treatment development and interventions, informing and engaging our patients and their families and carers, and critical in reducing blame and stigma associated with eating disorders.

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18

Cognitive-Behavioral Theory of Eating Disorders

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Cognitive-behavioral models of anorexia nervosa (AN) and bulimia nervosa (BN) were proposed independently more than 30 years ago (Fairburn, 1981; Garner & Bemis, 1982). At the time, their convergent explanations of restrictive and bulimic behavior differed markedly from dominant views; over subsequent decades, they have had a strong influence on how eating disorders (EDs) are conceptualized and treated, most notably through the success of cognitive-behavioral therapy (CBT) for BN (see Chapter 56). According to both models, the complex symptom pictures seen in these apparently dissimilar disorders emerge from a common core disturbance: the overvaluation of weight, shape, and dietary control. While cognitive-behavioral theory and treatment continue to evolve, subsequent versions conform closely to the original proposals, varying principally in emphasis and the elaboration or addition of specific elements. This chapter will outline the initial models separately, noting how each has developed over time, then describe the merging of both into a "transdiagnostic" account of EDs (Fairburn, Cooper, & Shafran, 2003). The strong evidence base for many postulates of cognitive-behavioral theory will be reviewed and several areas of persistent deficiency identified.

Cognitive-Behavioral Models of AN

The first detailed cognitive-behavioral accounts of AN focused on the proximal variables that initiate and maintain symptoms rather than remote causal factors (Bemis, 1983; Garner & Bemis, 1982, 1985; Garner, Garfinkel, & Bemis, 1982; see also Slade, 1982). AN was viewed as a multiply determined disorder that could arise from interactions among different predisposing variables within and across patients. No assumptions were made about pathogenic family patterns or childhood experiences, although neither was excluded as a possible contributor in individual cases.

The distal vulnerability factors featured most prominently in cognitive-behavioral models are the cluster of temperamental traits linked to AN. This emphasis is by no means unique. The association between AN and characteristics such as perfectionism, obsessionality, and

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hypersensitivity was discerned by early clinical observers (Bruch, 1978; DuBois, 1949) and confirmed through subsequent research (see Chapter 32). Because virtually all models implicate these traits in the etiology of AN, the growing evidence of their significance and genetic basis cannot be claimed as specific support for one interpretation. Cognitive-behavioral models have contributed several more distinctive hypotheses about how these general traits may interact with sociocultural influences to produce ED symptoms, as well as their role in facilitating self-starvation and setting its reward value.

In the interval preceding onset of symptoms, these anxious, self-critical individuals usually encounter circumstances that threaten their precarious sense of competence and control. The episode may be precipitated by an external stressor such as interpersonal conflict or entering a new school, or by developmental changes related to physical maturation, sexuality, or increasing expectations for independence. Concern about eating and weight may be triggered by teasing by peers, comments from parents, coaches, or physicians, classroom instruction on "healthy eating," or rapid weight gain (Smolak & Levine, 1996). In other instances, however, diffuse distress settles on the size and shape of the body in the absence of specific prompts. The idea that personal worth can be enhanced by eating lightly and weighing less is well established in contemporary culture, and especially salient to the demographic groups at highest risk for EDs (see Chapter 29). In addition to these positive social connotations, control over eating and weight may hold particular appeal to individuals with the personality profile linked to AN. Hard-core, long-haul dieting requires and rewards precisely those traits they have to excess: persistence, attention to detail, conscientiousness, and the ability to suppress or defer hedonic impulses.

If it is easy to understand why someone might turn to dieting as a self-help solution during periods of distress, the transition to severe AN seems to defy rational explanation. In both popular and scientific accounts, the disorder is frequently characterized as "mysterious" and "enigmatic." According to the cognitive-behavioral model, however, anorexic symptoms do make sense in the context of both the beliefs these individuals hold and the functional relationships that develop to sustain self-starvation. Most of what appears to be irrational and purposeless behavior derives predictably from the central premise that weight is the pre-eminent index of personal worth. Once formed, this dominant idea influences people with AN to engage in stereotypic eating behaviors, to adopt elimination tactics such as laxative abuse and strenuous exercise, to respond to increasingly eccentric reinforcement contingencies, to process information through predictable cognitive biases, and, eventually, to be affected by starvation-induced psychological and physiological changes—all of which serve to strengthen the underlying premise. A host of secondary beliefs about food and the determinants of weight loss or gain start to devolve from the central tenet, and ruminations about intake and output come to dominate most of the individual's private experience.

Effects of Starvation

A solid background in the phenomenology of starvation is viewed as essential to decoding AN (Garner, 1997). Patterns that emerge whenever normal people are chronically deprived of calories cannot be construed as ED-specific psychopathology— or even as abnormal—when they appear in underfed, underweight people with AN.

Data from diverse contexts attest that behavior is profoundly and predictably altered by food deprivation (Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950; Vitousek, Manke, Gray, & Vitousek, 2004). Most of these changes represent adaptive responses to the stress of starvation, which evolved to reduce energy expenditure and/or focus the hungry organism's attention on

securing something to eat. Starved individuals become depressed, irritable, perseverative, self-centered, socially withdrawn, and sexually indifferent. In addition, they develop characteristic peculiarities in eating-related attitudes and behaviors—notably, a towering preoccupation with food and an obsessive interest in minutiae about how and when to conserve or consume it. Familiarity with other instances of human starvation clarifies that many of the strange things AN patients think, feel, and do are direct effects of eating and weighing too little.

Clearly, starvation does not account for all features seen in the presenting picture of AN; however, it substantially reduces the number for which disorder-specific explanations are required. It also provides the context for understanding AN symptoms that are not fully explained by starvation alone. For example, the rigidity and obsessiveness that appear when normal individuals are food-deprived can interact with patients' pre-existing beliefs and stable traits in unfortunate ways (Fairburn, Shafran, & Cooper, 1999; Vitousek & Hollon, 1990). Ideas about the importance of weight become increasingly resistant to change; amplified obsessionality supports more drastic restraint. Moreover, individuals who were temperamentally avoidant and detail-oriented prior to onset may welcome starvation effects that others would find aversive, such as the blunting of emotional experience and the narrowing of concerns.

Biased Information Processing

Cognitive-behavioral theory posits that ED individuals develop organized cognitive structures (called schemas or schemata) concerning weight and its implications for the self (Vitousek & Hollon, 1990; Williamson, Muller, Reas, & Thaw, 1999). Schematic processing can contribute to symptom maintenance in a relatively automatic fashion, by shaping the ways in which information is perceived, interpreted, and remembered. Biased processing is not in itself pathological; indeed, because people can attend to and retain only a subset of the data to which they are exposed at any moment, all information processing is necessarily selective, influenced by learning history, mood state, and current concerns. New parents notice more child-related stimuli in the environment; anxious individuals are primed to detect subtle threat cues and to construe neutral or ambiguous situations as dangerous.

In a similar manner, ED symptoms may be maintained in part by automatic biases toward weight and food cues. Patients may attend selectively to signals of "fatness" (such as inner thighs touching), attach weight-related meaning to a variety of unconnected events (e.g., attributing a social snub to weight gain), and ignore or distort conflicting information (e.g., rejecting evidence of their own thinness) (Vitousek & Hollon, 1990). Starvation effects also skew cognitive processing, highlighting the salience of food and blocking attention to social cues and other environmental stimuli. As a result of these attentional and interpretive biases, the everyday experience of ED individuals seems to validate their beliefs about the centrality of eating and weight issues, while isolating them from alternative perspectives.

Positive and Negative Reinforcement

Although some of the consequences that maintain AN are automatic and unmotivated, the cognitive-behavioral model proposes that symptoms also serve valued functions for these individuals. Three categories of instrumental functions are identified:

- those based on *negative reinforcement*, including the avoidance of unpleasant affect or pressures to succeed in other domains, as well as relief from conditioned anxiety about feared foods and "fatness";

- those based on *positive reinforcement*, such as feelings of self-control, virtue, and accomplishment;
- *simplifying* or *organizing* functions that contain elements of both positive and negative reinforcement for individuals who are daunted by ambiguity and prefer effortful, rule-bound pursuits, such as the rigorous management of eating and weight.

To a greater extent than other models, cognitive-behavioral accounts stress the positively reinforcing and simplifying functions of AN, postulating that these explain its most unusual features—as well as its resistance to change—better than the avoidance-based patterns that are also influential.

Individuals with AN are unquestionably afraid of eating and weighing "too much," and their relief when they avoid these dreaded outcomes contributes to symptom maintenance. Yet patients with AN are not more fearful than patients with anxiety disorders, most of whom benefit rapidly and substantially from exposure therapy and medication—interventions that appear to be much less effective in the treatment of AN (see Chapter 59). Indeed, unlike individuals with obsessive-compulsive disorder (OCD) or panic disorder, many anorexic patients seem to be invested in retaining their distress. This dynamic is evident in the cultivation of anxiety about fattening foods, as well as in the alarm many feel if their fears start to erode. As one of our weight-restored patients observed: "I feel like a shift has happened where I'm not quite as uptight and worried [about eating and weight] as I was before—and that really scares me."

Moreover, individuals with AN are not simply afraid, nor are they merely relieved when feared outcomes are avoided (Bemis, 1983). In association with successful restriction, they often describe feeling elated, powerful, and proud. Some use striking metaphors to convey the intensity of the experience:

> It's like winning the Nobel Prize or something. It's like you get a kingdom or become a goddess. (Patient quoted in Way, 1993, p. 69)

> I'm proud of my stoic, Spartan existence. It reminds me of the lives of the saints and martyrs … [AN] has become the most important thing I've ever done. (Ciseaux, 1980, p. 1468)

Phenomena such as pride in symptoms, a sense of "specialness," moral certitude, and the cultivation of fear are not well explained by an avoidance paradigm. They do make sense, however, when dietary restriction and weight control are recognized as effortful, highly valued behaviors that are maintained by elements of both approach and avoidance. Although culture supplies the positive valence associated with thinness and restraint (see Chapters 21, 29, & 67), consensual validation does not extend to equating a body mass index (BMI) of 14 (e.g., 81.5 lb [~37 kg] at 5′4″ [1.63 m]) with Nobel Prizes or divinity. According to the cognitive-behavioral model, the potent reinforcement value of AN depends on interactions between predisposing traits, general beliefs, and the strategies required to sustain starvation.

Cognitive-Behavioral Therapy for AN

As the distinctive features of AN appear to make a disproportionate contribution to the challenges that both patients and clinicians face in treatment, CBT is organized around efforts to address them (see Chapter 56). Because of the interaction between physical and psychological elements, efforts to restore normal nutritional status are essential to the clinical agenda (see Chapter 61).

Because of the valued quality of symptoms, considerable attention is paid to engaging patients' interest in the prospect of change and translating it into action. CBT for AN specifies a blend of psychoeducational, experimental, functional, and values-related elements designed to enlist patients in re-examining the relationship between anorexic symptoms and their own goals and ideals. In the later stages of therapy, attention shifts to more general aspects of self-concept.

Development of the Cognitive-Behavioral Model of AN

Subsequent discussions by the originators of the model represented elaborations of specific elements rather than reformulations of the basic approach. Vitousek and Hollon (1990) discussed the role of schematic processing and hypothesized a linkage between a cognitive style that favors certainty and patients' investment in symptoms. Other papers postulated a relationship between ED psychopathology and general views of the self (Garner, Vitousek, & Pike, 1997; Vitousek & Ewald, 1993) and highlighted motivational issues (Vitousek & Gray, 2005; Vitousek, Watson, & Wilson, 1998).

Several cognitive-behavioral models of AN have been described by other ED experts. Most are at least broadly consonant with the initial account, proposing shifts in emphasis or identifying additional contributing mechanisms. Some suggested that the original model was too narrow, paying insufficient attention to interpersonal variables and "deep" aspects of the self (Leung, Waller, & Thomas, 1999); others considered the model too broad, allocating unnecessary attention to these issues, and recommended a more focused approach (Fairburn et al., 1999). Fairburn et al. (1999) also highlighted the significance of control over eating as the predominant focus for a subset of patients and elaborated the role of perfectionism. Other models advocate increased attention to "deeper" core beliefs and self-schemas, interpersonal variables, and/or the emotion-regulating functions of AN symptoms (Schmidt & Treasure, 2006; Waller, Kennerley, & Ohanian, 2004; Wolff & Serpell, 1998).

Cognitive-Behavioral Models of BN

The cognitive-behavioral analysis of BN was developed by Fairburn (1981, 1985, 1997; Fairburn, Cooper, & Cooper, 1986; Fairburn, Marcus, & Wilson, 1993). As noted earlier, cognitive-behavioral models of AN and BN hold that both are "essentially cognitive disorders" (Fairburn, 1997, p. 210) deriving from a distinctive core psychopathology: overvaluation of weight and shape and their control. Analyses of BN emphasize that almost all of its key features are linked to this central disturbance, including stringent efforts to limit food intake, use of various forms of compensatory behavior, and development of body checking behaviors.

Binge eating is the one diagnostic feature of BN that does not follow directly from the core psychopathology; rather, it is viewed as a byproduct of patterns that do. Binge episodes result from patients' efforts to practice extreme, inflexible forms of dietary restraint (Fairburn, 1997, 2008; Fairburn et al., 1986). Like patients with AN, these individuals self-impose strict rules specifying the amounts and kinds of foods they may eat at designated times and under certain conditions. Compared to their restricting AN counterparts, however, those who develop BN experience more frequent failures in following the regimen and/or respond differently to transgressions. Even minor violations are viewed as losses of control and signs of personal weakness. People with BN react by abandoning all dietary constraints. Binge episodes intensify concern about eating and weight, leading to redoubled efforts at restraint and more experiences

of failure. Patients attempt to undo these lapses by vomiting or taking laxatives or diuretics. Their belief that such methods eliminate the calories consumed is misguided (Fairburn, 1995). Nonetheless, compensatory tactics further reinforce the bulimic cycle by lowering the perceived cost of binge eating and temporarily relieving anxiety (Fairburn, 2008).

Cognitive-Behavioral Therapy for BN

Fairburn's original CBT protocol for BN was designed to address each of the key elements identified in the model. The standard intervention was a structured, manualized approach focused on the establishment of regular eating patterns, reintroduction of avoided foods, interruption of the binge-purge cycle, and correction of inaccurate beliefs about weight, eating, and compensatory behaviors (Fairburn, 1981; Fairburn et al., 1986). Compared to the CBT model described for AN, less emphasis was placed on cognitive techniques, perceived symptom functions, or more general views of the self.

Development of the Cognitive-Behavioral Model of BN

The initial model included no speculations about individual characteristics or life experiences that increase susceptibility to BN. Moreover, it gave little attention to variables unrelated to eating and weight that could influence symptoms after onset, except to note that triggers for binge episodes often involve negative mood states and/or interpersonal difficulties. As with AN, a close focus on proximal maintaining variables and presenting symptoms rather than remote causes and "deeper" concerns is characteristic of cognitive-behavioral interventions in general. Even within this framework, however, Fairburn's model of BN was viewed by some CBT experts as excessively stripped down (Garner et al., 1997; Hollon & Beck, 1994; Meyer, Waller, & Waters, 1998; Vitousek, 1996). CBT for depression and anxiety—as well as for AN—usually extends to a wider range of topics, such as interpersonal problems and low self-esteem, when these are deemed relevant to individual cases.

It should be noted that because little was known about BN when the initial theory was outlined, there were few grounds for evidence-based hypotheses about vulnerability factors or exacerbating conditions. Moreover, the focused treatment derived from this minimalist model proved effective in reducing or eliminating symptoms in many BN patients (see Chapter 56). In response to accumulating knowledge about BN and the recognition that some individuals did not benefit from exclusively symptom-focused CBT, revisions of the model included a wider range of contributing variables.

- **Perfectionism and low self-esteem.** The first revision concerned tendencies toward negative self-evaluation and high standard-setting in some patients (Fairburn, 1997; Fairburn et al., 2003; Wilson, Fairburn, & Agras, 1997). In this subgroup, fundamentally negative views of the self can be construed as "a second core cognitive characteristic" (Fairburn, 1997, p. 211). Such patients may develop particularly intransigent ED patterns that do not resolve unless their more general beliefs are addressed.
- **Mood regulation.** Another change was increased attention to the role of affect. Observers from within and outside CBT noted that for some patients, ED behaviors are maintained principally or in part by their mood-regulating functions (Heatherton & Baumeister, 1991; Meyer et al., 1998; Stice, 2002). For most individuals with BN, negative mood states lower the threshold for interruptions in dietary restraint (Fairburn et al., 1986). In a subset, however,

marked sensitivity to emotional states and intolerance for intense affect dominate the clinical picture (see Chapters 54 & 57). Bulimic behaviors may be used by these patients to regulate mood in a manner that parallels their reliance on other harmful coping tactics such as substance abuse and self-injury. The enhanced model proposed by Fairburn et al. (2003) specified supplementary techniques for addressing these more complex mood-related contributors.

- **Interpersonal processes.** The revised analysis of BN also included more material on interpersonal factors (Fairburn et al., 2003). Since the model was proposed, substantial evidence had amassed on the influence of interpersonal and sociocultural variables (Keel & Forney, 2013; see also Chapters 21 & 31). Moreover, the demonstrated effectiveness of interpersonal psychotherapy for BN (see Chapter 62) suggested that CBT might be enhanced by attending to interpersonal domains that appeared to affect symptoms in certain cases.

Fairburn's conceptual and treatment models have dominated cognitive-behavioral perspectives on BN. Several alternative accounts have been proposed, such as a model by Cooper, Wells, and Todd (2004) that includes hypotheses about the development of the disorder, and an analysis by Waller et al. (2004) that emphasizes self-schemas and emotional avoidance. In addition, some recent proposals blend elements of the cognitive-behavioral model with other theoretical and therapeutic principles (Wonderlich et al., 2011).

Transdiagnostic Cognitive-Behavioral Models of EDs

Over the past three decades, many experts have proposed that EDs should be merged into a single diagnostic category, arguing that the high percentage of unclassifiable cases, the instability of subtype diagnoses, the similarity of symptoms and distribution patterns, and the evidence of cross-transmission of familial risk all affirm the operation of common mechanisms (Beumont, Garner, & Touyz, 1994; Holmgren, Humble, Norring, & Roos, 1983). Cognitive-behavioral theorists have consistently advanced this perspective (Fairburn & Garner, 1988), while noting that some aspects of the treatment approach vary in important ways according to patients' current weight status and motivation for change (Garner et al., 1997).

In 2003, Fairburn and colleagues proposed that this conceptualization be formally represented through adoption of a transdiagnostic model for understanding and treating EDs (Fairburn, 2008; Fairburn et al., 2003). The transdiagnostic theory encompasses the key processes identified in cognitive-behavioral accounts of AN and BN (i.e., overvaluation of eating, weight, and shape expressed in a variety of specific behaviors) and extends this analysis across all forms of EDs. It also specifies four maintaining mechanisms that interact with the central psychopathology in some individuals (perfectionism, core low self-esteem, mood intolerance, and interpersonal difficulties), proposing that a "broad" form of CBT should be used to address these problems when they are judged likely to present impediments to change.

Evidence Base for Cognitive-Behavioral Models of EDs

Cognitive-behavioral analyses of the EDs have generated multiple lines of quantitative and qualitative research. Virtually all of the predictions investigated to date have been affirmed, and the strong evidence favoring CBT for BN provides additional indirect support; however, a number of important elements of the conceptual model remain untested more than 30 years after they were initially proposed.

Nature and Centrality of Concerns about Weight, Shape, and Eating

Despite the prevalence of weight concern and dieting in the general population, studies confirm that people with AN and BN are distinguishable from normal individuals in the content, intensity, and absolutism of their beliefs about eating and weight. These findings are based on a variety of assessment strategies, including semistructured interviews, attitude questionnaires, self-statement inventories, and thought sampling. Scores on measures of eating, weight, and shape concern have been shown to correlate with other ED symptoms and with severity, to decrease over the course of treatment, and sometimes to predict symptom persistence, treatment response, and relapse (Cooper, 1997, 2005; Vitousek, 1996; Williamson, White, York-Crowe, & Stewart, 2004). Risk factor research consistently identifies weight concerns (as well as negative emotionality and perfectionism) as powerful predictors of ED symptoms (Keel & Forney, 2013; Stice, 2002).

General Beliefs and Self-Schemas

Individuals with EDs also differ from normal and dieting comparison groups in the endorsement of negative self-schemas and core beliefs unrelated to eating, weight, and shape (Cooper, 2005). Perfectionism is strongly associated with both AN and BN, and appears to be a predisposing factor for the emergence of ED symptoms (Shafran, Cooper, & Fairburn, 2002; Stice, 2002). Such maladaptive beliefs, negative views of the self, and harsh standards for self-evaluation are associated with a wide range of other psychiatric disorders. This lack of specificity does not rule out an important role for such constructs, which presumably interact with other influences to shape diverse symptom patterns. In most instances, however, proposed causal relationships between these phenomena and the specific psychopathology of EDs have not been established.

Information Processing Biases and Styles

The prediction that ED patients will show systematic biases in processing food- and weight-related information has been extensively investigated and generally affirmed (Brooks, Prince, Stahl, Campbell, & Treasure, 2011; Cooper, 1997, 2005; Dobson & Dozois, 2004; Lee & Shafran, 2004). Effects are stronger for attentional biases, measured by paradigms such as the modified Stroop, than for selective memory effects, and variable findings have been obtained for biases in judgment. Biased responding is more consistently demonstrated in clinical samples than in dieting, restrained, weight-concerned, or short-term food-deprived comparison groups. AN and BN may be associated with different patterns of selective attention to food and/or weight cues. In general, the evidence suggests that information processing biases decrease or disappear as a function of treatment (Shafran, Lee, Cooper, Palmer, & Fairburn, 2008). There is also inconsistent evidence of biased responding to general threat cues and emotional stimuli (Cooper, 2005; Treasure & Schmidt, 2013; Zhu et al., 2012).

Information processing research has become substantially more rigorous over time with reference to controlling for the properties of stimuli, examining contextual cues and other task parameters, and including more instructive comparison groups. Nonetheless, concerns about the conceptual and clinical significance of this line of inquiry have persisted (Cooper, 1997, 2005; Lee & Shafran, 2004; Vitousek, 1996). While most findings are consistent with the cognitive-behavioral model, researchers have not yet assembled a convincing case for some of

its most distinctive hypotheses, such as the postulate that biased information processing plays an important role in maintaining ED symptoms, rather than simply reflecting current concerns. The use of new investigative techniques, including those examining the neural correlates of exposure to food and weight cues, may help to clarify how these processes operate (Friederich, Wu, Simon, & Herzog, 2013; Zhu et al., 2012). It should be noted, however, that such methods pose formidable interpretive problems of their own.

Cognitive-behavioral models contend that AN symptoms are valued and sustained in part because of the potent schematic properties of both an obsessive focus on weight and starvation effects. These serve to narrow the attentional field, reduce ambiguity, facilitate judgments and predictions, and provide a parsimonious set of premises for guiding behavior and gauging self-worth (Merwin et al., 2011; Vitousek & Hollon, 1990). Recent neurocognitive research has provided data supporting some of these premises. AN patients are characterized by extreme attention to detail, set-shifting difficulties, "top-down" processing, and high levels of self-control in non-ED domains (Steinglass et al., 2012; Tchanturia et al., 2012; Wagner et al., 2007). Most of these features are especially pronounced in acutely ill adults, but some persist in attenuated form after weight restoration. It is unclear whether these tendencies precede or follow starvation, and their contributions to onset and maintenance remain conjectural.

Attitudes Toward ED Symptoms and Recovery

Researchers have recently begun to solicit patients' own views of their symptoms and the prospect of change. In the case of a disorder marked by "overvaluation" and resistance to change, knowing what individuals think they value and want to retain is likely to be instructive, even though a complete and accurate account of functional relationships cannot be expected from these (or any other) informants.

Qualitative studies confirm that patients ascribe multiple benefits to AN, including avoidance-based advantages (such as a sense of "safety") and positive assets (such as a sense of "specialness") (Nordbo, Espeset, Gulliksen, Skarderud, & Holte, 2006; Serpell, Treasure, Teasdale, & Sullivan, 1999). Individuals with BN tend to identify negatively reinforced functions and to attribute relatively more costs to the disorder (Serpell & Treasure, 2002). Similar patterns differentiate AN and BN groups on self-report measures (Gale, Holliday, Troop, Serpell, & Treasure, 2006). As predicted, individuals with AN are also more likely to construe their symptoms in positive terms compared to other psychiatric groups, including samples with agoraphobia, specific phobia, OCD, alcohol dependence, stimulant drug abuse, and BN (Vitousek, Gray, & Goodyear, 2013).

In the past 15 years, there has been a surge of research activity on the assessment and enhancement of motivation for change (see Chapter 63). The emphasis on motivational variables is certainly consonant with the cognitive-behavioral perspective; however, much of the work in this area has been based on stage models and the construct of "readiness," often examining the utility of brief, discrete interventions designed to boost motivation prior to treatment. Neither the emphasis on "readiness" nor the use of pretreatment motivational modules reflects the CBT approach (Vitousek & Gray, 2005; Wilson & Schlam, 2004).

Treatment Response

The success of a treatment does not validate the theory on which it is based, although some of its assumptions may be supported indirectly. That is the case for Fairburn's focal CBT, which is closely bound to a concise model of symptom maintenance in BN. Manual-based CBT

produces greater symptom reduction more rapidly and reliably in a higher percentage of BN patients than any other psychological or pharmacological modality (Wilson, Grilo, & Vitousek, 2007; see also Chapter 56). The disorder remits completely in one third to one half of treated cases, and many of the remainder improve. CBT also reduces other psychiatric symptoms, improves general functioning, and increases self-esteem. Of particular relevance to the theoretical model, decreases in dietary restraint partly mediate reductions in bulimic behavior (Wilson, Fairburn, Agras, Walsh, & Kraemer, 2002; see also Chapter 24), while persistent overvaluation of weight and shape after behavioral symptoms cease predicts subsequent relapse (Fairburn, Peveler, Jones, Hope, & Doll, 1993; see also Chapters 55, 56, & 64).

The picture is quite different with reference to the treatment of AN. Although most briefly ill adolescent patients respond well to several tested treatments, no pharmacological or psychological intervention, including CBT, has achieved comparable success in adult patients. The database is limited because AN has proven exceptionally challenging to study as well as to treat (Wilson et al., 2007; see also Chapter 12). Several treatment trials have produced uninterpretable findings or broken down completely—often due to astronomical rates of treatment refusal and dropout from the weak interventions to which CBT was being compared (Wilson et al., 2007).

Recent uncontrolled case series using the transdiagnostic CBT protocol suggest that the approach is at least moderately effective for adults with AN (Byrne, Fursland, Allen, & Watson, 2011; Fairburn et al., 2013). Because of the high rates of dropout and withdrawal in the AN group, however, intent-to-treat analyses repeat the general pattern of less favorable outcomes for AN relative to other EDs, despite the longer course of treatment specified for these patients.

The poor-to-middling success rate of all tested treatments for established AN is clinically troubling. From a cognitive-behavioral perspective, however, it is predictable and instructive with reference to the valued nature of core AN beliefs and behaviors. Ironically, a particularly convincing refutation of the cognitive-behavioral analysis would be the discovery of any intervention that rapidly and reliably reversed AN in virtually all of the patients who received it—unless the method worked similarly well when applied to the modification of other highly valued patterns such as religious extremism or ultrarunning. Yet while the model predicts that no spectacularly effective treatments will emerge, from within or outside CBT, it also assumes that a clearer understanding of functional relationships can inform relatively more effective treatments that benefit a higher percentage of patients.

Conclusions and Future Directions

Over the three decades since cognitive-behavioral analyses of AN and BN were proposed, they have had profound effects on how the EDs are conceptualized and treated. The idea that patients' beliefs about weight and eating form the core psychopathology of these disorders, once radical, is now commonplace, represented in diagnostic criteria and built into standard symptom inventories. The proposal that symptoms could be reversed by directly, and sometimes narrowly, targeting these beliefs and associated behaviors has been affirmed with reference to BN and at least modestly supported for AN. The cognitive-behavioral perspective is also represented in one of the most effective prevention programs tested to date, which is designed to develop dissonance between endorsement of the thin ideal and other valued beliefs (Stice, Shaw, Becker, & Rohde, 2008; see also Chapter 44).

Active research continues across these topic areas, with particular emphasis on incorporating additional variables into the conceptual model, supplementing and strengthening the treatment approach, and disseminating evidence-based CBT more widely. At the same time, future efforts should be directed toward the investigation of basic elements of the model that have been persistently and peculiarly neglected.

Getting Back to Basics

One welcome development is renewed research interest in core cognitive and behavioral elements of ED psychopathology. During the first decade after the models were proposed, numerous studies were undertaken to examine ED thoughts and beliefs and to chart patients' experience over the course of binge-purge episodes. By 2005, however, one expert observed that "detailed analyses [of basic cognitive and behavioral phenomena] have been curiously lacking in recent years" (Cooper, 2005, p. 526). Six years later, Walsh (2011) noted that "the eating behavior of individuals with AN has received surprisingly little attention" (p. 527).

The value of focusing on the defining characteristics of EDs (see Chapter 1) is illustrated by the analysis of body checking behavior. A series of descriptive and experimental studies (e.g., Shafran, Lee, Payne, & Fairburn, 2007) yielded new insights about the role of checking and avoidance in maintaining ED symptoms, insights that guided development of additional clinical techniques that are wholly compatible with focused CBT. Systematic study of other variables such as eating patterns and self-motivational tactics in AN might yield comparable benefits.

The revival of interest in basic cognitive and behavioral features may owe much to advances on other fronts in the ED field. Current enthusiasm for biological models (see Chapters 17, 28, & 30) has stimulated several lines of research relevant to cognitive-behavioral theory. Neuropsychological and brain imaging studies have found preliminary evidence consistent with the view that AN patients process information in rule-bound, detail-oriented ways. The cognitive-behavioral model posits that this disposition may help to explain the appeal of extreme dietary restriction, the ability to sustain it over time, and the resistance to changing established patterns of thought and behavior (Vitousek & Hollon, 1990). Neurocognitive data provided the impetus for supplementary treatment techniques, including Cognitive Remediation Therapy (Tchanturia, Lloyd, & Lang, 2013), designed to develop more flexible thinking, and Attention Bias Training (Renwick, Campbell, & Schmidt, 2013), which attempts to modify automatic orienting to cues related to food, weight, and general threat. Although both could have been derived directly from the cognitive-behavioral model, neither had been pursued within a CBT framework.

In the future, instructive information on these topics is likely to come from collaborative efforts between neurocognitive and cognitive-behavioral experts. In particular, the development of investigative techniques that are less dependent on self-report (Thomas, Hartmann, & Killgore, 2013; Vitousek, Daly, & Heiser, 1991) would be enhanced by combining the neurological and technical expertise of brain researchers with cognitive-behaviorists' skills in systematic observation and functional analysis.

Thinking More Critically and Creatively About the EDs

If some elements of the cognitive-behavioral model have been neglected by cognitive researchers, others have been actively avoided by the field as a whole. Since AN was first described, clinicians' observations and patients' accounts have repeatedly alluded to the sense

of accomplishment and virtue that individuals derive from their symptoms and to the diligent effort applied to sustain them. Both of these distinctive features have problematic implications, on multiple levels. Understandably, researchers, clinicians, and advocates who want the EDs recognized as serious, disabling, biologically influenced conditions are reluctant to highlight features that might jeopardize this goal and stigmatize patients. All find it easier to feel compassion for the suffering that patients endure when it is perceived as "pure" (i.e., devoid of any elements of reward or gratification) and wholly involuntary. In fact, there is substantial, convergent evidence that anorexic individuals do value many aspects of the devastating disorder from which they simultaneously suffer, and work extraordinarily hard to keep their symptoms running. These themes are prominent in the cognitive-behavioral analysis of AN, yet neither has been investigated as intensively as their hypothesized significance merits. In particular, some of the most provocative elements, such as competitiveness over symptom severity and feelings of moral superiority, remain virtually unstudied.

Moreover, while considerable research attention in the ED field has been paid to the questions of "who" and "why," there has been astonishingly little interest in the question of "how." Part of the reason for the frequent characterization of AN as "baffling" and "mysterious" is the implicit recognition that it is fiendishly difficult to persist in self-starvation when ample food is available. Although investigators continue the quest for an elusive biological mechanism that suppresses appetite in AN, as well as a drug to reverse it, compelling observational evidence suggests that patients restrict despite the fact that most remain, as one put it, "ravenously, desperately, savagely hungry" (Johnston, 1993, p. 30). Highly convergent indicators that self-starvation is effortful for individuals with AN (e.g., observations that they conduct research on dieting techniques, seek out inspirational images and slogans, self-impose punishments for "eating too much," and cultivate fear of fattening foods) are so consistently skipped over in theoretical discussions and omitted from research agendas that it suggests a collective blind spot. Seeing these distinctive features more clearly does not mean viewing the EDs less compassionately. Indeed, a better understanding of how the demands of self-starvation shape patients' experience and constrain their options can increase accurate empathy, while suggesting additional ways to assist in modifying patterns of thought and behavior that keep individuals trapped in EDs.

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19

Feminist Theories of Eating Disorders

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There are many different types of feminism and feminist theory, ranging from more moderate to extremely radical forms. Perhaps the most commonly held feminist beliefs in the United States are those of liberal feminism, which is based in the same theory as the foundations of modern principles of liberty and democracy. Liberal feminism asserts that women and men are equal and all should share the same rights and have the same opportunities (Henley, Meng, O'Brien, McCarthy, & Sockloskie, 1998). Another type of feminism is cultural feminism, which focuses less on the equality of men and women, and more on the equality of what cultural feminists term masculine and feminine values, stating that "women's values" such as peace and caring for others should be valued equally with "masculine values" such as aggressiveness. A more extreme type of feminism is radical feminism, which views women's oppression by men as the oldest and most universal type of oppression. Other types of feminism focus on the concerns of women of color ("womanism") or those of lesbian women (Henley et al., 1998; Simoni, Henley, & Christie, 1999). The common element among these and other varieties of feminism is that all strive for some form of equality between men and women, whether or not that is the primary theoretical focus of each school of feminism.

Feminist theorists have also provided important perspectives on the development and maintenance of eating problems among women (Heinberg, 1996). In particular, feminist theorists have attempted to explain the gender differential in the occurrence of eating psychopathology, as lifetime prevalence for anorexia nervosa (AN), bulimia nervosa (BN), and binge eating disorder (BED) is 1.75 to 3 times higher among women than among men (Hudson, Hiripi, Pope, & Kessler, 2007; see also Chapters 5 & 27). Eating disorders (EDs) have become one of the most common problems among young females in Western countries (Grave, 2003). Approximately 10–15% of girls and young women who are tested in studies score above the cut-off point for probable disordered eating (Austin, 2000). Moreover, up to 59% of college women endorsed skipping meals to control their weight (Tylka & Subich, 2002).

Because of these startling statistics, feminist theorists and researchers have been especially concerned with explaining why a growing number of people, especially women, engage in behaviors that are so dangerous, counterintuitive, and even deadly. Smolak and Murnen (2004) offer one feminist explanation for EDs, attributing the gender differential of these

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problems to issues of power and objectification (see Chapter 27). For instance, patients with both AN and BN focus on *controlling* weight and shape, which Smolak and Murnen (2004) argue may reflect women's attempts to take back control of their bodies from such problems as objectification and sexual harassment, both of which often are perpetrated by men. These authors, therefore, argue that body image and eating problems are "gendered" problems and that it is important to focus on women's learned experiences in studying these problems. Other feminist theorists have argued that negative body image and eating pathology are "natural responses to pathological societal pressures to be thin" (Heinberg, 1996, p. 35).

This chapter will examine various theories that have sought to explain EDs and related issues from a feminist perspective. In addition, the idea that holding feminist beliefs may be protective against eating psychopathology will also be explored, along with potential implications of feminist theory for ED prevention (see Chapter 43).

Feminist Theoretical Explanations of Eating Psychopathology

This section will examine several diverse feminist theoretical explanations for EDs and related behaviors. The theories considered include Bordo's (1993/2003) emphasis on cultural messages about the body, Wolf's (1991) examination of the political and economic implications of EDs, Fredrickson and Roberts's (1997) views on the role of objectification of women in eating psychopathology, and Piran and colleagues' exploration of embodiment (Piran, 2010; Piran & Cormier, 2005; Piran & Teall, 2012).

Messages About the Body

Bordo (2003), a philosopher who first proposed her theory in 1993 and presented a revised edition in 2003, explains that all feminist theorists writing about EDs share "a prima facie commitment both to taking the perceptions of women seriously and to the necessity of systemic social analysis" (p. 10). In other words, feminist theorists focus on the impact of culture on EDs rather than focusing on individual or familial factors and also take into account women's lived experiences instead of a detached perspective. Rather than viewing the thoughts and behaviors that characterize EDs as solely the result of individual psychopathology, Bordo argues that they are instead symptomatic of the unrealistic and unhealthy messages transmitted to women from society about their bodies and embedded in our social structure (see Chapters 21 & 29). She emphasizes how the thin ideal—the perfect, slender, often unattainable image of a body that women are encouraged to strive for—has been a continually moving target, literally shrinking before our eyes. Indeed, research examining the shape of Miss America contestants and Playboy centerfolds has shown that these body weights steadily decreased over time and were significantly lower than those of the average American female (Garner, Garfinkel, Schwartz, & Thompson, 1980; Wiseman, Gray, Mosimann, & Ahrens, 1992).

Bordo (2003) sees the body in general, but especially the female body, as a "medium of culture" (p. 121) and emphasizes that the female body, in particular, is forced to rebel against its own development in the case of women with EDs. Like Smolak and Murnen (2004), she emphasizes that, in order to accomplish this task, women must exercise control over their bodies (see Chapter 27). Bordo reports accounts of women with AN who discuss how this control was its own reward, stating, "Anorexia could thus be seen as an extreme development of the capacity for self-denial and repression of desire (the work ethic in absolute control)" (p. 157), an attribute

seen as one to be admired by our achievement-driven society. Women who are overweight or obese, on the other hand, are seen as defying the norm, not following society's rules, and thus worthy only of being shunned.

Western culture repeatedly emphasizes that this control—both control of women by society and women's own self-control—is necessary not only for women to meet the thin ideal, but also so that women are confined sexually. For Bordo (2003), "the sexual act, when initiated by a woman, is imagined as itself an act of eating…women's sexual appetites must be curtailed and controlled, because they threaten to deplete and consume the body and soul of the male" (p. 73). Metaphors for female sexuality, such as "man-eater" or the more recent term "cougar," associate women's hunger and women devouring food not only with sexuality but with negative connotations. Taking these metaphors into account, Bordo (2003) draws parallels between today's EDs and Victorian hysteria. In both disorders, she explains, symptoms often occur in reaction to society discouraging women from engaging in career-focused goals, particularly in traditionally male fields, and striving to meet an overexaggerated, stereotypically feminine ideal. Indeed, the driving pathology for EDs—the drive for thinness, the drive to virtually disappear as patients lose more and more weight—is akin to the Victorian idea that women should not take up space in public society, but instead should confine themselves to the domestic sphere and within restricting corsets.

Bordo (2003) sees today's thin ideal as an attempt by society to fight against increasing female independence and power, a backlash of sorts encouraging a return to "traditional" values and gender roles, just as the corsets of the Victorian era were meant to confine their wearers. Women who suffer from EDs are paradoxically unconsciously protesting—a literal hunger strike against the curvy figure of a maternal, domestic woman—while punishing themselves, meeting societal ideals, and retreating from conscious protest. Recent research by Calogero (2013) found that self-objectification, or seeing oneself as an object as a result of internalization of the thin ideal (see Chapter 27), was associated with less engagement in feminist social activism and this relationship was mediated by system justification, a tendency to justify the status quo even if doing so is detrimental to the woman in question. Therefore, the pressures of society related to appearance do indeed seem to be linked to a retreat from active social protest.

For Bordo (2003), the importance of culture for the development of AN and BN cannot be overestimated:

> Psychopathology, as Jules Henry has said, "is the final outcome of all that is wrong with a culture." In no case is this more strikingly true than in that of anorexia nervosa and bulimia, barely known a century ago, yet reaching epidemic proportions today. (p. 95)

Bordo points out that dieting and obsession with fat have become normal in the United States, an assertion that Silberstein, Striegel-Moore, and Rodin (1987) echoed when they pointed out that dissatisfaction with one's own body is so common, even in nonclinical populations, that it may be a "normal part of the female experience" within Western culture (p. 89), a phenomenon that has been referred to as "normative discontent." Bordo (2003) also emphasizes that research on AN and BN often either ignores the importance of cultural pressures or, if acknowledged, almost apologizes by commenting that not everyone exposed to these pressures develops an ED. This latter argument ignores the fact that these cultural pressures interact with all the other aspects that make up a woman's life, such as genetics, ethnicity, and family (see Chapter 67). Bordo (2003) also says that ignoring the importance of culture downplays its role in creating an environment in which disordered eating behavior can thrive. In fact, work by Keel and

Klump (2003) suggests that BN, in particular, may be so tied to Western cultural pressures that it should be considered a "culture-bound syndrome" much in the same way that amok, a disorder whose symptoms include a sudden, murderous rage in normally nonviolent individuals, is considered a culture-bound syndrome in Malaysia. To Bordo (2003), culture—and women's protest against it, conscious or not—is the root cause of EDs.

The Politics and Economics of Eating Disorders

What Bordo (2003) describes with careful academic detail, social critic and political activist Wolf (1991) expresses and elaborates upon with righteous anger, infusing a personal touch and drawing links to both economics and politics. Wolf also likens the actions of those with EDs to hunger strikes by prison inmates. She sees eating disordered individuals as girls and women who are attempting to avoid having their choices limited to those afforded to women—certain career paths, motherhood, and so forth—by preventing their bodies from taking on a womanly shape. Like Bordo (2003), however, she recognizes that these disorders are in fact limiting rather than freeing and are foisted upon women by the prevailing culture under the guise of personal decisions about one's appearance.

Wolf (1991) sees a direct relationship between women's suffrage and the cult of dieting in Western culture, pointing out that the concept of fat as bad and dieting to fit a thin ideal as good began during the years 1918–25, just as women gained the right to vote. She sees a similar pattern arising following the women's movement of the 1960s and 1970s. In Wolf's (1991) opinion, the push towards a focus on weight and appearance "arose…to save magazines and advertisers from the economic fallout of the women's revolution" (p. 66). She points out that women became obsessed with the idea of losing 10 to 15 pounds in order to be their best selves, which she terms the "One Stone Solution," named for the British weight equivalent of 14 lb (p. 186). In order to reach this solution, women had to change their bodies in unnatural ways, resulting in self-hatred for those who could not meet this goal just when they should have been feeling empowered and confident.

In Wolf's (1991) opinion, this goal is not only about thinness, hunger, or power; it is also about money. At the time of Wolf's writing in 1991, she reported that the diet industry was worth $33 billion a year. Recent reports show that the value of the diet industry has now ballooned to $265 billion worldwide in 2012, which is roughly the gross domestic product of Hong Kong (ReportsnReports, 2013). Weight control is big business; this idea is one of the crucial premises of Wolf's (1991) argument about societal and economic pressures for women regarding their bodies. It is big business not only for those who make diet pills or perform liposuction, but it is also a money maker for those who depend upon the revenue from the advertising of these products: TV, movies, online sites, and especially women's magazines (see Chapter 29). Kilbourne (1999) explains the power of these advertisements: "Advertising doesn't cause eating problems, of course…However, these images certainly contribute to the body-hatred so many young women feel and to some of the resulting eating problems" (p. 135; see also Chapter 29). Having focused much of her career on the power of advertisements on influencing women, Kilbourne (1999) argues that advertisers are not purposefully trying to make women develop eating disorders, but instead, are reflecting societal messages about weight and the fact that these messages are indeed profitable. However, as she points out, these ads still shape women's attitudes about what an ideal body should look like and how their own bodies compare, thus

increasing the risk for disordered eating, regardless of whether advertisers are purposefully trying to invoke these symptoms (see Chapter 29).

It is not just self-hatred or economic pressure that women experience related to their weight. Women who fail to meet the thin ideal are also ridiculed and shamed by society at large (see Chapters 21 & 27). Wolf (1991) believes there is a reason why we care so much about other women's weight: "A cultural fixation on female thinness is not an obsession about female beauty; but an obsession about female obedience" (p. 187). She argues that by forcing women to diet, which she believes is in fact a state of "self-inflicted semistarvation" (p. 193), Western culture forces women to become smaller not only physically but also to take on a smaller, quieter role in society. Wolf (1991) points out that women who suffer from AN end up diminished, weak, and with difficulty focusing. The physiological consequences of AN are well known, can be life-threatening, and include cardiac problems, growth impairment, and osteoporosis (Katzman, 2005; see also Chapters 14 & 52). Wolf (1991) also expresses concerns about the effect of self-starvation on the ability of women and girls to think clearly and thus fully participate in their educations and professions. Research from the field of neuropsychology supports this assertion, as patients with AN show poorer recall, reaction time, and motor speed, as well as difficulties concentrating and with interrupting ongoing activities (Green, Elliman, Wakeling, & Rogers, 1996; Southgate, Tchanturia, & Treasure, 2005).

Wolf (1991) argues that the physiological and cognitive consequences of eating disorders make it difficult for women to have energy not only to engage in their professions, but also to protest against any of the difficulties they are experiencing. Being in a state of starvation effectively silences women even in an era when their voices should be loudly heard. Again, Wolf says that this marriage of appearance and silence is political, not just personal—a convenient way for society as a whole to maintain the status quo and keep women so distracted by their appearance that they have difficulty being fully engaged citizens. She emphasizes that theories that focus only on the impact of individual difference variables or family dynamics can never fully explain eating disorders when 60–80% of women are dieting or engaging in disordered eating behaviors. As she explains, speaking of her own experience with AN, "The youngest victims, from earliest childhood, learn to starve and vomit from the overwhelmingly powerful message of our culture, which I found no amount of parental love and support strong enough to override" (p. 205).

Kilbourne (1999) echoes these concerns, pointing out that advertisements about women's weight are about fears about women's power. She gives examples of ads that literally say, "Soon, you'll…be taking up less space" (p. 137). Focusing on women's weight in advertising and other societal messages both distracts women from being able to focus on the task at hand and trivializes women's empowerment, as in the case of an ad for low-calorie bread where women sing about the "taste of freedom" not because they can participate in their own governance, but because they can now eat carbs.

Wolf (1991) believes that the only way to combat what she terms an epidemic of EDs in Western society is by changing the fabric of society as a whole, rather than focusing on individual difficulties. She emphasizes the importance of building communities among women and discussing issues of eating disorders. She calls for an introduction of more representative media images so that girls and women can see what successful women from a range of ages look like instead of only seeing manipulated images of teenage girls. She also calls for acceptance of all body shapes and types, and she believes that only when women free themselves from the "One Stone Problem" and more severe eating psychopathology will they be able to fully engage in their lives—personal, professional, and political.

Objectification Theory

Focusing on the same issues of representations and examinations of women's bodies that anger Wolf (1991), psychologists Fredrickson and Roberts (1997) proposed objectification theory as a feminist explanation for the disproportionately greater numbers of women who suffer from psychological issues such as depression, sexual issues, and especially EDs (see Chapters 21 & 27). This theory argues that women are often regarded as objects by society—specifically, male members of society—with the focus of representations of women being placed on all or part of their bodies in a sexual context rather than on their abilities. As a result, the woman is seen not as an entire person of substance, but is instead considered to be represented entirely by her body or by one or several portions of her body. Fredrickson and Roberts (1997) described this experience as "being treated *as a body*" (p. 174). Women and their bodies are thus objectified, that is, regarded as objects for consumption by others, usually men. While men do the active looking and objectifying of women, according to this theory, women are forced to be passive objects to be acted upon by men in society. The media, which are generally visually based and full of images that focus only on women's bodies or often a specific body part, may intensify this objectification (see Chapter 29). Like Bordo (2003), Fredrickson and Roberts (1997) state that factors such as age or ethnicity may impact how each woman experiences objectification, but they emphasize that "Bodies exist within social and cultural contexts, and hence are also constructed through sociocultural practices and discourses" (p. 174).

According to Fredrickson and Roberts (1997), this sexual objectification of women is dangerous because women then internalize this objectification, or *self-objectify*. Fredrickson and Roberts (1997) draw on the ideas of art historian Berger (1972), whose writings on how we look at art clearly describe the experience of self-objectification. As Berger articulates:

> A woman must continually watch herself. She is almost continually accompanied by her own image of herself.…From earliest childhood she has been taught and persuaded to survey herself continually.
>
> One might simplify this by saying: *men act* and *women appear*. Men look at women. Women watch themselves being looked at. This determines not only most relations between men and women but also the relation of women to themselves. The surveyor of woman in herself is male: the surveyed female. Thus she turns herself into an object. (pp. 46–47)

Fredrickson and Roberts (1997) argue that this process of turning oneself into an object is dehumanizing and dangerous. Self-objectification can lead to feelings of despair, shame, anxiety, or helplessness, which, in turn, can lead to increased probability of mental health problems such as depression and EDs.

Research has supported these claims. Muehlenkamp and Saris-Baglama (2002) found that self-objectification is directly related to restrictive eating, bulimic symptoms, and symptoms of depression. A test of all of the proposed relationships in Fredrickson and Roberts's (1997) original theory by Tiggemann and Williams (2012) confirmed that self-objectification predicts body shame and appearance anxiety, which in turn predict disordered eating. Their model accounted for an astonishing 93% of the variance in disordered eating. Objectification can be manipulated experimentally, as Fredrickson, Roberts, Noll, Quinn, and Twenge (1998) did in an innovative study in which participants were asked to try on either a swimsuit (objectifying condition) or a sweater (nonobjectifying condition) in a room with a mirror and then fill out surveys. The women in the objectifying condition exhibited more body shame, more restrained

eating, and higher levels of negative affect than women wearing the sweater. A more recent study by Calogero and Pina (2011) activated self-objectification via a scrambled sentence task with objectifying words and found that women in the objectified condition experienced more guilt about their bodies and endorsed higher levels of restrained eating. This research is consistent with the writings of Wolf (1991), who emphasized the importance of guilt that arises when women believe that they eat too much.

Calogero, Davis, and Thompson (2005) found women in an eating disordered population had markedly higher levels of self-objectification than women in non-eating-disordered populations. This finding is consistent with Fredrickson and Roberts's (1997) theory that EDs are perhaps one of the most obvious consequences of self-objectification and with assertions by other feminist theorists (Bordo, 2003; Smolak & Murnen, 2004; Wolf, 1991) that eating disorders are primarily about control. As Fredrickson and Roberts (1997) explain, women in today's society who wish to either meet cultural ideals or rebel against them must do so with their bodies. Like Bordo (2003), Fredrickson and Roberts (1997) also argue that these disorders may be a form of protest against sexual objectification and women's powerlessness, albeit a protest that is both passive, rather than active, and harmful to the woman making said protest. As Fredrickson and Roberts soberly explain, "Having a female body, then, gives girls and women plenty to worry about and little to control" (p. 188) except, that is, for the very target of the objectification: their own bodies.

Embodiment Theory

Another theory that focuses on women's experiences with their own bodies and builds upon the theories previously discussed—the developmental theory of embodiment—was codified by Piran and Teall (2012), although Piran began work on this theory earlier (Piran, 2010; Piran & Cormier, 2005; see also Chapters 27 & 43). Piran and colleagues argue that models and theories that attempt to explain EDs need to be as inclusive as possible, taking into account not only the silencing of women by the political sphere emphasized by Bordo (2003) and Wolf (1991), but also the ramifications of seeing oneself as a sexual object articulated by Fredrickson and Roberts (1997). Piran sees the common thread among these problems in women's detachment from their own body. As she explains:

> internalized gender-based social discourses can disrupt women's body-self-connection and eating patterns. For example, it has been suggested that internalizing the negative constructions of women's sexual desire may have an adverse impact on women's body image and experience of bodily needs (Tolman & Debold, 1994), or that social discourses limiting women's roles in the public sphere have led to women's monitoring of the physical space they take in public (Bartky, 1988). (Piran & Cormier, 2005, p. 556)

Expanding upon this idea of women feeling disconnected from their bodies, Piran articulated her theory. Rather than talking about body satisfaction or body dissatisfaction, Piran speaks of "disembodiment," a detachment that goes beyond mere unhappiness with one's body. The term "embodiment" was developed by philosophers to describe the mind-body connection. Moreover, the term also encompasses the way that the body connects to the world around it. Piran and Teall (2012) argue that girls and women receive social messages about their bodies and how these bodies should interact with the world through what they term "three core pathways" (p. 169): physical experiences, messages about social expectations, and social power.

Piran and Teall's (2012) theory is a developmental one, emphasizing the role transition from childhood to adolescence with which girls struggle. Piran and Teall note the increased pressures that emerge at this time, including exposure to media, peer comments, and increasing awareness of gender inequality. It also has greater breadth than theories that merely focus on body image. For Piran and Teall, embodiment goes beyond dissatisfaction with one's body to focus on all of one's experiences with one's body within the context of a society that is focused upon female appearance. Disembodiment, then, includes body dissatisfaction, but also encompasses alexithymia, or a lack of awareness of one's own emotions and internal physiological states (see Chapter 17); self-harm (see Chapter 15); anxiety surrounding participation in physical activities; and risky sexual behaviors, all of which, in turn, have been associated with disordered eating. For example, Myers and Crowther (2008) found that lack of awareness of emotions but especially of feelings of hunger and satiety is a strong predictor of disordered eating and mediates the relationship between self-objectification and disordered eating attitudes. Piran and Teall (2012) emphasize how the experience of self-objectification makes it nearly impossible to have a positive connection to one's body.

For Piran (2010), risk factors for EDs go beyond messages about how the body should look and include the fact that these messages also tell girls and women that taking care of their appearance is a way to gain social power, making gender itself a risk factor for eating pathology. Echoing Fredrickson and Roberts (1997), she points out that girls and women experience more sexual harassment, sexual violence, and restriction on what types of activities are acceptable to do. Indeed, Piran and Teall (2012) emphasize that Objectification Theory draws upon views of embodiment that reflect the connection between the body and the world around it. Self-objectification becomes a strategy not of narcissism, but of self-protection in a world where women learn by observation that the only way for them to garner social power is to focus upon their appearance. In fact, Piran and Teall use the concept of disembodiment to explain why some girls and women with EDs may not express body dissatisfaction at all. They cite the example of a case of a girl with AN who starved herself after being harassed by a group of boys in order to make herself invisible as a means of escape. This patient did not express body dissatisfaction, and many survivors of sexual abuse or sexual assault exhibit disordered eating behaviors in the absence of distorted body image. Instead, eating psychopathology becomes one of a number of self-harm behaviors used as a coping mechanism by these survivors. Piran and Teall also cite cross-cultural examples of women with EDs who express powerlessness but not fear of fat (see Chapters 16 & 23).

In order to address the problem of disembodiment and thus the problem of disordered eating, Piran (2010) argues that therapists and those implementing prevention programs need to address all of the ways the connection between the body and the self is disrupted in women's lives. She gives examples of addressing such diverse issues as substance abuse and lack of participation in sports in addition to more obvious targets such as plastic surgery or body dissatisfaction. Piran and Teall (2012) emphasize that these programs need to include messages from adults and schools about girls connecting positively with their bodies and the physical environment around them, as well as becoming critical toward societal messages that encourage disembodiment. In addition, girls have to feel safe in their own bodies, which can be accomplished with strictly enforced policies against harassment and abuse. Piran and Teall argue that by addressing all of these aspects of disembodiment directly, we can move girls and women toward embodiment, which includes feelings of agency and

power as well as engaging in a level of care for oneself with which disordered eating is inconsistent.

Common Themes Among Feminist Theories of Eating Disorders

Feminist theorists and researchers have clear views on why EDs are diagnosed among girls and women. In particular, there are several themes that the feminist theories discussed above share in their explanations of disordered eating. These commonalities include:

1 Emphasizing the role of environmental pressures to conform to an often unnaturally thin body type in the development of EDs.
2 Clearly tying disordered eating to sexism experienced by women.
3 Focusing on how disordered eating is often an overt manifestation of the covert struggles that women experience.

One common theme of these diverse feminist theories, as articulated clearly by Bordo (2003), is that each emphasizes the role of environmental pressures to conform to an often unnaturally thin body type in the development of EDs. Fredrickson and Roberts's (1997) objectification theory is the obvious example with its focus on the sexual representation of women as objects and the ramifications of the internalization of these messages. Piran and Teall (2012) also discuss how disruptive are societal messages about the body, naming these messages as a cause of disembodiment. All of the theorists emphasize the importance of environmental pressures to a certain extent, rather than placing the blame on the family unit or the individual patient who has developed AN or BN. These theories argue that we live in a society that constantly tells girls and women that they need to look and act a certain way, whether directly or in more subtle ways, as Kilbourne (1999) discusses in her dissection of the portrayal of women in advertising, and that these messages are a large part of why girls and women develop disordered eating behaviors.

Another common theme among these theories is clearly tying disordered eating to sexism experienced by women in society as a whole (see Chapter 27). The fact that, as Wolf (1991) points out, pressures on women to be thin have increased after both the first and second waves of feminism shows how much women's voices and protests are seen as a threat by society as a whole. In addition, Piran and Teall's (2012) explanation of all of the ways in which women experience sexism (harassment, discrimination, etc.) underscores how disordered eating fits into the larger problems with gender in Western society. These theorists, in particular, see gender discrimination and an attempt to navigate it as a root cause of eating psychopathology, even in the absence of body dissatisfaction, which is usually assumed to be a driving force behind these disorders.

These feminist theories also share a common thread of focusing on how disordered eating is often an overt manifestation of the covert struggles that women experience (see Chapter 43). Piran and Cormier (2005) emphasize that the choice of women to remain silent, often called self-silencing, is clearly tied to disordered eating both theoretically and empirically. Indeed, Fredrickson and Roberts (1997) include self-silencing in their list of potential consequences of self-objectification along with EDs. More than that, however, both Bordo (2003) and Wolf (1991) see disordered eating behaviors as a way for women who have been silenced, or prevented from fully participating in overt protest on their own behalf, to communicate and protest against the sociopolitical and economic circumstances that all women experience.

Feminist Beliefs as a Protective Factor

Because feminist theory criticizes and rejects the thin ideal and women's need to conform to it, for the past 20 years feminist researchers have suggested that belief in feminist ideas may allow women to reject this thin ideal and thus to have a more positive body image and fewer problems with eating pathology (MacKay & Covell, 1997; Myers & Crowther, 2007; Myers, Ridolfi, Crowther, & Ciesla, 2012; Ojerholm & Rothblum, 1999; Rubin, Nemeroff, & Russo, 2004; Tiggemann & Stevens, 1999). Correlational, laboratory, ecological momentary assessment, and prospective studies in this area have produced various results. For example, in an experimental manipulation, MacKay and Covell (1997) examined the relationship between viewing advertisements with sexually objectified women and acceptance of feminism and found that women who were shown the objectifying advertisements during the experimental session had lower levels of acceptance of feminism. In a cross-sectional study, Tiggemann and Stevens (1999) examined attitudes towards feminism, weight concern, and self-esteem across the life span (women ages 18–60) and found a negative relationship between attitudes toward feminism and weight concern—but only for women in the 30–39 and 40–49 age groups. These researchers argue that the importance of appearance is so ingrained and so highly socially reinforced that even though younger women may hold positive attitudes toward feminism, these beliefs do not serve as a protective factor against weight concern.

In another correlational study with college women, Ojerholm and Rothblum (1999) found a small but significant negative correlation between attitudes toward feminism and attitudes toward other people's weight and body shape, indicating that more feminist women were less likely to judge other people's bodies negatively when those other people did not conform to the thin ideal. However, these researchers found no significant correlations between attitudes toward feminism and body dissatisfaction, which consists of dysfunctional, negative beliefs and feelings about one's weight and shape (Garner, 2002).

In a qualitative study, Rubin and colleagues (2004) talked to groups of college women who self-identified as "feminist" about their experiences. One finding was that feminist beliefs appear to provide "an alternate way to understand cultural messages and reframe negative thoughts" about body image for these women (Rubin et al., 2004, p. 27). The researchers also found that there is a conflict between these women's feminist beliefs and their feelings about their own appearance such that even though feminist women may "know" that they should not judge themselves based upon their own appearance, they still sometimes find themselves being critical of their own bodies. These findings are consistent with Rothblum's (1994) assertion that women may reject traditional gender roles, but continue to worry about their appearance.

One methodological problem with some of the previous studies (MacKay & Covell, 1997; Ojerholm & Rothblum, 1999; Tiggemann & Stevens, 1999) is their use of the Attitudes Towards Feminism and the Women's Movement Scale (FWM; Fassinger, 1994). This scale measures attitudes toward the women's movement, not necessarily feminism per se; for example, one item on the questionnaire is: "The leaders of the women's movement may be extreme, but they have the right idea" (Fassinger, 1994, p. 395). It is questionable whether the FWM is relevant to today's college women, particularly given recent findings that suggest many undergraduate women may be reluctant to self-identify as feminist (Zucker & Bay-Cheng, 2010). In fact, none of the aforementioned studies that used this scale as a measure of feminism found significant results for college-aged women (MacKay & Covell, 1997; Ojerholm & Rothblum, 1999; Tiggemann & Stevens, 1999), which suggests it may not be applicable to female undergraduates.

A scale does exist, however, to measure feminist attitudes and agreement with the beliefs of different types of feminism: Henley et al.'s (1998) Feminist Perspectives Scale (FPS), which contains subscales to measure conservative, liberal feminist, radical feminist, socialist feminist, cultural feminist, and womanist perspectives, with an additional "Fembehave" subscale to measure feminist behavior. More recent research has used the FPS to examine the role of feminist beliefs as a potential moderating or protective factor against body image disturbance, a known risk factor for eating disorders. For example, using a correlational methodology, Myers and Crowther (2007) used the FPS to examine feminist beliefs as a moderator between media exposure and internalization of the thin ideal and found that, although women have high levels of thin ideal internalization with high levels of media exposure regardless of their level of feminist beliefs, those with higher feminist beliefs have lower levels of thin-ideal internalization in the presence of higher media exposure than do those with lower feminist beliefs.

Myers and colleagues (2012) further examined the role of feminist beliefs as measured by the FPS in the relationship between comparing one's appearance to others and body image disturbance. Myers et al. used an ecological momentary assessment methodology, which entails gathering information at multiple time points during the participants' daily lives. They found that following an upward, appearance-focused social comparison, participants experienced virtually the same amount of body dissatisfaction regardless of whether they were low or high on feminist beliefs. However, following these same upward, appearance-focused comparisons, participants with higher levels of feminist beliefs experienced lower levels of engagement in body checking, a behavioral component of body dissatisfaction that involves obsessively checking areas of one's body (Reas, Whisenhunt, Netemeyer, & Williamson, 2002), than did participants with lower levels of feminist beliefs. These findings are cause for optimism, as body checking has been shown to be clearly associated with ED symptoms (Shafran, Fairburn, Robinson, & Lask, 2004). These findings suggest that higher levels of feminist beliefs may give women a different lens through which to interpret this information and are consistent both with the previous qualitative work of Rubin et al. (2004) and the theoretical work of Rothblum (1994). In addition, a meta-analysis by Murnen and Smolak (2009) considered the hypothesis that feminist women would experience positive body image more than nonfeminist women. They found that there was a protective relationship between feminist identity and body attitudes that was even stronger when women's studies students or older women were examined.

Implications for Prevention

Shisslak and Crago (1994) laid out guidelines for creating feminist ED prevention programs (see Chapter 43). One of their primary suggestions was that prevention programs based on feminist theories and ideals should focus on educating women about the societal pressures for women's appearance and the motivations behind them. These programs should also focus on helping girls and women to recognize these messages and how unrealistic they are. Many school-based prevention programs (e.g., Smolak, Levine, & Schermer, 1998) have done just that already by incorporating media literacy (see Chapter 45), which teaches girls and women about how unrealistic media representations of the female body are. These programs often incorporate other elements such as body image, healthy eating habits, and the symptoms of EDs.

In keeping with the overarching themes of the feminist theories examined in this chapter, Neumark-Sztainer et al. (2006) argue that a critical part of any prevention strategy will be to target the environment, specifically sociocultural messages and practices that objectify women, both at home and in the media. Such programs may benefit those outside the purview of the program or at later times due to the ripple effect of body-positive changes in an environment. Neumark-Sztainer and colleagues acknowledge that these programs will likely be difficult and expensive but point to encouraging findings for substance abuse as well as EDs (see Chapter 47). Similarly, Piran (2010) argues that feminist prevention programs need to recognize and address environmental factors related to gender discrimination and inequality in general in order to prevent self-body disconnection.

Emphasis on challenging the environment around women and their interpretation of this environment has also been used in ED prevention programs designed for undergraduate women. In particular, Becker and colleagues (Becker, Hill, Grief, Han, & Stewart, 2013; Becker, Smith, & Ciao, 2005; see also Chapter 44) have developed a program to work with women in sororities, who are known to be at high risk for eating disorders. This program emphasizes discussion of cultural pressures for thinness and ways to combat this pressure. The researchers have found that this prevention program leads to reduction in self-objectification, thin-ideal internalization, negative affect, body dissatisfaction, and disordered eating. These changes were largely maintained at 8-month follow-up. These results suggest that addressing the environment, specifically societal pressures that are linked to EDs, leads to a reduction in disordered eating behaviors.

Another goal emphasized by Shisslak and Crago (1994) for feminist prevention programs was empowering women. As the theorists examined in this chapter have all emphasized, one of the consequences of women's focus on thinness and their self-starvation is a loss of power, leaving them smaller, quieter, and less able to engage in their own education and work lives. One example of a prevention program that fits this goal is Piran's (1999) feminist intervention at an elite and highly competitive ballet school. The program hoped to decrease emphasis on body weight and shape by again implementing an environmental change, restructuring the way the school itself looked at puberty, growth, and diversity of body types. Students met and guided the direction of small discussion groups, where they learned to evaluate their own dissatisfaction with their bodies in both a cultural and a dance context, learned healthier eating practices, discussed feminist ideas, and suggested needed environmental changes at the school. Piran (1999) found that fewer of the students were dieting and more had realistic concepts of their own body image after this program was implemented. There was a significant and consistent decrease in binge eating, vomiting to purge, laxative use, and restrictive dieting. The participatory model upon which this program was based is a prime example of feminist action and ideals (Piran, 2010).

Future prevention programs should continue to address the cultural issues at the root of feminist theories on EDs as well as to use empowering feminist techniques to implement these programs, in keeping with Shisslak and Crago's (1994) guidelines. In addition, future feminist prevention programs should focus on other goals proposed by Shisslak and Crago (1994), such as addressing the socialization of gender roles for adolescent girls in order to help them find their voice, literally and figuratively, in the world and to avoid the quiet and small role that EDs create for girls and women in society. Goals proposed for prevention more recently by Levine and Smolak (2006) emphasize critical analysis of issues such as power and objectification, encouraging direction from the participants in the program by creating safe spaces where girls and women can develop their own activism, and working to change the school or other setting to promote a healthy, rather than harmful, environment.

Conclusions and Future Directions

In conclusion, feminist theories offer a perspective that challenges current biomedical explanations of eating disorders by implicating Western culture and ideals in the etiology of eating psychopathology. By examining these theories and recent supporting research, we allow ourselves a much more comprehensive view of disordered eating as a whole that suggests proactive possibilities for understanding, treating, and preventing these devastating disorders.

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20

Psychodynamic Theory of Eating Disorders

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Psychodynamic theory in the 21st century is a pluralistic field, enabling clinicians, researchers, and educators to understand and to assist patients in their recovery with a variety of tools and techniques. These tools include empathic understanding, clarification, confrontation, and interpretation of the individual's suffering that has ensnared them in often beguiling and intertwining symptoms that thwart full engagement in life.

Most commonly psychodynamic theory is employed in a one-to-one interaction between patient and clinician in an office setting, but it may also be adapted to therapeutic groups, especially in residential and intensive outpatient settings. The principles of psychodynamic theory are particularly helpful in consultations to individual clinicians or multidisciplinary teams when the patient has not responded to trials of standard treatments or when a stalemate has occurred in the team. For example, a therapist or one or more members of a multidisciplinary team (see Chapter 50) may feel confused, overwhelmed, overinvested, defeated, or simply unsure about what steps to take next. Psychodynamic theory asserts that these reactions provide invaluable information about the patient and what is happening beneath the surface manifestations of the specific problem area. The patient may be unable to articulate in words a feeling or an important part of the history that has been projected into or contained by the clinician(s) involved in the treatment. It is the goal of consultation using psychodynamic ideas to entertain possibilities about what has transpired and why, to begin to formulate key dynamics that may guide the treatment going forward, and to offer in a benign and confidential setting some pragmatic suggestions or course corrections to the clinician(s).

This chapter aims to briefly describe those core features of psychoanalytic theory that, when applied to patient care, offer an important perspective in understanding the patient and his or her symptoms in the unique context of that person's life. Focus is placed on the role of transference and countertransference (i.e., the therapeutic relationship); development and family dynamics; contemporary research; psychological meanings of the eating disorder (ED) symptom; symbolic roles of food and body image; resistances to improvement in a substantial number of ED patients; and the necessary establishment of a core sense of self during treatment to promote full recovery.

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Key Principles of Psychodynamic Theory That Guide Treatment

The keystones of psychodynamic theory were developed and expanded upon by Sigmund Freud and his followers throughout the 20th century and continue to the present. The three principles are (a) the role that the dynamic unconscious plays in emotional life; (b) the repetition of psychological and behavioral themes based on the dynamic unconscious that ensnare the individual in cumulative, refractory, and self-defeating patterns of living; and (c) the importance of the therapeutic relationship in understanding the patient and guiding the treatment (Zerbe, 1993a, 1995, 1998, 2001, 2008).

Emphasis in contemporary psychodynamic theory is also placed on understanding the person as a unique human being, whose symptoms arise from a variety of factors, including but not limited to attachment problems in infancy, childhood maltreatment, deprivation, trauma, family dynamics and secrets, problems in modulating affect, the culture and society, unconscious conflicts, and obstacles to moving successfully through the essential and expectable phases of the developmental life cycle (Banker, 2012; Brody, 2002; Farrell, 2000; Zerbe, 1993a, 1995, 2007, 2010). Many tenets of psychodynamic theory, such as "the subconscious," are now so ubiquitous in the culture as to no longer be considered novel or radical (think of how many times one sees or hears the term "subconscious" alluded to in a movie or print format) and have been widely and unabashedly incorporated into other therapeutic techniques. Clinicians employ them without being aware that we are doing so (Kandel, 2012; McWilliams, 2004; Tobin, 2012; Zerbe, 2010).

The Therapeutic Relationship

In practicing from a psychodynamic perspective, the clinician will place particular emphasis on what transpires in the patient-therapist relationship. That is, the clinician will help the patient observe how what happens with the therapist in session is a new edition of what has occurred in past or current relationships (i.e., analysis of the transference relationship) and encourage the patient to bring their thoughts and feelings about the therapeutic relationship directly to the session for mutual study and understanding. The therapist will also make use of her or his own feelings, thoughts, and fantasies that emerge when working with the patient to glean additional appreciation of the patient's unique conflicts, strengths, deficits, and defenses (Cloak & Powers, 2010; Kernberg, 1995; Zerbe, 1998, 2008). Traditionally, what the therapist brings to the therapeutic encounter and how she or he responds to what the patient brings in is called the countertransference.

Countertransference responses engendered in the therapist will not usually be shared with the patient but made use of more implicitly to guide the treatment. For example, the therapist may feel interest in but also frustrated by a patient with bulimia nervosa (BN) who has made gains in the treatment but suddenly seems to be in a stalemate or plateau phase. Openness to countertransference feelings enables the therapist to consider if this patient has had a setback or is on the cusp of revealing something new about hidden shame or secrets regarding purging rituals (Zerbe, 2008) or is preparing to take a next developmental step forward, such as beginning graduate school or risking greater honesty with thoughts and feelings in personal relationships.

In contemporary psychoanalysis, the interplay of transference and countertransference responses is viewed as a ubiquitous, ongoing, and evolving aspect of the therapeutic alliance. Transference and countertransference are part of a system, in which each person has influence on the other by bringing their "unique, idiosyncratic…[and] universal" (Lachmann, 2000, pp. 192–193)

reactions and feelings to the new relationship. In this way, the therapeutic encounter is an open system that is bidirectional, meaning that each participant contributes to what transpires in the therapeutic field. The fact that the psychotherapeutic relationship is coconstructed (Lachmann, 2000) still leaves room for what the therapist may glean about the patient from the therapist's own thoughts, feelings, and even physical reactions that emerge in a session (countertransference responses) but is enlarged upon by what the therapeutic pair create together that engenders change and personal development.

Contemporary Research on Psychodynamic Therapy of Eating Disorders

Research on the application of psychodynamic theory to the treatment of persons with EDs is limited but growing (Bachar, Latzer, Kreitler, & Berry, 1999; Dare, 1997; Dare, Eisler, Russell, Treasure, & Dodge, 2001; Thompson-Brenner, Weingeroff, & Westen, 2010; Tobin, 2012; Tobin, Banker, Weisberg, & Bowers, 2007). This research is particularly important because most studies in the ED field have a short-term focus on symptom relief alone and do not address the quality of life concerns of patients and their loved ones. A significant minority of patients may recover with a short-term, behaviorally based or educational intervention, but a majority will continue to struggle with maintaining a reasonable BMI, refraining from purging, basing an excessive amount of their self-worth on attaining and then maintaining a particular shape and weight, and finding a sense of personal identity outside the one they derive from having an ED.

Getting at the issues that underlie the ED and attempting to rectify them are key components of what psychodynamic theory aims to help clinicians do. Moreover, there is an expanding database in child and adult psychiatry to support the contention that longer, more intensive psychotherapy based on psychodynamic principles, derived from psychoanalytic theory, is cost effective and may afford a cost offset in conditions such as depression, anxiety disorders, personality disorders, somatic and pain disorders, and substance abuse disorders, even though this form of treatment may take longer than short-term or manualized therapies (Berghout, Zevalkink, Katzko, & de Jong, 2012; Dancyger, Krakower, & Fornari, 2013; Lazar, 2010). More intensive therapy is not, as Lazar, Sledge, and Adler (2010) opine, inexpensive and may not necessarily save money in other treatment costs. There is increasing evidence, however, that psychotherapy works and:

> can at times provide a significant cost offset in other medical and hospital expenses, and is not overused or abused by those truly in need…[Psychotherapy] does provide effective medical help at a cost acceptable to society, both in comparison with other effective treatments for the same condition and in comparison with medical treatments for other classes of medical disorder. (Lazar et al., 2010, p. 10)

Given that EDs cause chronic medical illness, place patients at risk for sudden death, and incur long-term expense because of the medical oversight needed to treat the physical consequences of starvation, purging, yo-yo dieting, and so forth (see Chapters 14 & 52), society may be particularly well served by investing in intensive psychotherapy for these conditions. EDs are the quintessential psychosomatic disorder that pits mind against body (Zerbe, 1993a, 1995); even though the curative factors of psychotherapy have not as yet been delineated, intensive psychotherapy has the potential to reduce suffering, reduce cost, increase quality of life, and, in some cases, save life.

To date there have been three trials of psychodynamic therapy for anorexia nervosa (AN) (Bachar et al., 1999; Dare et al., 2001; Treasure et al., 1995), one for BN (Bachar et al., 1999), and one for BED (Tasca et al., 2006). The importance of attachment to the therapist (transference), reduction in internal self-criticism, positive support for the development of the core self, and scrutiny of maladaptive interpersonal relationships were factors that emerged as potentially ameliorative of entrenched ED symptoms and improved quality of life. These findings support the clinically observed phenomena of insecure or dismissive attachment patterns (Candelori & Ciocca, 1998; Tasca, Ritchie, & Balfour, 2011; Treasure, Corfield, & Cardi, 2012; Ward et al., 2001), a relentlessly punitive superego (Bers, Blatt, & Dolinsky, 2004; Teusch, 2012), and sadomasochistic enactments in relationships (Zerbe, 1993b) as core issues that a psychodynamic approach addresses. Patients with AN and BN struggle with psychosomatic dissociation from their bodies that leads them to disavow the life-threatening nature of their symptoms and to engage in repetitive, cyclical, but ultimately self-destructive relational patterns that are ameliorated within the safe, encouraging, and consistent presence of the therapist.

The majority of clinicians who use psychodynamic methods in their practice will combine them with other forms of contemporary treatments, including motivational (see Chapter 63), cognitive behavioral (see Chapter 56), psychopharmacological (see Chapter 59), and spiritual approaches (Tobin, 2012; Tobin et al., 2007; Zerbe, 2008, 2010). Longer, more intensive treatments (greater than 25 sessions) for patients whose symptoms do not readily improve with a more limited intervention tend to be more psychodynamic in scope (Tobin, 2012). Those patients who do not improve with a more limited, behaviorally based intervention are inclined to have histories of trauma (see Chapter 34), comorbid problems with anxiety, depression, dissociation, or psychophysiologic illnesses (see Chapters 15 & 54), interpersonal and family of origin conflicts (see Chapter 62), self-esteem, self-perception, and self-regulation disturbances (see Chapter 57), and ego deficits. Achievement of a sense of effectiveness, mastery over intense and heretofore unmanageable affects, understanding the roots of self-loathing, and promoting greater competence in regulating interpersonal relationships are fertile soil for psychodynamic methods (Thompson-Brenner & Westen, 2005).

Further research on the efficacy of psychodynamic treatments for EDs is warranted (Dancyger et al., 2013; Thompson-Brenner et al., 2010). This research must include naturalistic studies as well as controlled trials and should address quality of life concerns (Cloak & Powers, 2010; Noordenbos, 2011; Tobin, 2012). Substantial gains in psychodynamic psychotherapy appear to be generated by assisting patients to face split off denial of the ED symptoms and disavowed aspects of the self while enhancing the capacity to form secure attachment relationships and to use words instead of action (symbol formation; Tasca, Ritchie, & Balfour, 2011). Secure attachment develops within the nexus of the therapeutic relationship where previous problematic relationships are explored and the patient is emboldened to integrate various self representations, process complex emotions (especially those related to getting better), and struggle with normative ambivalence toward others, especially the therapist.

The Symptom as Expression of Emotional Pain

Psychodynamic theory looks at the patient's ED symptoms as an expression of emotional suffering that the individual resists becoming conscious of and addressing directly and fruitfully by taking stock of his or her personal situation. Instead, these individuals cope with life's

inevitable travails through restriction, binge eating, purging, and the like. For example, the intense fear of gaining weight that anorexic patients experience may be motivated by drives, needs, expectations, and hopes that the person is not aware of consciously. When patients can begin to discuss their personal history in depth and consider why they have placed so much importance on body shape and size, disturbing emotions frequently arise.

Few individuals wish to face what is painful and potentially dysregulating to the personality structure, so they resist doing so. Psychodynamic theory stresses that the way out of what causes emotional pain is to face it directly in the context of a safe and sustaining relationship with the therapist. Keeping psychodynamic principles in mind in clinical practice offers the patient a perspective that the ED symptom has meaning and, while behavioral methods of care may temporarily help control it, only by facing one's conflicts, developmental deficits, traumatic events, and abandonments can health be ultimately restored and psychological development proceed more normally.

This treatment principle derives from "a fundamental feature of human psychology and behavior" (Auchincloss & Samberg, 2012, p. 226) that psychoanalysts call the repetition compulsion. Freud (cited in Auchincloss & Samberg, 2012) observed that human beings have an "uncanny way of repeating the same unhappy scenarios, as if fate itself were conspiring to make the person unhappy" (p. 226). In clinical work with ED patients, clinicians are often mystified and may feel defeated after serious efforts have been made to educate patients about the life-threatening aspect of EDs and to effect change by motivational and cognitive-behavioral strategies. The compulsion to repeat in an ED patient can be as concrete as engaging in repeated episodes of vomiting even though the individual knows that the symptom does not, for example, lead to weight loss or ablate angst or anger for very long. Repetition may also take the form of a more symbolic or dramatic recreation that is part of the individual's personal history. For example, a patient refuses to gain weight after much effort on the part of therapists and intensive residential or outpatient treatments. At a pivotal point in adolescence, she was told that her developing body would lead to a significant loss, such as a curtailing her gymnastic career or tantalizing those outside of her immediate family nexus. Despite efforts to help the patient understand over time that loss is an inevitable fact of life or that her family members loved her but held distorted views about sexuality, gaining weight is eschewed because it symbolically means excommunication from a profound attachment.

The clinical chestnut that a "therapist must give something to the patient before the patient can give up anything" derives from a simple understanding of the compulsion to repeat, or more colloquially, how all human beings hold on to patterns, even self-destructive ones, to avoid (in the unconscious mind) something worse from happening. This is one reason that the working through process, illustrated by consistent, significant symptom reduction, often takes a very long time.

Psychodynamic theory asserts that an entrenched symptom is one way an individual may attempt to master a painful situation from the past, especially after neglect or trauma, and, furthermore, is "a defense against remembering and a form of remembering through action" (Auchincloss & Samberg, 2012, p. 226). Hence, clinicians applying psychodynamic theory to a patient who has not improved in treatment using other modalities would ask themselves questions such as: Is there experience of trauma or maltreatment that is being acted out repeatedly in the here and now? Does the patient hope to gain mastery of a situation of which they are not consciously aware? Is the emphasis placed on being a particular weight or size a communication from the past wherein the patient attempts to

overcome loss, capitulate to caretaker wishes, emancipate from insidious caretaker demands, or punish the self out of guilt and/or shame? Working through aspects of the repetition compulsion in the treatment setting enlivens the transference/countertransference situation and is essential for therapeutic action.

The Principle of Multiple Function: One Symptom, Many Roles

Another basic tenet of contemporary psychodynamic theory is that there is never a "one size fits all" approach to treatment or to a full comprehension of how the patient's difficulties began and why they are often tenacious. As the therapist works with the patient and resistances are overcome, both members of the dyad are struck that there is a multiplicity of forces or causes that led to the problem.

This principle of multideterminism or multiple function is one of the most complex but useful to emerge in psychoanalysis and has considerable implication for thinking about eating disorders. For example, a patient with AN may struggle with concerns about becoming a separate individual from her parents, as Bruch (1973, 1974, 1978) demonstrated decades ago. This useful clinical observation has led to unfortunate simplification and stereotypes. Instead of taking into account the variety of childhood, adolescent, and adult historical facts that may be implicated in the etiology of the eating problem, the primary caretaker, usually but not always the mother, is accused of being enmeshed with the patient. Mother is viewed as thwarting growth for her own needs. Consequently, the patient-to-be fails to successfully traverse the crucial periods of separation/individuation in the toddler and early adolescent periods. Little wonder that psychodynamic theory has been viewed as "blaming the mother" for the patient's difficulties; nowhere in this picture are the father, siblings, peer, or group relationships considered!

Personal history as it emerges in treatment is viewed as a narrative approximation of historical truth that changes and often deepens over time. Thus, the clinician cannot rely on only one formulation when listening, as the patient's story unfolds in a treatment process that often takes several years. Rather, classical Freudian ideas of drive, defense, and conflict (Druck, Ellman, Freedman, & Thaler, 2011; Rothstein, 2010; Zerbe, 2001), ego psychology (Goldberger, 1996; Pine, 1990; Wilson, 1992), object relations (Hughes, 2004; Zerbe, 1993a, 1995), feminism (Prozan, 1992; Zerbe, 1995, 2008), self psychology (Teicholz, 1999; Zerbe, 1998, 2007, 2008), intersubjectivity and affect theories (Fosha, 2000; Lachmann, 2000; Orange 2011; Zerbe, 2010), and Lacanian perspectives (Hamburg, 1999; Ruti, 2009, 2011) are synthesized and amalgamated to arrive at a more holistic and elaborate formulation of the individual patient's quagmire with the ED.

Each psychological explanation uncovered in the therapy is a part of the whole and important to appreciate for a more comprehensive understanding. For example, applying ego psychology, the clinician will look at symptoms as compromise solutions for a burdened ego that found tenuous but salutary relief in disturbed eating and then assist that patient in learning healthier coping mechanisms. Making use of object relations theory and studies based on attachment research in infancy, the patient referenced above may also find during a psychodynamically oriented therapy process that there were struggles with primary caretakers or siblings that played a role in the etiology of the problem. Turning to restrictive eating may have served the function of quelling the stirring of sexual or aggressive drives or feeling states that had heretofore been unconscious and thus rendered unmanageable.

Combining principles and clinically focused techniques of conflict and affect theory, the clinician works to make the unconscious conscious and to transform into words the affects that were previously submerged and distorted.

What is essential to underscore is that neither the patient nor therapist will know at the outset of treatment all of the possible psychological explanations, motivations, or defensive needs that are part of the manifest symptoms, but as treatment proceeds they will arrive at a fuller understanding of them. With such appreciation can come greater self-acceptance, forgiveness of oneself and others for past failure, acknowledgement of powerful feelings, an increased capacity to manage those feelings, and an enhanced sense of one's unique selfhood and well-being. In psychological terms, the patient may be said to have developed a coherent sense of self that will not be dominated by the ED as a way of life and will enable sufficient "interactions that affirm individual self-coherence and at the same time create or consolidate necessary bonds between individuals" (Teicholz, 1999, p. 57).

As feminist psychodynamic psychotherapist Charlotte Prozan (1992) wisely declared, "You can't have change without struggle. You can't plant seeds without turning over the earth" (p. 334). Psychodynamic theory aims to uproot well-established but pathological modes of coping in order to sow sufficient seeds for the individual's ultimate growth. Patients will have made significant progress when they can bear disparate feelings states such as sadness, fear, futility, isolation, anger, and joy in the presence of another with minimal dysregulation. They will also have a greater sense of their own boundaries, limits, and limitations and a lesser need to strive for perfection or to seek the unattainable from others. In summary, they lose their quest for an ideal but find the real.

The Principle of Complemental Series: Valuing the Roles of Nature and Nurture

As much as psychoanalysis is sometimes lampooned as a specious method that constructs beliefs about the mind that can never be proven, in fact the discipline has never left its original roots in natural science and evolutionary biology and attempts to integrate them with the humanities and the prevailing cultural milieu (Kandel, 2012; Sulloway, 1979). As a neurologist, Freud championed the method of investigation that takes into account observable and reproducible facts. He also believed that all human behavior and symptoms derive from a combination of constitutional factors (e.g., heredity, biology) and experience (e.g., family, events, culture). This "principle of complemental series" continues to the present in psychoanalysis; it maintains that there is a complex layering of factors in any illness, and that the patient is well served only when "the interactions between biology and the environment in shaping behavior" (Auchincloss & Samberg, 2012, p. 38), including temperament, culture, caretaker responses, early and later childhood events, childhood or adult maltreatment, deprivation or trauma, and development over the entire life cycle, are taken into account in the treatment formulation. Psychodynamic theory sidesteps the ongoing nature/nurture debates in contemporary psychiatry and instead maintains that both are important and essential for understanding and helping the patient as an individual (see Chapter 67).

With respect to EDs, psychodynamic theory takes heed of the biological bedrock of the starvation state, the irrefutable evidence of neuroscience in assessing the brain-based differences between patients with an ED and controls (see Chapter 17), and the number of studies that demonstrate a robust hereditary factor for the illness among some patients (see Chapters 28 & 30). While these

factors play a necessary role in planning and executing comprehensive therapy, they are not considered sufficient to adequately explain etiology and facilitate change because they leave out a congeries of personal, family, and cultural factors in which the illness came to fruition.

For example, a patient whose personal and family history is highly comorbid for obsessive-compulsive disorder (OCD) will be given short shrift in treatment if the meaning and role of the symptoms are not thoroughly sorted out. In such a case, the patient's AN and OCD may be highly biologically determined and modified by pharmacologic and behavioral treatments after target weight is restored. If this person's symptoms remain refractory (as they often do), psychodynamic theory would propose that the meanings and roles of the symptoms in the patient's life also be deconstructed. Often in such cases, for example, the obsessive defenses are found to serve psychological functions wherein the patient binds anger and aggression or attempts to control powerful impulses and maintain a sense of order in what is experienced as an unmanageable and disorganizing environment.

It is also increasingly common to find EDs occurring among different generations in the same family. In the 1960s and 1970s, EDs were considered to be largely illnesses of adolescents or young adults who wanted to be thin and collude with a cultural ideal shape. Etiology is now viewed as much more complex, and as women have aged into their 50s, 60s, and 70s, a new population of EDs in middle-aged and older adults is entering treatment (Lapid, Chen, Rummans, McAlpine, & Zerbe, 2013; Zerbe, 2002, 2008; see also Chapter 36). The clinical histories that these patients tell are often complicated and challenging to unravel. For example, a grandmother, mother, father, aunts, and children in a family may share features of AN or BN. Although genetic and epigenetic studies suggest that a hereditary or identifiable biological factor is singularly important in the etiology of the problematic eating, interviews with each family member may trace the disturbance to a preoccupation with size and shape that began in a previous generation. Each person's account of the family's history will be different and nuanced but a theme that emerges in the therapy process is how seriously the prevailing cultural ideal or demand by a powerful figure in the family to be a certain weight and size was incorporated by all who developed their own body image and eating problems. Consequently, there appears to be a psychological and cultural pressure that augments any biological bedrock toward disordered eating and body image distortion in this example.

Psychodynamic theory, by its inclusion of the complemental series principle, takes the genetic, environmental, and family history roles into account and uses them to engage the designated patient in treatment. The therapist often finds that identification with figures in one's family of origin may be a key contributor to the symptom, as when one wishes to "look like" or "be just like" an admired figure. Sometimes the process of identification is conscious but it usually remains unconscious until well into a psychodynamic therapy process. Loss may play a role with circular and obsessive thoughts serving the function of avoiding guilt, regret, and grief. Eating rituals and avoidance of intake may occur because mealtime was a source of tension at home or the primary caretakers had their own conflicts with food preparation, the messiness of the feeding situation with infants and children, or a history of imposed starvation in their youth (e.g., survivors of torture, holocaust, or severe poverty situations).

Understanding the Meaning of Food and Eating for the Individual

Feeding is a central activity from birth through all phases of the life cycle; it has enormous psychological significance because in the earliest infant-caretaker relationship eating not only reduces physical hunger pangs but also brings with it psychological gratification, comfort, and

pleasure. In the unconscious, food is equated with love (Glucksman, 1989; Zerbe, 1993a, 1995), and the "unconscious equation" of mother and food "remains intact" (Glucksman, 1989, p. 151) even as separation and individuation proceed through toddlerhood and well into adulthood. Psychodynamic theory attempts to take a closer look at the meanings of eating and body image and the role food may itself play in the life of the person who has an ED. In essence, food and eating are important topics of conversation and an essential resource for gaining access to the unconscious life, memories, conflicts, developmental strivings, and family dynamics of the patient.

For example, one patient with BN explained in session that she turned to food for self-care (and actually self-survival) because her parents had to leave her with babysitters in her youth because they each worked two jobs to support an otherwise impoverished family. Staples of various foods were readily available in the babysitters' homes where she was otherwise ignored or dismissed as she watched hours of TV alone and with few playthings to provide comfort. This patient eventually attempted to rid herself of the anxiety and shame she felt by overeating, vomiting, and exercising excessively. She married early to a man who was "a great cook" because she equated his lavish meal preparation with love but she continued to purge. When her husband discovered her long-held secret ritual, he insisted she enter treatment. Learning about her food, eating, and purging rituals became an illuminating source of information about her internal sense of self-depletion and inner emptiness. Compulsion to repeat her self-destructive eating rituals gradually diminished as buried feelings and impulses were faced with greater maturity and sense of mastery.

Development of a Core Self

Historical Perspectives

Pediatrician and psychoanalyst D. W. Winnicott's (1965) concepts of the True Self and the False Self are well known to mental health practitioners of diverse clinical and theoretical backgrounds. These are not only highly evocative concepts that students intuitively grasp, they are ubiquitously played out in everyday life and clinical encounters.

As a practicing physician, Winnicott observed thousands of mother–child interactions over his long career, and he wrote and spoke to lay audiences in Great Britain in the 1940s through the 1960s about healthy practices in childrearing. Winnicott (1965) observed how the natural life force of the individual can be dampened down and even completely disrupted if the environment does not welcome and help elaborate the individual's "spontaneous gestures." Parents are called upon to be resilient when their child moves away from them or becomes temporarily rejecting. If the parent provides a "good enough" environment, the child will develop a "True Self" that feels recognized, is creative and imaginative, and feels alive (Bornstein, 2013; Phillips, 1988; Winnicott, 1965). When a child is deprived of these psychological nutrients, a "False Self" may develop that "has three functions: it attends, within severe limitations, to the mother; it hides and protects the True Self by complying with environmental demands; and it is also a "caretaker…taking over the caring function of the environment that has failed" (Phillips, 1988, pp. 133–134).

The concepts of True and False Self have been applied to understanding many different kinds of psychopathology in the late 20th and early 21st centuries. As Winnicott observed, disruption to the sense of safety or continuity in the mother-infant dyad leads to later

breakdown of mind or body; hence, his theory has considerable applicability in understanding ED from a psychoanalytic perspective. For example, the body symptoms of restriction, purging, overexercise, overemphasis on physical perfection, and so forth, can be formulated in some cases as False Self phenomena. When speaking about aspects of the False Self in this way, we are actually veering away from seeing the concept as a useful metaphor of the mind or an explanation for faulty psychological functioning alone and extending the concept to the soma.

ED symptoms provide a self-protective function for many patients; physical symptoms are experienced as necessary to protect the individual from dissolution and thereby create a false sense of security. When these adaptations to life are disrupted, as when a physical illness occurs, what ensues is psychological breakdown or a return to more overt and potentially lethal symptoms (Zerbe, 1992). In fact, ED patients are actually exceedingly anxious about making emotional contact with others and experiencing normal human dependency needs (Sands, 2003). Patients can sometimes also become attached to the therapist in a symbiotic, enmeshed fashion and are experienced by the therapist as intrusive and demanding. All the while, the patient may make limited progress, yet treat the therapist as if essential to that patient's life.

Patients with EDs who struggle with a False Self will have significant difficulty in negotiating the inevitable tension between autonomy and mutuality in relationships. The therapist must help the patient to normalize the yearning for relationship and integrate this with a separate sense of self. In this way, the False Self may gradually give way over time, and the patient can establish a sustained sense of separateness in the presence of another person. The False Self may become a shroud for a sense of death in life that is responsible for countertransference reactions of ennui, despair, deadness, and psychophysiological reactions. One psychodynamic caveat is to focus on ED symptoms and the consequences of poor self-care. As Sands (2003, p. 108) has commented, "Because the patient's deepest needs are sequestered in her body, *the therapist must remember the body.* If we forget the patient's body, we forget the child in the patient" [italics in original].

Contemporary Views

All persons strive to have and to maintain a sense of a cohesive or core self. Paradoxically, there are those times when we experience a sense of discontinuity from our core self. For example, one notices how one speaks differently about the same issue or concern to one's significant other than one does to a colleague, friend, or child. Especially at those times when we must face the inevitable losses that accompany life or undergo a significant personal or professional transition, we rely upon these "seemingly discordant portrayals of the self" (Mitchell, 1991, p. 125) because our sense of self is under a significant strain and simultaneously called upon to function at a high level. Persons with a cohesive sense of self can weather these inevitable storms of life reasonably well and often creatively because we call upon the resources in our environment to shore up our subjective experience of strain. Kohut (1984) called these environmental resources "healthy selfobjects" after observing clinically how individuals use the arts and their empathic, sustaining relationships with others to buttress a flagging sense of self-worth. The important emphasis is that all individuals need and use selfobjects in our daily existence to function at the levels that we do.

ED patients are prone to episodes of dissociation or multiple, contradictory self states more often than others, and this causes them significant angst and problems in relationships. At the beginning of treatment they do not have the resources that Kohut found essential to

emotional health and well-being. They cannot sustain a sense of a core self because the healthy selfobjects in the environment are found wanting. Hence, the therapist is called upon to fulfill this role by empathic resonance with the patient that will, over time, help build a more cohesive sense of self by capitalizing on the patient's "search for the real self" (Masterson, 1988), development of a "true self" (Winnicott, 1965), or quest for a "new beginning" (Balint, 1968). These psychoanalytic terms connote the highly complex but profound importance placed on diminishing the frequency of dysregulating dissociative episodes and negative affective experiences in order to enable a healthy core self to emerge and to mature.

The establishment of a core self is not a static process. Contemporary psychoanalytic theory is informed by neuroscience and by empirical analyses of the caretaker–infant interaction. Integrating this research into practice has led to an understanding that the therapeutic relationship is an open system in which the "disruption and repair" cycles that go on between patient and therapist may be the most important moments for growth of the core self (Beebe & Lachman, 2002). In actuality, both participants in the therapeutic dialog are likely to change over time because a bidirectional and mutual interaction facilitates a flourishing of the sense of a cohesive self.

Because the case histories of persons with EDs include instances of trauma, maltreatment, misunderstanding, and dysregulation of affect, therapeutic openness to error and jointly constructed narratives may be the facilitator for creating change. A humble demeanor and willingness to admit error and make course corrections is an attitudinal shift wherein the therapist can withstand the experience of "not knowing" and, instead, enable "heightened affective moments" and "a joint construction of a new ongoing regulation" (Beebe & Lachman, 2002, p. 197) to take center stage. A sense of a core self emerges more readily because mistakes in understanding are welcome, and the therapist eschews perfectionism and idealization. Therapeutic errors leading to feelings of rage, defeat, and despair occur but "the meaning of the rupture" that leads to repair is central to ongoing engagement and "increased self-regulatory capacity" (p. 206) for the patient.

This interactive model of therapeutic change links neuroscience to psychoanalysis in a manner that is exceedingly plausible and sanguine for patient care and its scientific study. Beebe and Lachman (2002, p. 228) summarize how the core self develops out of an interactive psychotherapeutic model of mind:

> Equal emphasis is placed on both sides of an interactive model; the brain influences behavior, but experience alters the brain. Tremendous neural diversity, with variability in size, shape, type, and connections of cells, insures that every brain is different…One implication of this model is that there is no fixed schema or representation of a stimulus. The representation of a stimulus is always being updated, "reassembled" as a function of organism arousal, context, and experience. The mind updates its maps. (p. 228)

Conclusion and Future Directions

Psychoanalytic ideas derive from a theory of the mind that informs how symptoms develop and are understood in a unique human being. Psychoanalytic theory emphasizes that all symptoms have conscious and unconscious meanings that, when grappled with by the individual, lead to mastery and improved control over that specific symptom. With respect to etiology of

EDs, childhood maltreatment and abuse, struggles in development and aging, problems with self-regulation, and navigation of relationships with others are some of the major areas that psychoanalytic research and case studies have found crucial in ascertaining what may lie beneath the manifest symptoms of weight preoccupation, body image, and sense of self-worth.

Contemporary psychoanalytic theories of EDs converge to assist the patient in a process of self-transformation. The aim of the clinician is to reduce suffering brought about by the symptom in order for the individual to feel more alive and at peace, particularly with his or her body. Newfound optimism, hopefulness, and creative expression are byproducts of the development of a cohesive sense of oneself, but in psychodynamic psychotherapy, as in life itself, a certain amount of human pain is accepted as a given.

More research in EDs is needed to understand better how change occurs over time by any method of treatment or set of theoretical lenses. With respect to psychoanalytic theory, what is additionally necessary is demonstration of which patients may particularly benefit from such a time-intensive mode of healing that makes it a reasonable cost for our society to bear. ED patients who are able to forge healthier lives that thereby reduce their medical costs and strengthen their capacity to work and to love (e.g., reduction of manifest symptoms; improved quality of life; development of cohesive sense of self) offer robust parameters for the efficaciousness of the psychoanalytic method that must be demonstrated by time-consuming, naturalistic, and expensive clinical outcome studies.

The neurophysiological and neuroanatomical concomitants of personal growth (e.g., as suggested by the emerging research field of neuropsychoanalysis) may be of particular importance in showing how different emotional systems of the brain benefit from psychodynamic psychotherapy or psychoanalysis. An additional benefit of this research for understanding and treating EDs will be to localize the cortical and subcortical brain systems that interact with genetic and epigenetic factors to produce lasting and often overwhelming symptoms. Psychotherapy could then be tailored to address not only any historical, traumatic precipitants but also the tendency toward emotional reactivity and the inability to soothe that are so ubiquitous among patients with EDs. Extant follow-up studies of patients with regulatory difficulties, such as seen in EDs, provide an opportunity to illustrate that the brain is modifiable and that greater affect tolerance, enhanced ability to process information, and attainment of a degree of mastery over historical antecedents of the manifest symptom are central factors of enduring therapeutic change.

As the ability to self-regulate expands, an individual becomes an active agent in the process of change rather than a passive victim of her or his symptoms. This concept is synchronous with emerging neurobiological models of the mind; such a mind can "update its maps" because it is an open system susceptible to positive input in the coconstructed therapeutic relationship. Flexibility and resilience are outgrowths of a psychodynamic approach to ED treatment that eventuate in greater self-acceptance and self-cohesion. These two bulwarks of the human quest are essential for the subjective, creative experience of living life fully.

The application of psychodynamic theories to the treatment of EDs thus provides much more than insight into the etiology of the symptoms or education about past trauma or developmental failure. Psychodynamic treatments aim to make the life of the person more vital and meaningful. This process only transpires over time. This "tincture of time" allows the patient a new opportunity to weather inevitable change and loss, to nurture the capacity to feel happiness and joy, to experience the therapist as bearing witness to the inevitable exigencies of life, to withstand setbacks, and to embrace positive steps forward, any of which may cause dysregulation and a temporary recrudescence of a symptom.

The complexities of psychodynamic theory and practice are well illustrated in the cinematic version of the *Wizard of Oz* (and consequently usefully shared with students or patients attempting to grasp essential points as they embark on learning more about its applications). Dorothy's trip down the Yellow Brick Road led to many detours into the unknown and warnings to turn back! With courage and the help of her friends (in psychoanalytic parlance, her good internal objects and external supports), she pressed forward. She learns on her journey that the Wicked Witch and the Flying Monkeys (her bad internal objects or exploitative, traumatic relationships) cannot defeat her and that Oz is not a wizard but only a man (embodying the human tendency to idealize a person, find a quick fix, adhere to utopian principles, or persist in fallacious symptoms that appear to solve a problem but actually lead to a dead end). In the penultimate scene, Glenda, the Good Witch of the North (representing the good internal object, mother, or psychotherapist) reminds Dorothy that she "has the Ruby Slippers" and has always had them but she needed to undertake her journey in order to unlock her power (i.e., find her True Self). Glenda further explains to Dorothy that without the struggle she would never believe she had the capacity to face all that she did. When Dorothy returns home, she is transformed. Although she has arrived safely and the movie is over, the viewer knows that Dorothy is getting a fresh start and actually starting a new chapter. As in the tale, so in life and in psychotherapeutic practice: The end of one era ushers in new beginnings for the patient's ongoing, enfolding life story.

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21

Sociocultural Theories of Eating Disorders

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A substantial body of research has documented the powerful role that sociocultural influences have on the development and maintenance of eating and body image disturbances (Thompson, Heinberg, Altabe, & Tantleff-Dunn, 1999; Tiggemann, 2012). "Sociocultural" is something of a sponge word, in that it has absorbed many meanings over the years. When we speak of "sociocultural," we mean environmental factors that include the proximate and contextual experiences that an individual encounters on a daily basis that have an impact on how that person views themself physically (body image), how these experiences might modify interpersonal social comparisons (appearance comparisons), and on how that person might modify their thoughts regarding acceptance of prevailing social norms of appearance (internalization of societal ideals). Additionally, these factors can be thought to produce changes in behavior (restriction, purging, etc.) designed to produce a physical body more acceptable to the individual and/or those significant others in the person's life. Sociocultural factors may or may not be similar across countries, genders, or ethnic backgrounds; thus, evaluation of such issues is an important emerging research area (see Chapters 16, 23, 25, & 27).

The particular sociocultural factors that have most often been considered in the area of body image and eating disorders are media and interpersonal variables (Carlson-Jones, 2012; Levine, 2012; see also Chapters 29 & 31). Studies examining media influence have traditionally focused on examination of appearance ideals espoused in magazines, TV, and movies. However, more recently, the images and messages contained on the Internet and social media have begun to be evaluated (Hussin, Frazier, & Thompson, 2011). Interpersonal influences can theoretically emerge from anyone encountered in a social context (e.g., the sexualized gaze of a stranger); however, typically these instances come from family, friends, peers, teachers, coaches (see Chapter 35), or other known associates. These communications may consist of appearance-related comments, modeling of appearance concerns (e.g., a mother who verbalizes her own body image issues to a child), or direct information regarding body concerns or modification (e.g., a friend who suggests a fad diet).

Contemporary media and interpersonal influences exist in a larger cultural context and thus may vary across culture and time. In some Westernized societies, such as the United States, it

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can be argued that the prevailing focus on beauty and thinness for women has been present for roughly the last 50 or so years, with a more current focus on muscularity ideals for men for perhaps 15 years or so (see Chapters 29 & 37). In other non-Westernized countries, such appearance ideals may be virtually nonexistent. Finally, in countries with more exposure to Western culture (Becker's classic studies of the effect of Western media on Fijian body image ideals), the prevailing notions of beauty are in a state of flux (Edmunds, 2012; see also Chapters 6 & 23).

There are literally hundreds of studies that have investigated the role of sociocultural factors on body image and eating disturbances. In recent years, theoretical models have evolved to postulate how these influences lead to body image and eating disturbances, either directly or indirectly by producing changes in dispositional factors such as internalization and social comparison. In this chapter, we review some of the primary theories and indicate directions for future research, along with noting how the findings from these studies have an impact on prevention and treatment strategies.

Tripartite Influence Model of Body Image and Eating Disturbance

The most widely tested and well-validated sociocultural model is the Tripartite Influence Model of body image and eating disturbance (TIM; Thompson et al., 1999; Figure 21.1). According to this model, there are three primary sociocultural sources of influence—parents, peers, and media—that exert their influence on body dissatisfaction both directly and via the mediational processes of appearance comparison (i.e., the tendency to compare one's appearance with that of others) and internalization of the thin ideal (see Chapters 26, 29, & 31). Body dissatisfaction, as the model posits, is linked to dietary restriction and bulimic symptoms. The original model also includes a directional link from restrictive eating to bulimic symptoms, which is linked to psychological functioning (e.g., self-esteem, depression). As noted by Stice (2001b), one of the strengths of this model is its ability to integrate distal sociocultural factors with proximal individual difference factors.

There is a great deal of empirical support for the TIM. In a series of two covariance structure modeling (CSM) studies, Thompson, Coovert, Richards, Johnson, and Cattarin (1995) examined the relationships among maturational status, level of obesity, perceived weight,

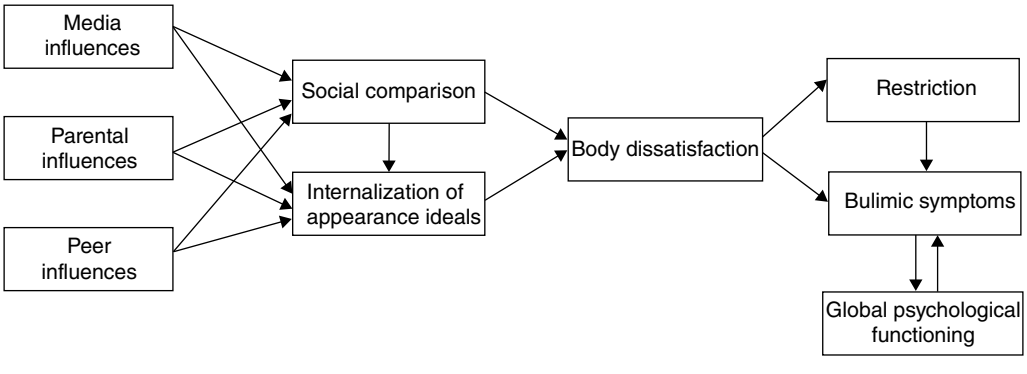

Figure 21.1 Tripartite Influence Model of body image and eating disturbance. Adapted from Thompson, Heinberg, Altabe, and Tantleff-Dunn (1999). Reproduced with permission of APA.

teasing history, body image, eating disturbance, and global psychological functioning (i.e., self-esteem, depression, anxiety). Results from Study 1 suggested a directional effect of teasing history on body image and eating disturbance. Study 2 provided additional support for the effect of teasing and body image on restrictive eating and initial support for the link between (a) bulimic symptoms and restriction, and (b) bulimic symptoms and overall psychological functioning. A subsample of participants from Study 1 completed a 3-year follow-up (Study 3), the results of which provided support for an interactive effect of obesity and teasing on the development of body image disturbance.

The mediational role of appearance comparison was first examined in a study by van den Berg, Thompson, Obremski-Brandon, and Coovert (2002). Results indicated that appearance comparison mediated the effects of family and media influences on body dissatisfaction in college women. Body dissatisfaction, in turn, influenced eating disturbances in the form of restrictive and bulimic behaviors. Unlike family and media influences, peer influences were directly related to restriction.

The TIM has also been supported in preadolescent (Shroff & Thompson, 2006) and adolescent female (Keery, van den Berg, &Thompson, 2004) samples. In an attempt to replicate and extend findings from the Keery et al. study, which utilized a composite sociocultural influence variable, Shroff and Thompson (2006) compared the original model to a modified version of the model in a different sample of adolescent females. In the modified version, each of the components of sociocultural influence (i.e., peer, parent, and media) was considered separately. While peer and media influences were significantly associated with the mediator or outcome variables, parental influences were not. This finding is inconsistent with results from a more recent study of Australian sister pairs (18–25-year-olds; Coomber & King, 2008) that examined pressure and modeling by multiple family members (i.e., sister, mother, and father) and provided evidence for the roles of sisters and mothers as important modeling agents. Interestingly, while sister pressure was linked to eating disturbance via body dissatisfaction, sister modeling had a direct effect on bulimic and restrictive behaviors.

Males

An examination of a modified version of the TIM suggested that parent, peer, and media influences predicted muscle-building techniques (e.g., weight lifting, steroid use) in adolescent boys directly and via social comparison (Smolak, Murnen, & Thompson, 2005). The TIM has also been extended to explain body dissatisfaction in college men. Given the societal emphasis on male muscularity, male body dissatisfaction in these studies is commonly assessed via scales that measure drive for muscularity (e.g., Drive for Muscularity Scale; McCreary & Sasse, 2000) as opposed to drive for thinness (see Chapter 37). Similarly, internalization of the thin ideal is replaced with internalization of mesomorphic ideals (e.g., muscularity, physical fitness). Structural equation modeling (SEM) results provided support for the mediational role of both internalization and social comparison in explaining the relationship between social influences and muscularity-oriented body dissatisfaction (Karazsia & Crowther, 2009).

Karazsia and Crowther (2010) conducted a follow-up evaluation of the TIM with the goal of examining influences on males' engagement in risky body change behaviors (e.g., overtraining). Results indicated that social influences were linked to internalization of the ideal, which predicted muscularity-oriented body dissatisfaction. Body dissatisfaction, in turn, was associated with engagement in body change behaviors (e.g., drinking weight-gain or protein shakes, consuming as many calories as possible per day). In this study, social influences

were conceptualized as mother, father, sibling, and peer encouragement to lift weights or diet to enhance physical appearance. Consistent with the TIM, social comparison served as a partial mediator of the relationship between social influences and internalization. Social comparison, however, was not directly linked to muscularity body dissatisfaction. This finding is inconsistent with theoretical (Thompson et al., 1999) and empirical (Karazsia & Crowther, 2009) work that supports both direct and indirect links between social comparison and body dissatisfaction.

Recently, the male version of the TIM was further refined to (a) include dating partners as a source of social influence, and (b) examine dual body image pathways to body change behaviors: muscularity and body fat concerns (Tylka, 2011; Tylka & Andorka, 2012). Direct pathways between muscularity-oriented body dissatisfaction and muscle-building behaviors, and between dissatisfaction with body fat and disordered eating, were predicted. Internalization of the mesomorphic ideal, but not social comparison, was examined as a mediator between social influences and body dissatisfaction.

The "quadripartite" model fit the data and provided further evidence for the mediational roles of internalization, muscularity-oriented dissatisfaction, and body fat dissatisfaction in explaining the relationship between social influences and body change behaviors in men (Tylka, 2011). Results further supported dual body image pathways to body change behaviors and direct pathways between (a) internalization of the mesomorphic ideal and muscularity enhancement behaviors, and (b) pressure from dating partner to be mesomorphic and disordered eating behaviors, such as dieting, preoccupation with food, and bulimic behaviors. Prior research by Karazsia and Crowther (2010) also supported a direct path between internalization and change behaviors aimed at increasing muscularity. These findings suggest that internalization of the mesomorphic ideal, even in the absence of muscularity body dissatisfaction, is sufficient to promote men's engagement in behaviors to enhance muscularity (Tylka, 2011). Conversely, the relationship between internalization and men's engagement in disordered eating behaviors does not exist in the absence of body fat dissatisfaction (Tylka, 2011).

Tylka and Andorka (2012) also examined an expanded "quadripartite" model in a sample of gay men. This most recent manifestation of the TIM was revised to include gay community involvement as a fifth social pressure variable. Pressures to be muscular versus pressures to be lean were examined individually. Findings provided support for dual body image pathways to body change behaviors. Interestingly, the link between muscularity dissatisfaction and muscularity enhancement behaviors was stronger in the sample of homosexual versus heterosexual men (Tylka, 2011; Tylka & Andorka, 2012). The authors concluded that "gay men may feel more of an incentive to engage in behaviors that promote their muscularity if they feel that they are scrawny and underdeveloped" (Tylka & Andorka, 2012, p. 64).

Non-U.S. Samples

The TIM has also been examined in non-U.S. samples. A slightly revised version of the model supported relationships between social influences and body dissatisfaction, as mediated by internalization of the thin ideal and/or social comparison, in a sample of Japanese undergraduate females (Yamamiya, Shroff, & Thompson, 2008). The model also provided a good fit to data obtained from Australian and French undergraduate females (Rodgers, Chabrol, & Paxton, 2011). Results suggested that the Australian women perceived greater pressure from peers and the media, endorsed higher levels of social comparison and internalization, and reported more bulimic symptoms than their French counterparts (Rodgers et al., 2011).

In perhaps the most recent cross-cultural examination of the TIM, researchers tested a version of the model—revised to include BMI and weight perception and exclude social comparison and bulimic behaviors—in Hungarian boys versus girls (ages 10–16 years; Papp, Urbán, Czeglédi, Babusa, & Túry, 2013). While results provided general support for the model in both samples, significant gender differences emerged. Girls were more likely than boys to perceive themselves as overweight; they were also more likely to engage in dietary restriction than their male counterparts. Boys reported lower levels of body dissatisfaction than girls and expressed a desire to either lose weight or gain weight (see Chapter 37). In the male sample only, body dissatisfaction was not associated with thin-ideal internalization. In both samples, sociocultural influences were directly related to internalization and restriction, and the relationship between BMI and body dissatisfaction was fully mediated by weight perception (Papp et al., 2013). In contrast to prior research in the area (Rodgers et al., 2011; Shroff & Thompson, 2006), the relationship between restriction and self-esteem was not supported in the Hungarian samples.

Dual Pathway Model of Bulimia Nervosa

Stice (1994) developed the Dual Pathway Model (DPM) as an extension of the pre-existing sociocultural model (Striegel-Moore, Silberstein, & Rodin, 1986) by including mechanisms of action hypothesized to explain how a subset of women exposed to sociocultural pressures ultimately develops bulimia nervosa (BN). According to this model (Figure 21.2), individuals receive messages from sociocultural institutions (peers, family, media) that reinforce and perpetuate the thin ideal, the centrality of appearance for females, and the importance of appearance for success. When these messages are internalized, the proposed result is body dissatisfaction. As its name implies, the DPM posits that body dissatisfaction is linked to BN via two pathways: dietary restraint (i.e., restriction in the TIM) and negative affect. Dietary restraint is also linked to negative affect via a unidirectional path.

The DPM is supported by cross-sectional and longitudinal research. An initial test of the model—including perceived pressure from family, friends, and dating partners—revealed that it accounted for 71% of the variance in bulimic symptomatology in female undergraduate students (Stice, Nemeroff, & Shaw, 1996). Consistent with the proposed model, BMI was positively associated with perceived pressure and body dissatisfaction. Perceived pressure was linked to body dissatisfaction both directly and via internalization of the thin ideal. Both pathways to BN, dietary restraint and negative affect (e.g., guilt, anxiety, depression), were significant, as was the pathway between restriction and negative affect. An unexpected direct path between perceived pressure and dietary restraint was revealed. This finding is particularly salient, as it suggests that sociocultural pressures to be thin can lead to restriction even in the

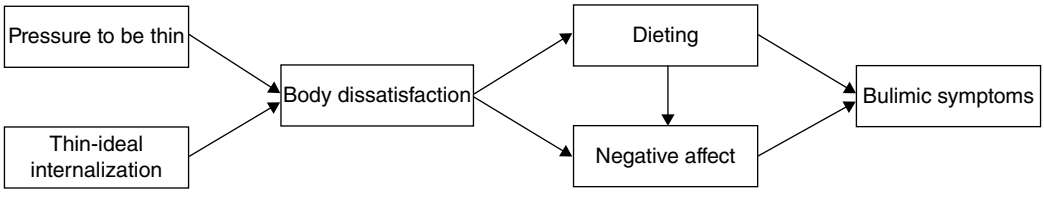

Figure 21.2 Dual Pathway Model of bulimia nervosa. Adapted from Stice, Nemeroff, and Shaw (1996).

absence of internalization and/or body dissatisfaction. Similar results have been obtained in a female sample of high-school and university students when different measures were used to assess dietary restraint (Shepherd & Ricciardelli, 1998).

Although the first empirical test of the model was cross-sectional in nature, prospective tests of the DPM have since been conducted to provide evidence of temporal precedence and elucidate the direction of effects. Stice, Shaw, and Nemeroff (1998) measured predictor variables at Time 1 (i.e., BMI, pressures, internalization, body dissatisfaction, dietary restraint, and negative affect) and bulimic pathology at Time 2 (9 months later) in a sample of adolescent females. Both the restraint and negative affect pathways evidenced prospective effects on bulimic symptoms. A second 2-year prospective test of the model suggested that baseline pressures to be thin and internalization were associated with growth in body dissatisfaction (Stice, 2001a). Additionally, baseline body dissatisfaction predicted increases in dieting and negative affect, and initial dieting and negative affect predicted increases in bulimic pathology in adolescent girls.

The proposed relationships are less straightforward in adolescent boys. Research suggests that adolescent boys "aspire to two contrasting and seemingly opposite body size ideals" (Ricciardelli, & McCabe, 2001, p. 1317; see also Chapter 37). For boys who wanted a thinner body, the relationship between body dissatisfaction and bulimic behavior was fully mediated by negative affect. For boys who wanted a bigger body, body dissatisfaction and dietary restraint were unique predictors of bulimic behavior. The authors concluded that this group of boys may engage in dietary restraint and binge eating in an attempt to achieve the mesomorphic ideal.

The DPM has also been applied to samples with varying levels of bulimic symptomatology (Stice, Ziemba, Margolis, & Flick, 1996). An evaluation of the model in three female samples suggested that those with BN reported higher levels of perceived sociocultural pressure, internalization, body dissatisfaction, negative affect (particularly sadness and guilt), and dietary restraint than controls (i.e., women who did not report binge eating, compensatory behaviors, or concern with body shape or weight), despite no significant differences in BMI. These findings provide support for both the model and the continuity hypothesis, which posits that bulimic symptomatology exists on a continuum—the endpoint of which is BN.

In a more recent study, the original model versus an extended version of the model was tested in a nonclinical sample of adolescent girls and a clinical sample of women with eating disorders (van Strien, Engels, van Leeuwe, & Snoek, 2005). In this study, the model was extended to include interoceptive awareness and emotional eating as mediators of the relationship between negative affect and overeating/binge eating. Both models fit the adolescent data well, with the original versus extended model explaining 9% versus 12% of the variance in binge eating. Only the extended model exhibited a good fit in the clinical sample; it accounted for 61% of the variance in binge eating. Results suggested that, in clinical samples, a lack of interoceptive awareness and emotional eating mediate the relationship between negative affect and binge eating. The negative affect pathway, but not the restraint pathway, was supported in both samples. Although the results do not appear to support the continuity hypothesis (see above), it is unclear whether the differences between the samples are attributable to age or patient status (van Strien et al., 2005). Support for the extended model has also been provided by research that has operationally defined overeating as the amount of food consumed during a taste-test (Ouwens, van Strien, van Leeuwe, & van der Staak, 2009).

Objectification Theory

Objectification theory (Fredrickson & Roberts, 1997; Figure 21.3) draws upon sociocultural and feminist perspectives of body image to offer a framework for understanding how the cultural construction of gender roles may contribute to the observed high levels of body dissatisfaction and disordered eating among women in Western societies. Objectification theory posits that Western societies socialize young women and young men differently with regards to the importance of their physical appearance. While males are taught the importance of strength, confidence, and intelligence, females are taught that appearance and sexual attractiveness are the primary determinants of their social value (Calogero, Tantleff-Dunn, & Thompson, 2011; see also Chapters 19 & 27). Messages emphasizing the primary importance of a woman's sexuality are communicated and reinforced throughout the culture but may be most evident in interpersonal interactions (e.g., catcalls, leering at women's bodies, sexual comments, and sexual harassment) and media images highlighting and hypersexualizing the female form (Calogero et al., 2011).

The theory proposes that young girls and women, steeped in a culture of sexual objectification, internalize pervasive objectifying messages, learn to view themselves from the male

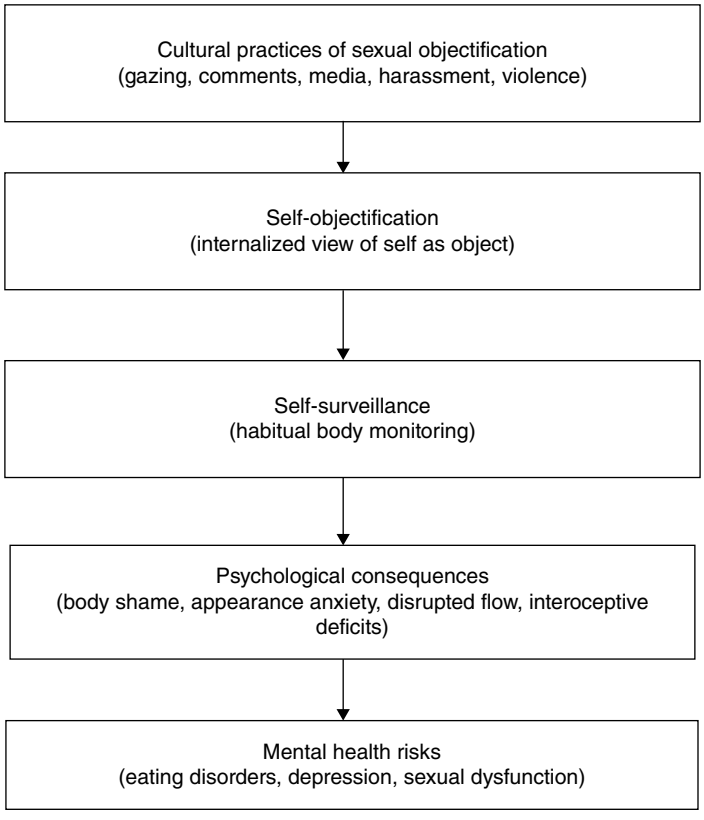

Figure 21.3 Objectification Theory Model. From Calogero, Tantleff-Dunn, and Thompson (2011). Reproduced with permission of APA.

observer's perspective and to conceptualize their bodies as sexual objects to be enjoyed by others (termed self-objectification). Furthermore, women who have assumed this self-objectifying perspective will experience increased self-consciousness about how their bodies are perceived by others, which manifests behaviorally in the habitual monitoring of their appearance (termed self-surveillance). The continual surveillance and evaluation of one's own appearance is then thought to lead to a host of negative psychological outcomes known to disproportionately affect women: body shame, appearance anxiety, safety anxiety, reduced ability to concentrate and become fully absorbed in tasks (i.e., flow experiences), and reduced awareness of internal bodily states (e.g., hunger and satiety signals). Fredrickson and Roberts (1997) further suggest that the accumulation of such experiences may contribute to the development of several psychological disorders disproportionately experienced by women, including eating disorders.

To date, the majority of empirical support for objectification theory comes from cross-sectional studies utilizing primarily Caucasian undergraduate samples. Such research indicates that while men and boys may experience objectification, their female counterparts report significantly higher levels of objectifying experiences and more negative consequences of such experiences (Bryant, 1993; Kozee, Tylka, Augustus-Horvath, & Denchik, 2007; Murnen & Smolak, 2000; see also Chapter 27). Within female samples, research has demonstrated associations between self-objectification (or its behavioral signal: body surveillance) and body shame, appearance anxiety, body dissatisfaction, and disordered eating (Calogero, 2009; McKinley 1998; McKinley & Hyde, 1996; Tiggemann & Slater, 2001; Tylka & Hill, 2004).

Generally, researchers have focused on body shame as a mediational link between self-objectification and disordered eating. A handful of correlational studies have attempted to test this mediational model, with results supporting the hypothesized links between self-objectification, body shame, and disordered eating (Calogero, 2009; Moradi, Dirks, & Matteson, 2005; Tiggemann & Slater, 2001).

There is considerably less research examining the role of appearance anxiety, reduced flow, and reduced awareness of internal bodily states in mediating the relationship between self-objectification and disordered eating. Extant findings provide only mixed support for these proposed mediators (Tiggemann, 2011). Longitudinal data collected from undergraduate women over a 10-year period indicate that levels of self-surveillance, body shame, dieting, and restricted eating decrease over time (McKinley, 2006a, 2006b). Notably, McKinley did not analyze the temporal sequence of changes in these variables, so it is not clear whether changes in self-surveillance preceded changes in other variables, as the theory would suggest.

Experimental studies examining the causal links between self-objectification and proposed consequences provide further support for the theory. Such experiments have utilized a common protocol wherein female participants were assigned to try on either a swimsuit, which was thought to increase one's body consciousness (objectification condition), or a sweater (control condition). Women who donned swimsuits reported increased levels of state self-objectification, self-consciousness, body shame, anxiety, fearfulness, feelings of humiliation, lower self-esteem, and diminished cognitive functioning compared to women who wore the sweater (Gapinski, Brownell, & LaFrance, 2003; Hebl, King, & Lin, 2004). Importantly, the negative effects of these objectification experiences appeared to last beyond the actual experience itself; women in the objectification condition continued to have increased body-related thoughts even after they had got dressed in their regular clothes. Moreover, the amount of body shame experienced during the objectifying experience mediated the relationship between the objectifying condition and postexposure body-related thoughts (Quinn, Kallen, & Cathey,

2006). Other experiments have utilized deception (i.e., telling participants that they would be interacting with and viewed by a male stranger) or sentence-scrambling tasks to manipulate state self-objectification. In these studies, women in the objectification condition experienced increased negative emotions, body shame, and appearance anxiety (Calogero, 2004; Roberts & Gettman, 2004).

Researchers have further utilized the swimsuit paradigm to examine eating behavior following an objectifying experience. After wearing either a swimsuit or sweatshirt, participants got dressed and were asked to take part in a sham taste test of cookies, chocolate drinks, or chocolate bars. One study found that women in the objectification condition exhibited higher levels of restrained eating, with levels of body shame predicting restrained eating (Fredrickson, Roberts, Noll, Quinn, & Twenge, 1998). However, a study by Hebl and colleagues (2004) found no effect on eating behavior. In sum, although correlational data provide consistent support for an association between self-objectification and disordered eating, which is most commonly mediated by body shame, experimental data provide only mixed support for the immediate impact of increased state self-objectification on dietary restraint (one facet of disordered eating).

As noted above, most studies of objectification theory have utilized convenience samples of undergraduate women. Recent work has attempted to examine the model in more diverse samples in order to clarify potential group differences and moderating factors. For example, studies have begun to examine the moderating influence of age, ethnicity, sexual orientation, level of physical activity, and degree of recovery from an eating disorder. Preliminary evidence offers mixed findings, highlighting the need for further research in these important areas (Moradi & Huang, 2008).

In the 16 years following Fredrickson and Roberts's original publication of objectification theory, a rich field of research has developed to examine the basic tenets of the theory. A large body of evidence now supports the proposed relationship between self-objectification and disordered eating, with body shame operating as a mediator between these two constructs. Other proposed mediators (e.g., appearance anxiety, reduced flow) have received only mixed support. While the association between self-objectification and disordered eating has been demonstrated in numerous studies, the extant literature is limited by a heavy reliance on largely Caucasian undergraduate samples. Future work should aim to examine the proposed relationships within various racial/ethnic groups (e.g., African American women, Latinas) and across a broad age range (e.g., children/adolescents, older adults).

Elaborated Sociocultural Model of Disordered Eating

Fitzsimmons-Craft (2011) recently proposed an elaborated sociocultural model of disordered eating in college women (Figure 21.4). Fitzsimmons-Craft asserts that extant sociocultural models do not adequately explain the processes by which internalization of the thin ideal leads to body dissatisfaction and disordered eating. The elaborated model draws strongly on the TIM, shifting the sequence of influence slightly (thin-ideal internalization is now proposed to precede appearance comparisons) and incorporating two additional variables: body surveillance and motives.

The concept of motives is derived from Uses and Gratifications Theory (Rubin, 2002), which is concerned with how individuals *choose* to expose themselves to certain forms of media and how they *choose* to behave in response to those messages. Uses and Gratification Theory

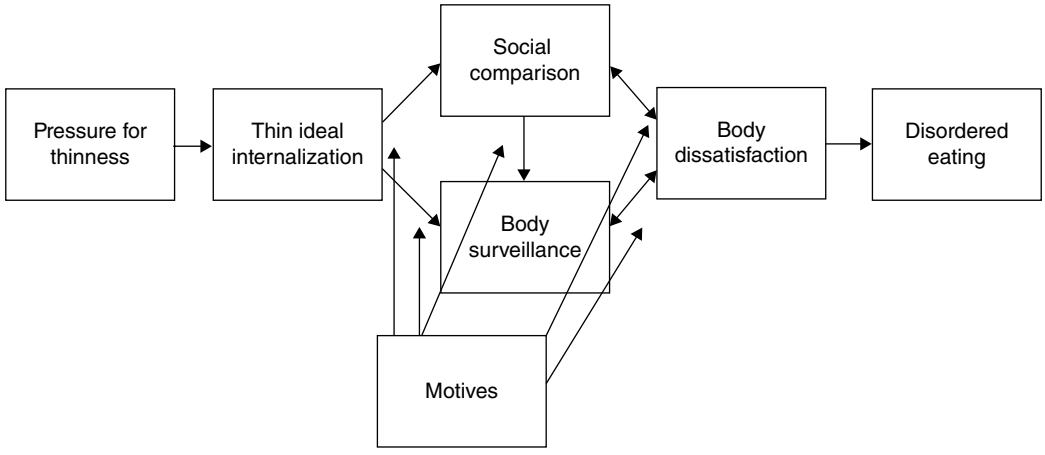

Figure 21.4 Elaborated Sociocultural Model of disordered eating. From Fitzsimmons-Craft (2011). Reproduced with permission of Elsevier.

therefore suggests that individuals are not simply passive recipients of sociocultural messages, but rather, that individuals differ with regard to their active engagement with media, selection of media content, motivation for seeking out such content, interpretation of media messages, and behavioral responses to those messages (Hesse-Biber, Leavy, Quinn, & Zoino, 2006; Levine & Smolak, 1996). While some women may use appearance-related media content (e.g., fashion magazines) for diversion, relaxation, or entertainment, other women may use the medium as a source of information regarding appearance standards and methods of meeting the appearance ideal (Fitzsimmons-Craft, 2011; Tiggemann, 2003). To date, relatively little research has directly examined the tenets of uses and gratification theory as it relates to disordered eating. However, extant research suggests that incorporating motivation for appearance media use into etiological models of disordered eating may have utility. For example, one study found that exposure to two separate forms of media were differentially associated with antifat attitudes; amount of exposure to magazines, but not amount of exposure to television, showed a positive association with negative beliefs about overweight individuals and a fear of becomming fat (Lin & Reid, 2009).

Fitzsimmons-Craft's elaborated model suggests that a woman's motive for exposing herself to appearance media and the manner in which she chooses to interact with those media will moderate the impact of the sociocultural pressures on proposed downstream outcomes such as body dissatisfaction and eating pathology. Thus, exposure to appearance media and media pressures for thinness may more negatively impact a woman who intentionally uses that information in an inappropriate manner, for example, using images of underweight models as "thinspiration." To our knowledge, the elaborated sociocultural model has not yet been tested empirically. Future work should examine this model among college women, as well as more diverse samples.

Implications for Prevention and Treatment

In parallel with the evidence that has accrued over the past two decades supporting sociocultural approaches, are treatment and prevention efforts focused on similar sociocultural influences. For instance, Haines and colleagues developed and tested an effective program

called Very Important Kids, which particularly targets teasing from peers (Haines, Neumark-Sztainer, Perry, Hannan, & Levine, 2006; see also Chapter 43). O'Dea and Yager (2012) also recently reviewed school-based psychoeducational approaches, many of which include some type of media literacy component (see Chapter 45). In addition, Stice, Becker, and others have found success with a dissonance-based program designed to reduce internalization of the thin ideal (Stice, Becker, & Yokum, 2013; see also Chapter 44). Tylka and Augustus-Horvath (2011) recently outlined strategies to prevent or reduce self-objectification (see Chapter 33). Clearly, theoretical work on sociocultural mechanisms is being translated into strategies designed to prevent or address body image and eating problems.

Conclusions and Future Directions

One historical limitation in the conceptualization of theoretical models in the area of eating disorders is the lack of an integrative approach that contains sociocultural and biological or genetic variables (see Chapter 67). Recently, however, Suisman and colleagues (2012) evaluated the role of genetic and environmental factors (shared, nonshared) in understanding thin-ideal internalization (a key component of sociocultural models, in particular, the TIM) (see Chapters 28 & 30). In their study of 343 postpubertal twins, they found a significant effect of genes and nonshared environment on internalization, but a nonsignificant effect of shared environment. Shared environment consists of factors, such as media, that both sets of twins might experience, whereas nonshared might consist of differential peer or parent pressures to lose weight. This study indicates the need for future conceptualizations of sociocultural models to move forward and consider the inclusion of a genetic component, and potentially, biological factors, such as pubertal timing, in the reformation of existing models.

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Section V

Risk and Protective Factors and Correlates

22

Body Image Disturbance and the Development of Eating Disorders

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Slade (1994) described body image as the "picture we have in our minds of the size, shape, and form of our bodies" (p. 497) and the feelings we have concerning these characteristics and our constitutional parts. From such phenomena as phantom limb, eating disorders (EDs), and body dysmorphia, we know that body image is based on the physical self but is not always synonymous with it. The translation from the physical to the mental representation of the body is a complex and emotionally charged developmental process.

A definition of body image as a multidimensional construct was first outlined by Cash and Deagle (1997) and still predominates today (Ahrberg, Trojca, Nasrawi, & Vocks, 2011; Cash & Smolak, 2011). According to this definition, body image is comprised of three main components: cognitive-affective, perceptual, and behavioral. The cognitive-affective component of body image refers to thoughts and feelings about one's body and is comprised of self-evaluation of the body and the influence of the body's appearance in self-esteem (Fairburn, Cooper, & Shafran, 2003). Disturbance in the cognitive-affective component of body image includes body dissatisfaction (negative evaluation of one's own body). It also includes overvaluation of weight and shape in one's self-image, resulting in undue influence of body image in overall self-image. The perceptual component of body image refers to the mental representation of one's own body shape and size. Disturbance in this area involves a perceptual distortion of having an erroneous estimate of the shape and weight of one's own body (e.g., body size overestimation; Cash & Deagle, 1997).

The behavioral component of body image refers to behavioral correlates of thoughts and feelings about one's body, such as body checking and avoidance. Body checking may include frequent looking in the mirror, asking for reassurance from others, and pinching or measuring of one's body parts. On the other end of the spectrum, avoidance behaviors may include wearing baggy clothes to cover up perceived imperfections or avoiding certain people or places (Cash & Smolak, 2011).

Numerous variables have been shown to affect body image disturbance (BID), including age of menarche, teasing (Menzel et al., 2010), pressures from family, friends and media (Stice, 2002; see also Chapters 26, 29, & 31), physical and sexual abuse (Kearney-Cooke &

The Wiley Handbook of Eating Disorders, First Edition. Edited by Linda Smolak and Michael P. Levine.

Ackard, 2000; Kearney-Cooke & Striegel-Moore, 1994; see also Chapter 34), and personality traits such as perfectionism (Polivy & Herman, 2002; see also Chapter 32). Sociocultural factors such as glorification of slenderness by mass media and stigmatization of body fat and obesity affect body image (Wertheim, Paxton, & Blaney, 2009; see also Chapter 21). Living in a culture that objectifies women's bodies and teaches them that the way to attain a greater feeling of control and self-confidence is through weight reduction also plays a role in body image disturbance and disordered eating (Calogero, Tantleff-Dunn, & Thompson, 2011; Stice, 2002; Stice & Agras, 1998; see also Chapters 19, 21, & 27).

Body image disturbance affects feelings, thoughts, perceptions, and behaviors related to the body and has long been recognized as a clinical feature of critical importance in the development and maintenance of anorexia nervosa (AN) and bulimia nervosa (BN; see Chapters 2, 3, 8, 9, 55, & 67). Emerging evidence also supports the role of BID in binge eating disorder (BED; see Chapter 10). As Hilde Bruch (1962) originally argued, amelioration of a dysfunctional body image is necessary for effective treatment of EDs. BID may engender attrition from treatment, and its persistence following otherwise successful treatment is a predictor of relapse (Fairburn, Peveler, Jones, Hope, & Doll, 1993; Rosen, 1990; see also Chapter 55). BID is also a correlate in additional psychological disturbances including depression, social isolation, anxiety, and low self-esteem (Kearney-Cooke, 1989; Wertheim et al., 2009).

In this chapter, we will discuss the role of BID in the development and treatment of EDs. We will examine questions such as why, despite high levels of body dissatisfaction that exist among women (Klemchuk, Hutchinson, & Frank, 1990), the actual incidence of EDs is relatively low (Striegel-Moore & Cachelin, 2001). We will also discuss the role of emotion regulation in BID and its relationship with the development and maintenance of EDs. Integrating research and clinical experience, we will discuss the implications of research on the prevention and treatment of BID.

Body Image Disturbance in Anorexia Nervosa and Bulimia Nervosa

Body image disturbance is a strong predictor of the use of weight control behaviors, including dietary restriction and purging (self-induced vomiting, laxative misuse, and compulsory exercise; Neumark-Sztainer, Paxton, Hannan, Haines, & Story, 2006). In those with the requisite biological and temperamental risk factors, BID and weight control behaviors are associated with the development and maintenance of AN and BN (Cash & Deagle, 1997; see also Chapter 55). Stice (2002) conducted a meta-analytic review of longitudinal studies examining the role of body dissatisfaction and other risk factors to predict increases in eating pathology and confirmed that negative body image is an independent predictor of disordered eating. Due to the strong association between BID and disordered eating behaviors, BID is incorporated into the *DSM-5* diagnostic criteria for both AN and BN (*Diagnostic and Statistical Manual of Mental Disorders*, 5th edition; American Psychiatric Association, 2013; see also Chapters 8 & 9).

According to *DSM-5* diagnostic criteria, a diagnosis of AN includes satisfaction of three criteria, one of which (Criterion C) is "disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or persistent lack of recognition of the seriousness of the current low body weight" (*DSM-5*, 2013, p. 339). Cash and Deagle's (1997) aforementioned delineation of body image disturbance maps particularly well onto this diagnostic criterion for AN. That is, perceptual and attitudinal

aspects of body image disturbance, for example, body dissatisfaction, body image investment, and distortion or misperception of body size, represent core features of AN. A diagnosis of BN requires satisfaction of five criteria; of most relevance to BID is Criterion D ("Self-evaluation is unduly influenced by body shape and weight"; p. 345), which highlights the role of body image disturbance in this disorder (*DSM-5*, 2013). It is important to distinguish BID from simply disliking or being dissatisfied with one's body (Cash & Smolak, 2011). Body dissatisfaction is only one aspect of the attitudinal component of body image disturbance. BID in individuals with EDs is characterized by preoccupation with appearance that is time-consuming, distressing, and life-interfering (Bell & Rushforth, 2008).

Several theories have been posited to explain the connection between BID and EDs. The Cognitive-Behavioral Therapy–Enhanced (CBT-E) model for EDs (Fairburn et al., 2003; see also Chapters 18 & 56) states that the primary maintaining factor for EDs is the overvaluation of body shape and weight and its control. That is, although most people evaluate themselves based on a number of domains, those with EDs evaluate themselves predominantly in terms of body shape, weight, and perceived control over their own bodies. This "core psychopathology" outlined in the CBT-E model explains that overvaluation and preoccupation with the body leads these individuals to invest mental, emotional, and behavioral resources in dietary control and pursuit of thinness. Behavioral correlates of BID such as food restriction, other forms of weight control behavior, and body checking and avoidance may be understood as part of the development and maintenance of the ED (Fairburn et al., 2003). Other studies support the importance of overvaluation as a risk factor for EDs. For example, studies have demonstrated that among individuals who are dissatisfied with their bodies, overvaluation of body image significantly differentiates those with EDs from those without EDs, including individuals with binge eating disorder (Goldfein, Walsh, & Midlarsky, 2000; Masheb & Grilo, 2003).

Objectification theory (Fredrickson & Roberts, 1997) argues that the dominant culture and cultural socialization in the United States promotes the "thin ideal" and sexualization of women's bodies (see Chapters 19, 21, & 27). Through a process termed "self-objectification," women who internalize this repeated cultural objectification and the importance of physical attractiveness begin to objectify their own bodies by focusing on and overvaluing their outward appearance. Research demonstrates the link between sociocultural pressures and self-objectification (Stice, 2002; see also Chapter 27). Women who display self-objectification are more concerned with how the body looks (outward appearance) than what it can do (nonobservable characteristics). These women tend to be more dissatisfied with their bodies and, consequently, are more likely to exhibit disordered eating behaviors (Calogero et al., 2011; Fredrickson & Roberts, 1997; Noll & Fredrickson, 1998). Noll and Fredrickson (1998) found support for the connection between self-objectification, body shame, and disordered eating behaviors, including symptoms of both AN and BN. Slater and Tiggemann (2010) tested a mediational model of self-objectification theory in a sample of Australian boys and girls aged 12–16. They hypothesized that self-objectification, as measured by a body surveillance scale, leads to mediators of body shame and appearance anxiety, which are significantly associated with disordered eating. Results supported the model for both girls and boys, demonstrating that even in early adolescence, the process of self-objectification leads to negative affect and disordered eating behavior (Slater & Tiggemann, 2010).

In addition to CBT-E and objectification theory, emotion regulation theory can be used to understand the relationship between BID and EDs. This topic will be discussed in further detail in later sections of the present chapter.

Moderators of the Relationship Between EDs and Body Image Disturbance

Numerous studies support the strong association between BID and EDs (Brannan & Petrie, 2011; Cash & Deagle, 1997; Polivy & Herman, 2002). Although BID is a necessary component in the development and maintenance of EDs, research demonstrates that such disturbances (body dissatisfaction, in particular) are somewhat normative—especially among women in Western cultures (Rodin, Silberstein, & Striegel-Moore, 1984). However, the prevalence of EDs is comparatively low (Tylka, 2004; see also Chapters 5–7). Therefore, it is important to investigate the factors that moderate the relationship between BID and EDs.

Tylka (2004) examined a number of variables thought to moderate the relationship between EDs and body dissatisfaction. Results suggested that variables of body surveillance, neuroticism, and presence of a family member with an ED significantly strengthened the relationship between body dissatisfaction and ED symptomology. Body surveillance (see Chapter 26), which involves paying particular attention to the appearance of certain parts of the body through behaviors such as body checking, accounted for the most variance in the relationship between body dissatisfaction and ED symptomology (Tylka, 2004). Research has also shown that increases in dieting and negative affect may help to explain the link between body dissatisfaction and disordered eating (Stice & Shaw, 2002).

Comparatively fewer studies have investigated protective factors that moderate the relationship between BID and EDs (see Chapter 33). In one such study, Brannan and Petrie (2011) examined four variables associated with psychological well-being hypothesized to buffer the association between body dissatisfaction and BN: self-determination, optimism, satisfaction with life, and self-esteem. Consistent with previous findings, results demonstrated a significant correlation between body dissatisfaction and symptoms of BN in a sample of female undergraduates. Further analyses demonstrated that, as hypothesized, all four of the aforementioned variables associated with psychological well-being served to moderate the relationship between high levels of body dissatisfaction and symptoms of BN (Brannan & Petrie, 2011).

Treatment of BID in AN and BN

An understanding of the relationship between BID and EDs can facilitate treatment of EDs as well as guide preventive efforts. Despite the well-researched link between BID and the development and maintenance of EDs, treatment studies in this area are relatively limited, and treatments that specifically target BID are lacking (Cash & Deagle, 1997; Farrell, Shafran, & Lee, 2006). Even following successful treatment for EDs, residual BID has been found to be a significant predictor of relapse, specifically in BN (Fairburn et al., 1993; Freeman, Beach, Davis, & Solyom, 1985; see also Chapter 55). As such, it is critical to investigate what works in decreasing BID and how these techniques can be incorporated into existing empirically sound ED-related treatments.

Clausen (2004) analyzed the time course of different ED symptoms in a sample of patients with AN, BN, and eating disorder not otherwise specified (ED-NOS) who were treated at an eating disorders center in Denmark. Results demonstrated that among categories of behavioral, physical, and psychological symptoms of EDs, the psychological symptoms (i.e., disturbed body perception, obsession with weight and shape, and fear of gaining weight) were the last to remit. Furthermore, all three psychological symptoms were present in patients with AN and BN. This is notable because a diagnosis of BN does not require "fear of gaining weight," but evidence from the study suggested that it is present in both diagnostic groups (Clausen, 2004).

Researchers have compared the efficacy of a number of empirically investigated treatments focused on improving BID (Farrell et al., 2006; Rosen, 1996). In general, existing evidence supports the usefulness of cognitive-behavioral interventions for BID; cognitive-behavioral therapy (CBT) seems to be more effective than treatments from other theoretical frameworks (Farrell et al., 2006; see also Chapter 56). Furthermore, CBT that contains a targeted body image component has generated more support than CBT without such components (Danielsen & Rø, 2012; Rosen, 1996). CBT that directly addresses body image tends to involve cognitive restructuring, behavioral experiments, and size perception training—see Cash and Smolak (2011) or Rosen (1997) for detailed examples of specific treatments. However, many of these treatment studies have not been replicated and may not be generalizable to all diagnostic categories or levels of care. For example, Legenbauer et al. (2011) conducted a CBT for a body image group for ED outpatients and found that negative body-related thoughts and feelings decreased postintervention. However, this promising intervention has not been studied within the context of individual therapy or inpatient settings.

Bhatnagar (2010) investigated a cognitive-behavioral intervention aimed at improving body image in women with EDs. The intervention consisted of cognitive restructuring, psychoeducation, and mirror exposure. Results showed that participants in the treatment group reported a number of significant changes, including greater appearance satisfaction, decreased appearance investment, decreased discrepancy between actual and ideal appearance, and decreased body-avoidance behaviors. Notably, participants in the treatment group also showed significantly decreased levels of disordered eating and symptoms of depression.

Danielsen and Rø (2012) studied a transdiagnostic sample, comprised of individuals with AN, BN, and ED-NOS, in an inpatient eating disorders treatment program to investigate the way in which changes in body image are associated with changes in eating disorder symptomology. The inpatient program in which the patients participated included body image-specific interventions. The Body Attitude Test (BAT; Probst, Pieters, & Vanderlinden, 2008) was used at intake to identify different aspects of body image problems and to measure any changes in body image at the conclusion of inpatient treatment. The Eating Disorder Inventory-2 (EDI-2; Garner, 1991) was used as a measure of eating disorder symptomology. Results showed that BAT scores emerged as the strongest predictor of change in EDI-2 scores, as compared to changes in measures of general psychopathology and measures of personality characteristics. Of the four subscales of the BAT, results also showed that "increased familiarity with one's body" was most predictive in improvement in ED symptoms. Importantly, these results demonstrate that the ability to accurately perceive and interpret interoceptive information has a significant relationship with ED symptom improvement. However, the directionality of this relationship remains unknown (Danielsen & Rø, 2012).

Guided imagery has also been investigated as an intervention for BID. For example, Kearney-Cooke (1989) found that using guided imagery in the body image component of an intensive ED treatment program caused significant changes in the Body Cathexis Scale (BCS; Secord & Jourard, 1953) and the Color A Person Test, signifying a decrease in overvaluation of body image in one's self-concept. These results were maintained at 1-year follow-up. Esplen, Garfinkel, Olmstead, Gallop, and Kennedy (1998) found that guided imagery had substantial effects on the reduction of binge eating and purging episodes. Importantly, the guided imagery group also demonstrated improvements on measures of attitudes concerning eating, dieting and body image in comparison to the control group.

The Role of BID in Binge Eating Disorder

Binge eating disorder (BED) is defined in *DSM-5* (2013) as recurring episodes of eating significantly more food in a short period of time than most people would eat under similar circumstances, with episodes marked by feelings of lack of control (see Chapters 4 & 10). BED episodes are associated with at least three out of five behavioral indicators (e.g., eating until feeling uncomfortably full). The person may have feelings of guilt, embarrassment, or disgust and may binge eat alone to hide the behavior from others. BED is associated with marked distress and occurs, on average, at least once a week over a period of 3 months. No systematic countermeasures for weight regulation are undertaken following these binging episodes, such as self-induced vomiting, intake of diuretics, laxatives, or appetite suppressants, or excessive physical exercise. Spitzer et al. (1992) found that the most frequent comorbidity of BED is obesity, with 30–40% of patients with BED being overweight.

DSM-IV-TR (American Psychiatric Association, 2000) described BED in an appendix; it was not considered a distinctive diagnosis, and clinicians were forced to assign the catch-all diagnosis of ED-NOS to patients with BED. The *DSM-5* (2013), for the first time, recognizes BED as a specific diagnosis and as a treatable behavior pattern. Whereas for AN and BN one of the main diagnostic features is BID, the diagnostic criteria for BED (see Chapter 10) do not refer to BID. The discussion of whether BID is a core symptom of BED and therefore should be integrated into the diagnostic criteria is controversial (Hrabosky, Masheb, White, & Grilo, 2007; Masheb & Grilo, 2003).

Concerning the perceptual component of BID, no evidence exists that persons with or without BED differ in terms of perception of their own body size. Individuals with BED have been found to report significantly greater degrees of shape and weight concerns than both overweight and normal weight individuals without BED. However, their BID levels are similar to those of individuals with AN and BN (Goldschmidt et al., 2010).

Emerging research focusing on the role of BID in BED has shifted to overvaluation of shape and weight. Hrabosky et al. (2007) found that 58% of a sample of BED patients seeking treatment endorsed clinically significant overvaluation of body image. This subset of patients reported greater disordered eating and general psychopathology than those with subclinical levels of overvaluation. Grilo et al. (2008) found that a higher level of weight-shape overvaluation in BED is associated with the greatest body dissatisfaction, eating pathology, depressive symptomology, and significantly poorer treatment outcomes. Grilo et al. (2009) also found that patients with BED accompanied by overvaluation had significantly higher levels of eating psychopathology than patients with BED without overvaluation. According to Grilo (2013), overvaluation demonstrates concurrent validity within BED. This suggests that overvaluation warrants consideration as a diagnostic specifier because it signals greater severity with BED, but not as a required criterion because that would result in exclusion of many individuals with clinically significant eating pathology.

Overall, BID seems to be a symptom of BED that still requires further exploration. Despite the absence of body-related criterion for BED in the *DSM-5* (2013), a burgeoning body of research has shown that overvaluation of shape and weight is associated with greater severity of eating-related pathology and psychological distress, as well as having negative prognostic significance in this population (Grilo, 2013). Assessment of overvaluation of weight and shape is essential with BED, and body image treatments focusing on this cognitive component must be included in treatment for BED to increase the rates of treatment success.

The Role of Emotion Regulation in BID

Research consistently shows that the capacity for adaptive emotion regulation (ER) is necessary for mental health and well-being. In particular, ER deficits have been found to play a role in the majority of psychological disorders (Gross & Levenson, 1997; Linehan & Chen, 2005; Pelletier, Dion, Slovinec-D'Angelo, & Reid, 2004). ER has been defined as the ability to modulate one's emotions in order to respond to environmental demands. Successful ER involves general awareness, understanding, and acceptance of emotions, and the capacity to act effectively despite the presence of difficult and/or strong emotions (Gratz & Roemer, 2004; Linehan, 1993).

Although limited research exists in this area as it relates to BID, we believe that the capacity for ER plays a major role in BID. In a sample of undergraduate women, Ackard, Croll, and Kearney-Cooke (2002) found that greater difficulties in ER and affective disturbances were related to a higher frequency of both dieting and body image variables such as body dissatisfaction and drive for thinness. In a community sample of 533 boys and girls, Hughes and Gullone (2011) found that ER moderated relationships between body image concerns and both bulimic and depressive symptoms. However, they did not find significant relationships between ER deficits and drive for thinness or anxiety symptoms.

Bydlowski et al. (2005) investigated emotional processing deficits in individuals with EDs compared with controls. They used measures of both alexithymia and emotional awareness and found that compared to controls, individuals with EDs were characterized by a global deficit in emotional processing, which was characterized by impaired ability to identify their own emotions, as well as impairment in understanding others' emotional experiences (see Chapter 16). Lavender and Anderson (2010) found that ER difficulties were associated with disordered eating in a sample of male undergraduates and also contributed to body dissatisfaction in men. Difficulties in ER were found to account for a unique amount of variance in both disordered eating and body dissatisfaction independent of variance accounted for by BMI and negative affect. Furthermore, it has been found that behaviors associated with BN lead to a reduction in anger; the effect was particularly pronounced in individuals with a strong tendency to avoid expressing their anger (Milligan & Waller, 2000).

Conclusions and Future Directions

Because BID is so closely linked to the prognosis of EDs, it is imperative that ED treatments incorporate interventions specifically designed to address this aspect of the illness (Cash & Smolak, 2011). BID has been found to predict both the onset and maintenance of EDs. It is a complex and multidimensional construct that is often the last to remit in the course of ED treatment (Stice, 2002). Although a number of programs have been designed to address BID, consistent empirical evaluation is still needed. In addition to the promising new interventions that specifically target BID, it is also important for treatment to focus on bolstering protective factors that may serve to reduce the connection between BID and ED symptomology (Brannan & Petrie, 2011).

We agree with the recommendation mentioned in similar reviews that interventions that address BID should be integrated into existing evidence-based treatments for EDs in order to be most efficacious (Danielsen & Rø, 2012; Farrell et al., 2006). Continuing to investigate and develop multidimensional treatments for BID is essential. Research supports that deficits

Case Example

Nancy, a 23-year-old woman with AN, comes into group one day after an assignment that asked each member of the group to eat three meals with others in the next week and shares the following story:

> I planned to have dinner with my sister and her husband on Friday night; they were going to pick me up at 6:00 PM. I kind of got excited about going out with them because I spend so much time alone. I was even looking forward to eating at my favorite Greek restaurant. I try not to get excited about getting together with others or about food; it leaves me feeling vulnerable and out of control. There have been so many disappointments in my relationships. It was 6:15, and they didn't pick me up. I thought they might be running late. Then it was 6:30, and I knew they weren't coming. Why did I let myself get so excited about this? I am getting out of control. I changed into my workout clothes, skipped dinner, and stayed on the treadmill for 2 hours. I got on the scale. Lost 2 pounds since the last weigh-in—felt 100% better. I felt back in control and no longer upset about my sister and brother-in-law forgetting our dinner date.

Johnson and Connors (1987) proposed that from birth, we humans develop feelings of personal mastery by gaining control of our bodies. From the time we are able to reach out and grab things, crawl, walk, gain sphincter control, ride a bicycle, and so on, a massive feedback loop exists between control of the body and self-esteem. The more we feel in control of these things going on in our bodies, the greater our feelings of personal mastery in general.

The body is the only object in a person's perceptual world that simultaneously is perceived and is part of oneself (Fisher, 1986; Schilder, 1950). The unique closeness of the individual's body to their identity maximizes the likelihood that the body reflects and shares in a person's most important preoccupations (Kearney-Cooke & Striegel-Moore, 1997). Fisher (1986) wrote that the body, like all significant objects, can become a "screen" onto which one projects one's most intense concerns. For example, patients with EDs may project their experiences of emotion dysregulation and/or interpersonal struggles that they feel inadequately equipped to deal with onto their body. "I feel out of control," would be projected onto the body as, "My stomach is out of control and fat. I must lose weight and restrict all day."

Engaging in dietary restraint (skipping dinner) and over-exercising enabled Nancy to substitute a sense of control or mastery in the appearance domain for a lack of capacity to handle emotions and relationships with others. Nancy regulated her terrifying feelings of needing people or food as being out of control by projecting them onto her body: moving from "I'm out of control," to "My body is out of control—it's fat, I hate how it looks." By skipping the meal and overexercising, she was able to regulate her emotions.

in emotion regulation play a role in BID and disordered eating (Bydlowski et al., 2005). Dialectical behavior therapy (DBT; see Chapter 57) is an example of a treatment designed to address pervasive emotion dysregulation by teaching clients skills including mindfulness, emotion regulation, interpersonal effectiveness, and distress tolerance (Linehan, 1993; Salsman & Linehan, 2006). Teaching skills to increase the capacity for self-regulation

(including findings from emerging research on willpower and self-control) in prevention and treatment programs is essential. Such skills can address difficulty in identifying emotions (i.e., alexithymia) and teach individuals to connect internal cues to emotions—rather than to problems with their bodies.

Expressive therapies like guided imagery are also powerful tools through which negative body experiences, such as abuse, teasing, and rejection, can be brought to the surface and worked through. Guided imagery can help patients reclaim their bodies and selves by imagining a future self and ways to have a more positive body image, regardless of weight or shape. Promising results have been found in the use of guided imagery in the treatment of BID and EDs (Esplen et al., 1998; Kearney-Cooke, 1989), and further development is warranted. Tools such as "decoding sheets" teach patients how to pay attention and respond to interoceptive cues. Decoding sheets allow patients to keep track of negative thoughts about their bodies and what these thoughts may be distracting them from. For example, "I feel fat when I am around my father," eventually may be translated into, "I get confused about how I feel when I am with my father." This decoding process helps patients work through interpersonal problems and maladaptive emotion regulation mechanisms underlying their BID.

BID is especially amenable to prevention and outreach programs (Levine & Piran, 2004). As such, we believe it is time to think outside the box with regard to the content of prevention programs for EDs. The majority of the ED prevention programs have largely targeted body image and excluded additional related factors. Body dissatisfaction and overvaluation of shape and weight are powerful risk factors for the development and maintenance of EDs and continue to be an important target for prevention (see Chapter 41). However, additional risk factors need to be addressed in prevention and outreach programming. Focus on increasing protective factors such as presence of adaptive coping skills, self-regulation skills, self-determination, and goal-setting skills will increase the effectiveness of future prevention programs. This type of programming can be achieved through outreach efforts targeted toward at-risk groups (e.g., undergraduate women high in body dissatisfaction, early adolescent girls, and high-school students; see Chapter 45). Objectification theory also emphasizes the importance of preventive outreach programming in an effort to reduce sexual self-objectification within a dominant culture that promulgates the "thin ideal." Adrienne Rich (1976, p. 75) wrote, "women have either become their bodies, complying blindly and slavishly with male theories about them or try to exist in spite of them." Both prevention programs and treatment programs should focus on teaching individuals to reclaim their bodies and selves, listen carefully to their internal cues, and use their voices, not their bodies, to competently communicate what's really going on inside (see Chapter 42).

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23

Cultural Similarities and Differences in Eating Disorders

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Eating disorders emerge at a specific historical moment. Rates of incidence vary cross-culturally and within a single nation in predictable ways. Finally, symptoms differ systematically in different contexts (Anderson-Fye & Becker, 2003; see also Chapters 21 & 35–37). In these ways, we know that cultural context matters in eating disorders (EDs) and disordered eating. This chapter describes the primary ways culture matters in the prevalence and symptomatology of EDs and disordered eating behavior. We first explain the overarching trends that have the greatest impact, and then summarize regional differences.

Concern about disordered eating has continued to expand, both around the world and throughout all subgroups in the United States (see Chapters 5–7). EDs remain among the deadliest of psychiatric disorders and can debilitate individuals across the lifespan, but especially in youth (see Chapters 15 & 56). Like other complex and widespread disorders, such as depression, that have biological, psychological, and social underpinnings, these disorders are very difficult to model, prevent, and treat across diverse groups (see Chapters 41–65). The data are now overwhelming that culture needs to be taken seriously in our models of assessment, treatment, and prevention, although as yet there is no widely agreed way of doing so.

This chapter draws on decades of work to outline the major cultural dynamics important to disordered eating across national contexts. While there has been an enormous increase in research output investigating cultural factors in EDs, the aggregate findings raise more questions than they solve about the myriad unknowns of how culture impacts EDs and how we can better intervene. They also underscore the importance of expanding methodologies as we expand our etiological models. Mixed methods including a qualitative or ethnographic component are particularly useful to the study of culture.

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Factors that Matter Everywhere

Examination of global and national data reveals important broad sociocultural factors that impact development, prevalence, and symptoms of EDs everywhere. These important categories include cultures of gender, sexuality, physical activity, acculturation, class, race and ethnicity, and life cycle groupings. Often, in the EDs literature, "culture" is used as a static variable, and frequently as a proxy for racial or ethnic group (though there are notable exceptions, e.g., Levine & Smolak, 2010). Here, we describe cultural context in a more dynamic way.

Cultural context matters centrally in the risk, development, and manifestation of EDs and disordered eating behavior. What is "normal" or "typical" is heavily context-dependent, so what is abnormal or atypical also varies by culture. For example, type of diet and frequency of meals during a day can range substantially from the pattern typically represented in mainstream U.S. culture: breakfast upon waking, noon lunch, and early evening dinner. What food is available, consumed, and considered a "healthy" diet also differs widely across national contexts and within them. In some areas of the world, a full caloric intake is equal to that of a highly restrictive diet in the United States. Similarly, physical activity, appearance ideals, and food meanings can vary widely. These norms contribute to recognition of when eating behaviors and related psychological states are considered "abnormal" or "disordered."

While eating-disordered behavior can be inventoried reliably, its meanings are locally dependent. A classic example of this point is found in Le Grange, Luow, Breen, and Katzman's (2004) article on disordered eating among impoverished black South Africans. Young people of both genders in Cape Town schools showed high levels of disordered eating as measured by standardized tests such as the Eating Attitudes Test (EAT-26). That is, they endorsed high levels of symptoms such as food preoccupation and binge eating. For more than a decade researchers had concluded that these South African young people had high rates of disordered eating related to psychiatric distress. Yet in qualitative follow-up, the researchers discovered that the indicators actually related to poverty and low food availability, rather than the anorexia and bulimia-like symptoms the tests were intended to measure.

It is important to recognize culture as complex and multilevel. Culture is historically grounded and symbolic as well as extant at all levels of human existence, from macrosocial to intrapersonal. Culture is both structural and relational, meaning that it influences both human groups en masse and the particular circumstances in which group members find themselves. We therefore must expand consideration of the implications of culture in EDs beyond the ethnic and racial categorizations we often think of as constituting "cultural" groupings. This broader approach is important because culture is at work not only with respect to who develops EDs cross-culturally, but also with regard to which people within a population are more vulnerable to developing them and what circumstances contribute to that vulnerability. This section examines some of the cultural factors and dynamics that literature indicates have bearing on EDs between and within groups generally.

Gender Culture and Eating Disorders

It is well documented that EDs are highly gendered conditions (see Chapter 27). Since EDs first became recognized as pathological conditions in the 19th century (see Chapters 2 & 3), their prevalence has been far higher among females than males. This pattern holds true almost everywhere they have been studied and is documented most thoroughly in North America, Western Europe, and Asia (Gordon, 2000; Makino, Tsuboi, & Dennerstein, 2004). The heavily gendered difference in prevalence of EDs for some time was attributed to what was believed to be females' inherently vain and emotionally unstable *nature* (Brumberg, 2000;

Sentilles & Callahan, 2012). However, decades of study of the environmental and relational circumstances of EDs' risk, reports of EDs among a substantial minority of men in some studies (Striegel-Moore et al., 2009; see also Chapter 36), and identification of particular activity-based groups among whom weight matters, such as wrestlers (Chatterton & Petrie, 2013; see also Chapter 35) and jockeys (Baum, 2006) provide convincing evidence that culture affects gendered prevalence rates and symptoms (Anderson-Fye & Becker, 2003; Olivardia, 2001).

Almost everywhere, the common positioning of the female body is as the *object* of culture (emphasis on ornamentality/appearance) while the male body is the *subject* of culture (emphasis on instrumentality/performance). This divide contributes to different incidences and manifestations of EDs between males and females. Cross-culturally, male bodies index *power over*—over others and over circumstances. Thus, the relevant male bodily preoccupation is muscularity rather than thinness per se. The rising prevalence of body-related disorders among adult males (Cash, Winstead & Janda, 1986; McCreary & Sasse, 2000; see also Chapter 37) more often takes the form of muscle dysmorphia (a form of body dysmorphic disorder) and binge eating than anorexia nervosa (AN; Hudson, Hiripi, Pope, & Kessler, 2007). The increase in muscle dysmorphia is commonly attributed to the valuation of male physical power (represented by muscularity), coupled with the growing sensibility over recent decades that a muscular and sculpted, fat-free body is *the* desirable male presentation, that there is a unitary body ideal to which males should and do aspire (Pope, Olivardia, Gruber, & Borowiecki, 1999; see also Chapter 28). When a particular presentation of a masculine body (i.e., lean and muscular) becomes more important socially, levels of body dissatisfaction and prevalence of EDs also tend to rise. Some have speculated that as female occupational and earning power increases, male ornamental appeal becomes more important (Olivardia, 2001).

For females, body dissatisfaction and restrictive ED are most often characterized by a drive for thinness. However, as Lee and others have demonstrated (Lee, Lee, & Leung, 1998), that drive is not always accompanied by a fear of fatness (see Chapter 16). Rather, studies suggest that among females, distorted body perception and disordered eating may reflect a body-as-object orientation. For the individual, this object-body symbolizes a conditional *power to*—power to attract a partner, to achieve a particular economic or social status, or to distance the object-body from the self in situations of conflict or abuse (Thompson, 1994). That power exists if one is able to exert control over food intake or the effects of food on the body (e.g., through vomiting; Cassin & von Ranson, 2005).

Structural gender inequality is likewise thought to be a factor in binge eating disorder (BED; Harrington, Crowther & Shipherd, 2010), with periodic, excessive food consumption being used to cope with other types of physical or emotional deprivation or trauma (Beauboeuf-Lafontant, 2009; Harrington et al., 2010; Thompson, 1994). Perhaps one of the most significant commonalities regarding the gendered nature of restrictive EDs is the sufferers' common (mis) perception that their efforts are in accord with the norms of sexual attractiveness. It is common for persons with EDs to believe that the gender ideals to which they subscribe demand someone who is more of what they strive to be, be it thinner or more muscular (Bergstrom, Neighbors & Lewis, 2004; Herzog, Newman & Warshaw, 1991; Jacobi & Cash, 1994; Smith, Hawkeswood, Bodell, & Joiner, 2011). Not only can achieving a certain bodily presentation be seen as essential to partner selection, but bodily presentation also is a marker of class status, another common mate selection criterion (Katzman, Hermans, Van Hoeken, & Hoek, 2004; McLaren 2007). Cultures of gender matter in EDs, then, because gender shapes and is shaped by shared norms concerning what constitutes a "good" body, and how those norms are applied.

Sexual Culture and Eating Disorders

Like gender culture, sexual culture (i.e., the institutions, practices, norms, and modes of representation that orchestrate and establish meanings for sexuality; http://cssc.berkeley.edu/) figures prominently in EDs. Sexual culture affects beliefs about who is susceptible to EDs and among whom and how EDs are manifested.

Sexist and heterosexist assumptions about who develops EDs once made eating disordered behavior among nonheterosexuals a neglected area of research and clinical practice. However, the perception that EDs are unique to young, white, middle class, heterosexual females is increasingly recognized as inaccurate and obsolete. More recent studies have documented the growing prevalence of these conditions in other population subgroups, leaving almost no one invulnerable. This extensive documentation has challenged long-held presumptions about the singular etiology of EDs, as well as a unitary risk factor profile. The data have shown a relationship between sexual minority culture (indeed, minority cultures in general) and EDs, although that relationship is not always well understood. For example, studies of sexual minorities tend to use measures of disordered eating (involving psychometric instruments like the EAT-26), rather than EDs (using *Diagnostic and Statistical Manual of Mental Disorders* [*DSM*] criteria; Feldman & Meyer 2007) as the outcome of interest. Such studies have generated a conventional wisdom that gay and bisexual men display more disordered eating symptomatology (DES) than men who identify as heterosexual (Beren, Hayden, Wilfley, & Grilo, 1996; Kaminski, Chapman, Haynes, & Own, 2005), and that lesbian and bisexual women experience disordered eating less often than do heterosexual women. Such findings suggest a primarily sociocultural etiology for EDs among nonheterosexuals, that risk for EDs is inherent to male sexual minority culture because of the value attached to thinness and muscularity, and that protection against EDs is inherent to female sexual minority culture because thinness and mainstream gender body ideals are less valued.

Sexual culture does account for some of the within-gender difference in ED prevalence. For example, rates of restrictive EDs among gay and bisexual males are 6% compared to a 1% rate among heterosexual males (Austin et al., 2009). Moreover, a high drive for thinness or fixation on pursuit of a muscular male body ideal has been found. Binge eating is also more frequent among gay and bisexual males than among heterosexual males (Austin et al., 2009).

However, recent research has challenged the notion that lesbians and bisexual women are "culturally" protected from EDs by existing in subcultures where pressures creating EDs among heterosexual women cease to exert influence. Feldman and Meyer (2007) found that rates of EDs among lesbian and bisexual women were comparable to those found among heterosexual women across all categories of the disorders. Other studies have shown that adolescent and young adult lesbian and bisexual females were more likely to report binge eating and purging than their heterosexual peers (Austin et al., 2009). These findings call into question the assertions of "cultural" protection conveyed by lower drive for thinness and nonadherence to mainstream body ideals among lesbian and bisexual females, and suggest that the existence of these subcultural norms is not necessarily a guard against development of EDs. Overall, the existing literature on EDs in sexual minority groups underscores that a holistic consideration of culture and minority status, not just identification of group-level norms, matters everywhere when it comes to understanding health and illness in populations.

Physical Activity Culture and Eating Disorders

Because physical activity is widely acknowledged as being able to both make and remake bodies, physical activity norms and behaviors have profound bearing on EDs. Physiognomy is frequently associated with particular occupational or recreational activities—an individual may be said to be "built like" a runner, dancer, model, or player of a particular sport. Build is commonly associated with ability to perform (or inability to perform) in a particular arena. For example, the preferred body type for female figure skaters and ballet dancers is similar at the macro level: petite, slender, and lithe with no excess flesh but also little visible musculature. Given that success in these sports, which includes the assessment that one is capable of performing excellently, is strongly predicated on bodily presentation (e.g., an ornamental orientation), it is not surprising that restrictive ED rates within these subgroups are substantially higher than in the general population (Sundgot-Borgen &Torstveit, 2004). Participation in *instrumental* sports, that is, sports where performance matters regardless of appearance (basketball, soccer, etc.), has been found to be associated with lower rates of EDs (Sundgot-Borgen &Torstveit, 2004; Zucker, Womble, Williamson, & Perrin, 1999).

Male athletes and performers also experience pressure to shape their bodies in particular ways. Increased international attention to male bodily appearance in sport over the past few decades (perhaps best exemplified by David Beckham's status as "the body" of soccer), has contributed to the growing association between male athleticism and muscle dysmorphia or EDs (see Chapter 35).

Physical activity can be a symptom of EDs more generally. Overexercising is commonly observed in persons with restrictive EDs and can also be a gateway to disordered eating behavior, especially among college students. The nature of the physical activity tends to reflect the bodily preoccupations of the individual. Cardiovascular exercise is preferred among those with a strong drive for thinness, and weightlifting is preferred by those with a strong drive for muscularity. Physical activity is also a relevant consideration in binge eating. Compared to adolescents exhibiting no binge eating behaviors, those exhibiting binge eating behaviors tended to neglect physical activity in favor of more extreme weight control measures such as use of laxatives or diet pills (Neumark-Sztainer et al., 2006). Much remains to be learned about the context of the disinclination toward physical activity among this group, particularly given the broad social approval afforded to physical activity engagement in developed nations.

In short, current scholarship on this subject suggests that additional research targeting the interactions of culture, physical activity, and EDs would yield significant insights. Little is known about the contexts in which a largely beneficial pursuit like physical activity can become obsessive and dysfunctional. Even less is understood about how culture operates in the neglect of physical activity in favor of more extreme and harmful weight loss behaviors among those who engage in binge eating. Close exploration of the experience of physical activity in the context of disordered eating and EDs is needed, especially given the population prevalence of BED relative to other EDs.

Acculturation, Acculturative Stress, and Eating Disorders

Few experts in the field now subscribe to the notion that EDs are "culture-bound," that they are peculiar to "Western" cultures (Anderson-Fye & Becker, 2003). Over the past 20 years, study after study has documented a rise in EDs in the developing world (Edmonds, 2012). In almost every case, however, that rise has occurred in the contexts of modernization (the shift

from a subsistence economy to a market economy) and "Westernization" (contact with the dominant cultures of North America and Western Europe as a result of migration, media exposure, or minority status; Anderson-Fye, 2011; Becker, 2004; Lester, 2004). Even if EDs are not unique to modern, Western cultures, aspects of modern, Western culture tend to be adopted in cross-cultural encounters and contribute to a rising prevalence of EDs in the receiving groups.

Acculturation refers to a change in norms, dispositions, and practices resulting from cross-cultural encounters. Acculturation is presumed to occur in proportion to exposure to a new culture, with cultures of developed nations exerting greater influence than the local culture in the developing nation. Acculturation is thought to be variable in its manifestation between groups, progressive (from low to high), and correlated with risk for EDs (Lester, 2004). This schema, of relative exposure and intrinsic resistance combining to produce a degree of acculturation and therefore a degree of ED risk, has been applied to U.S. minority cultures and around the globe with varying degrees of success. For example, despite substantial exposure to majority-race body norms, African American females for some time have been viewed as largely unacculturated to majority race body norms and therefore less likely to develop restrictive EDs. However, recent studies found few differences in the prevalence of EDs between African Americans and European Americans, with African Americans showing higher rates of bulimia nervosa (BN), and no differences being found on other disorders (Marques et al., 2011). U.S. Latina females appear to be simultaneously more and less acculturated; subscription to majority race body norms has been documented, along with adherence to appearance values more consistent with their cultures of origin (Rubin, Nemeroff, & Russo, 2004). In an interesting reversal of conventional trends, native Korean women actually exhibit a greater drive for thinness and disordered eating than Korean immigrant women in the United States (Jackson, Keel & Lee, 2006).

If, as the data suggest, the relationship between the rise of EDs and Westernization is meaningful (though not isomorphic), how can we make sense of these observed inconsistencies between acculturation effects and the risk and manifestation of EDs? One answer lies in our conceptualization of acculturation and its effects. The recent study by Becker et al. (2010) of acculturation in Fiji makes a strong argument for acculturation as a noncategorical, nonlinear phenomenon. That is, acculturation is not a unitary, incremental, and discrete process. Rather, Becker and colleagues found that acculturation manifests in multiple ways and life dimensions, and does so heterogeneously within groups. Similarly, Anderson-Fye (2004) found that local interpretation of transnational messages guided internalization of body ideals and disordered behaviors, which mitigated acculturative effects in Belize. Given these findings, it makes sense that acculturation has been inconsistently associated with development of EDs.

Acculturative stress is a reduction in health status resulting from inadequate resources and strategies for coping with acculturation processes and effects (Berry, Kim, Minde, & Mok, 1987). The degree of *acculturative stress*, however, has been identified as a moderator of the relationship between body dissatisfaction and ED symptoms among African American females. Acculturative stress also predicts bulimic symptoms among African American, Asian American, and Latina women (Kroon et al., 2013; Perez, Voelz, Pettit, & Joiner, 2002).

The identification of acculturative stress as salient to the development of EDs in ethnic minority populations is consistent with research that has identified structural inequality as a likely factor in the development of EDs (Brennan, Craig, & Thompson, 2012). Structural inequality and power differentials are inherent to the processes of acculturation and economic development. Whether the group in question is a longstanding minority population, migrant

population, formerly colonized population, or an economically struggling nation, power, resource, and/or opportunity gradients relative to a dominant group are involved. In addition, local variation in acculturative stress is created by local ethnopsychologies and individual differences (Anderson-Fye, 2004). Understanding acculturation and acculturative stress as dynamic and heterogeneous means that Western cultural influence matters everywhere, but not in the same way for everyone.

Class Culture and Eating Disorders

Structural dynamics within societies are also factors in ED prevalence and etiology. EDs were first identified among young, upper middle-class females, and through much of the 20th century they were thought to be more common among elite classes (Brumberg, 2000). Currently, the literature shows that no class group is immune to EDs, and that upward mobility is likely more important than class per se in risk for disordered eating and full-scale EDs (Anderson-Fye, 2011). Class-based body aesthetics are a significant factor in the development of EDs and are especially notable in situations of class mobility and in contexts of cultural change. Upward mobility and acculturative stress can dovetail to explain some of the high risk of EDs among migrant populations and populations experiencing globalization-influenced cultural change.

Body size is an indicator of social status and general health in many parts of the world (Anderson-Fye, 2012). Investigations of the historical and current meanings of body size cross-culturally consistently support this assertion. Thinness (or lean muscularity) is emblematic of middle-class status in Western societies, and "middle class" is what many in the developing world aspire to attain. Middle-class status connotes an abundance of resources, including a level of food security sufficient to make bodily energy storage (in the form of fat) unnecessary. Accompanying this resource abundance are social role obligations that include presenting a body size that conveys those life circumstances (Anderson-Fye, 2012).

Class-associated body aesthetics can figure particularly prominently in the drive for thinness among people who both exhibit disordered eating and are upwardly mobile (Thompson, 1994). The interaction between aspiration to higher social status, drive for thinness, and disordered eating has been documented in several qualitative studies of EDs in nonmajority groups. Katzman et al. (2004) offer this analysis in their case-control study of AN among mixed-race women in Curaçao:

> The women who developed anorexia on Curaçao reported that a thin body was indeed equated with greater success and belonging. Its attainment not only complied with appearance expectations but was worn as a blazer for those of an elite (wealthier) club. (p. 473)

Other studies have reported similar findings, affirming that body size concerns speak not only to cultural aesthetics, but are also a form of social validation (Popenoe, 2004; Puoane, Tsolekile, & Steyn, 2010; Thompson, 1994). The aspiration to higher social status can be accompanied by a felt pressure to achieve and maintain a particular bodily presentation for purposes of recognition as well as identity. Katzman et al.'s (2004) participants spoke of the difficulties of being mixed-race and identifying more with Whites than Blacks. Similar issues of not fitting neatly into recognized social categories or of resisting expected social roles are part of the narratives of upwardly mobile persons diagnosed with EDs (Thompson, 1994). Like acculturation, the relationship between upward mobility and EDs appears to be critically

important, yet neither linear nor orthogonal. Examining the combined effects of acculturation (or acculturative stress) and upward mobility may yield important insights into the etiology of EDs, particularly in racial, ethnic, sexual, class, or religious minority populations.

Cultural Change

Another recognized contextual concern in ED etiology is cultural change. The set of studies published as Volume 28, Issue 4 of *Culture, Medicine and Psychiatry* (2004), arguably represent the seminal ethnographic work in this area in the past 20 years. The papers by Pike and Borovoy (2004), Becker (2004), Anderson-Fye (2004), Katzman et al. (2004), and Le Grange et al. (2004)—all in that volume—give important theoretical and practical insights regarding how contexts of cultural change can play a role in the onset and presentation of EDs within populations. Theoretically, these studies suggest that it is not cultural change in and of itself (cultures are not static) that influences the development of EDs, but the pace and nature of that change. Rapid cultural change in the form of markedly increased exposure to U.S. entertainment media affected perceptions of what constituted an economically successful body in both Fiji and Belize, but did so in markedly different ways (Anderson-Fye, 2004; Becker, 2004). In Fiji, the slender body ideal was incorporated into the existing concept of individual bodies representing the collective. Just as robust bodies had traditionally been viewed as a symbol of belonging and being cared for, slender bodies came to be viewed by some of Becker's participants as emblematic of economic progress and success, and EDs were on the rise. In roughly the same time period in Belize, an ethos of self-protection and self-care along with perceptions of the body as "God given" and fixed rather than malleable seem to have contributed to a much lower incidence of EDs, despite similar levels of Western media exposure.

Practically, these studies provide great insight into cultural change as a multilevel phenomenon that must be approached as such if research is to be both accurate and meaningful. Pike and Borovoy demonstrated that evolving Japanese internal cultural dynamics concerning gender roles and beauty ideals were as much a part of the development of EDs in that country as the international dynamics that drove industrialization and urbanization. Le Grange et al. (2004) documented how a focus on changed policy and structure on a national level (the postapartheid South African educational system) without concomitant attention to social processes (educational efficacy and food insecurity) can contribute to mischaracterization of particular eating behaviors as disordered.

Cultural change is ongoing in all these countries, and, as is the case with the rest of the world, the pace of change has not slowed. Unfortunately, the prevalence of EDs has risen in all of them. However, the insights offered by this group of studies are that (a) rapid cultural change matters everywhere; (b) rapid cultural change is associated with an increase in EDs generally; and (c) the local context and meaning of that change *also* matters everywhere and is essential to our understanding of EDs as a cross-cultural phenomenon.

Ethnic and Racial Cultures

Ethnicity and race are difficult concepts to define. Frequently, in ED research the two concepts get conflated, even though they are distinct. Racial groups are socially constructed and vary by cultural and national context. Ethnic groups involve nuances of history, shared values, practices, and orientations that do not necessarily line up neatly with racial groups. For example, in the United States, African Americans are considered a racial group, though this group

involves many people of mixed racial descent and myriad ethnic groups. Similarly, "Latino" or "Hispanic" is considered a racial group, although the ethnicities that compose that group are many. Some ethnic groups, such as the Garifuna, may cross racial lines such as being African- and Native Carib-descended but Spanish speaking. Racial and ethnic group construction and norms are important to eating and its disorders, although, as with other variables, the relationships can be complex.

In much of U.S. EDs research, race has been considered irrespective of the cultures described previously. Such a collapsing of complexity does not reflect clinical or ethnographic realities. Recent work has shown the immense variation within racial groups glossed as unitary in the literature (McClure, Poole, & Anderson-Fye, 2012; Thompson, 1994). Understanding intragroup variation, especially when those groups are culturally constructed, is essential to prevention and treatment. Moreover, conflating race and ethnicity does a disservice to the empirical realities of risk and prevention of EDs.

Race and ethnicity both matter, though not in the same ways around the world. Foods considered desirable and undesirable, times and ways of eating (or fasting), and ritual food consumption can be shaped by ethnic groups. Racial categories profoundly impact structural inequalities but, yet again, are not isomorphic with them. A danger in U.S. research is to draw on small, relatively homogeneous populations that can be categorized into a single racial group and make generalizations about all people belonging to that racial group. A poignant example is the conventional argument that African American women value larger bodies than White women in the United States and therefore have fewer EDs. One of the corollaries of this overgeneralization was a clinical assumption that African American girls do not suffer EDs, which led to gross underdiagnosis and the finding that by the time African American girls are diagnosed, their disorder is more severe (Taylor, Caldwell, Baser, Faison, & Jackson, 2007). Others have written extensively on disordered eating as patterned by race and ethnicity (e.g., Franko, Becker, Thomas, & Herzog, 2007; see also Chapter 25).

Life Cycle Cultures

Life cycle matters for EDs everywhere. While disordered eating can span the entire human life cycle from infancy through old age, it is most heavily concentrated in adolescence and early adulthood (see Chapter 13). That said, rates of EDs have been increasing during other periods of human development as well (Rosen & The Committee on Adolescence, 2010). Developmental and institutional changes help explain the life-stage patterns, as do demographic factors such as life expectancy, infant mortality rates, and total fertility. For example, under conditions of longer life expectancy and lower fertility, adolescence is lengthened; it is in these contexts, both "Western" and "Eastern," that we see higher rates of EDs among adolescents. Macrolevel factors such as disease burden, educational system, and economic base also matter.

Adolescents and youth have particularly high rates of disorders. Developmentally, the cognitive, emotional, social, and identity development that occurs in adolescence attunes young people to peer comparisons, cultural ideals, and figuring out where they fit in relation to others (Erikson, 1978). The expansion of formal schooling around the world has age-segregated young people into peer cohorts in a new way. In peer-based social groups at school, social comparison and sharing of transnational media can flourish in a way that intensifies social pressure for bodies portrayed as ideal in Western media (Becker et al., 2010; see also Chapter 29). In the United States, school cultures continue to matter in terms of social

pressure, activity offerings, and intervention for problems. Specific subcultures can attenuate risk. For example, in Belize, as in much of Latin America and the Caribbean and beyond, high-school dances represent an extremely important ritual. Girls spend enormous resources on their outfits (e.g. one sixth of per capita income at one rural high school) and diet extensively in order to look a certain way in their dresses for that one night. After the event, eating is uncontrolled. A similar cycle ensues for the nation's abundant beauty pageants. This extreme cyclic dieting for the purposes of one "look" continues into adulthood where community celebrations become focal events.

For youth, the transition to college has been a notable time for the onset of disordered eating. Particularly for residential students, it can be the first time they are managing food consumption without supervision. Transition to college is also known to be generally stressful, with high rates of psychiatric illness, including EDs (Anderson-Fye & Floersch, 2011). Exercise is both lauded and a physiologically powerful way to manage stress. However, in college, it often functions as a gateway behavior for EDs. As mentioned earlier, sports teams that focus on the ornamentality of the body can further foster disordered eating. Sororities and fraternities have been found to contribute to behaviors both risky (e.g. social contagion of bulimia) and protective (e.g. social support) (see Chapter 44).

Other times of life also bring developmental and institutional pressures and buffers. For example, pregnancy is a poignant time for women's body image with some intensifying their concern and others finally breaking free of restrictive eating (Skouteris, 2012). In cultures where signs of aging are denigrated rather than respected, a resurgence of body image concerns can emerge during middle age and beyond. In sum, the developmental and physiological changes over the life cycle, combined with the institutions and subcultures where individuals spend their time, structure systematic differences in disordered eating. These differences have less to do with age, and more to do with subcultural groupings and culturally appropriate age-graded practices.

Intercultural and Intracultural Similarities and Differences: Context Matters

Several recent reviews have summarized regional differences in EDs around the world (Anderson-Fye, 2011; Anderson-Fye & Becker, 2003; Levine & Smolak, 2010). It is important to note that these trends are incredibly dynamic, changing regularly and sometimes dramatically. Recently, one of the most concerning trends includes an upward spike in the prevalence of body dysmorphia among males in all regions of the globe. These trends are also rooted in local economies and situations. For example, upwardly mobile black women in South Africa do not aspire to the slender ideal. Such a body could indicate positive HIV status, a highly stigmatized condition. Nor do they value certain fat distributions that would indicate antiretroviral (ART) treatment for HIV. Rather, they value an athletic body type with muscular definition that indicates work on the body (and the resources to do so), which also is a body difficult to attain with HIV (Puoane et al., 2010). Micro levels of cultural context help to define ideals and behaviors, even within one culture, region, or racial group.

Around the world, rates of disordered eating are consistently high in North America, Europe (both Western and Eastern), Australia and New Zealand, and the Middle East. Rates are largely unknown throughout Latin America, though the data that do exist suggest that the prevalence could be consistent with the aforementioned regions (Bojorquez & Unikel, 2004).

Disordered eating seems to be segmented by upward mobility and class status in the Caribbean (Anderson-Fye, 2004; Katzman et al., 2004), though there is rising concern about more widespread problems. Rates of disordered eating are possibly highest in parts of Asia, a diverse region that is showing cause for significant concern. In particular, Korea seems to be home to rates higher than in the United States and Western Europe among females, although the reasons for this trend are incompletely understood. Others speculate that Korea is not alone in Asia in its strong valuation of thinness and potentially a stigma against fat (Brewis, 2011). Sub-Saharan Africa, long thought to be immune from EDs, appears to yield mixed data that vary substantially based on the mix of cultural factors, including local cultures of gender, upward mobility, and disease burden described above. The Pacific islands are being investigated more fully, not only to understand a surprising emergence of disordered eating among young women (Becker et al., 2010), but also an increase in body dysmorphia and drive for male muscularity.

Undoubtedly, cross-national regional variation—where people may (or may not) share religion, language, food traditions, physical activity patterns, medical beliefs, body ideals, and ritual practices—depends on many other factors and varies across time. Yet, there is also consistency across these large territories in some patterns, and they warrant notice. For example, despite an enormous range of cultures and findings, concern about disordered eating across South and East Asia is supported by the literature. Specific studies that can be linked across regions are invaluable in understanding contemporary patterns.

Within the United States, there is enormous diversity in prevalence and symptoms of eating disorders as related to cultures of gender, sexuality, physical activity, acculturation, class, race and ethnicity, and life cycle. In addition, findings and patterns are constantly shifting and changing over time. Many have written about them. While the contemporary findings provide critically important data points, metareviews show that it is still difficult to draw many wide brush strokes that explain exactly *how* culture impacts disordered eating, although the data are incontrovertible that culture *does* matter. Filling in the many gaps and understanding what they mean when considered together is critically important in better attuning treatment and prevention to issues of culture. That said, some patterns are emerging clearly. For example, BED appears to be more prevalent among those in situations of structural inequality, such as people living in poverty and people of color, particularly women who are ethnic and racial minorities (Harrington et al., 2010; Reagan & Hersch, 2005). Understanding such trends, the reasons underpinning them, and the dynamics of expression of disorder is a crucial step in the process of improving outcomes.

Conclusions and Future Directions

This challenge of understanding is one we believe is best addressed ultimately by multidisciplinary and mixed-methods research. Qualitative and ethnographic research helps to explain—not just describe—patterns and is invaluable in helping to generate models that can then be quantitatively tested. Qualitative and ethnographic research tends to be limited by sample size but can dig deeply into lived experience and contextual factors important to models of disorder. Quantitative research can then test how widely applicable the findings are (Anderson-Fye & Lin, 2009). Conversely, when a surprising finding emerges in quantitative data, qualitative data can help to explain it (Anderson-Fye, 2004; Le Grange et al., 2004). In ED research, mixed-methods approaches often provide an ideal design to uncover patterns, understand how widespread they are, and gain insights into their meanings and dynamics.

Taken together, the findings discussed here point to the need to continue examination of variation both across and within subcultural groups. While much is known, many gaps and questions remain as diverse populations suffer from EDs. Significant differences exist in prevalence, symptoms, and dynamics of EDs based on cultural factors. These sorts of data can help us work at both the public health level and the clinical level for these serious disorders. For example, until the overwhelming data showing that ethnic and racial minorities were not immune from EDs, African American girls with AN went undiagnosed until much later due to clinician bias, among other factors. In Mexico, a country where family interdependence tends to be highly prioritized, ED treatment centers based on individualistic models developed in the United States tended to fail. Programs that included the family in culturally appropriate ways served clients better (Lester, 2007). These cases illustrate the critical difference research on cultural factors can make.

Taken together, the data indicate that culture matters significantly in EDs from macro levels to micro interactions. Sociocultural context plays an important role in almost every aspect of EDs. Cultural factors of gender, sexuality, physical activity, acculturation, class, race and ethnicity, and life cycle seem to have particular impact. We encourage a focus on intersectionality to understand intragroup variation and note that recent studies have paid increasing attention to how various cultures interact. We also note an increasing willingness to conceptualize that even within the same ED category, such as AN, enormous variation exists in the manifestation of symptoms and dynamics involved (see Chapters 2, 8, & 12). Such acknowledgement has led to better treatment (Lester, 2007). It is our hope that as nuances and meaningful variation are better understood and represented in the literature, our models of treatment and prevention will continue to improve—and a multiplicity of models may be our best course of action at this historical moment. We especially appreciate mixed-methods examinations that reach across disciplinary boundaries and see these as necessary to understand the operation of cultural dynamics—that is, where culture matters.

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24

Dieting as a Risk Factor for Eating Disorders

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Dieting is one of the most widely studied risk factors for eating disorders. Dieting refers to intentional and sustained restriction of caloric intake for the purposes of weight loss or maintenance (Wilson, 2002). Dietary restriction must result in a negative energy balance for weight loss or a balance between intake and output for weight maintenance. About 60–75% of dieters combine reduced caloric intake with increased exercise, though some report unhealthy weight control behaviors, such as fasting, vomiting, or laxative use (French, Perry, Leon, & Fulkerson, 1995). The duration of weight loss diets appears to range from less than a week to 6 months (French, Jeffery, & Murray, 1999). Polivy and Herman (1985) argue that the chronic hunger experienced by dieters increases the risk of binge eating, and that a reliance on cognitive control over eating leaves dieters vulnerable to uncontrolled eating when these cognitive processes are disrupted. Binge eating theoretically precipitates dietary restraint and the use of compensatory weight control techniques (e.g., vomiting), possibly escalating into a binge-purge cycle.

Determining whether dieting increases risk for eating disorders is crucial because 30% of young women report dieting (Field et al., 2003) and low-calorie diets are prescribed to treat obesity, often resulting in significant weight loss and improved health (Heilbronn et al., 2006). Yet, debate remains as to whether dieting increases risk for eating disorders and whether it is a helpful treatment for both eating disorders and obesity. Indeed, given the evidence that self-reported dieting is associated with increased risk for future onset of eating disorder (ED) symptoms and disorders, numerous researchers and clinicians assert that dieting is causally related to bulimia nervosa (BN; Fairburn, 1997; Neumark-Sztainer et al., 2006; Polivy & Herman, 1985), with some calling for a moratorium on dieting (Bacon et al., 2002; Polivy & Herman, 1992).

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Prospective Studies of the Relation of Dieting to Future Eating Pathology

Numerous prospective studies have found that young women with elevated scores on dietary restraint scales, those who report frequent weight loss dieting, and those endorsing any dieting are at increased risk for future onset of binge eating and other ED symptoms (Field et al., 1999; Goldschmidt, Wall, Loth, Le Grange, & Neumark-Sztainer, 2012; Neumark-Sztainer et al., 2006; Stice, Presnell, & Spangler, 2002), future increases in ED symptoms (Johnson & Wardle, 2005; Stice, 2001; Wertheim, Koerner, & Paxton, 2001), and onset of threshold and subthreshold BN (Killen et al., 1996; Stice, Davis, Miller, & Marti, 2008). Adolescent girls who have elevated scores on dietary restraint scales, report frequent dieting, or self-identify as dieters are at increased risk for future increases in measures of overall ED symptoms (Leon, Fulkerson, Perry, Keel, & Klump, 1999) and future onset of any ED (i.e., anorexia nervosa [AN] and BN; Patton, Selzer, Coffey, Carlin, & Wolfe, 1999; Santonastaso, Friederici, & Favaro, 1999; Stice, Marti, & Durant, 2011), though this latter effect has sometimes not emerged (Jacobi et al., 2011). The effect sizes from these methodologically rigorous studies (i.e., often utilizing long follow-up periods and validated diagnostic interviews) are typically large. For instance, adolescent girls with elevated dieting scores showed a 3.6-fold increase in risk for onset of any ED (odds ratio = 4.3; Stice et al., 2011).

Experiments Examining the Relation of Short-Term Caloric Deprivation to Lab-Based Eating

Only two experiments have tested whether acute dietary restriction is a maintanence factor for binge eating. Telch and Agras (1996) found that 6-hour caloric deprivation did not result in significantly greater self-reported binge eating episodes among women with BN, binge eating disorder (BED), or obesity relative to a no-deprivation control condition. Agras and Telch (1998) found that 14-hour caloric deprivation, relative to a no-deprivation control condition, produced significant increases in investigator coded binge eating, but not self-labeled binge eating, in women with BED, suggesting that longer, but not shorter periods of abstinence from caloric intake may serve as a maintenance factor for binge eating.

Experiments that examined the effects of abstinence from caloric intake on subsequent caloric intake in the lab (vs. binge eating) have also produced mixed findings. Short-term caloric deprivation relative to no-deprivation control conditions resulted in elevated caloric intake among women and men without EDs in two experiments (Mauler, Hamm, Weike, & Tuschen-Caffier, 2006; Spiegel, Shrager, & Steller, 1989), but this effect did not emerge in three other experiments (Hetherington, Stoner, Andersen, & Rolls, 2000; Schachter, Goldman, & Gordon, 1968; Spiegel et al., 1989). Short-term caloric deprivation also produced significantly elevated caloric intake among women with BN (Hetherington et al., 2000; Mauler et al., 2006), but this effect did not replicate in young women with AN or in healthy controls (Hetherington et al., 2000). Assignment to longer periods of dietary restriction resulted in significantly elevated acute caloric intake in young women following consumption of high-calorie food and during stress (Wardle & Beales, 1988), but this effect did not replicate in three other experiments (Lowe, 1992; Lowe, 1994; Lowe, Foster, Kerzhnerman, Swain, & Wadden, 2001). Note that certain reports describe results from multiple experiments (Speigel et al., 1989) or multiple subpopulations (Hetherington et al., 2000).

Several studies have experimentally manipulated caloric deprivation to examine the influence on responsivity of reward, attention, and gustatory brain regions to food stimuli. Functional magnetic resonance imaging (fMRI) experiments indicate that activation in regions that have been implicated in attention (anterior cingulate cortex), reward valuation (amygdala), and homeostatic feeding (hypothalamus) was significantly greater in response to pictures of palatable foods versus nonfood control images after caloric deprivation versus a no-deprivation condition (Fuhrer, Zysset, & Stumvoll, 2008; LaBar et al., 2001; Leidy, Lepping, Savage, & Harris, 2011), though null effects also emerged in studies with very small samples (Siep et al., 2009; Uher, Treasure, Heining, Brammer, & Campbell, 2006). One experiment found a greater response in taste- and reward-regions (amygdala, orbitofrontal cortex [OFC], insula, and striatum) to pictures of high-calorie versus low-calorie foods after a period of caloric deprivation versus a nondeprivation condition (Goldstone et al., 2009), suggesting that caloric deprivation increases the reward value of high-calorie foods more than low-calorie foods. Similarly, greater response in the primary taste cortex (anterior insula, frontal operculum) and regions associated with reward (medial prefrontal cortex) in response to intake of food (chocolate milk, chicken soup) after caloric deprivation versus a nondeprivation condition was observed (Uher et al., 2006). A study that examined self-imposed dietary restriction found that duration of acute caloric deprivation correlated positively with activation in regions implicated in attention, reward, and motivation (e.g., anterior cingulate cortex, OFC, striatum, and precentral gyrus respectively) in response to images, anticipated receipt, and receipt of palatable food (Stice, Burger, & Yokum, 2013). In response to the same stimuli, youth in a longer-term negative energy balance, assessed by highly standardized repeated measurements of weight, showed greater activation in similar attention, visual processing, and reward regions relative to those in neutral or positive energy balance (Stice et al., 2013).

A potential mechanism for the effects observed in the fMRI studies comes from animal experiments. In vivo dialysis experiments show higher food-induced dopamine release in fasted versus satiated rats (Wilson, Nomikos, Collu, & Fibiger, 1995). Intracerebroventricular injection of the dopamine receptor subtype 2 (DR_2) agonist quinpirole produced a more pronounced striatal neuronal activation in caloric-restricted rats versus ad lib fed controls (Carr, Tsimberg, Berman, & Yamamoto, 2003). Accumbens dopamine levels increase in response to caloric intake more following longer versus shorter caloric deprivation periods (Yoshida et al., 1992) and in response to caloric deprivation weight loss diets versus baseline (Avena, Rada, & Hoebel, 2008). Data collectively suggest that abstinence from caloric intake increases DR_2 sensitization, which could explain the greater reward value of food after caloric deprivation.

Thus, experiments indicate that short-term abstinence from caloric intake did not have consistent effects on binge eating among eating-disordered participants or on general caloric intake among eating disordered and nondisordered participants. These studies have generally been methodologically sound, although the moderate samples sizes may have limited the ability to detect small effects, and possibly social desirability biases may have influenced participants' eating. However, experimental and nonexperimental brain imaging studies suggest that abstinence from caloric intake does increase the reward value of food, particularly high-fat and high-sugar palatable foods. The fMRI data are more immune to self-presentation biases because they rely on objectively measured oxygen utilization to reflect brain regions that respond to experimentally manipulated presentation of stimuli (e.g., tastes of palatable food). These latter data imply that dieting characterized by acute abstinence from dietary intake may be bound to fail, as it increases the reward value of energy-dense food with every passing hour

of dietary abstinence. Results imply that rather than completely abstaining from dietary intake for weight control purposes, it might be more effective to simply replace intake of unhealthy energy-dense food with healthy foods that are less energy dense.

Experiments Examining the Relation of Longer-Term Dieting to Eating Disorder Symptoms

Experimental psychopathology trials have examined the effects of weight loss diet interventions on ED symptoms among nonobese young adults. Presnell and Stice (2003) found that young women assigned to a 6-week weight loss diet intervention, which resulted in significant weight loss, showed significantly greater reductions in binge eating and bulimic symptoms during the dieting intervention. These findings were replicated in women assigned to two types of 6-week weight loss interventions, one aimed at increasing frequency of small meal consumption (Groesz & Stice, 2007).

One randomized controlled prevention trial (see Chapter 44) found that a weight maintenance diet intervention that promoted lasting gradual reductions in caloric intake and increases in exercise and significantly reduced risk for weight gain and onset of obesity over 3-year follow-up, also produced significant reductions ED symptoms and risk for future onset of threshold and subthreshold EDs, relative to controls (Stice, Marti, Spoor, Presnell, & Shaw, 2008). A second randomized prevention trial found that a refined version of this intervention that incorporated health behavior change principles from nutrition science, which significantly reduced increases in weight gain through 6-month follow-up, likewise reduced increases in ED symptoms and risk for onset of threshold and subthreshold EDs through 2-year follow-up (Stice, Rohde, Shaw, & Marti, 2012).

Trials have also examined the effects of long-term weight loss diets on binge eating and ED symptoms among overweight individuals. One found that assignment to a 20-week low-calorie weight loss intervention produced significantly greater decreases in binge eating among overweight women (Klem, Wing, Simkin-Silverman, & Kuller, 1997). However, another trial did not replicate this effect (Redman, Martin, Williamson, & Ravussin, 2008).

Randomized treatment trials have also examined the effects of weight loss dieting interventions among individuals with BN or binge eating disturbances. Two controlled trials found that assignment to a low-calorie weight loss diet resulted in significantly greater decreases in binge eating for overweight and obese women who endorsed initial binge eating, but did not result in weight loss (Goodrick, Poston, Kimball, Reeves & Foreyt, 1998; Reeves et al., 2001). Another trial found that assignment to a 6-week weight loss diet intervention, which resulted in significant lasting weight loss, produced significantly greater reductions in binge eating and compensatory behaviors among women with threshold or subthreshold BN than observed in wait-list controls (Burton & Stice, 2006). Another trial found that assignment to a behavioral weight loss treatment resulted in significantly greater reductions in weight relative to cognitive-behavior therapy for obese individuals with binge eating disorders, but that cognitive-behavioral therapy produced significantly higher abstinence rates from binge eating (Grilo, Masheb, Wilson, Gueorguieva, & White, 2011).

In sum, there is considerable support from both randomized controlled trials and noncontrolled interventions that low-calorie weight loss or weight maintenance diets resulted in significantly greater reductions in binge eating and bulimic symptoms relative to assignment to a control condition. Importantly, results from some of these trials suggest that weight

maintenance dieting may represent an efficacious prevention intervention for eating pathology (see Chapter 44). Although there was also evidence that behavioral weight loss interventions can reduce binge eating and bulimic symptoms, this type of intervention has not consistently produced reductions in both eating disordered behavior and excess weight. However, there is some possibility that demand characteristics inherent to trials of weight loss or maintenance interventions have contributed to the reported reductions in binge eating and ED symptoms. Only a few of these studies utilized active alternative interventions, which should have reduced risk for this possibility. For instance, one trial found that a weight maintenance intervention produced significantly greater reductions in binge eating and bulimic symptoms relative to an alternative intervention (Stice, Shaw, Burton, & Wade, 2006); this same intervention produced significantly less weight gain than observed in two credible alternative interventions at 3-year follow-up (Stice, Marti, et al., 2008).

Possible Explanations for the Inconsistent Findings

Thus, numerous prospective studies have found that individuals who report dieting are at increased risk for future onset of binge eating, bulimic pathology, and EDs. However, several experiments have also suggested that assignment to weight loss diets results in significantly greater reductions in binge eating and bulimic symptoms and greater weight loss than assignment to assessment-only control conditions and in some cases alternative interventions. There are several potential reasons for this pattern of findings.

One possible explanation for the contradictory findings between the prospective and experimental studies is suggested by the fact that these two types of research designs differ in their inferential power. The key weakness of a prospective design is that it is always possible that some omitted third variable accounts for any prospective effect observed in a longitudinal study. That is, some confounding factor might cause both elevated dieting and elevated ED symptoms. In contrast, randomized experiments were developed to rule out third variable alternative explanations. Researchers randomly assign participants to conditions to create groups that are equivalent on all potential confounding factors (known or unknown), which theoretically allows the investigator to isolate the effect of the one variable that is manipulated (because potential confounding factors should be uncorrelated with treatment condition). Although random assignment can fail in creating initially equivalent groups on all potential confounding factors, this is the best available tool to rule out third variable confounding factors, even when these potential confounding factors are unknown. Thus, because randomized experiments are particularly effective for ruling out third variable explanations and prospective studies are not, the positive relation of self-reported dieting to increases in eating pathology may have emerged because some third variable increases the risk for both variables. However, one troubling aspect of this explanation for the inconsistent findings is that the findings from the prospective and experimental studies are consistently in the *opposite direction*. Typically, when prospective effects are due to an omitted confounding variable, experimental studies that manipulate the independent variable would not produce effects on the dependent variable.

Another possible explanation for the inconsistent results from the prospective and experimental studies is that the weight loss diets evaluated in the experiments involve more extreme dietary restriction than real world weight loss dieting. Although this explanation holds intuitive appeal, it does not appear to be a satisfactory account for the inconsistent findings, for several

reasons. First, if self-initiated diets are simply less effective than the weight loss diets evaluated in the experiments, then one would predict that the effects from the prospective studies would be smaller than the effects from the experiments involving prescribed diets, but in the same direction; yet, the effects from the prospective studies are consistently in the *opposite direction* relative to those from the experimental studies. Second, both lower intensity weight maintenance interventions (Klem et al., 1997; Stice, Marti, et al., 2008) and higher intensity weight loss interventions (Groesz & Stice, 2007; Presnell & Stice, 2003) produce significant reductions in binge eating and ED symptoms. This pattern of findings implies that even more modest weight control diets reduce ED symptoms. Third, the weight loss observed in the experimental trials that evaluated prescribed weight loss diets was small. These trials observed that participants lost an average of 0.14 kg (0.3 lb) per week. Fourth, the weight loss and weight maintenance diets from the experiments were similar in duration (mode = 1.5 months, range = 1 month to 18 months) to the reported duration of weight loss diets practiced in the real world (mode = 1 month, range = 1 week to 6 months; Emmons, 1992; French et al., 1999; Williamson, Serdula, Anda, Levy, & Byers, 1992). These considerations imply that it is unlikely that the experimental studies produced different findings from the prospective studies because the weight loss interventions evaluated in the former were more extreme than the typical weight loss diets used in the real world.

A third possible explanation for the conflicting findings is that the prospective studies that found dieting predicted future onset of or increases in eating pathology may have used invalid measures of dieting. Consistent with this notion, studies that have used objective measures of caloric intake reveal that none of the widely used dietary restraint scales used in the literature showed the expected inverse correlations with caloric intake during a single eating episode (Hetherington et al., 2000; Jansen, 1996; Ouwens, van Strien, & van der Staak, 2003; Stice, Fisher, & Lowe, 2004; Sysko, Walsh, & Wilson, 2007), as suggested by the original validity studies that relied on self-reported caloric intake (e.g., French, Jeffery, & Wing, 1994; Neumark-Sztainer, Jeffery, & French, 1997). Other studies have found that dietary restraint scales did not correlate with objectively measured caloric intake during multiple eating episodes (Jansen et al., 2003; Martin et al., 2005; Rolls et al., 1997; Stice, Cooper, Schoeller, Tappe, & Lowe, 2007; Sysko, Walsh, Schebendach, & Wilson, 2005). Additional studies found that individuals with high versus low scores on various dietary restraint scales do not consume fewer calories over 2–12-week periods per double-labeled water estimates of habitual caloric intake or unobtrusively measured food purchases (Bathalon et al., 2000; Stice et al., 2007; Stice, Sysko, Roberto, & Allison, 2010).

These validity findings seem to provide a compelling explanation for why the prospective studies and experimental studies have produced inconsistent findings; although the experiments were largely examining the effects of confirmed calorie-deficit diets, the prospective studies were examining individuals who do not appear to be on a caloric-deficit diet necessary for weight loss. However, one puzzling aspect of this explanation is why individuals who are either unable to achieve a true caloric-deficit diet or who want to give the impression that they are on such a diet are at increased risk for onset of binge eating and bulimic pathology. If the dietary restraint measures are invalid, such that individuals with high dietary restraint scores do not eat less than those with low dietary restraint scores, it seems that the prospective studies should simply have produced null findings with regard to the relation between dietary restraint scales and future eating pathology. However, the fact that the prospective studies have observed significant positive relations between initial scores on dieting measures and future increases in eating pathology implies that these scales assess a latent construct, other than energy-deficit

dieting, which increases the risk for future onset of eating pathology. That is, while it is tempting to dismiss the findings from the prospective studies because they appear to have used invalid measures of dietary restriction, one fact remains: these scales have strikingly consistent predictive validity for future development of eating pathology.

This analysis suggests that self-reported dietary restriction is a *proxy risk factor* for eating pathology, but that the nature of the true latent construct that is tapped by these scales has yet to be identified. A proxy risk factor is a variable that predicts a pathological outcome not because it has any causal relation to the development of pathology, but because it correlates with a true causal risk factor for the pathology (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001).

One other intriguing implication of the validity findings is that dietary restraint scales may assess *relative dietary restriction* rather than *absolute dietary restriction*. That is, these scales may be identifying people who are curbing an overeating tendency, but who are not actually achieving the negative energy balance necessary for weight loss. Because these individuals are eating less than they normally eat or less than they desire, they may perceive this relative restriction as dietary restraint despite the fact that they are not achieving the negative energy balance necessary for weight loss. This interpretation is consistent with the evidence that (a) intermittent dieters temporarily arrest a weight gain trajectory while they are attempting to engage in a weight loss diet, but do not show weight loss (Presnell, Stice, & Tristan, 2007); (b) individuals with elevated dietary restraint scores consumed significantly more calories than those with low dietary restraint scores in one study, but did not feel that they had overeaten (Jansen, 1996); and (c) dietary restraint scores often increase when individuals are placed on low-calorie diets relative to controls who are not placed on weight loss diets (Groesz & Stice, 2007; Williamson et al., 2007). This analysis suggests that a tendency toward overeating may give rise initially to efforts to reduce this overeating and eventually lead to binge eating onset.

There are two other possible alternative explanations for the inconsistent findings that warrant further research. First, it is possible that the reductions in ED symptoms in the trials are a product of the demand characteristics of experiments in which participants are assigned to a weight loss or weight maintenance diet condition. It is worth exploring this possible explanation with experiments that use credible placebo control conditions, confederate reports of ED symptoms, or objective measures of these symptoms.

A second potential alternative explanation for the inconsistent findings between the prospective and experimental studies is that a subset of dieters engages in a particularly unhealthy form of dieting, such as extreme fasting, which increases risk for bulimic symptoms for this subset of dieters. This interpretation is suggested by the fact that only a small subset of dieters eventually develops binge eating and BN. Consistent with this speculation, one prospective study found that self-reported fasting significantly increased risk for future onset of recurrent binge eating and threshold/subthreshold BN over a 5-year follow-up, with predictive effects that were consistently larger than the predictive effects of self-reported dieting (Stice, Davis, et al., 2008). These relations have been replicated in a study of adolescents at high risk for onset of EDs by virtue of body dissatisfaction (Stice & Marti, 2013). The evidence that abstinence from caloric intake shows a stronger relation to future onset of binge eating and bulimic pathology than elevated scores on dieting scales converges with the brain imaging studies noted above that suggest that acute abstinence from dietary intake increases the reward value of food, particularly energy-dense foods that are typically consumed during binge eating episodes.

Conclusions and Future Directions

Experiments from multiple labs that evaluated weight loss and weight maintenance diet interventions suggest that neither cause eating pathology. This suggests that obesity prevention and treatment interventions that prescribe weight loss dieting do not produce iatrogenic effects (see Chapters 44 & 49). Indeed, these experiments imply that weight maintenance dieting is an efficacious method of preventing ED onset and have provided some evidence that weight loss dieting is an efficacious treatment for binge eating and BN. Ironically, to our knowledge the only ED prevention program that has been found to significantly reduce the incidence of future ED onset in multiple trials is an intervention that promotes gradual reductions in energy-dense foods and increases in physical activity that are designed to bring caloric intake into balance with caloric expenditure. These data strongly suggest that the dietary restraint model of eating pathology is in need of refinement. One remarkable feature of this literature is the confidence that has been placed in the assertion that dieting causes bulimic pathology in the absence of any experimental evidence that actual dietary restriction increases bulimic symptoms (Fairburn, 1997; Neumark-Sztainer et al., 2006; Polivy & Herman, 1985).

The studies reviewed here also illustrate the importance of documenting the validity of measures with methods that are less subject to distortion by social desirability. It is often useful to re-evaluate assumptions when confronted with findings that appear to be inconsistent, such as whether the scales we use actually assess what they purport to assess.

Further, this review underscores the hazards of relying solely on prospective studies when making etiologic inferences. The fact that it is impossible to rule out third-variable alternative explanations for prospective findings from longitudinal studies serves as a potent reminder of the importance of using randomized experiments that manipulate putative etiologic factors to confirm causal relations suggested by prospective studies. Only through the use of the most rigorous research designs available can we hope to develop valid etiologic models for psychopathology and design optimally effective prevention and treatment interventions.

This review suggests several directions for research. First, researchers should seek to identify the third variable that is tapped by dietary restraint scales that truly increases risk for onset of bulimic pathology, because this will advance our understanding of the etiologic processes that cause EDs. Future studies should attempt to experimentally manipulate any potential third variables that are identified to allow more rigorous inferences. Second, it will also be important to conduct experiments that manipulate specific weight loss behaviors. As noted, it appears that a subset of individuals with elevated dietary restraint scores engage in extended abstinence from caloric intake (i.e., fasting), which apperas to increase risk for binge eating. We think it vital to determine which particular dieting behaviors increase risk for binge eating. For ethical reasons, it would be best to reduce these unhealthy behaviors experimentally. Third, future research should develop valid measures of dietary restriction. Without valid measures of dieting it will be impossible to draw accurate inferences regarding the consequences of dieting. Given the power of self-presentation biases, we suspect it may be necessary to resort to objective measures of whether participants are in a negative energy balance or not, as assessed by standardized repeated anthropomorphic assessments. Finally, given the evidence that weight loss dieting curbs binge eating and ED symptoms in prevention and treatment trials, it will be useful to develop even more effective interventions that promote weight maintenance diets for individuals at a healthy weight and weight loss diets for overweight individuals. The increasing prevalence of obesity suggests that assisting individuals with healthy weight control is a particularly pressing need from a public health perspective. After all, obesity is responsible

for considerably more morbidity and mortality than EDs. Prevention programs that reduce risk for both EDs and obesity clearly have greater public health significance than programs that only affect one of these outcomes (see Chapter 49).

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Ethnicity as a Risk Factor for Eating Disorders

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The idea that ethnicity might play a role in the etiology of eating disorders (EDs) has received considerable attention in the academic and clinical literatures. Early accounts postulated that Caucasian females were at increased risk for EDs relative to members of "minority" ethnic groups (e.g., African American individuals), but recent work suggests a more complex relation between ethnicity and EDs (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004; Striegel-Moore & Bulik, 2007). In this chapter, we review evidence that ethnicity is a risk factor for EDs. Following the guidelines of Jacobi et al. (2004), we conceptualize ethnicity as a potential *fixed marker* of EDs, that is, "a risk factor that cannot be changed or change spontaneously" (p. 20). However, as detailed later in the "Conclusions and Future Directions" section, a multifaceted conceptualization of ethnicity that includes fixed (e.g., race) and variable (e.g., acculturation, socioeconomic status, experiences of discrimination and harassment) components might have greater utility for advancing models of ED prevention and treatment.

Methodological Considerations

Before evaluating evidence that ethnicity is a risk factor for EDs, it is important to consider the ways in which limitations in study methodology might have influenced research findings. Below we highlight two areas that are especially relevant to the current review: (a) measurement issues, and (b) traditional definitions of "caseness."

Measurement Issues

The way in which psychological constructs are measured can have a significant impact on research findings. Thus, it is important to examine the psychometric properties of ED measures in different ethnic groups (see Chapter 40).

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Reliability Reliability analyses in the ED field have focused primarily on measurement of internal consistency reliability and test-retest reliability and stability. Collectively, this work indicates that internal consistency estimates are similar across U.S. ethnic minority and nonminority women for a variety of ED measures (Bardone-Cone & Boyd, 2007; Fernandez, Malacrne, Wilfley, & McQuaid, 2006; Franko et al., 2012; Henrickson, Crowther, & Harrington, 2010; Warren et al., 2008). In addition, data suggest that test-retest reliability and stability of ED measures generally are good to excellent in U.S. ethnic minority women (Bardone-Cone & Boyd, 2007; Franko et al., 2012). Nevertheless, results from one study showed that test-retest reliability for the Three Factor Eating Questionnaire (TFEQ)-Restraint scale was lower in African American women compared to Caucasian women (Bardone-Cone & Boyd, 2007). Bardone-Cone and Boyd (2007) also found that subjective binge eating did not demonstrate significant rank-order stability in African American women. In sum, available data indicate that reliability is not a serious measurement concern in ethnic minority research in the ED field; however, additional work examining the psychometric properties of restrained eating and subjective binge eating episodes in ethnic minorities may be warranted. There also is a need for research on the reliability of ED measures in ethnic groups outside of the United States.

Validity Although the majority of ED self-report measures demonstrate adequate reliability across U.S. ethnic groups, this does not imply that ED measures are equally valid in ethnic minority and Caucasian individuals. Importantly, if ED measures are differentially valid across ethnic groups, there is a risk that ethnic minority individuals might score lower than their Caucasian counterparts, despite having similar levels of disordered eating.

A rigorous test of differential validity is multiple-group analysis (MGA) (Brown, 2006). MGA is a form of structural equation modeling that can be used to determine if a measure is invariant across groups. Evidence for factor structure variance suggests that a measure is not assessing the same latent construct between groups. MGA proceeds in multiple steps. The first step, *configural invariance*, tests the degree to which the model fits the data in each group separately. Subsequent steps evaluate *metric invariance*, which tests whether items comprising a scale have similar factor loadings, intercepts, and residual error variances among groups, and whether the latent means for the scale vary between groups.

To our knowledge, three studies have applied MGA procedures to test whether common ED measures are invariant between ethnic minority and Caucasian persons in the United States, and one study has examined whether a measure of body dissatisfaction, a construct that is highly correlated with EDs, is invariant across individuals from different countries and cultural groups. For example, Warren et al. (2008) found that a one-factor model for the Body Shape Questionnaire (BSQ) demonstrated a good-to-excellent fit in Euro-American and Hispanic-American women, which provides evidence for configural invariance. However, constraining the factor loadings to be equal for the BSQ resulted in a modest decrement in model fit, indicating between-group differences in the factor loadings (i.e., metric variance). An important strength of Warren et al.'s study was that they used evidence for factor variance to derive a set of items that met criteria for strict metric invariance between groups. Such information will be useful to future researchers and clinicians seeking to assess body dissatisfaction in Latina women.

In contrast to the findings of Warren et al. (2008), two studies using MGA documented configural variance (i.e., differences in model fit) of common ED measures among different U.S. ethnic groups. Fernandez et al. (2006) compared the factor structure of the Bulimia Test-Revised (BULIT-R) in African American, Asian American, Caucasian American, and

Latina American college women. Results were somewhat difficult to interpret because model fit was poor in all groups (including the Caucasian group), but based on the fit indices reported, it appeared that the initial five-factor model for the BULIT-R had the worst fit among African American women. Similarly, Kelly et al. (2012) examined configural and metric invariance for the Binge Eating Scale (BES), Eating Disorder Diagnostic Scale (EDDS), Eating Attitudes Test-26 (EAT-26), and BULIT-R in a well-powered sample of African American and Caucasian female undergraduate college students. Results showed that only the BES demonstrated an acceptable fit to the data, and several measures had the worst fit among African American participants. Collectively, these findings suggest that the majority of ED self-report assessments are measuring different constructs in African American and Caucasian women, which has important implications for the interpretation of risk factor research using these instruments.

Finally, Fuller-Tyszkiewicz et al. (2012) used MGA procedures to test configural and metric invariance for the five-item version of the Body Change Inventory (BCI) in large independent samples of adolescent males and females from Australia, Chile, Greece, Indigenous Fiji, Malaysia, Indo-Fiji, and Tonga. The BCI was chosen because it was specifically developed to test body image between genders and among cultures in adolescents. Results showed that the BCI had configural and metric invariance between genders within each culture. The BCI also demonstrated configural invariance in the combined sample (which included both males and females across cultures), and evidence for partial metric invariance; specifically, factor loadings were invariant among cultural groups, but item intercepts were noninvariant. Within a MGA context, the intercept refers to the predicted value of an observed variable when the latent variable is zero (Brown, 2006). In other words, these results mean that the predicted score for a variable (or set of variables) on the BCI varied among cultural groups who had the same latent level of body image concerns. Although it is somewhat unclear exactly how cultural groups differed in their [biased] responses to questionnaire items, a notable strength of this study was the rigorous examination of both configural and metric invariance, as well as the inclusion of gender as a potential source of body image invariance. Together, results show that certain body image items demonstrate small to moderate evidence for systematic cross-cultural bias among adolescents.

Traditional Definitions of "Caseness"

Apart from differences in how ED behaviors are measured, there may be variance in ED symptom expression among ethnic groups. Cross-cultural and cross-ethnic differences in EDs are covered in detail in Chapters 6, 7, 16, and 23. However, several issues that have relevance to the interpretation of risk factor research are noted below.

First, studies have indicated that ED presentations in non-Western environments differ from diagnoses included in leading nosological schemes, for example, the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2013), which could impact efforts to evaluate the relative prevalence of EDs in different ethnic groups. For example, in a series of patients receiving treatment in Hong Kong, Lee, Ho, and Hsu (1993) described a phenotype of "non-fat-phobic" anorexia nervosa (AN) characterized by low weight and amenorrhea in the absence of a fear of weight gain or becoming "fat." Cross-cultural research also has documented a form of ED characterized primarily by the use of indigenous Fijian herbal purgatives that has not been observed in Westerns samples (Thomas, Crosby, Wonderlich, Striegel-Moore, & Becker, 2011).

In addition to studies conducted in non-Western countries, research in the United States has found ethnic differences in ED presentation that have implications for risk factor work. For example, Bennett and Dodge (2007) reported that Asian Americans and Native Americans were more likely than other ethnic groups to report embarrassment associated with binge eating episodes, whereas Hispanic Americans were more likely than African Americans and Caucasians to report fear of loss-of-control during binge eating episodes. Finally, nationally representative epidemiological research suggests that the temporal course of eating pathology may vary among ethnic-racial groups, with African Americans demonstrating an earlier age of onset and a shorter duration of illness when compared to primarily Caucasian samples (Franko, 2007).

Evidence that Ethnicity is a Risk Factor for EDs

With methodological considerations in mind, we now review findings from several lines of research that help to explicate whether ethnicity is a risk factor for EDs. We begin by reviewing data from recent epidemiologic studies and studies conducted in large community samples that have compared rates of EDs in different ethnic groups, an approach that is consistent with the notion that ethnicity is a *fixed marker* of ED risk (Jacobi et al., 2004). Next, we present longitudinal research examining the effect of ethnicity on the future expression of EDs. Third, we review studies that have examined ethnic differences in putative risk factors for EDs (e.g., internalization of thin ideal) to identify mechanisms by which ethnicity might differentially influence ED risk. Finally, we close by considering the effects of within-group heterogeneity on the relation between ethnicity and EDs.

Epidemiologic Studies and Studies in Large Community Samples

One approach to evaluating the impact of ethnicity on ED risk is to compare rates of ED symptoms and diagnoses in different ethnic groups (Jacobi et al., 2004). Research in this area has advanced significantly in the last 10 years, with the publication of data from nationally representative epidemiologic studies, as well as several large-scale community samples.

Epidemiologic Studies We identified four studies that have examined the relative prevalence of ED behaviors in ethnic minorities and Caucasian individuals using data from nationally representative samples in the United States. Collectively, this work challenges the prevailing notion that only European American women are at risk for the development of EDs.

For example, Marques et al. (2011) documented rates of AN, bulimia nervosa (BN), binge eating disorder (BED), and any binge eating in Latino, Asian, and African American women and men relative to non-Latino White women and men using pooled data from the National Institute of Mental Health Collaborative Psychiatric Epidemiology Studies. No ethnic differences in current (past 12 months) or lifetime prevalence estimates of AN or BED were observed in the sample overall or in men and women separately. Moreover, in the sample overall, lifetime and 12-month prevalence of BN was higher in Latinos and African Americans compared to non-Latino Whites. Lifetime prevalence of BN also was higher in Latino men than in non-Latino White men. Finally, lifetime prevalence estimates for any binge eating were higher in Latinos (overall and in men and women separately), Asians (overall), and African Americans (overall and in women separately) than in non-Latino Whites; in addition, 12-month prevalence

of any binge eating was higher in Latinos and African Americans relative to non-Latino Whites (overall and in women separately).

Swanson, Crow, Le Grange, Swendsen, and Merikangas (2011) reported similar findings in a study examining the prevalence and correlates of AN, BN, BED, and subthreshold AN and BED in a nationally representative sample of adolescent girls and boys aged 13–18 years. Using data from the National Comorbidity Survey Replication–Adolescent Supplement, Swanson et al. found a significant effect of ethnicity on lifetime prevalence of BN. Rates of BN were highest in Hispanic adolescents followed by "other" ethnic groups, non-Hispanic Blacks, and non-Hispanic Whites, respectively. There also was a trend toward higher rates of BED (threshold or subthreshold) in Hispanic, non-Hispanic Black, and "other" ethnic groups relative to non-Hispanic Whites. Conversely, lifetime prevalence of AN was higher in non-Hispanic Whites than in the rest of the sample, at a trend level; lifetime prevalence of AN was 0.4% in non-Hispanic Whites compared to 0.2% in Hispanics, 0.1% in non-Hispanic Blacks, and 0% in other ethnic groups. The potential effects of sex on ethnic differences in ED prevalence were not examined.

Striegel-Moore et al. (2011) documented rates of ED behaviors and diagnoses in a nationally representative sample of American Indian/Native American, Native Hawaiian, and Alaska Native (referred to herein as AI/NA) young adults compared to non-Hispanic Whites. Consistent with findings from other epidemiologic reports, AI/NA women were significantly more likely than White women to meet study criteria for binge eating and report having ever been diagnosed with an ED. There were no significant differences between AI/NA men and White men in rates of ED psychopathology, but loss-of-control over eating was more prevalent in AI/NA men at a trend-level (2.4% in AI/NA men vs. 1.2% in White men, $p = .09$).

Finally, Chao et al. (2008) examined ethnic differences in weight control practices among adolescents using data from the Youth Behavior Surveillance System Survey conducted by the Centers for Disease Control and Prevention. Results showed significantly higher rates of dieting and use of diet products and exercise for weight control in White and Hispanic females compared to Black females. Rates of purging (i.e., self-induced vomiting or laxative misuse) also were higher in Hispanic females than in Black females. Finally, data in males showed a slightly different pattern, with Hispanic males more likely than White and Black males to report dieting and exercise for weight control; Hispanic males also were more likely than White males to endorse the use of diet products, purging, and exercise for weight control.

Large Community Samples Findings from recent studies in large community samples converge with nationally representative data in suggesting that ED risk is elevated across multiple ethnic groups. For example, in a diverse sample of 1,225 adults living in Los Angeles, CA (United States), Regan and Cachelin (2006) found that binge eating was equally common in Asian, Hispanic, Black/African American, and White men and women. Moreover, rates of self-induced vomiting and the use of laxatives, diuretics, and diet pills to control weight were higher in Black, White, and Hispanic women compared to Asian women. Of note, Black women were significantly more likely than White, Hispanic, and Asian women to report using laxatives, diuretics, and diet pills to control weight.

Similar results emerged in a study of 16,978 middle-school students in the state of Massachusetts (United States). Specifically, S. B. Austin et al. (2011) reported that the odds of engaging in "disordered weight control behaviors" (i.e., vomiting or the use of laxatives or diet pills to control weight in the past month) were 2–10 times higher in most "minority"

ethnic groups relative to Whites. These results echo findings from earlier community-based research, which documented equivalent or higher rates of ED behaviors in non-White compared to White adolescents (e.g., Croll, Neumark-Sztainer, Story, & Ireland, 2002; Neumark-Sztainer et al., 2002).

Although most large-scale community studies focusing on ethnic differences in ED prevalence have been conducted in the United States, two reports from other countries are noteworthy. In a sample of 895 South African high-school and college students, Le Grange, Louw, Russell, Nel, and Silkstone (2006) found similar rates of "possible binge eating or bulimia" (operationalized as a score ≥25 on the Bulimic Investigatory Test, Edinburgh [BITE]) in White, Black African, and multiracial individuals (5.2%, 5.6%, and 2.8%, respectively). However, rates of possible AN (operationalized as a score ≥20 on the EAT-26) were significantly higher in Whites compared to Black African and multiracial students (17.5%, 11.9%, and 8.7%, respectively). Finally, in a representative sample of 601 women from the Province of Alberta, Canada, Boisvert and Harrell (2009) reported that bulimic behavior (as measured by a single item on the EDI) was more common in Hispanic women compared to White, Aboriginal, and Asian women.

In sum, studies examining the relative prevalence of EDs and disordered eating symptoms across multiple ethnic groups, primarily in the United States, generally have failed to support the notion that Caucasian ethnicity is a risk factor for EDs. Furthermore, although a few reports have suggested that membership in specific ethnic groups might protect against the emergence of particular ED symptoms (e.g., Chao et al., 2008; Le Grange et al., 2006; Neumark-Sztainer et al., 2002), the preponderance of evidence indicates that ED risk is equal or higher in non-White individuals compared to Whites. Of note, epidemiologic data indicate that Hispanic, African American, and AI/NA ethnicity increases risk for binge eating and BN in females (Marques et al., 2011; Striegel-Moore et al., 2011; Swanson et al., 2011), which could have important implications for future research on the etiology and prevention of bulimic syndromes.

Longitudinal Research

A small number of longitudinal studies have examined the impact of ethnicity on the future emergence of ED symptoms. With notable exceptions (Leon, Fulkerson, Perry, & Early-Zald, 1995; The McKnight Investigators, 2003), this work does not support the idea that ethnicity is a risk factor for EDs (Jacobi et al., 2011; Nicholls & Viner, 2009; Stice, Presnell, & Spangler, 2002). However, longitudinal studies typically have conceptualized ethnicity as a dichotomous variable (i.e., White vs. non-White), and as data from epidemiologic and community samples illustrate, the relation between ethnicity and EDs is more complex.

Notably, findings from the one longitudinal study that utilized a more nuanced approach to classifying ethnicity (viz., White, Black, Hispanic, and "Other") suggest that Hispanic ethnicity might be a risk factor for the development of bulimic EDs, specifically, full or partial-syndrome BN and BED (The McKnight Investigators, 2003). It is important to acknowledge that this effect was observed only at one site (Arizona, United States) in a two-site (Arizona and California) trial. Nevertheless, among 590 girls initially assessed in grades 6–9 and followed prospectively for 4 years, Hispanic ethnicity emerged as a significant predictor of bulimic ED onset. Hispanic ethnicity also moderated another ED risk factor in the Arizona sample, such that the effect of thin body preoccupation and social pressure on the development of bulimic EDs was stronger in Hispanic girls than in non-Hispanics.

Ethnic Differences in the Salience of ED Risk Factors

In addition to examining direct effects of ethnicity on the development of EDs, a large body of work has sought to determine whether ethnic differences exist in the salience of established ED risk factors. The idea that membership in a non-majority ethnic group might be a "protective factor" against sociocultural influences on eating pathology has garnered particular attention (Warren, Gleaves, Cepeda-Benito, del Carmen Fernandez, & Rodriguez-Ruiz, 2005, p. 242). However, research findings have been mixed.

In a sample of 300 women enrolled at large universities in the United States and Spain, Warren et al. (2005) found that awareness and internalization of the thin ideal were significantly higher in European American women than in Mexican American women. Moreover, relationships among thin-ideal awareness, thin-ideal internalization, and body dissatisfaction were significantly stronger in European American women compared to Mexican American and Spanish women (who did not differ on these analyses). Warren et al. concluded from these findings that ethnicity may serve as a moderator of the effects of thin-ideal awareness and internalization on the expression of ED symptoms.

In contrast, Shaw, Ramirez, Trost, Randall, and Stice (2004) found no evidence that ethnicity (categorized as Asian, Black, Hispanic, or White) moderated the relations between ED risk factors (including perceived pressure to be thin, thin-ideal internalization, and body dissatisfaction) and ED symptoms in a sample of 745 American females aged 11–26 years. Furthermore, of the seven ED risk factors examined, only one was characterized by ethnic differences. Specifically, internalization of the thin ideal was higher in Asian and White females than in Black or Hispanic females.

Other research has found elevated levels of thin-ideal internalization and body dissatisfaction in members of minority ethnic groups compared to Caucasian individuals. For example, in a large sample ($n = 1{,}212$) of children aged 11.2 ± 1.0 years living in the United States, Xanthopoulos et al. (2011) showed that body dissatisfaction was significantly higher in Asians compared to Caucasian, African American, Hispanic, and "other" ethnic groups; moreover, these effects remained after controlling for sex and relative weight status. Similarly, a study by Hermes and Keel (2003) found that "non-Caucasian" girls in the United States had higher thin-ideal internalization than their Caucasian peers despite no between-group differences in awareness of the thin ideal. Finally, in a cross-cultural study of body dissatisfaction, Mellor et al. (2013) found higher levels of overall body dissatisfaction in Malaysian Chinese adolescents relative to Malaysian Malays, Chinese, and Australia adolescents.

Studies examining the relative "fit" of sociocultural models of ED risk in members of non-White ethnic groups relative to Whites also have produced mixed findings, with some reports indicating that these models apply equally well in Whites and non-Whites (e.g., Mitchell & Mazzeo, 2009; Nouri, Hill, & Orrell-Valente, 2011), and others suggesting a better fit in Whites (e.g., Fitzsimmons-Craft & Bardone-Cone, 2012). Notably, two studies found strong support for the sociocultural model of disordered eating among Hispanic girls living in Mexico and Guatemala, respectively (J. L. Austin & Smith, 2008; Vander Wal, Gibbons, & del Pilar Grazioso, 2008). Nevertheless, most studies examining ethnic differences in ED risk factors have utilized cross-sectional designs (for a notable exception, see Fitzsimmons-Craft & Bardone-Cone, 2012); thus, between-group differences in the influence of sociocultural risk factors on the expression of ED symptoms cannot be established. In addition, the assessment instruments used and the models tested have varied across studies, which could explain differences in findings. Finally, the salience of sociocultural models of disordered eating

may differ between ethnic minority groups (e.g., African American vs. Asian), as well as within members of the same group.

Within-Group Differences in the Relation between Ethnicity and EDs

A potential limitation of studies comparing ethnic groups on ED prevalence and risk is that "race" and "ethnicity" are treated as *the* key independent variables of interest. Yet, there is growing evidence for within-group variability in ED risk among members of ethnic groups. For example, cross-cultural studies have indicated that ethnic differences in body dissatisfaction can be accounted for, at least in part, by differences in socioeconomic status, religious influences, ideal body size, rural versus urban setting, media exposure, and messages from parents and peers (McCabe et al., 2011; Swami et al., 2010). In the following paragraphs, we focus on two constructs, *ethnic identity* and *acculturation*, that have been hypothesized to moderate ED risk in ethnic minority persons (Phinney, 1996).

Ethnic identity refers to feelings of belonging, and ethnic cultural values, social practices, language (when applicable), and attitudes that are components of one's socially constructed self-identity. This definition suggests that all individuals, including Caucasian persons, have an ethnic identity. *Acculturation* is the process of social and psychological change that occurs as a result of interacting with individuals and groups from another culture. Berry (1990) has described four outcomes that result from the process of acculturation:

1 *Assimilation*—"letting go" of one's original cultural identity, and incorporating an identity that is consistent with another culture.
2 *Integration*—simultaneously keeping one's original cultural identity, while incorporating another cultural identity.
3 *Separation*—maintaining original cultural identity, and not incorporating another cultural identity.
4 *Marginalization*—no cultural or psychological connection to original culture or another culture.

Ethnic identity and acculturation are not identical constructs; however, both concepts play an important role in sociocultural models of EDs, which posit that increased "Westernization" and assimilation are risk factors for EDs, whereas traditional ethnic identity is protective from the development of eating pathology (Stice, 1994). The sociocultural hypothesis is based on the idea that Western cultures place greater value on a thin body image ideal (see Chapters 21 & 29), which may lead to body dissatisfaction, weight/shape concerns, and disordered eating behaviors. Thus, if an individual has low ethnic identity and high assimilation, they would be thought to be at increased risk for eating pathology.

An alternative hypothesis, called *culture clash,* postulates that the pressure to assimilate increases ED risk due to stress resulting from the acculturation process (Katzman & Lee, 1997; Lake, Staiger, & Glowinski, 2000; Mumford, Whitehouse, & Platts, 1991). Similar hypotheses suggest that individuals with a "marginalized" outcome should demonstrate the highest levels of eating pathology, due to conflicts and other challenges experienced in navigating the process of acculturation (Katzman & Lee, 1997; Perlick & Silverstein, 1994; Steiner-Adair, 1991).

No clear findings resolving these hypotheses have emerged. Some studies have found that internalization of Western beauty ideals, low ethnic identity, and high acculturation are directly

related to increases in eating pathology among ethnic minority persons (Stojek, Fischer, & Collins, 2010; Wood & Petrie, 2010). For example, in the longitudinal study of ED risk factors described above (The McKnight Investigators, 2003), the authors speculated that differences between the Arizona and California sites with respect to the influence of Hispanic ethnicity on ED onset might have been due, in part, to differences in ethnic identify. Specifically, Hispanic girls living in California (who were not at increased risk for ED onset) had higher rates of non-English-speaking households and were more likely to identify themselves as being Latin American than were Hispanic girls living in Arizona (who were at increased risk for bulimic EDs). Likewise, a study conducted in Japan found that adolescents with "pro-modern" and "pro-Western" ideals were more dissatisfied with their bodies than "anti-modern" and "traditional" adolescents; pro-Western adolescents also were more likely to be attempting weight loss than were the other acculturative groups (Brokhoff et al., 2012; see also Chapter 6).

However, numerous other reports have found that high ethnic identity, low acculturation, and traditional ethnic values are associated with *increased* risk for eating pathology both in the United States (Tsai, Curbow, & Heinberg, 2003) and in other countries (e.g., Australia, Korea; Humphry & Ricciardelli, 2004; Jackson, Keel, & Lee, 2006; Jennings, Forbes, McDermott, & Hulse, 2006; Lake et al., 2000). In addition, several studies have found no association between acculturation and disordered eating (Barry & Garner, 2001; Haudek, Rorty, & Henker, 1999; Kuba & Harris, 2001; Shuttlesworth & Zotter, 2011) or no association between ethnic identity and disordered eating (Barry & Garner, 2001; Baugh, Mullis, Mullis, Hicks, & Peterson, 2010; Bisaga et al., 2005; Jennings et al., 2006; Shuttlesworth & Zotter, 2011).

Finally, some studies have reported *indirect* relations between acculturation, ethnic identity, and disordered eating. For example, Henrickson et al. (2010) found that the relation between ethnic identity and disordered eating was moderated by expectancies regarding the role that thinness might play in affect management and life improvement. Specifically, individuals with low ethnic identity and high positive expectancies regarding thinness were at increased risk for development of maladaptive eating behaviors. Bettendorf and Fischer (2009) reported that for individuals low in "familialism," which represents a "deeply ingrained sense of the individual being inextricably rooted in the family" (p. 431), stronger levels of acculturation were associated with increased rates of disordered eating. However, at high levels of familialism, these effects were null or reversed. The results of these moderation studies may be useful for future research testing the sociocultural and culture-clash hypotheses, as these findings may help to explain inconsistent relationships between acculturation, ethnic identity, and eating pathology.

Conclusions and Future Directions

The interaction among ethnicity, culture, and ethnic identity is important for many mental disorders. However, these concepts may be of even greater importance to the ED field, given the role that social constructs, such as body image, play in the etiology and maintenance of EDs. Several directions for future research will be important for better characterizing these relations, and we summarize these considerations below.

First, many studies examining ethnicity as a risk factor for EDs are hampered by extremely small sample sizes that are underpowered for statistical tests. This is particularly problematic when researchers test hypotheses in adequately powered Caucasian samples and underpowered ethnic minority samples within the same study. As with any statistical analysis, inadequate power may lead to biased parameter estimates, or null or inconsistent findings.

Second, given the potential for cohort effects to impact relations between ethnicity and ED risk (particularly for cultures in which there has been recent social change or novel cultural influences), more studies examining the longitudinal course of disordered eating in ethnic minorities are needed (see Chapter 55). Third, researchers should consider controlling for the effects of potential confounding variables that might influence associations between ethnicity and ED risk. These include: (a) *setting effects* (e.g., rural, suburban, or urban residence), which may represent an additional source of within-group variability among ethnic minorities, and (b) *ethnic differences in body mass index* (BMI), which are a concern because the psychometric properties of certain self-report measures of binge eating and restraint are poor in overweight and obese individuals (Bohrer, Forbush, & Hunt, 2013), and ethnic differences exist for BMI.

Fourth, research would benefit from a continued balance between examining ethnicity as an independent variable and better characterizing within-group relations among ethnic identity, acculturation, and disordered eating. Related to this point, although ethnicity currently is regarded as a *fixed* risk factor for EDs (Jacobi et al., 2004), it may be more appropriate to conceptualize ethnicity as a multifaceted construct comprised of fixed *and* variable components. The fixed aspects of ethnicity, such as genetic background, likely are familiar to most professionals in the EDs field. However, variable aspects of ethnicity, such as differences in religious beliefs, gender roles, the experience of discrimination and harassment, and the meaning of food and eating, may be at least as important in explaining ethnic differences in disordered eating. Moreover, identifying potential moderators and mediators of the relation between ethnicity and EDs would have important implications for prevention and treatment.

Fifth, few studies have examined the interaction of ethnicity, sex, and ED risk, despite the fact that minority males do experience disordered eating (Marques et al., 2011). Finally, improved assessment using valid measures of disordered eating is crucial, given that differential validity may bias research findings and lead to underestimates of correlations between ED variables and other substantive psychological constructs.

Theory and research on the role of ethnicity as a risk factor for EDs have advanced significantly since the days when EDs were thought to be rare in non-White individuals. We now know that EDs and ED symptoms are present in a broad range of U.S. ethnic groups, and emerging data indicate that ED risk factors such as body dissatisfaction and internalization of the thin ideal are present in non-Western contexts. However, the specific mechanisms that contribute to the emergence of ED symptoms remain elusive. Through future research examining the role of ethnicity in EDs, the field has an opportunity to gain new insights that can be used to improve early identification of individuals suffering from eating pathology, and develop culturally informed prevention programs and treatments for individuals who experience disordered eating.

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26

Familial Risk Factors and Eating Disorders

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Theory and research have focused on the role of familial factors in the development and maintenance of eating disorders (EDs). Early theoretical conceptualizations focused on dysfunctional family relationships characterized by enmeshment, overprotectiveness, and rigidity (Minuchin, Rosman, & Baker, 1978) and dysfunctional familial environments characterized by the presence of conflict, lack of cohesion, and disorganization (Kog & Vandereycken, 1989). More recently, theory has focused on the family as one source of influence through which socioculturally defined body ideals and the ways in which these ideals are achieved may be transmitted (Stice, 2001; Thompson, Heinberg, Altabe, & Tantleff-Dunn, 1999; see also Chapter 21). The dual-pathway model (Stice, 2001) proposes that pressure to be thin from family, peers, and the media contributes to body dissatisfaction, a major risk factor for EDs. The Tripartite Influence Model (Thompson et al., 1999; see also Chapter 21) posits that messages about body shape and weight from parents, peers, and the media are influential in the development of body image and eating disturbance. Such theoretical work has generated multiple lines of empirical research on familial risk factors for EDs.

To provide context for this review, we draw upon a series of case-control studies that examined potential risk factors for anorexia nervosa (AN; Fairburn, Cooper. Doll, & Welch, 1999; Pike et al., 2008), bulimia nervosa (BN; Fairburn, Welch, Doll, Davies, & O'Connor, 1997), and binge eating disorder (BED; Fairburn et al., 1998; Striegel-Moore et al., 2005). A significant strength of these studies is that each utilized a psychiatric control group, thus enabling the identification of risk factors that distinguished between psychiatric disorders in general and EDs more specifically. Compared to psychiatric controls, individuals with a current diagnosis of AN had greater exposure to problems in the parenting domain, including familial discord and parental demands, and more frequent exposure to critical comments regarding shape, weight, and eating (Pike et al., 2008). Individuals with a history of AN also reported greater exposure to other problems in the parenting domain, particularly with respect to low parental contact, high parental expectations, and underinvolvement, as well as more frequent exposure to familial dieting and critical comments regarding shape, weight, and eating (Fairburn et al., 1999). Similarly, compared to psychiatric controls, individuals with BN had greater exposure to parental problems, particularly low parental contact and high parental

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expectations; they also had greater exposure to a history of parental obesity, the presence of dieting among family members, and critical comments by family about shape, weight, and eating (Fairburn et al., 1997). Finally, compared to psychiatric controls, individuals with BED had greater exposure to low parental contact; they also had greater exposure to the dieting vulnerability subdomains, including critical comments by family about shape, weight, and eating (Fairburn et al., 1998). Striegel-Moore et al. (2005) replicated some of these findings among samples of Caucasian and African-American women with BED, noting greater exposure to familial overeating, familial discord (which included low parental contact), and parental demands (which included parental criticism). In contrast, one study failed to find differences in general familial functioning between patients with AN and BN and patients with obsessive-compulsive disorder (OCD; Erol, Yazici, & Toprak, 2007). Given these findings, this chapter will provide a review of the literature in three familial domains: general familial functioning, including dysfunctional familial characteristics and parental relationships; parental modeling of overconcern with weight, dieting, and maladaptive eating habits; and negative familial communications regarding shape and weight and encouragement or pressure to diet.

Familial Dysfunction

General Family Functioning

Studies have found that families of individuals with EDs commonly evidence familial dysfunction (Di Paola, Faravelli, & Ricca, 2010; Humphrey, 1988; Kog & Vandereycken, 1989; McNamara & Loveman, 1990; Steiger, Liquornik, Chapman, & Hussain, 1991; Stern et al., 1989). These families may exhibit conflict, a lack of cohesion, poor communication and problem-solving skills, poor behavioral control, higher levels of expressed emotion (i.e., criticism, hostility, and/or emotional overinvolvement by family members), and low levels of affective responsiveness and expressiveness (the latter defined as the extent to which individuals are encouraged to openly and directly express feelings). Numerous studies have found that individuals with BN perceive more conflict, less cohesion, less expressiveness, and higher achievement orientation within their families compared to controls (Bonne et al., 2003; Johnson & Flach, 1985; Stern et al., 1989). Similarly, individuals with AN tend to perceive their families as lacking cohesion (Cunha, Relvas, & Soares, 2009). While there is some evidence that suggests families of individuals with AN are more avoidant of conflict compared to controls (Latzer & Gaber, 1998), other evidence has not supported this finding (Lattimore, Wagner, & Gowers, 2000). Low levels of expressiveness may be a particularly salient feature of families of individuals with EDs (Stern et al., 1989). Among adolescent girls without a specific ED diagnosis, high compulsive eaters had lower levels of family cohesion, organization, and expressiveness than low compulsive eaters on the basis of their mothers' self-report (Attie & Brooks-Gunn, 1989).

Research examining differences in family functioning between ED subtypes has yielded discrepant findings. For example, Kog and Vandereycken (1989) found that families of individuals with AN reported more cohesion compared to BN families, and individuals with BN perceived more disorganization compared to those with AN. In contrast, other studies examining family functioning among individuals with AN and BN reported nonsignificant findings (Benninghoven, Tetsch, Kunzendorf, & Jantschek, 2007; Stern et al., 1989). In a study of 74 mother, father, and adolescent daughter triads, Humphrey (1989) found that parents of individuals with AN communicated a "double message" of affection and neglect, whereas

individuals with BN and their mothers evidenced hostile enmeshment (e.g., mutual belittling, blaming, sulking, and appeasing). Di Paola et al. (2010) found that perceived expressed emotion was higher among individuals with AN, BN, and BED compared to controls, but there were no differences in expressed emotion among the ED subgroups. In a study of AN subtypes, Casper and Troiani (2001) assessed family functioning among adolescents with restricting or bulimic type AN (AN-R and AN-BP, respectively). Findings indicated that adolescents with AN-BP reported perceiving their families as more impaired, having more disturbances in affective expression, involvement, and control compared to controls, whereas those with AN-R did not differ from controls. Although there is some evidence that suggests normal-weight individuals with BN and AN-BP report more family nurturance problems compared to individuals with AN-R (Humphrey, 1986, 1989), other research (Wonderlich & Swift, 1990) has failed to replicate such differences. Furthermore, in a cluster analysis of aspects of family functioning among different ED subtypes, results indicated there were seven clusters of families, though the ED subtypes were distributed throughout the clusters (Kog, Vandereycken, & Vertommen, 1989). Taken together, there is little conclusive evidence to suggest that specific ED subtypes are associated with specific aspects of family functioning.

Parent-Child Relationships

In addition to general family functioning, the possible influence of parent-child relationships in EDs has been examined. Studies using the Parental Bonding Instrument (Parker, Tupling, & Brown, 1979), which assesses the perceived quality of parental relationships (Vincent & McCabe, 2000), have generally found that samples of individuals with AN, BN, and mixed ED diagnoses perceive less parental care and more parental overprotection compared to non-ED controls (Bulik, Sullivan, Fear, & Pickering, 2000; Fassino, Amianto, Rocca, & Daga, 2010; Panfilis, Rabbaglio, Rossi, Zita, & Maggini, 2003). However, neither parental care nor parental overprotection emerged as a significant predictor of bulimic tendencies in adolescent girls and boys (Vincent & McCabe, 2000).

A substantial body of literature has investigated the relationship between attachment processes and disordered eating. Attachment refers to a child's bond with her or his parents and the extent to which parents provide a secure base and encourage exploration from it (Bowlby, 1969, 1973, 1980). Attachment styles (Ainsworth, Blehar, Waters, & Wall, 1978), which can be broadly categorized as "secure" or "insecure," are determined by interactions between infants and their caregivers. Bowlby suggested attachment styles determine internal working models of predictions about oneself and others' responses to one's needs. Therefore, an individual's style of attachment is believed to provide a framework for developing later interpersonal relationships and emotion regulation processes, both of which have been implicated in the development and maintenance of EDs (Fairburn, Cooper, & Shafran, 2003; see also Chapters 22, 57, & 62). It should be noted that, although attachment styles theoretically are developed early in life, attachment measures have been developed for use among adult populations (O'Shaughnessy & Dallos, 2009).

To date, reviews of the evidence regarding the possible impact of attachment in EDs (O'Shaughnessy & Dallos, 2009; Ward, Ramsay, & Treasure, 2000) indicate there is a strong association between EDs and insecure attachment styles, with some research suggesting the prevalence of insecure attachment among ED samples to be as high as 100% (Ringer & Crittenden, 2007; Zachrisson & Kulbotten, 2006). However, the evidence regarding the relationship between particular attachment styles and ED subgroups is inconclusive

(O'Shaughnessy & Dallos, 2009; Ward et al., 2000), in part because many studies have used samples consisting of heterogeneous ED diagnoses (Abbate-Daga, Gramaglia, Amianto, Marzola, & Fassino, 2010; Barone & Guiducci, 2009; Demidenko, Tasca, Kennedy, & Bissada, 2010) and studies vary in the use of terminology to describe attachment styles and the assessment of attachment constructs. Though some research has suggested AN is associated with an "avoidant/dismissing" style (Candelori & Ciocca, 1998; Ward et al., 2001) and BN is associated with a preoccupied/entangled style (Candelori & Ciocca, 1998), other research has not found differences in attachment styles between ED subgroups (Tereno, Soares, Martins, Celani, & Sampaio, 2008). Rather, it may be that attachment style is related to symptom severity rather than the specific ED diagnosis (Broberg, Hjalmers, & Nevonen, 2001; Troisi, Massaroni, & Cuzzolaro, 2005; Zachrisson & Kulbotten, 2006).

Generally, research suggests that individuals with EDs either perceive or experience greater familial dysfunction and parental relationships of poorer quality than controls. However, given that this literature primarily is cross-sectional in nature and few studies utilize non-eating-disordered psychiatric samples as controls, it seems likely that these constructs are risk factors for psychological problems in general. Nevertheless, this early literature laid the foundation for the exploration of other familial constructs that may be more closely related to the development of EDs. Given that case-control studies have identified familial overeating and dieting and familial criticism regarding weight, shape, and eating as potential risk factors for EDs, these familial variables may warrant additional consideration.

Modeling and Negative Communication

Researchers have focused on two mechanisms through which family members, particularly parents, transmit sociocultural messages regarding the nature and importance of the socioculturally defined body ideal and the methods that may be used to achieve it. The first mechanism is parental modeling of attitudes toward weight and shape and specific dieting and eating behaviors. Modeling has been conceptualized as having an indirect influence (Abraczinskas, Fisak, & Barnes, 2012), since the attitudes and behaviors modeled by parents have to be observed and adopted by their children. The second mechanism is familial communication regarding their child's weight, shape, and eating behaviors, which involves verbal messages in the form of negative statements regarding the child's appearance, criticism regarding a child's weight and shape, teasing, and encouragement to diet in order to lose or control weight (Kichler & Crowther, 2001; Rodgers & Chabrol, 2009; Thompson et al., 1999). Familial communication has been conceptualized as an "active" or direct influence (Abraczinskas et al., 2012; Rodgers & Chabrol, 2009), because such communications generally are directed toward the child or occur in his/her presence. For this chapter, we choose to focus on those studies that examined associations between modeling and familial communication and disordered eating; thus, studies that solely examined body image disturbance or dieting as dependent variables are not included here (see Rodgers & Chabrol, 2009, for a review).

Modeling

Research examining the associations between modeling and disordered eating has yielded mixed results. Among studies that examined parental dieting, research has found positive associations between maternal dieting and unhealthy weight control behaviors among girls and

boys (Keery, Eisenberg, Boutelle, Neumark-Sztainer, & Story, 2006; Neumark-Sztainer et al., 2010); maternal dieting and bulimic symptomatology among girls but not boys (Rodgers, Faure, & Chabrol, 2009a); and maternal dieting and bulimic symptomatology in girls who have had their first menstrual cycle (Wertheim, Martin, Prior, Sanson, & Smart, 2002). Interestingly, although girls and boys are equally likely to have parents who engaged in dieting (Wertheim et al., 2002), girls are more likely to report maternal modeling of weight loss than boys (Vincent & McCabe, 2000). However, these and other studies also have found nonsignificant associations between maternal dieting and maladaptive eating attitudes and behaviors among college-aged women (Kichler & Crowther, 2001) and paternal dieting and unhealthy weight control behaviors in girls (Neumark-Sztainer et al., 2010) and bulimic symptomatology in adolescent girls and boys (Rodgers et al., 2009a; Wertheim et al., 2002).

With respect to parental variables other than dieting, Yanez, Peix, Atserias, Arnau, and Brug (2007) found that girls whose mothers exhibited maladaptive eating attitudes and behaviors were nearly three times more likely to have abnormal eating attitudes. Cooley, Toray, Wang, and Valdez (2008) found that mothers' thin-ideal internalization and eating symptomatology were positively associated with their college-aged daughters' eating symptomatology. While Baker, Whisman, and Brownell (2000) found nonsignificant associations among the scores of parents and those of their children on the Eating Attitudes Test (EAT) and nonsignificant associations among perceived maternal and paternal modeling and daughters' EAT scores among females, they found significant associations between perceived paternal modeling and sons' EAT scores. Among those studies that compare females who already exhibit disordered eating to those who do not, Pike and Rodin (1991) found that mothers of daughters with disordered eating had a longer dieting history and higher levels of ED symptomatology than mothers of women without disordered eating, while Kanakis and Thelen (1995) found nonsignificant differences on mothers' and fathers' dieting and weight history, body importance, and eating behaviors. Canals, Sancho, and Arija (2009) found that at follow-up, the mothers' drive for thinness was associated with increased risk for an ED.

Family Communication

Negative Comments In contrast to the literature on modeling, findings regarding the association between familial communication and disordered eating are more consistent. It is important to note that negative familial communication appears to be quite common. For example, in a sample of young adults, 35.8% of females and 22.0% of males reported receiving hurtful weight-related comments from family, while 64.3% of females and 58.3% of males reported weight-related teasing from family members or peers during earlier phases of the study (Eisenberg, Berge, Fulkerson, & Neumark-Sztainer, 2012). In a younger sample of middle-school girls, 23% reported parental teasing about their appearance and 29% reported appearance teasing from a sibling (Keery, Boutelle, van den Berg, & Thompson, 2005). In a female high-school sample, 45.2% of daughters reported encouragement to diet from their mothers, and 36.4% reported encouragement from their fathers (Neumark-Sztainer et al., 2010).

Regardless of whether familial communication is defined as negative or critical comments regarding weight and shape, teasing, or encouragement to diet or lose weight, research indicates positive associations between negative familial communication and maladaptive eating attitudes and disordered eating (Annus, Smith, Fischer, Hendricks, & Williams, 2007;

Ata, Ludden, & Lally, 2007; Baker et al., 2000; Keery et al., 2005; Levine, Smolak, & Hayden, 1994; Neumark-Sztainer et al., 2010; Unikel, Von Halle, Bulik, & Ocampo, 2012; Vincent & McCabe, 2000; Wade & Lowes, 2002), even after adjusting for previous disordered eating (Eisenberg et al., 2012). To our knowledge, only one study that examined the effects of child gender and maternal and paternal encouragement to diet on bulimic symptomatology reported nonsignificant findings (Wertheim et al., 2002); however, for girls, regardless of whether or not they had had their first menstrual cycle, greater encouragement to diet from mothers and fathers was associated with higher bulimic symptomatology; for girls who had had their first menstrual cycle, these relationships remained significant even when controlling for child size.

One issue raised by this research involves the relative contribution of modeling and familial communication to disordered eating. When only the bivariate relationships are considered, the effect sizes for the association between modeling and disordered eating range from small to medium, and between familial communication and disordered eating range from small to large (for studies with data permitting conclusions regarding effect size). However, several studies have included both modeling and familial communication in their analytic models rather than considering these variables in isolation, thus allowing examination of the relative contribution of these variables. For example, Cooley et al. (2008) found that mother's thin-ideal internalization ($\beta = .25$), daughters' perceptions of their mothers' weight concerns ($\beta = .30$), and mothers' negative comments about daughters' weight ($\beta = .29$) all emerged as significant predictors of daughters' eating symptomatology. Baker et al. (2000) found that perceived criticism regarding eating and appearance from mothers was more strongly associated with scores on the EAT than perceived modeling by mothers for undergraduate men and women; comparable analyses examining perceived criticism and perceived modeling by fathers were nonsignificant. Similarly, although Rodgers et al. (2009a) found that maternal modeling emerged as a significant predictor of drive for thinness in adolescent girls and that paternal modeling was a significant predictor of body dissatisfaction in adolescent boys, familial communication variables emerged as important to bulimic symptomatology. Specifically, although a wide range of parental variables accounted for 22% and 4% of the variance in bulimic symptomatology for girls and boys, respectively, negative maternal and paternal comments and maternal importance and comparison comments emerged as significant predictors of bulimic symptomatology for girls, while paternal importance and comparison comments were significant predictors of bulimic symptomatology for adolescent boys. Vincent and McCabe (2000) also conducted a series of multiple regression models that included BMI, peer, and parental variables including maternal and paternal encouragement, modeling, negative commentary, and discussion about weight loss, as predictors of bulimic tendencies. Among the parental variables, maternal encouragement to lose weight and maternal and paternal negative commentary were significant predictors of bulimic tendencies for adolescent girls; no parental variables emerged as significant predictors of bulimic tendencies for boys.

In contrast, to our knowledge, only one study found greater support for modeling. In their study of 89 mother-daughter pairs, Benedikt, Wertheim, and Love (1998) found that maternal body dissatisfaction and extreme weight loss behaviors, but not maternal encouragement to lose weight, emerged as significant predictors of daughters' extreme weight loss behaviors. Given the relative strength of previous findings regarding negative commentary and teasing (e.g., Neumark-Sztainer et al., 2010; Rodgers et al., 2009a; Vincent & McCabe, 2000), it is possible that these forms of familial communication have more potent effects than encouragement to lose weight.

Positive Comments As this literature indicates, there has been considerable emphasis on negative familial communication, yet it seems possible that positive comments from family members might serve to protect their child from disordered eating (Gross & Nelson, 2000; Kichler & Crowther, 2009). In their research on elementary-school-aged children, Kichler and Crowther (2009) found that daughters reported that their mothers and fathers provided significantly greater positive communication than negative communication. In their adolescent sample, Rodgers et al. (2009a) found that girls reported receiving significantly more positive comments from their mothers than did boys. However, research on the associations between positive comments and disordered eating has yielded mixed findings. Gross and Nelson (2000) reported that positive maternal messages were associated with less disordered eating, Rodgers et al. (2009a) reported that positive maternal comments were associated with greater bulimic symptomatology among sons but not daughters, and others found a nonsignificant relationship between positive communication and maladaptive eating attitudes and behaviors (Kichler & Crowther, 2009) and bulimic symptomatology (Rodgers, Paxton, & Chabrol, 2009b). Clearly, additional research is needed to clarify the nature of positive maternal and paternal comments that may serve a protective function, including how they are perceived and interpreted by a child.

Other Family Influences Two additional variables that have received considerable attention in the literature are familial pressure and familial influence. Familial pressure has been defined in several ways, including the child's perception of the importance of his/her weight to parents (Rodgers et al., 2009a) and of pressure to be thin or lose weight (Shisslak et al., 1998; van den Berg, Thompson, Obremski-Brandon, & Coovert, 2002). Given these definitions, the extent to which familial pressure involves modeling and negative communication is unclear. However, research indicates positive associations between perceived maternal pressure and disordered eating, including weight control behaviors (Shisslak et al., 1998) and bulimic symptoms (Peterson, Paulson, & Williams, 2007; Rodgers et al., 2009a) in girls. For the two studies that included male participants, maternal pressure was positively associated with bulimic symptoms in one (Peterson et al., 2007), while neither maternal nor paternal pressure were significantly associated with bulimic symptoms in the other (Rodgers et al., 2009a). Moreover, daughters who met criteria for BN and subclinical BN reported more pressure from their mothers, but not their fathers, to diet, restrict their food, and exercise than daughters without an ED (Kanakis & Thelen, 1995).

Familial or parental influence often is a composite variable that may incorporate elements of modeling and/or familial communication, as well as constructs such as pressure to be thin or lose weight, parental investment in a child's appearance or thinness, and familial concern or preoccupation with weight and dieting (Abraczinskas et al., 2012; Keery, van den Berg, & Thompson, 2004; The McKnight Investigators, 2003; van den Berg et al., 2002; Young, Clopton, & Bleckley, 2004). Although one study indicated that parental influence was not a risk factor for the development of an ED (The McKnight Investigators, 2003), other research has found that familial or parental influence is significantly associated with bulimic symptomatology (Abraczinskas et al., 2012; Keery et al., 2004; Shroff & Thompson, 2006), even when other variables such as peer influence, body dissatisfaction, depression, and parental expectations are included in the model (Young et al., 2004). Among studies examining the Tripartite Influence Model (see Chapter 21), familial or parental influence has been conceptualized as a distal variable that impacts body dissatisfaction and, subsequently, bulimic symptomatology through internalization of the thin ideal and appearance comparison with others (Keery et al., 2004; van den Berg et al., 2002).

Integrating the Familial Environment with Direct and Indirect Influences

Just as research that examined both modeling and familial communication facilitates our understanding of the relative importance of these mechanisms to the development of disordered eating, research that examines these mechanisms in the context of specific familial environments also facilitates our understanding of the relative contribution of each to the development of disordered eating. It has been argued that dysfunctional familial environments may create a vulnerability to psychopathology but that specific family-related experiences related to body image, food, and eating may be important to the development of disordered eating (Crowther, Kichler, Sherwood, & Kuhnert, 2002; Fairburn et al., 1997; Kluck, 2008; Laliberte, Boland, & Leichner, 1999; see also Chapter 67). Additionally, it is possible that specific characteristics of the familial environment increase the likelihood that negative food-related experiences occur. We would argue that these family-related experiences may encompass modeling, familial communication, familial pressure, and familial influence. To further explore these propositions, we would like to briefly describe four studies.

Laliberte and colleagues (1999) proposed that "family climate for eating disorders variables" (p. 1021) would be stronger predictors of eating disturbance than "more general family process variables." Using principal components analysis, they identified two factors: a Family Dysfunction factor, which measured traditional constructs such as family conflict, cohesion, and expressiveness, as well as constructs derived from measures they created such as family esteem, family impulsivity, and family depression; and a Family Appearance/Achievement factor, which measured family appearance orientation, family body dissatisfaction, and family achievement emphasis. Using a sample of undergraduate women and their mothers, they found that both factors emerged as significant predictors of eating disturbance; however, the Family Appearance/Achievement factor explained 19% of the variance while the Family Dysfunction factor explained only 4% of the variance. Drawing on clinical samples of patients with EDs and depression, planned comparisons indicated that compared to controls, the two clinical groups exhibited significantly greater family dysfunction but did not differ significantly from each other. However, the patients with EDs differed from the remaining two groups on the Family Appearance/Achievement factor. While Laliberte et al. (1999) note that one explanation for this finding is that having an ED may distort perceptions of one's family, they also suggest that a "family climate for eating disorder" (p. 1021) may increase risk for an ED in an individual whose familial environment may increase their vulnerability to psychopathology.

Crowther and colleagues (2002) examined three domains of familial variables, including general family dysfunction, family eating attitudes and behaviors, and negative familial communication, as predictors of bulimic symptomatology. Using hierarchical multiple regression analysis, they found that familial conflict, external control of food intake, rules related to family mealtime, and negative familial communication were significant predictors of bulimic symptomatology, explaining 3.0, 2.0, 3.0, and 10.3% of the variance, respectively. Although, as they argued, "the presence of familial conflict may provide the foundation for the appearance of other family mealtime and communication behaviors that increase risk for bulimic symptomatology" (p. 148), these findings suggest that negative familial communication is relatively more potent than the other familial influences. Similarly, Vincent and McCabe (2000) concluded that it is processes such as parental modeling, negative commentary about their child's body, discussion about weight loss, and encouragement to lose weight rather than the quality of the familial environment that impact disordered eating.

Finally, in a comprehensive investigation of multiple familial domains, Kluck (2008) examined whether family food-related experiences mediated the relationship between familial dysfunction and disordered eating. Familial dysfunction was a latent variable comprised of general family dynamics (adaptability and cohesion) and maternal and paternal dynamics (maternal and paternal caring, control, and communication). Family food-related experiences, also a latent variable, were comprised of maternal modeling, parental negative commentary, and familial focus on appearance and eating. Structural equation modeling yielded an adequate model fit. Kluck (2008) concluded that, since family food-related experiences mediated the relationship between family dysfunction and disordered eating, it is these variables (e.g., modeling, teasing, and criticism) that increased risk for disordered eating.

Conclusions and Future Directions

In summary, research suggests that familial variables, including family dysfunction, familial modeling, and familial communication, are important to our understanding of the development of disordered eating. However, to put the contribution of familial variables in perspective, existing research suggests that familial variables generally explain a relatively small percentage of the variance in eating psychopathology. For example, Neumark-Sztainer et al. (2010) found that after entering sociodemographic variables, the variables of parental dieting behaviors, family weight talk, and family weight-related teasing explained an additional 15% of the variance in binge eating and 10% of the variance in extreme weight control behaviors. Rodgers et al. (2009a) found that parental variables explained 22% of the variance in bulimic symptomatology in girls, but only 4% of the variance in boys. These studies suggest that large percentages of the variance may be explained by variables other than the familial variables examined in these studies, thus supporting the examination of risk factors drawn from multiple domains (e.g., genetic factors, characteristics of the individual as well as familial and peer influences; see Chapters 28, 31, & 32).

Future research may want to consider how familial variables interact with other variables in the development of EDs and the ways in which familial variables have their influence. For example, Kichler and Crowther (2001) found that at high levels, but not low levels, of negative familial communication, there was a significant relationship between maternal modeling and maladaptive eating attitudes and behaviors. Moreover, in a cross-sectional study, Annus et al. (2007) found that women's expectancies regarding the benefits of thinness and dieting mediated the relationships between maternal modeling and disordered eating behavior and familial teasing and disordered eating behavior.

Given the relatively consistent findings regarding negative familial communication, future research should consider the impact of familial communication more broadly. While we have focused on negative familial communication directed toward the child, other forms and patterns of familial communication may be important. For example, Neumark-Sztainer et al. (2010) assessed parent "weight talk," the frequency with which mothers and fathers talk about their own weight. They found that "weight talk," particularly by mothers, was prevalent and associated with binge eating and extreme weight control behaviors in girls. In a different vein, Gross and Nelson (2000) found that daughters' perceptions of their father's messages to their mother regarding their mother's weight was significantly associated with daughters' weight preoccupation. They suggested that young women may learn from their father's criticism of their mothers that it is important to be thin.

Finally, the vast majority of studies reviewed here are cross-sectional in nature, which limits causal inferences regarding the role of familial factors. In their 7-year prospective study, Field et al. (2008) incorporated several familial variables that examined predictors of the onset of at least weekly binge eating and purging in adolescents from the Growing Up Today Study. Their findings suggest the potential importance of paternal weight-related variables, specifically the importance of daughters' weights to their father for adolescent girls and paternal negative comments regarding sons' weights for adolescent boys, as predictors of the onset of disordered eating. Also of importance is the possibility that individuals who develop disordered eating or EDs may elicit dysfunction in their families over time. In support of this assertion, Lehoux and Howe (2007) found that individuals with BN rated their parents as more controlling compared to their sisters, who also reported their parents were more controlling toward their sister with BN. This study highlights the possibility that just as family factors may influence eating psychopathology, the presence of eating psychopathology may elicit dysfunctional family responses. Furthermore, some evidence suggests that eating psychopathology may influence familial functioning (Archibald, Linver, Graber, & Brooks-Gunn, 2002). For example, in their longitudinal study, Archibald et al. (2002) found that adolescents' unhealthy eating behavior had a direct effect on parental relationships over time, but not vice versa. Clearly, additional prospective research is needed.

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Gender and Eating Disorders

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Gender differences in eating disorders are so commonly accepted as pervasive and immutable that gender has been identified as a "fixed risk factor" for the development of eating disorders (Jacobi, Hayward, deZwaan, Kraemer, & Agras, 2004; Striegel-Moore & Bulik, 2007). This perspective particularly reflects the fact that the rates of both anorexia nervosa (AN) and bulimia nervosa (BN) are substantially higher among girls and women than boys and men (see Chapter 5).

In this chapter, we examine research that seeks to explain gender differences in rates of eating disorders (EDs) and related problems. Given that body dissatisfaction, particularly regarding weight and shape concern, thin-ideal internalization, and dieting are all good predictors of the development of EDs and ED symptoms and that they show gender differences (Smolak, 2012b), we include such attitudes and behaviors in our analysis. We first consider the epidemiology of gender differences in EDs. We then discuss the difference between sex and gender, as well as why gender should not be considered a fixed variable. Biological explanations of gender differences are considered next. Finally, Piran's (Piran & Teall, 2012; Smolak & Piran, 2012) Adverse Social Experiences Model is used to organize the social and cultural forces that might account for the gender differences.

Gender Differences in Eating Disorders

The overall gender ratio for eating disorders may be as high as 10 women for every man, though the ratio may be smaller in childhood and adolescence, perhaps as low as 3:1 (Watkins, 2011). AN seems to be particularly rare among men. For example, a six-country study of rates of EDs in a stratified probability sample of over 4,000 community members found no lifetime cases of AN among men (Preti et al., 2009). This is not to say that men cannot develop AN; a U.S. study with a nationally representative sample of nearly 3,000 people found a lifetime prevalence rate for AN of 0.3% in men and 0.9% in women (Hudson, Hiripi, Pope, & Kessler, 2007). BN may be less rare among men than AN is, but it is still remarkably more common among women. Hudson et al. (2007) reported a BN lifetime prevalence of

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0.5% among men and 1.5% among women in the United States, while Preti et al. (2009) reported approximately eight times the number of BN cases among European women than men. A study by Striegel-Moore and colleagues (2009) also reported that women were more likely than men to report bulimic symptomology. Specifically, women were more likely to report loss of control over eating, as well as more binge eating, vomiting, and fasting. The effect sizes were small, however.

Both AN and BN involve a component of weight control attempts, often marked by dieting and food restriction, as well as weight-related body dissatisfaction. A third eating disorder, binge eating disorder (BED), does not have weight control attempts or body image disturbances as diagnostic criteria (see Chapters 4 & 10). Interestingly, there may be a smaller gender difference in cases of BED than in AN and BN. Preti et al. (2009) reported that about 15% of the cases of BED they identified occurred among men, whereas Hudson et al. (2007) reported lifetime prevalence rates of BED as 2.0% of men and 3.5% of women.

AN and BN are rare among prepubescent children (Watkins, 2011). At least two other eating-related problems occur among children (see Chapter 13). One, food avoidance disorder, is about four times more common in girls than boys, while the other, selective eating, may be about four times more common among boys.

It is also noteworthy that the majority of adults diagnosed with EDs also meet the criteria for other gendered psychiatric syndromes (Hudson et al., 2007; see also Chapters 15 & 54). Anxiety disorders and mood disorders are particularly common comorbid diagnoses. For example, Hudson et al. (2007) reported that 47.9% of AN, 80.6% of BN, and 65.1% of BED cases also met the criteria for an anxiety disorder, while 42.1% of AN, 76.7% of BN, and 46.4% of BED cases could be diagnosed with a mood disorder. Preti et al. (2009) also reported high rates of comorbidity between EDs, mood disorders, and anxiety disorder. Like EDs, depression and anxiety are more common among women than men (McLean, Asnaani, Litz, & Hofmann, 2011). While it is unclear how the comorbid problems are related, it is possible that gender differences in eating disorders are at least exacerbated by the presence of anxiety or mood disorders.

Currently, the same ED criteria are applied to men and women (see Chapters 8–12). This assumes that the disorders are similar in men and women. Yet, many questions about gender differences in risk and protective factors and the course of the disorders remain unanswered. This is partly because men are simply omitted from much ED research on the assumption that they are rarely affected.

Sex and Gender

In order to discuss variables important in explaining the links between gender and EDs, we first distinguish between the terms sex and gender. In everyday language people might use the term "sex" (as in, e.g., "what sex was her baby?") to refer to a biological distinction between females and males. In the usual course of development males and females can be distinguished by their sex chromosomal pattern; by their internal gonads that start the circulation of varied amounts of particular types of hormones; and by their external genitals (Fausto-Sterling, Coll, & Lamarre, 2012). Thus, there are biological variables associated with sex, although these might not unambiguously assign people to the two categories of male and female.

There are, of course, many sociocultural variables associated with the categories "male" and "female," and the term "gender" can be used to refer to a distinction that can encompass this

range. In the process of development children usually identify with the gender they are labeled (typically, "girl" or "boy") and start to monitor their behavior accordingly (Martin & Ruble, 2004). There are clear gender stereotypes in U.S. culture such that men are associated with agency and industriousness, and women with emotional expressiveness and communality (Diekman & Eagly, 2008; Rudman, Moss-Racusin, Phelan, & Nauts, 2012). In the usual course of development children learn these stereotypes easily and early and might internalize them, using them to guide behavior. Engaging in gender-stereotyped behaviors allows people to experience congruence between their behavior and expected roles (Diekman & Eagly, 2008) and to avoid backlash (Rudman et al., 2012). Further, particular situations call forth gender-stereotyped behaviors, and social role theory (Eagly, Wood, & Johannesen-Schmidt, 2004) explains that gender-stereotyped social roles are likely to elicit behaviors that help reinforce the stereotypes.

Gender is sometimes treated as if it represents just one immutable construct, that is, a fixed marker risk factor (Jacobi et al., 2004). However, given the complexity of variables involved in sex and gender, it is more accurate to think of gender as a summary or composite variable that is associated with many different factors that might interact with one another. The interaction makes it difficult, if not impossible, to isolate the effects of any one variable associated with gender to investigate the single cause of a gendered behavior. Furthermore, different experiences are associated with each gender.

Biological Variables Associated with Gender and Eating Disorders

Biological risk factors are increasingly invoked as explanations for the development and maintenance of eating disorders (see Chapters 17, 24, 28, & 30). Much of the recent research focuses on neurobiology (including neurocircuits and neurochemistry) and genetics. Like all research on EDs, the samples in these studies are often restricted to women, limiting their usefulness for understanding gender differences. Furthermore, it is noteworthy that there are very few prospective or experimental genetic or neurochemical data. Thus, although there is face validity to the argument that genetics or neurobiology might be crucial in understanding the etiology of ED, the currently available data do not clearly demonstrate this (Levine & Smolak, 2014; see also Chapter 67).

Neurobiology

There is substantial evidence indicating that the brains of women in the acute phases of AN and BN function differently than those of women who are not ill with ED (Kaye, Wierenga, Bailer, Simmons, & Bischoff-Grethe, 2013). While there is some normalization of brain functioning as the ED symptoms subside, some differences remain. The precise differences are beyond the scope of this chapter and are discussed in detail in Chapter 17. We want to make two points about these data.

First, there seems to be an underlying assumption that women's brains are more susceptible to EDs than men's brains. Such vulnerability would account for gender differences in the disorders and is reflected in the reliance on female samples. Limited research shows that women's serotonin systems are more sensitive to calorie-restrictive dieting (Cowen, Clifford, Walsh, Williams, & Fairburn, 1996). It has further been suggested that hormones, which affect brain functioning, covary with disordered eating. For example, in a small sample study, Racine and

colleagues (2012) found that estradiol was negatively associated and progesterone levels positively associated with body dissatisfaction and drive for thinness. Body dissatisfaction and drive for thinness were highest in the midluteal/premenstrual phase of the menstrual cycle. Dietary restraint did not show a clear relationship to menstrual cycle. Similarly, a study by Klump and colleagues (2013) found that emotional eating was highest in the midluteal phase and was associated with an interaction of estradiol and progesterone. Hormonal fluctuations predicted changes in emotional eating but not the reverse. Edler, Lipson, and Keel (2007) reported similar findings in a sample of BN clients. These findings need to be replicated.

This brings us to our second point. While it is premature to pinpoint a neurobiological vulnerability that might contribute to the gender differences in ED, there are some suggestive data that one might exist. The big question, then, is what creates the vulnerability? When people hear about a biological vulnerability, they often assume that the vulnerability is innate or hereditary. This is certainly possible, but it is not likely. At the very least, characteristics such as thin-ideal internalization might represent one outcome of an interaction between biological and environmental factors. It is also likely that the brain is shaped by experiences and exposure to various environmental factors. Neuroscientists have clearly established that experiences shape the extent and nature of neuronal connections (Cichetti & Curtis, 2006). Sociocultural factors, including media (Murray et al., 2006), are likely influencing brain functioning. As noted above, dieting affects neurochemistry. Whether early or frequent dieting has a more dramatic effect that might underlie ED is a question for future research, but it is a reasonable hypothesis. Thus, rather than being a mysterious distal cause of EDs, neurobiological factors may be a mediator between ED and measurable sociocultural factors that affect females differently than males. Models of such relationships, with appropriate empirical tests, are desperately needed (see Chapter 67).

Genetics

Numerous studies have suggested a sizeable genetic component in EDs such that correlations for various disordered eating behaviors are higher in identical than in fraternal twins (see Chapter 28). Some of this research addresses the issue of gender differences. First, a few studies have included men as well as women (Suisman & Klump, 2011). These studies indicate that men show similar or lower estimates of genetic components than women do. Some of this may be attributable to the use of measures that were developed for women. If there is a difference in genetic vulnerability, it is unclear what precisely constitutes the risk. It is possible, for example, that women more commonly than men have personality characteristics (see Chapter 32) or emotional characteristics (e.g., negative affect) that have substantial genetic components and contribute to the overall clinical picture of ED.

Thin-ideal internalization provides a good example of this possibility. Thin-ideal internalization is one of the best predictors of the development of ED (Stice, 2002). Recent research has found that a genetic component accounts for nearly half of the variability in thin-ideal internalization in women (Suisman et al., 2012). The researchers do not argue that thin-ideal internalization itself is heritable. Rather, they, like sociocultural theorists (see Chapters 21 & 29), suggest that some personality characteristic, such as perfectionism or social comparison tendency, mediates the relationship between sociocultural influences (media, peers, parents) and ED. It is that unmeasured personality characteristic that might be heritable. Future research should evaluate candidate characteristics.

A second line of research focuses on heritability and puberty. Klump et al. (2010) have found that, prior to puberty, girls' ED symptoms show little heritability and appear to be heavily influenced by shared environmental factors. The situation is reversed postpubertally in girls. This shift does not occur in boys, who show a steady genes:shared environment ratio throughout adolescence (Klump et al., 2012). This gender difference, combined with the work suggesting relationships between ovarian hormones and some disordered eating behaviors, has led Klump and her colleagues to suggest that ovarian hormones mediate the onset of genetically based components of ED.

Caveats Although critiques of the genetic research concerning ED have been offered elsewhere (Levine & Smolak, 2006, 2014; Smolak, 2012a), three points should be noted here. First, much of the research uses a sample from the Michigan Twin Study Sample. The findings need to be replicated with other, more diverse samples. Second, genetic studies of EDs generally use an additive model (genetics + shared environment + unique environment; Klump et al., 2010; Suisman et al., 2012). Behavioral geneticists and developmental psychologists have long acknowledged that interactive models more accurately depict development. In the additive model, gene–environment interactions are assigned to the genetic component, thereby inflating this variance estimate. Finally, there are as yet no longitudinal studies examining how environmental factors might affect the expression of genes.

Conclusions

Biopsychiatric research on EDs presents an interesting new direction for etiological and treatment models (see Chapters 17 & 67). However, it is in its infancy, and so far we do not have experimental or longitudinal data examining neurobiological or genetic factors. While it makes sense that these variables play a role in ED development and maintenance, we do not have the data to establish that. Thus, we need to proceed cautiously (see Chapter 67). That said, researchers need to be aware of the possible influence of both sociocultural and biopsychiatric components in EDs, and explain the gendered nature of EDs.

Sociocultural Variables Associated with Gender and Eating Disorders

Biopsychiatric research has only rarely considered the gendered nature of ED. In contrast, many sociocultural variables consistently explain gender variability in ED attitudes and behaviors. Piran and colleagues' ecologically oriented Adverse Social Experiences Model (ASEM; Piran & Teall, 2012) can be used to frame this body of research (see Chapter 43).

Adverse Social Experiences Model (ASEM)

The ASEM is derived from the more general Developmental Theory of Embodiment (Piran & Teall, 2012; see also Chapter 43) in which embodiment, the "experience of engagement of the body with the world" (Allan, 2005, cited in Piran & Teall, 2012, p. 169), occurs if people experience physical freedom (as opposed to physical corseting), mental freedom (vs. mental corseting), and social power (vs. social disempowerment). For example, an embodied woman might be someone who engages in physical pursuits with her body (such as skiing) and perceives

them as enjoyable, is able to reject harmful societal prescriptions about the body such as the unrealistically thin ideal (see Chapter 21), and is in a social position free from the experience of discrimination. In contrast, according to the ASEM (see Chapter 43), there are disruptions to embodiment that can occur in each of the three domains: (a) the experience of prejudice based on social position; (b) internalization of harmful stereotypes; and (c) violations of body ownership. Each domain forms a direct path to disordered eating, and experiences in multiple domains may increase the likelihood of developing disordered eating (Smolak & Piran, 2012).

The ASEM is consistent with objectification theory (Fredrickson & Roberts, 1997), which has received much empirical support (Calogero, Tantleff-Dunn, & Thompson, 2011; Tiggemann & Williams, 2012; see also Chapter 19). Persistent sexual objectification of women leads women to internalize the objectification, leading to *self*-objectification which can be manifested as surveillance of the body. Persistent, if not obsessive, body monitoring is proposed to deprive women of peak emotional experiences and to disrupt "flow," to result in a lack of attention (and therefore awareness) of internal bodily states, and to create an increase in appearance anxiety and body shame. The latter have been found to mediate the relationship between self-objectification and eating disorders, depression, and sexual dysfunction (Calogero et al., 2011; Tiggemann & Williams, 2012).

A distinguishing feature of the ASEM compared to objectification theory is that the ASEM does not require body dissatisfaction as a component in the pathway to ED (see Chapter 43). Piran and Teall (2012) point out that EDs may also represent a girl's attempt to gain control over a traumatic experience, such as abuse (see Chapter 34), or other pressures attributable to prejudice and discrimination. Other theorists have long noted the importance of a sense of control in ED, particularly in AN (Bruch, 1978; see also Chapter 2). The ASEM also allows for the consideration of many different variables as important contributors to ED, including direct experiences and one's position in society. To begin our discussion of research supporting the ASEM, we start at the societal level.

Group-Based Prejudice: The Cultural Mandate of the Thin, Submissive Female Body

We conceptualize the cultural treatment of the female body as a form of group-based prejudice against girls and women. An unrealistically thin ideal body, often portrayed as the most important female attribute, is a dominant message in the Western mass media (see Chapters 21 & 29). Collins (2011) reviewed content analyses examining gender and media depictions, concluding that women were portrayed less often than men but, when portrayed, were very likely to be depicted as sexual objects and in submissive social roles. Greenwood and Lippman (2010) found that female characters in U.S. media were shown as more attractive, thinner, sexualized, and younger than male counterparts. Buote, Wilson, Strahan, Gazzola, and Papps (2011) conducted several studies showing that individuals in the United States are much more likely to be confronted with images of the female body ideal compared to the male body ideal. For example, they found that 72% of women appearing in magazines were depicted as very attractive, 76% were thin, 5% were curvy, 2.2% were fit, and 0% very muscular; the figures for males depicted were 39% very attractive, 9% thin, 0% curvy, 37% fit, and 2% very muscular.

Media exposure to the thin, sexualized, and objectified ideal has been linked directly with the experience of body dissatisfaction in women, according to meta-analyses of the data (Grabe, Ward, & Hyde, 2008; Hausenblaus et al. 2013; see also Chapters 19, 21, & 29). The few longitudinal studies on this topic show that in children the amount of media exposure

predicts subsequent increases in negative body image and disordered eating (Levine & Murnen, 2009). The media's role is prejudicial because they often symbolically exclude women and, when girls and women are included, their value is defined narrowly in terms of their appearance and their bodies, inviting the audience to join in the process of judging women as inadequate if they fail to meet the ideal.

The thin, sexualized ideal that is widely advertised for women is incorporated into a gender-stereotyped sexual role for women that is submissive to men's stereotyped sexual dominance. A "heterosexual script" exists in U.S. culture that encourages men to pursue sex without emotional commitment, and women to secure committed relationships with men (Kim et al., 2007). If women try to pursue the ideal, they are at risk for EDs through the process of self-objectification (see Chapter 19). The prominence of both the female sexual object role and the corollary male sexual actor role encourages the direct treatment of women as sexual objects, which also puts females at risk for ED (see below). The submissiveness associated with the ideal and its accompanying disempowerment might lead women to try to regain control through dietary restriction.

In addition, girls are likely to experience societal pressure about the body from many sources other than the media. For example, some girls are members of appearance-oriented friendship groups who share information about fashion and appearance, glorifying the slender ideal (Smolak, 2012a; see also Chapter 31). Appearance conversations with friends and appearance social comparison to friends, dieting by friends, and peer teasing have all been prospectively related to body dissatisfaction among adolescent girls (Jones, 2004; Paxton, Eisenberg, & Neumark-Sztainer, 2006).

Myers and Crowther (2009) found through meta-analysis that there was a stronger link between social comparison and body dissatisfaction in females compared to males. The feminine gender role likely encourages social comparison of the body (Mahalik et al., 2005). Further, in another meta-analysis, the association between weight teasing and body dissatisfaction was found to be greater in females than males (Menzel et al., 2010). Research on "fat talk," conversations in which girls criticize their own bodies (in part for social approval), shows that it is not benign, but instead linked to body dissatisfaction and eating-disordered attitudes (Clarke, Murnen, & Smolak, 2010; Salk & Englen-Maddox, 2011, 2012). Women in both the United Kingdom and the United States report greater pressure than men to engage in fat talk (Payne, Martz, Tompkins, Petroff, & Farrow, 2011), though men may engage in other forms of body talk, including commenting on muscularity (Englen, Sladek, & Waldron, 2013). Overall, girls and women may face more pressure to conform to unrealistic body standards and this difference may contribute to the gender differences in ED.

Internalization of Harmful Stereotypes

The second domain in the ASEM involves the internalization of harmful stereotypes such as the ones about women we just described. Research indicates that internalization of norms associated with femininity, including the thin ideal (Stice, 2002), self-objectification (Tiggemann & Williams, 2012), and self-silencing (Smolak & Munsterteiger, 2002), are related to negative body image and disordered eating (see Chapter 19). Indeed, a study by Piran and Cormier (2005) found that self-objectification and self-silencing were independent contributors to drive for thinness and bulimic symptoms. Stice (2002) concluded that thin-ideal internalization was a causal risk factor for the development of EDs, particularly BN and bulimic symptoms.

Cafri, Brannick, and Thompson (2005) used meta-analysis to examine the association between sociocultural factors and body image dissatisfaction in women. The strongest relationship was found between internalization of the media ideal and body image dissatisfaction (see Table 27.1). In their media effects meta-analysis, Grabe et al. (2008) found the strongest associations with media exposure and internalization of the ideal, followed by media exposure and body image dissatisfaction, and media exposure and eating-disordered attitudes behaviors (see Table 27.1). These data suggest an indirect relationship between media exposure and eating-disordered behavior that is mediated by internalizing processes.

Table 27.1 Effect sizes associated with the prediction of eating disorders and associated constructs, gender-related variables, listed in order of effect size.

Predictor Variable	*Data Type*	*Gender*	*d*	*k*	*Citation*
Predicting internalization of thin ideal					
Feminist identity	C	F	−0.30	9	Murnen & Smolak (2009)
Media exposure	C,E	F	0.39	23	Grabe et al. (2008)
Predicting body dissatisfaction					
Masculine traits	C	M	−0.32	15	Blashill (2011)
Feminist identity	C	F	−0.25	28	Murnen & Smolak (2009)
Perceived pressure thinness	E,P	F	0.18	6	Stice (2002)
Media exposure	C,E	F	0.28	90	Grabe et al. (2008)
Media exposure	E	F	0.31	43	Groesz et al. (2002)
Weight-related teasing	C	M	0.49	8	Menzel et al. (2010)
Social comparison	C	M	0.54	34	Myers & Crowther (2009)
Awareness of ideal	C	F	0.61	25	Cafri et al. (2005)
Weight-related teasing	C	F	0.80	35	Menzel et al. (2010)
Social comparison	C	F	0.83	146	Myers & Crowther (2009)
Perceived pressure media	C	F	1.09	7	Cafri et al. (2005)
Internalization thin ideal	C	F	1.15	18	Cafri et al. (2005)
Predicting eating-disordered attitudes or behavior					
Masculine traits	C	M	−0.32	8	Blashill (2011)
Ball game sports	C	F	−0.20	8	Metzger et al. (2010)
Masculine traits	C	F	−0.13	21	Murnen & Smolak (1997)
Feminine traits	C	F	0.14	23	Murnen & Smolak (1997)
Thin-ideal internalization	E,P	F	0.16	4	Stice (2002)
Endurance sports	C	M	0.19	13	Metzger et al. (2010)
Perceived pressure thinness	E,P	F	0.24	5	Stice (2002)
Body image dissatisfaction	E,P	F	0.26	12	Stice (2002)
Media exposure thin ideal	C,E	F	0.30	20	Grabe et al. (2008)
Dieting	E,P	F	0.30	13	Stice (2002)
Aesthetic sports	C	F	0.32	51	Metzger et al. (2010)
Child sexual abuse	C	F	0.37	30	Smolak & Murnen (2002)
Weight-related teasing	C	F,M	0.77	22	Menzel et al. (2010)

Note. Data "Type" refers to type of studies from which data were collected: C = correlational, E = experimental, P = prospective. "Gender" refers to the gender of the sample. Effect size values (d) were translated from correlation values (r) when necessary with formula $d = 2 \times (r/\sqrt{[1 - r^2]})$.

The gender difference in the role of internalization in body-related attitudes and behaviors is likely due to the fact that females are sexually objectified to a greater extent than males, and attention to physical appearance (especially maintaining thinness) is tied directly to feminine gender role prescriptions. Research looking at gender roles more broadly supports this idea. While the feminine gender role might encourage body concerns, the masculine role might be protective in encouraging a sense of agency that is antithetical to self-objectification. Rejection of the traditional feminine gender role in the form of feminist attitudes was negatively associated with body dissatisfaction in a meta-analysis conducted by Murnen and Smolak (2009). Women with a strong feminist identity were less likely to internalize the thin media ideal.

Violation of Body Ownership

In the third domain of the ASEM, violations of body ownership, there are gendered physical experiences that relate to ED (see Chapter 43). For example, the experience of child sexual abuse (CSA; see Chapter 34) represents a dramatic violation of body ownership. Girls are more likely than boys to be the victims of CSA (Smolak & Piran, 2012). A meta-analytic review of research on CSA and eating disorders among females found that when sexually abused individuals were compared with nonabused individuals there was a slightly higher risk of eating disorders (Smolak & Murnen, 2002). Additional research has established sexual abuse as a nonspecific risk factor for ED, especially BN and related disorders (Thompson & Wonderlich, 2004; see also Chapter 34). Sexual abuse has also been associated with body dissatisfaction and ED in men and boys (Brewerton, 2007; see also Chapter 37). While the greater likelihood of CSA in girls may help to explain the gender differences in BN, Brewerton's findings do suggest that when boys and men have experiences similar to those of girls and women, they develop similar problems.

Messman-Moore and Garrigus (2007) found increased predictability of eating problems by looking at multiple forms of abuse experienced during childhood, as well as experience with rape in young adulthood. In other research, rape and other forms of sexual violence have been found to be associated with ED, especially, again, BN (Mitchell, Mazzeo, Schlesinger, Brewerton, & Smith, 2012; see also Chapter 34). Mitchell and colleagues also reported that BED was associated with nonrape sexual assaults. Faravelli, Giugni, Salvatori, and Ricca (2004) found that rape was more strongly related to body dissatisfaction and disordered eating than were other traumatic experiences.

Sexual harassment may seem a less dramatic violation of body ownership, but it occurs frequently and may be viewed as a reminder of men's control over women's bodies (Sheffield, 1987) and hence is a threat to body integrity and ownership (see Chapter 43). Even though the rate of ever experiencing harassment is often found to be similar between the genders, around 80% (AAUW, 2001), girls experience more frequent and severe harassment (Hand & Sanchez, 2000), and are more negatively affected (Murnen & Smolak, 2000; Petersen & Hyde, 2013b). For girls, experiencing peer harassment is associated with self-objectification and body shame (Lindberg, Grabe, & Hyde, 2007; Petersen & Hyde, 2013a), which might help explain some of the gender difference in EDs. Indeed, the experience of harassment has been associated with disordered eating in a sample of working women (Harned, 2000; Harned & Fitzgerald, 2002). Experience with "everyday" sexist events, such as being treated like a sexual object, has been linked with eating-disordered attitudes (Sabik & Tylka, 2006), likely through encouraging self-objectification (Kozee, Tylka, Augustus-Horvath, & Denchik, 2007). Thus, violations of the body in the form of harassment, rape, and abuse that occur more often among girls and women than boys and men are related to the development of eating problems.

On the other hand, athletic participation may improve one's sense of body ownership, thereby serving as a protective factor (Menzel & Levine, 2011; Piran & Teall, 2012; see also Chapter 33). Sports participation is still stereotyped as more masculine than feminine (Daniels & Lavoi, 2013), and despite federally mandated gender equality in sport opportunity in schools there are still higher rates of boys participating in sports than girls in the United States (Sabo & Veliz, 2008); so in this realm, too, females are at a disadvantage. Additionally, instead of athletics being protective of eating concerns as hoped, some forms of athletic participation have been associated with increased risk (see Chapter 35). Meta-analyses have shown that elite female athletes participating in sports that emphasize leanness, such as gymnastics, are at greater risk for EDs than female nonathletes (Smolak, Murnen, & Ruble, 2000), as are female athletes in aesthetic sports in which an evaluation of form and appearance is made by others (Metzger, Murnen, & Smolak, 2010). Certain groups of male athletes are more at risk, too, such as men in endurance sports (Metzger et al., 2010). Females participating in sports that do not emphasize leanness, such as ball-game sports, were found to be at lower risk.

Although it theoretically makes sense for athletic physicality to lead to a greater connection between mind and body, some sports encourage a thin ideal, and there is much objectification involved in some women's athletic uniforms. Also, there is cultural sexualization of female athletes that can range from more subtle forms, such as focusing on female athletes' appearance in sports news and commentary more than for male athletes, and less subtle forms, such as depicting famous women athletes in very revealing clothing on the cover of mainstream magazines (Daniels & Lavoi, 2013). A sample of Australian girls who participated in sports reported teasing experiences in this domain, which were related to self-objectification and body dissatisfaction (Slater & Tiggemann, 2011). Girls and women who engage in sport or exercise for appearance enhancement (i.e., to be or appear thinner) are likely to experience self-objectification (Harrison & Fredrickson, 2003; Slater & Tiggemann, 2011), which is related to EDs. Thus, the possibility that sport and exercise might promote physical freedom for girls and women is compromised in a society that encourages female sexual objectification.

Finally, because women are sexual objects, some of the clothing and beauty practices they are encouraged to adopt highlight their sexuality and (consequently?) limit their physical functioning. For example, clothing designed to make women look sexy, such as high heels and short skirts, prohibits certain types of movement. In experimental tests of objectification theory, wearing a swimsuit has been associated with negative feelings about the body (Fredrickson, Roberts, Noll, Quinn, & Twenge, 1998; Gapinski, Brownell, & LaFrance, 2003; Quinn, Kallen, & Cathey, 2006). A recent Australian study found that women chose clothes primarily for comfort, assurance, and fashion (Tiggemann & Andrew, 2012). Those who chose clothing for fashion were higher in self-objectification, suggesting that the everyday experience of dressing as a "fashionable" woman might reinforce bodily concerns that are common among women. The recent phenomenon of dressing "tween" and teen girls in sexualized clothing (Goodin, Van Denburg, Murnen, & Smolak, 2011) likely increases girls' self-objectification, which puts them at risk for EDs.

Conclusions and Future Directions

In this chapter we have provided evidence that American culture (along with other Western cultures) encourages females to obsess over their bodies, objectifying them rather than living in and with them in ways that enhance function, physical pleasure, and empowerment. The

frequent media depiction of the thin female sex object and the direct treatment and exploitation of females as sex objects put females at risk for the development of body image problems and EDs. Table 27.1 shows a summary of the meta-analytic data we reviewed. Data from this table suggest that the variables most highly associated with eating disorders are weight-related teasing and child sexual abuse. Thus, direct experiences of the violation of body ownership are very problematic (see Chapter 43). Further, the development of body image dissatisfaction is highly correlated with developing an eating disorder, and one of the strongest predictors of body image dissatisfaction is internalization of the media's thin ideal. Thus, the media help create a problematic cultural climate (see Chapter 29).

The data suggest that we could perhaps mitigate the damage of cultural forces by reducing internalization or increasing mind-body connection opportunities, but the roots of the problem are found at the cultural level. The cultural treatment of females as sexual objects, which makes women's primary value contingent on their sexual appeal while defining that appeal based on very narrow standards (American Psychological Association, 2007), puts undue cultural pressure on girls and women. Piran (2010) pointed out that in eating disorders research we have been more focused on "causes of cases," or individual differences that put specific people at risk, rather than "causes of incidence," which put a whole population at risk (seeChapters 43 & 47). The data reviewed in this chapter suggest that while there are individual difference variables that help predict the development of EDs, including the possibility of biological variables associated with gender, cultural factors are of supreme importance and we need to "treat the culture" if we hope to prevent EDs and help individuals on a large scale.

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28

Genetic Risk Factors for Eating Disorders

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This chapter discusses the genetic risk for eating disorders. The state of the science of family, twin, linkage, and molecular genetic association studies for anorexia nervosa (AN), bulimia nervosa (BN), and binge eating disorder (BED) are reviewed. However, because BED is a relatively new diagnosis, less research has focused on the genetic basis for this disorder. Future directions and upcoming approaches are also discussed.

Family Studies

Family studies have provided strong evidence for the familial nature of eating disorders. For example, Strober, Freeman, Lampert, Diamond, and Kaye (2000) found that first-degree relatives of individuals with AN are 11 times more likely to have AN during their lifetime than relatives of unaffected controls. Relatives of individuals (probands) with either AN or BN also have significantly elevated risk of having AN and BN than relatives of unaffected controls (Lilenfeld et al., 1998; Strober et al., 2000). Further, BED also aggregates within families. In an initial exploratory study, Fowler and Bulik (1997) found that a significantly greater percentage of participants with BED had a first-degree relative with BED compared with control participants. A much larger family study interviewing overweight or obese individuals with BED and first-degree relatives confirmed these findings, indicating that BED is approximately two times more common in family members of probands with BED, and aggregates independent of obesity (Hudson et al., 2006). Although family studies are typically the first

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step in determining genetic etiology, family studies are unable to elucidate whether a disorder aggregates within families due to genetic or environmental factors.

Twin Studies

In contrast to family studies, twin studies are able to delineate the genetic and environmental contributions to familial aggregation by comparing the concordance rates of a disorder between monozygotic (MZ) and dizygotic (DZ) twin pairs. MZ twins are assumed to share 100% of their genetic makeup, and DZ twins are assumed to share, on average, 50% of their segregated genes. Therefore, if the observed correlation between MZ pairs is twice as strong as the correlation between DZ pairs, genetic factors are implicated. However, twin studies cannot identify the specific genes involved in vulnerability to a disorder.

Population-based twin studies corroborate the familial aggregation of AN, BN, and BED. Studies using varying definitions of AN estimated heritability to range from 28% to 74% (Klump, Miller, Keel, McGue, & Iacono, 2001; Kortegaard, Hoerder, Joergensen, Gillberg, & Kyvik, 2001). Heritability estimates for varying definitions of BN have ranged from 28% to 83% (Bulik, Sullivan, Wade, & Kendler, 2000). Two population-based twin studies have estimated the heritability of the *DSM-IV* (*Diagnostic and Statistical Manual of Mental Disorders*, 4th ed.) diagnosis of BED; heritabilities were estimated at 39% and 45% (Javaras et al., 2008; Mitchell et al., 2010).

Linkage Studies

Linkage studies are a molecular genetics approach used to identify specific regions in the genome containing genes that may predispose individuals to a disorder. They are particularly useful for narrowing down a search of the entire genome to specific areas. To date, linkage studies have only focused on AN and BN. Areas on chromosomes 1, 2, 4, and 13 have been identified as possible regions of interest for AN (Devlin et al., 2002; Grice et al., 2002); chromosome 1, which houses the delta opioid receptor (*ORPD1*) and serotonin receptor 1D genes, has been of particular interest. To date, only one linkage study has been conducted for BN; significant linkage was observed on chromosome 10, and a region on chromosome 14 met criteria for genome-wide suggestive linkage, defined as a p value of less than 10^{-8} (Bulik et al., 2003). Although linkage studies have revealed areas of the genome that are of potential interest for AN and BN, few replication studies have been conducted to determine whether these regions of interest do in fact house genes that may influence liability to eating disorders. Moreover, the linkage approach has fallen out of favor in modern genetics, giving way to more sophisticated approaches such as genome-wide association studies (GWAS).

Candidate Gene Association Studies

Candidate gene association studies explore the relationship between a disorder and a specific gene that is selected a priori on the basis of some prior knowledge of the biology of a condition or the function of the gene. If the gene and disorder are significantly correlated, an association is said to exist. Association studies can also include transmission disequilibrium tests, which investigate if a genetic variant, or polymorphism (one or more variations in gene sequence among individuals),

is transmitted from parents to affected offspring. If the variant is passed from parent to affected child at a higher rate than would be expected, it is said to be associated with the disorder.

Given that we understand precious little about the underlying biology of eating disorders (see Chapters 17 & 30), the candidate gene studies that have been conducted have relied on the "usual suspects," or genes known to influence appetite, mood, and weight regulation including the serotoninergic system (5-HT system), the dopaminergic system, and the opioidergic system. Many of these studies have not possessed adequate statistical power to draw firm conclusions; moreover, none of the studies to date have identified genes that are in any way specific to the etiology of any eating disorder. We review the studies below, but we caution the reader against overinterpretation of the findings.

The Serotoninergic System

The 5-HT system is frequently studied in eating disorders research, as it plays an integral role in mood, appetite, and weight regulation (see Chapter 30). Many studies have focused on the *5-HT2A* receptor gene and the 5HT-transporter-linked polymorphic region (*5-HTTLPR*). The –1438G/A polymorphism of the *5-HT2A* gene has been shown to be significantly associated with risk for AN in meta-analyses (Gorwood, Kipman, & Foulon, 2003; Martásková, Slachtová, Kemlink, Záhoráková, & Papezová, 2009). However, findings regarding an association between BN and the promoter polymorphism –1438G/A of the *5-HT2A* receptor gene have been inconclusive.

Findings are also mixed regarding the role of *5-HTTLPR* in risk for eating disorders. The short allele of *5-HTTLPR* has been associated with increased duration of illness in AN (Gervasini et al., 2012) and has been shown to be significantly associated with risk for AN in two meta-analyses (Calati, De Ronchi, Bellini, & Serretti, 2011; Lee & Lin, 2010). In regard to BN, meta-analyses have failed to show that any *5-HTTLPR* polymorphism increases risk for BN (Calati et al., 2011; Lee & Lin, 2010; Polsinelli, Levitan, & De Luca, 2012). However, women with AN who are carriers of the *5-HTTLPR* short allele are four times more likely to cross over (see Chapter 55) to a clinical picture of BN during the course of their illness (Castellini et al., 2012).

To date, limited studies have investigated the role of 5-HT polymorphisms in BED. One small study exploring the potential of *5-HTTLPR* in BED vulnerability observed that the long allele was more frequent in individuals with BED than in controls (Monteleone, Tortorella, Castaldo, & Di Filippo, 2006). However, the sample size was small, so results should be interpreted cautiously.

More recent work has explored the interaction between *5-HTTLPR* and environmental factors, such as parenting style and childhood maltreatment, in the risk for AN and BN. For example, a significant interaction was observed between the short allele of *5-HTTLPR* and parenting style for AN; carriers of the short allele who self-reported having experienced problematic parenting styles (e.g., parental criticism, control, and high expectations) were at increased risk for AN (Karwautz et al., 2011). Studies have also identified significant interactions between the short allele and childhood maltreatment (see Chapter 34) in the prediction of traits common to BN; women with BN who are carriers of the short allele and who experienced childhood maltreatment exhibited increased psychopathological traits such as sensation seeking, insecure attachment, and dissocial behavior (Steiger et al., 2007, 2008, 2009).

These findings highlight the fact that genetic factors likely do not work alone in increasing eating disorder vulnerability, but may interact with environmental factors in complex ways to influence risk (see Chapters 34 & 67). For example, consistent evidence from the animal literature suggests that environmental factors can indeed influence gene expression. A comparison

of rat pups exposed to responsive mothers (indicated by patient licking of offspring) and nonresponsive mothers (indicated by neglect of pups) showed that pups with a neglectful mother are more anxious (Meaney, 2010). Analyzing the brains of the pups showed distinctive differences in gene expression in the hippocampus. This variation suggests that the responsive mothers may have buffered their pups' genetic vulnerability to anxiety and stress. Therefore, it will be important to consider the role of the environment in both the risk and resilience against eating disorder development (see Chapter 67).

The Dopaminergic System

The dopaminergic system is involved in feeding modulation, thinking processes, motor activity, and reward-motivated, drug-seeking behaviors, making it a reasonable candidate for eating disorder vulnerability. Investigations have focused primarily on the *DRD2* and *DRD4* receptor genes. In a follow-up haplotype analysis—which is a statistical analysis examining a combination of genes located on a chromosome that are inherited together—of the linkage study conducted by Grice and colleagues (2002), two *DRD2* polymorphisms were specifically associated with AN binge-purge type, while two additional polymorphisms were associated with AN diagnosis in general (Bergen et al., 2005). *DRD4* polymorphisms have also been associated with AN and AN binge-purge type (Bachner-Melman et al., 2007).

No significant associations between *DRD2* and BN have been reported. However, significant interactions between *DRD2* polymorphisms and environmental factors have been observed in BN samples. For example, an interaction between the A1 allele of the *DRD2* Taq1A polymorphism and childhood sexual abuse was observed for sensation seeking in women with BN. Specifically, women with BN who were carriers of the A1 allele who also experienced childhood sexual abuse exhibited higher sensation-seeking scores (Groleau et al., 2012). A significant interaction was also identified between the hypofunctional, 7-repeat allele of *DRD4* and season of birth for maximum lifetime body mass index (BMI) in women with BN (Levitan, Kaplan, Davis, Lam, & Kennedy, 2010). Finally, a gene by gene interaction was reported between *DRD2* and *DAT* (dopamine active transporter) for BMI in women with BN, such that women possessing the alleles that code for less dopamine activity had higher BMIs (Thaler et al., 2012). These findings further illustrate the importance of the interplay between genetic and environmental factors in the risk for eating disorders and related traits.

Findings regarding the association between dopaminergic genes and BED have been unremarkable. A comparison of individuals with BED, normal weight individuals, and obese individuals without BED on several *DRD2* polymorphisms revealed no significant differences (Davis et al., 2008). However, in a follow-up study, significantly more individuals with BED had the G allele of the Taq1A polymorphism of the *DRD2* gene compared to obese controls (Davis et al., 2009). Finally, a larger investigation by Davis and colleagues (2012), which compared obese individuals with and without BED, found a significant association between the Taq1A A2 allele and the TT genotype of the C597T marker in individuals with BED. The authors conclude these findings may suggest that obese adults with BED are hypersensitive to reward compared with obese adults without BED.

The catechol-*O*-methyltransferase (*COMT*) gene is involved in the metabolism of dopamine and norepinephrine and has been posited to play a role in eating disorders by several reports. For example, women with AN are more likely to have the Val158 allele of the Val158Met polymorphism, and individuals homozygous for this variant have a two-fold increased risk for AN (Frisch et al., 2001). However, replications of this finding have not been consistent, and a

2012 meta-analysis did not reveal any significant effect of this polymorphism on AN risk (Brandys et al., 2012).

Findings regarding the association between *COMT* and BN or BED have been unremarkable. However, a gene by gene interaction has been observed between *COMT* and *DRD4* for self-harming behaviors in women with BN. Women possessing the *COMT* and *DRD4* alleles that result in lower levels of dopamine neurotransmission exhibited higher scores on a self-report measure of self-harm (Thaler et al., 2012).

Genes Involved in Food Intake and Weight

Ghrelin, produced primarily in the mucosa of the stomach, is a hormone best known for its role in food intake and meal initiation. Association studies investigating the relationship between the gene encoding ghrelin and eating disorders have revealed inconsistent findings, with most reporting no significant associations (Ando et al., 2006; Cellini et al., 2006; Kindler et al., 2011; Monteleone, Tortorella, Castaldo, Di Filippo, & Maj, 2006). However, some studies have identified significant associations. For example, exploring the ghrelin gene has shown that the 72Met variant of the Leu72Met polymorphism is significantly more common in individuals with AN binge-purge type than healthy controls or individuals with AN restrictive type (Dardennes et al., 2007). In addition, a ghrelin gene variant was shown to significantly predict weight recovery in AN. Those individuals homozygous for the TT genotype of the T3056C variant were more likely to achieve weight restoration (including crossover to another eating disorder or remission) (Ando et al., 2010). Finally, genetic variants of ghrelin *O*-acyltransferase (*GOAT*), a ghrelin-activating gene, have been implicated in AN risk. Individuals homozygous for the *GOAT* risk allele were approximately 1.5 times more likely to have an AN diagnosis (Muller et al., 2011).

Less work has been devoted to investigating the role ghrelin receptors have in the risk for BN and BED. However, one study reported a significant association between the 171C polymorphism of the ghrelin receptor (growth hormone secretagogue receptor: *GHRS*) gene and BN (Miyasaka et al. 2006). A significant positive association for the Leu72Met polymorphism of the ghrelin gene and BED has also been found and is associated with a moderate risk for developing BED (Monteleone et al., 2007).

Norepinephrine is also involved in food intake, affects the reward system, and plays a role in arousal. Transmission disequilibrium has been observed for the norepinephrine transporter gene, *SL6A2*, and AN (Urwin et al., 2002), but this association was not confirmed in a subsequent report (Hu et al., 2007). However, a gene by gene interaction has been observed between *SL6A2* and *MAOA,* the gene that encodes the enzyme monoamine oxidase A, such that individuals homozygous for the long allele of *SL6A2* who also had a *MAOA* long allele were at a two-fold increased risk for AN restrictive type (Urwin et al., 2003). To date, studies exploring the association between norepinephrine genes and BN and BED are in their infancy.

Cannabinoid receptors stimulate appetite and mediate the psychotropic effects of tetrahydrocannabinol. Transmission equilibrium tests of *CNR1* receptor gene revealed that the 14-repeat allele of the gene was more frequent in AN binge-purge type while the 13-repeat allele was more frequent in AN restrictive type (Siegfried et al., 2004). Further, additional *CNR1* polymorphisms and cannabinoid receptors have shown significant relations with AN (Ishiguro et al., 2011; Monteleone et al., 2009). In regard to BN, one study exploring the association between BN and polymorphisms of *CNR1* and the gene coding fatty acid amide hydrolase (FAAH) found that participants with BN had significantly higher frequencies of the A allele

and AG genotype of the *CNR1* single nucleotide polymorphism, rs1049353, as well as a significant association with a FAAH single nucleotide polymorphism and BN diagnosis (Monteleone et al., 2009). Little is known about the possible role of the cannabinoid receptors in BED.

Brain-derived neurotrophic factor (*BDNF*) plays an essential role in brain development, learning, and memory, and appears to have a role in mood, eating, and weight. The most frequently examined polymorphism of the *BDNF* gene, Val66Met, has been implicated in AN and BN risk. For example, the Met66 variant of the Val66Met polymorphism has been implicated in AN (Dmitrzak-Weglarz et al., 2007; Ribases et al., 2004) and AN restrictive type (Dmitrzak-Weglarz et al., 2013; Ribases et al., 2003, 2005). However, a meta-analysis indicated that the Val66Met polymorphism is not associated with vulnerability to AN (Brandys et al., 2013). Genetic variants of *BDNF* have also been implicated in BN. For example, two polymorphisms, 270C/T and Val66Met, have both shown significant associations with BN (Ribases et al., 2004). However, findings have been inconsistent.

Estrogens are integral for normal food intake, so they may play a role in eating disorder vulnerability. However, studies exploring the association between the estrogen receptors *ESR1* and *ESR2* and risk for AN or BN have yielded mixed results. For AN, a significant association has been observed between AN and both *ESR2* (Eastwood, Brown, Markovic, & Pieri, 2002) and *ESR1* (Versini et al., 2010). For BN, a significant association has been observed with *ERβ* (estrogen receptor beta gene) (Nilsson et al., 2004). However, findings have not been consistent (Eastwood et al., 2002); therefore definitive conclusions cannot be made about the estrogen receptors' role in eating disorder risk.

Because low body weight is a core feature of AN and being overweight is commonly observed in BED, studies have explored whether genes known to be involved in obesity are associated with risk for BED and AN. It is established that mutations in the melanocortin 4 receptor (*MC4R*) gene can cause severe, morbid obesity, so this gene has been targeted in eating disorder studies. One study exploring the role of *MC4R* in BED risk observed a genetic mutation in 24 severely obese individuals, all of whom had BED (Branson et al., 2003). Similarly, an investigation of 300 obese individuals undergoing laparoscopic gastric binding found that 19 individuals were carriers of the *MC4R* risk variant and that all 19 individuals met criteria for BED (Potoczna et al. 2004). In contrast, no significant associations have been observed between *MC4R* variants and AN (Brandys et al., 2010; Hinney et al., 1999).

The fat mass and obesity-associated gene (*FTO*) has been widely replicated as playing a significant role in obesity. Currently, results are inconclusive about the role of *FTO* in eating disorder risk. Specifically, studies have been mixed in regard to AN, while only one single nucleotide polymorphism has been significantly associated with BN (Muller et al., 2012).

The Opioidergic System

The opioidergic system influences pain, reward sensitivity, and food intake. It is also thought to affect vulnerability to addictive disorders, making this system a viable candidate for playing a role in eating disorders. For example, significant linkage for AN was observed on chromosome 1, which contains the opioid receptor delta 1 gene (*OPRD1)* (Grice et al., 2002). Follow-up analyses indicated that three polymorphisms of the *OPRD1* gene were significantly associated with AN. Additional significant findings have emerged with *OPRD1* and AN (Brown et al., 2007). However, work exploring the role of opioidergic system genes in BN or BED risk has been limited.

Genome-wide Association Studies

Molecular genetics is a rapidly evolving field, and the technology is constantly evolving. For example, advances in technology have enabled GWAS. GWAS are similar to association studies. However, GWAS allow for the comprehensive investigation of genes across the entire genome, rather than focusing on specific individual genes, sets of genes, or larger genomic areas. Therefore, GWAS may pinpoint novel genetic variants that are involved in eating disorder risk. Despite the fact that large-scale GWAS have been a fruitful approach in medicine and in other psychiatric diseases (e.g., schizophrenia—see below), only two GWAS attempts for AN have been conducted, and both possessed sample sizes far below those of other psychiatric disorders that have yielded significant findings (Collins & Sullivan, 2013). Therefore, these studies were likely underpowered to detect significant results. No GWAS for BN or BED have yet been performed.

Two GWAS for AN are noteworthy. The first study identified areas on chromosomes 1 and 11 that were significantly associated with AN (Nakabayashi et al., 2009). However, this investigation was restricted by the use of DNA pooling and including only 23,000 microsatellite markers, which limits the amount of the genome covered. The second investigation, by Wang et al. (2011), conducted GWAS in 1,033 female AN cases and 3,733 pediatric controls; however, no findings reached genome-wide significance, likely due to a small sample size. An additional GWAS has recently been completed (Boraska et al., 2014), and although no findings reached genome-wide significance, a comparison of the discovery results (i.e., the initial findings of the GWAS) to the replication results indicated that 76% of the effects were in the same direction—an observation highly unlikely to be due to chance ($p = 4 \times 10^{-6}$). This result strongly suggests that many true findings exist but that this sample, the largest yet reported, was still underpowered for their detection. Therefore, the accrual of large genotyped AN case-control samples should be an immediate priority for the field. Recent work underscores the importance of much larger sample sizes than originally conceived to enable identification of significant associations in psychiatry (Collins & Sullivan, 2013; Sullivan, Daly, & O'Donovan, 2012).

Conclusions and Future Directions

Family and twin studies have provided convincing evidence that AN, BN, and BED are likely influenced by genetic factors. Given our rapidly increasing knowledge in psychiatric genetics, it is clear that our job is to increase samples size before we can have confidence in any of our genetic findings in eating disorders. Our studies have routinely been underpowered, raising the risk of Type II error. There is considerable room for optimism given the rapid advances in gene discovery that have occurred in schizophrenia (Schizophrenia Psychiatric Genome-Wide Association Study (GWAS) Consortium, 2011) and bipolar disorder (Psychiatric GWAS Consortium Bipolar Disorder Working Group, 2011) as researchers have cooperated to increase sample size. Moreover, cross-disorder GWAS are allowing us to identify variants that contribute across psychiatric disorders (Cross-Disorder Group of the Psychiatric Genomics Consortium, 2013) —a highy sensible approach given the frequent symptom sharing across diagnostic categories and the high unlikelihood that the organization of the human genome in any way reflects the diagnostic schema of the *DSM*.

Of particular interest, larger GWAS outside of the eating disorders field have identified genetic variants that were not part of those commonly explored in candidate gene studies

(e.g., the role of *FTO* in obesity). This revolutionary technique has opened doors to discover new biological pathways that can influence disorder expression. Ultimately, the identification of genetic risk profiles may assist with developing more accurate predictions of an individual's risk for a disorder and assist us with understanding why some individuals are more vulnerable to environmental insults than others (see Chapter 67).

In sum, genetic factors appear to play an important role in the pathophysiology of AN, BN, and BED. Moreover, these genetics factors are likely involved in several aspects of these pernicious disorders such as increasing vulnerability towards the disorder, the severity of the disorder, and possibly individual response to treatment. For example, as described above, *5-HTTLPR* was significantly associated with diagnostic crossover from AN to BN, while a ghrelin gene polymorphism was significantly associated with weight restoration in AN, although both of these findings require replication. However, molecular genetic investigations are truly in their infancy. GWAS with larger sample sizes are necessary in order to further elucidate the genetic architecture of eating disorder risk and allow for an exploration of the genome in an unbiased fashion. A thorough understanding of the genetic architecture of eating disorders will have important implications for prevention and intervention efforts and may enrich our ability to explore environmental risk factors by placing them in a biological context.

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29

Media and Eating Disorders

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There is substantial empirical support for application of a sociocultural paradigm to understanding the causes of eating disorders (EDs; see Chapter 21). The mass media comprise one potentially important sociocultural factor in the development of EDs. Levine and Harrison (2009) have argued that mass media constitute a "meta-context" influencing various aspects of environments in the ecology of children, youth, and young adults, such as school, family, peer interactions, and athletics. In fact, prominent sociocultural theories, such as the tripartite influence model (Thompson, Heinberg, Altabe, & Tantleff-Dunn, 1999), McCabe and Ricciardelli's (2001) parents-peers-media model, and Stice's (1994) dual-pathway model of bulimia nervosa (BN), all propose that media operate with other sociocultural variables to increase risk for EDs.

There is no doubt that mass media play multiple, prominent roles in the lives of millions of people throughout the world. According to a Kaiser Family Foundation study (Rideout, Foehr, & Roberts, 2010), television remains the most frequently used media source among young people in the United States, followed by music, computers, and video games. Media use was up compared to 5 years prior, with adolescents spending nearly 7.5 hours a day with media. Given the use of multiple forms of media, this study estimated that the average young person spends 10.45 hours a day with media.

It is also indisputable that mass media contain many types of content that present, elaborate, and reinforce, in a salient fashion, the schematic ingredients, that is, the "nervosa," of severely disordered eating in girls and women. Unhealthy messages range from idealization of a level of thinness that is unattainable for most females without hurting themselves, to dichotomous, contradictory, and mystifying messages about passion, abandon, and indulgence versus control of hunger, sexuality, and other desires (Levine & Harrison, 2009; Levine & Murnen, 2009).

The question is whether mass media play a causal role in the development of EDs, and, if so, how. In this chapter, after considering relevant outcome variables, we summarize conclusions from large-scale reviews about the relationship between media exposure and EDs. We then turn to a sample of research updates and refinements that have occurred within the last 10 years, including more detailed study of mediating and moderating variables, new media, and

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media literacy. Following a discussion of findings in these areas in relation to gender, we conclude with suggestions for future research.

Media Effects: An Introduction

Effects on What?

In light of current theories of sociocultural influences on disordered eating (López-Guimerà, Levine, Sánchez-Carracedo, & Fauquet, 2010; Smolak & Murnen, 2004; Stice, 1994; Thompson et al., 1999), there is no reason to believe that exposure to mass media directly causes clinical syndromes. Rather, the focus is on the ways in which mass media increase the probability or strength of certain psychopathological processes, negative cognitive-emotional experiences, and cognitive-behavioral tendencies. Individually, these constitute risk factors for EDs. Together, in subsets, they become the components of EDs and therefore often make up the early (prodromal) stages of EDs. The processes include internalization of the unhealthy ideals, social comparison, self-objectification, and activation and elaboration of appearance-based schemas and/or self-ideal discrepancies (Levine & Harrison, 2009). Cognitive-emotional experiences include body dissatisfaction and body shame, negative affect, and anxiety about gaining weight and being or feeling fat. Cognitive-behavioral tendencies include variables such as beliefs about the positive, transformative effects of becoming thin, intentions to diet, severe calorie-restrictive dieting, and shape management via self-induced vomiting or other forms of purging.

What Does Causal Mean?

Drawing on the work of Kraemer et al. (1997) we have argued elsewhere (Levine & Murnen, 2009) that scientific confidence in the statement "media variable X is a *causal* risk factor for outcome Y" is increased to the extent that one can determine from correlational, experimental, and prospective research that there is a consistent, conditional relationship between the variables that can be explained by a theory of the process. Eight specific criteria that help determine the extent to which causality can be determined are listed in Table 29.1.

Size and Scope of Media Effects

Previous reviews of the link between the media and EDs have examined the large number of correlational studies, a large amount of experimental data, some cross-cultural data, some data from media literacy prevention programs, and a handful of prospective studies. Our review of published and unpublished data through 2007 indicated that evidence from research on the relationship between the media and ED meets most, but not all, of the Table 29.1 criteria. Consequently, we concluded that media are a variable risk factor for EDs rather than a causal one (Levine & Murnen, 2009). This means that the variable factor of exposure to and internalization of media messages increases the probability of a range of disordered eating. As noted previously, there is strong evidence that media convey problematic body image ideals and other unhealthy messages, and women and girls are exposed to and pay attention to this information. In addition, cross-sectional survey data and laboratory experiments clearly

Table 29.1 Criteria for establishing engagement with mass media as a causal risk factor for eating disorders.

1. Content	Salient, readily available media messages contain many different types of overlapping content that present, elaborate, and reinforce the schematic ingredients, that is, the "nervosa," of severely disordered eating
2. Exposure	The people at risk for disordered eating are frequently and intensively engaged with media such that they have many opportunities for both active and automatic processing of unhealthy information and incentives
3. Cross-sectional correlation	In a concurrent or retrospective cross-sectional design, extent of exposure to those types of media containing the potential unhealthy messages is significantly and positively correlated with the relevant negative outcome variable(s)
4. Prospective covariation	Relatively higher exposure and more intense engagement with unhealthy mass media messages precedes development of disordered eating
5. Experimental covariation in the laboratory	In a controlled experimental design, extent of exposure to those types of media containing the potential unhealthy messages causes a temporary or state increase in relevant negative outcomes. Conversely, manipulation of protective experiences before or after unhealthy media messages do reduce or eliminate negative effects
6. Experimental covariation in the field	In a controlled experimental design, preventive interventions providing media literacy training or other forms of a critical social perspective eliminate or reduce risk factors, mediating processes, and negative outcomes
7. People's experience of media influence	Surveys and qualitative research show that during periods of risk children, adolescents, and young adults report a desire to look like media "figures," and report the experience of various pressures from the media that correspond to the potentially unhealthy media messages
8. Construct-within-theory validity	Capacity to formulate and test theories that (a) integrate media influence with other causal risk factors and mediating processes in the development of eating disorders; and (b) provide guidance for developing and evaluating prevention and treatment components

Sources: Kraemer et al. (1997) and Levine and Murnen (2009).

support a link between media and both negative body image and disordered eating. It is also evident from the research data that it is the subjective experience of media pressure that influences development of EDs. However, it has not yet been established that media influence is a causal risk factor because the case has not yet been convincingly made that media exposure precedes and predicts the development of EDs, or that reducing media exposure or increasing critical awareness of problematic media ideals helps prevent EDs. If further research indicates that the latter, in particular, cannot be reliably demonstrated, then media influences would have to be considered what Kraemer et al. (1997) call a variable marker.

Table 29.2 summarizes data from 11 other meta-analyses that examined associations between sociocultural variables, including media, and ED symptoms. Four (Cafri, Yamamiya, Brannick, & Thompson, 2005; Groesz, Levine, & Murnen, 2002; Holmstrom, 2004; Stice; 2002) were

Table 29.2 Effect sizes associated with the relationship between media and eating disorders, listed in order of size.

Predictor Variable	*Data Type*	*Gender*	*d*	*k*	*Citation*
Criterion: Internalization of thin ideal					
Media exposure	C,E	F	0.39	23	Grabe et al. (2008)
Criterion: Body dissatisfaction					
Media exposure, control group	E	F,M	–.09	56	Hausenblas et al. (2013)
Media exposure, experimental group	E	F,M	0.03	57	Hausenblas et al. (2013)
Media exposure	E	M	0.14	19	Ferguson (2013)
Media exposure	C	F	0.14	93	Ferguson (2013)
Media exposure	C	M	0.14	25	Ferguson (2013)
Media exposure	P	F	0.18	16	Ferguson (2013)
Media exposure	P	M	0.20	8	Ferguson (2013)
Media exposure	E	F	0.35	140	Ferguson (2013)
Social comparison	C	M	0.54	34	Myers & Crowther (2009)
Awareness of ideal	C	F	0.61	25	Cafri et al. (2005)
Social comparison	C	F	0.83	146	Myers & Crowther (2009)
Perceived pressure media	C	F	1.09	7	Cafri et al. (2005)
Internalization thin ideal	C	F	1.15	18	Cafri et al. (2005)
Criterion: ED symptoms					
Media exposure, ED-NOS	E	F	0.04	7	Ferguson (2013)
Media exposure, restrictive eating	C	M	0.08	11	Ferguson (2013)
Media exposure, bulimia symptoms	C	M	0.14	5	Ferguson (2013)
Media exposure, bulimia symptoms	C	F	0.14	25	Ferguson (2013)
Thin ideal internalization	E,P	F	0.16	4	Stice (2002)
Media exposure, ED-NOS	C	F	0.20	21	Ferguson (2013)
Media exposure, ED-NOS	C	M	0.24	4	Ferguson (2013)
Media exposure, AN	C	F	0.24	16	Ferguson (2013)
Perceived pressure for thinness	E,P	F	0.24	5	Stice (2002)
Body image dissatisfaction	E,P	F	0.26	12	Stice (2002)
Media exposure, bulimia symptoms	E	F	0.30	4	Ferguson (2013)
Media exposure, restrictive eating	E	F	0.30	18	Ferguson (2013)
Media exposure thin ideal	C,E	F	0.30	20	Grabe et al. (2008)
Media exposure, restrictive eating	C	F	0.32	10	Ferguson (2013)

Note. *r* values were converted to *d* with formula $d = 2 \times (r/\sqrt{[1 - r^2]})$. Cohen's (1977) guidelines for effect sizes are that *d* values of 0.20 are small, 0.50 are moderate, and 0.80 are large. *k* = number of samples. Data type: E = experimental design, C = correlational design, P = prospective design, ED = eating disorders, AN = anorexia symptoms, ED-NOS = eating disorder not otherwise specified.

published prior to 2007, and seven were published after Levine and Murnen's (2009) manuscript was submitted (Barlett, Vowels, & Saucier, 2008; Blond, 2008; Ferguson, 2013; Grabe, Ward, & Hyde, 2008; Hausenblas et al., 2013; Myers & Crowther, 2009; Want, 2009). Collectively, these meta-analyses support four conclusions. First, experimental manipulation of media content produces stronger effects than the relationships found in correlational and prospective studies featuring survey-based assessment of dispositional variables. Exposure to idealized media images likely influences immediate body-related thoughts and feelings, which only sometimes get translated into long-term problems. Second, as predictors, specific measures of the processes involved in media influence (e.g., internalization and social comparison) yield stronger effect sizes than do measures of general media exposure, reinforcing the idea that there are important mediating processes. Third, with respect to the ED symptoms examined thus far, restrictive eating warrants closer examination because its effect sizes were the largest associated with media exposure, and these effects were moderate in size even in correlational studies of women. Fourth, effect sizes are generally larger in samples of women compared to men. As we discuss later, media effects are probably stronger among women than men. Nevertheless, it is likely that the variables most susceptible to media influence among men are not being measured, such as increases in body change strategies associated with muscularity concerns.

In several meta-analyses (Ferguson, 2013; Groesz et al., 2002; Want, 2009) the overall small-to-moderate negative effect of media was moderated by whether research participants had pre-existing body image dissatisfaction and/or EDs, with larger effect sizes generally limited to those with pre-existing issues. This appears to indicate that research designed to clarify the media's role as a causal factor should be abandoned. However, for three reasons, we reject this conclusion.

First, Want's (2009) meta-analysis of 75 effect sizes from 47 studies found that for those with high pre-existing levels of appearance concerns, experimental presentations of idealized media portrayals of beauty had a moderate-to-strong negative effect size ($d = -0.52$). Nevertheless, for those with medium or low levels of appearance concerns, the mean weighted effect sizes remained significant at –.37 and –.16, respectively. Second, Grabe et al. (2008) noted that the sizes of the effects found in their meta-analysis were similar to those found in investigations of the relationship between playing violent video games and aggressive behavior (effect size of $r = .28$ for combined correlational and experimental studies). Third, Becker et al. (2011) contend that, "because cumulative and collateral effects that characterize naturalistic media consumption have not been measured, laboratory-based findings very likely underestimate the true effects of media on eating pathology" (p. 43). For example, they measured Fijian girls' perception of their peers' access to technology permitting exposure to Western/global media, and found that this variable independently predicted the girls' own eating pathology (Becker et al., 2011). Another collateral effect is seen in Gurari, Hetts, and Strube's (2006) finding that media presentations of the slender beauty have negative effects on implicit self-image.

Based on the updated reviews (see Table 29.2), we do, however, stand by our conclusion that engagement with mass media is a variable risk factor rather than a causal one (Levine & Murnen, 2009). Consequently, we strongly support Ferguson's (2013) insistence on caution in drawing causal inferences from significant media effects. For example, Hausenblas et al.'s (2013) recent meta-analysis of effect sizes from 33 well-controlled experiments with pre- and post-exposure data found increases in depression and anger, and decreases in self-esteem, in experimental groups but not control groups, suggesting causal effects of the media. Interestingly, they did not find this pattern with body dissatisfaction, a well-established risk factor for eating disorders.

New Research on Mediating and Moderating Processes: The Example of Social Comparison

Mass media affect people differently, depending on factors operating in the transactions between people, the nature and content of the medium, and the context of use (Levine & Harrison, 2009). Understanding the dynamics of the person-context transaction is complicated because the same process may be a mediator and a moderator, such as when habitual activation of thin-ideal internalization or social comparison becomes a stable individual difference that directs selection of media and reactions to exposure. In line with previous research (Table 29.2), Dittmar, Halliwell, and Stirling (2009) found that presentation of thin images to young adult women resulted in negative body-related feelings. As predicted, this contrast effect was mediated by the extent to which the experimental conditions activated discrepancies between one's current self-perception of weight and shape versus one's ideal (anchored in the pursuit of thinness). Further, as predicted, this mediated effect was present only for those young women who already had internalized, as a personality trait, the slender beauty ideal as a personal goal. This pattern of mediation and moderation was found in two independent studies.

Nowhere are the complexities and challenges of understanding mediators and moderators more salient than in the matter of social comparison. "Surveys of elementary school girls, middle school girls, and college women confirm that females who compare themselves to the models in fashion magazines report greater body dissatisfaction and higher levels of disordered eating" (Levine & Harrison, 2009, p. 497; Levine & Murnen, 2009). Conversely, research has shown that women with higher levels of body dissatisfaction (Bessenoff, 2006) and disordered eating (Blechert, Nickert, Caffier, & Tuschen-Caffier, 2009) are more likely to engage in various forms of selective and intensive upward social comparisons. Although the process remains poorly understood, it appears that engaging in the social comparison process for the purpose of self-evaluation produces greater immediate negative effects, while the social comparison motivated by self-improvement eliminates these effects (Halliwell & Dittmar, 2005).

It is hard to imagine how the typical negative (i.e., "contrast") effect of experimental presentations of the slender beauty ideal could come about without some type of comparison of the self to the social ideal. Yet Want's (2009) meta-analysis of experiments found that explicit instructions to pay attention to the attractiveness or thinness of the model, which should facilitate a conscious social comparison, actually reduced the contrast effect significantly ($d = -0.24$), compared to no instructions ($d = -0.37$) or instructions to pay attention to some other aspect of the stimulus (e.g., how creative the advertisement is; $d = -0.47$). Want (2009) offered the testable hypothesis that the social comparison process involves two stages: a nonconscious automatic comparison, followed by a conscious elaboration, including the potential for defensive counterarguments that mitigate but do not eliminate the contrast effect. Thus, no instructions or instructions to pay attention to elements of the image unrelated to beauty activate the first process but do not encourage the second.

Wan, Ansons, Leboe, and Smeesters (2009) also hypothesized that automatic, implicit (nonconscious) social comparisons with media ideals are harmful to body satisfaction and self-perception, whereas explicit, more conscious social comparison facilitates counterarguments with media ideals, leading to more positive self-perceptions as well as increased body satisfaction. In support of this hypothesis, Wan et al. (2009) found that participants primed to think positively about fashion models (i.e., the idealized images) and to make implicit social

comparisons had the most negative self-perceptions, whereas participants in the explicit social comparison conditions were not affected by whether positive or negative model characteristics were primed. The proposal that there are two parallel interacting systems—one that is implicit, automatic, and impulsive, and another that is explicit, conscious, and reflective—deserves more attention from media effects researchers in the ED field. This theory has been empirically supported and useful in the general field of social cognition (Forehand, Perkins, & Reed, 2011). Its initial application to understanding how body image may be affected by exposure to thin or heavy models reveals that, even during automatic processing, negative or positive effects are determined by the nature of the standard and the type of knowledge accessed (Smeesters & Mandel, 2006).

The theory that two information processing systems, one implicit and the other explicit, are operating in social comparison is also consistent with the findings of a study by Tiggemann and Polivy (2010). Participants were asked questions that implicitly encouraged them to engage in social comparison with the models in media images, based on either appearance or intelligence. Compared to the control condition in which participants paid attention only to the advertisements' features, both manipulations led to decreases in mood compared to the control condition, but neither affected body dissatisfaction. Moreover, regression analyses indicated that, regardless of condition and consistent with previous work, individual differences in the tendency to make what were typically "upward" appearance-related social comparisons were positively related with increases in negative mood and body dissatisfaction following exposure to the slender beauty ideal (Thompson et al., 1999). Conversely, tendencies to make "downward" comparisons to the model's intelligence, education, and genuineness were negatively related to body dissatisfaction. This finding, coupled with the fact that there was some appearance processing from participants in all conditions, despite the manipulation, led the researchers to conclude that a focus on intelligence might require less automatic, more deliberate processing.

Thinking about Tiggemann and Polivy's (2010) findings in terms of the two-system theory of social cognition (Forehand et al., 2011), it may be that conscious (i.e., postautomatic) comparison with a highly dissimilar person (i.e., a very thin, glamorous fashion model) activates self-related dissimilarity information, heightening a contrast effect. Two recent studies have found the level of identification with models to be highly predictive of body dissatisfaction when the model chosen represented the thin ideal (Eyal & Te-eni-Harari, 2013; Shorter, Brown, Quinton, & Hinton, 2008). Conversely, it may be that in the Tiggemann and Polivy (2010) study the increased probability of thinking of one's self as more similar (or even superior) in intelligence to the model selectively activates more positive self-related information.

Want's (2009) two-stage theory is also supported by an experiment in which Tiggemann, Slater, Bury, Hawkins, and Firth (2013) examined whether the impact of exposure to idealized images on body dissatisfaction was moderated by an intervention highly recommended by media activists: labels warning that the image of slender beauty "has been digitally altered to smooth skin tone and slim arms and legs" (p. 47). The warning labels were found to have no effect on this link, leading Tiggemann et al. to conclude that social comparison seems to be a fairly automatic process. Ata, Thompson, and Small (2013) also found that neither a disclaimer nor a health warning moderated the traditional contrast effect of an increase in "state" body dissatisfaction in response to exposure to the slender beauty ideal (Groesz et al., 2002; Want, 2009). Future research should examine more about the automaticity of the process, and how upward social comparisons based on appearance might be interrupted.

New Media

Much of the cross-sectional data incorporated into meta-analyses and other reviews addresses the influence of appearance-oriented magazines and television on body dissatisfaction and EDs. Recently, theorists have been concerned about new forms of media such as the Internet. Use of new media by children and adolescents is of particular concern for some of the same reasons these media are so attractive and exciting (Brown & Bobkowski, 2011). New media are readily available, and the content and modes of presentation are basically unregulated and uncensored. New media also encourage individual choice in level of indulgence of one's interests, whether they be "getting a sexy makeover," becoming "fit," or even engaging in weight-and-shape-based "cyber bullying" and intimidation. In addition, some forms of new media promote a type of potentially unhealthy social interaction characterized by immediate, anonymous (or pseudonymous), and unreflective responding.

Tiggemann and Miller (2010) examined the extent to which Australian high-school girls consumed specific magazine, television, and Internet sources emphasizing appearance. Uses of each type of media were positively correlated with one another, supporting the idea that some girls are immersed in a media culture saturated with appearance-focused messages (Jones, Vigfusdottir, & Lee, 2004). Internet appearance exposure, particularly during use of social networking sites, was more highly correlated with internalization of the thin ideal, drive for thinness, and weight dissatisfaction than was television or magazine appearance exposure. As predicted by Thompson's tripartite model of sociocultural influences (Thompson et al., 1999), the link between Internet exposure and ED attitudes was partially mediated by thin-ideal internalization and appearance-based social comparison.

Tiggemann and Miller (2010) concluded that the Internet is an important medium to study, since it requires more choice and offers more active engagement than other forms of media. Tiggemann and Slater (2013) also found that Internet exposure was positively correlated with internalization, body surveillance, and drive for thinness in a large sample of Australian high-school girls ($N = 1{,}084$). Moreover, most of the girls reported using Facebook (an average of 1.5 hours per day), which offers many opportunities for social comparison. On the other hand, Bair, Kelly, Serdar, and Mazzeo's (2012) study of female undergraduates in the United States did not find differences in the size of relationships between body dissatisfaction and exposure to appearance messages on the Internet versus other sources. Possible age effects in the relationship between Internet exposure and body-related issues deserve further study. In the process, researchers should also develop measures that can test the hypothesis that newer forms of media lead to more engagement with media models and messages.

Pro-ED Websites

The typical Pro-ED website is developed by a woman with an ED who presents her anorexia nervosa (AN) or BN as a "lifestyle" or an "identity" to be proud of, rather than a debilitating psychiatric disorder (Norris, Boydell, Pinhas, & Katzman, 2006). Indeed, many professionals, parents, and young people are horrified by the glorification of symptomatology featured in popular pro-anorexia ("pro-ana") or pro-bulimia ("pro-mia") websites. Lewis and Arbuthnot (2012) reported that in 2011 pro-ED search terms were sought out more than 13 million times. These extreme "spaces" are plentiful, salient, and easily accessed. However, they should not blind us to the numerous other pro-ED websites that provide potentially helpful information about topics such as positive body image, healthy eating, harm reduction

while deciding whether or not to get help, stigma reduction, and the process of recovery, including the role of social support (Juarascio, Shoaib, & Timko, 2010).

On the other hand, if these sites, like the prototypical pro-ana and pro-mia sites, provide "triggering images" and actively encourage disorders by offering "tips and tricks" and other forms of inspiration for pursuing thinness ("thinspo"), they are potentially harmful (Lewis & Arburthnot, 2012). Borzekowski, Schenk, Wilson, and Peebles (2010) examined 180 websites active in 2007 and classified 25% in the highest harm categories, while 28% were considered "low harm" (but not necessarily helpful). Analysis of "tips and tricks" suggestions offered by pro-ana websites found that the most frequent pointers concerned dieting and calorie restriction, distracting oneself from hunger, deceiving healthcare providers, and burning calories (Harshbarger, Ahlers-Schmidt, L. Mayans, D. Mayans, & Hawkins, 2009).

Use of pro-ED websites is positively correlated with severity of ED symptoms (Harper, Sperry, & Thompson, 2008; Wilson, Peebles, Hardy, & Litt, 2006). It is difficult to know the causal nature of the association, though. College women exposed to a pro-ana site reported significantly lower affect, social self-esteem, and appearance self-efficacy than control participants (Bardone-Cone & Cass, 2007). Women in the pro-ana exposure condition also described their body mass indices (BMIs) as higher, which was not the case. Future research should look more closely at effects of exposure and involvement in pro-ED websites, among those with and without EDs. Even though efforts have been made to ban these sites, they continue to attract users, suggesting they serve a function. In the meantime, placement of warning text on pro-ana sites in The Netherlands has proven somewhat helpful in thwarting intended visitors (Martijn, Smeets, Jansen, Hoeymans, & Shoemaker, 2009).

Media Literacy

Advances in media literacy-based prevention programs can help establish a causal relationship between media and ED (Criterion 6, Table 29.1). Media literacy is a set of knowledge, attitudes, and skills that allow people critically to evaluate the nature of the mass media and one's relationship to them (Levine & Harrison, 2009). Theory and research converge in pointing to the value of this type of critical social perspective (see Chapters 19 & 27) in preventing disordered eating and EDs in females, especially if it emphasizes the power of multiple sociocultural factors (including mass media) to promote and reinforce a slender beauty ideal (Levine, 2014; Piran, 2010; see also Chapters 43–45).

With respect to prevention, the age of participants may turn out to be an important moderator variable. Using rigorous experimental methodology, Wilksch, Durbridge, and Wade (2008) evaluated the Media Smart program (see Chapter 45). This set of eight 50-minute media literacy lessons (e.g., analysis of advertising and methods of consumer activism) had no significant effects on shape and weight concern, internalization of media beauty ideals, dieting, and self-esteem of grade 10 Australian girls (mean age = 15) at 3-month follow-up, as compared to a control group. However, another well-conducted experiment by Wilksch and Wade (2009) demonstrated that a version of the same Media Smart curriculum, administered to grade 8 girls and boys (ages 13–14), produced desirable outcomes (with small to moderate effect sizes) for body dissatisfaction, weight and shape concern, dieting, ineffectiveness, and depression. These positive prevention effects were maintained 2.5 years after baseline assessment.

Media literacy programs may become even more important due to the increased rates of obesity among children, fueled in part by media messages promoting foods high in fat content

and low in nutritional value. Media literacy is one vitally important source of common ground for prevention specialists in the fields of EDs, obesity, and health promotion (Haines & Neumark-Sztainer, 2006).

Gender Differences

Although males have been less frequently included in studies of the link between media and EDs, meta-analyses indicate that gender tends to moderate effects, with larger effects among females (Hausenblas et al., 2013; Myers & Crowther, 2009). Regardless, since at least the 1970s there has been increased idealization of a lean and unrealistically muscular physique for male bodies in the media (Pope, Phillips, & Olivardia, 2000). This has been accompanied by increased rates of body dissatisfaction among males (Adams, Turner, & Bucks, 2005), suggesting that engagement with mass media also figures prominently in the body image issues and eating concerns of boys and men (see Chapter 37).

Studies of media effects on ED symptoms in males compared to females were subjected to meta-analysis by Ferguson (2013). The strength of correlations between media exposure and symptoms of BN or eating disorders not otherwise specified (ED-NOS) is similar in males and females, whereas associations with restricted eating appear greater among women compared to men. Media images likely exert different influences on women versus men in terms of body change strategies. While women are inclined and encouraged to think of restrictive eating in order to reduce body fat to emulate a thin ideal, men are socialized to manipulate exercise to create the muscularity associated with their ideal. Despite some differences in associations with outcome measures, models that link sociocultural factors, including mass media, with the risk factor of body dissatisfaction and with EDs in females have predictive validity in studies of males and females (McCabe & Ricciardelli, 2004; Smolak, Murnen, & Thompson, 2005; Smolak & Stein, 2010). Yet, the associations are likely to be stronger in women compared to men, as discussed next.

Objectification Theory

Objectification theory is an evidence-based approach to conceptualizing media effects on body image and disordered eating, including gender differences (Fredrickson & Roberts, 1997). According to this model, due to their subordinate economic, political, and social status in Western culture, women are frequently treated as sexual objects in the media and through interpersonal encounters with men and with women. This ubiquitous objectification leads them to internalize not only the slender beauty ideal, but also the "male gaze," that is, the very process of objectification. The resulting self-surveillance and self-objectification creates body shame, which increases the risk of EDs, depression, sexual dysfunction, and nonsuicidal self-injury. Some theorists have argued that because gay men are subjected to the male gaze, they will care more about their appearance (Hospers & Jansen, 2005) and pay more attention to media messages. Carper, Negy, and Tantleff-Dunn (2010) found that gay men (see Chapter 23) perceived more pressure from the media, which was positively correlated with their scores on drive for thinness and anxiety about appearance.

Content analyses of U.S. media show that women are underrepresented compared to men; are highly likely to be depicted as passive sexual objects; and are likely to be portrayed in

gender-stereotyped, domestic roles (Collins, 2011). The "heterosexual script" in American culture in general and in mass media in particular portrays men as obsessed with sex and with objectifying women, so the importance of women's appearance is paramount (Kim et al., 2007). Female characters across various types of media are shown as more attractive, thinner, more sexualized, and younger than male characters (Greenwood & Lippman, 2011).

This means that mass media establish, promote, and reinforce body image ideals for women and men that strengthen their stereotyped roles. The ideals and the ways they are promoted also perpetuate a patriarchal culture in which the thin, sexy adolescent girl and the thin, sexy adult woman of any age are viewed as an appropriate and ultimately submissive sex object, while the lean, muscular man is seen as dominant (Murnen & Don, 2012). Beginning at an early age, and certainly well before adolescence, girls are exposed to various forms of mass media that make it very clear that caring about attractiveness, specifically about thinness, is part of the normative gender role expectations for females (Mahalik et al., 2005). In contrast, while the powerful, muscular male shape embodies male gender role norms, such as dominance and prowess at sports, there are other ways for men to express dominance and prowess, such as inheriting a tremendous amount of money, high status work roles, and hard-earned, competitive achievement. In fact, it is inadvisable for a man to be too concerned with his appearance, for fear of being stigmatized as feminine or homosexual.

Once again, girls and women are more vulnerable to media messages about body ideals because the messages are very prevalent, the messages reinforce and extend sexual objectification, and the messages provide information very relevant, if not critically important, to women's social and financial success (see Chapters 19 & 27). Recent studies show that individuals in the United States are much more likely to be confronted with images of the female body ideal compared to the male body ideal (Buote, Wilson, Strahan, Gazzola, & Papps, 2011). And this ideal is bound up with weight and shape. Grieve and Bonneau-Kaya (2007) found that women's magazines were much more likely to focus on weight loss than men's magazines were to focus on weight gain (or weight loss).

Conclusions and Future Directions

Interpretation of the meta-analytic data in Table 29.2, using the criteria for determination of a causal risk factor that are listed in Table 29.1, continues to support five contentions from our previous reviews. First, engagement with mass media constitutes a variable risk factor, not a causal risk, for the psychopathology and symptoms of EDs. It remains the case that more longitudinal research, including media-literacy-based prevention studies, is needed to strengthen our ability to determine whether or not media exposure and/or certain types of engagement are a causal risk factor (Levine & Harrison, 2009; Levine & Murnen, 2009).

Second, in multiple ways, mass media definitely model, elaborate, and reinforce the raw materials for negative body image, weight and shape preoccupation, self-objectification, fear of fat, and other aspects of the "nervosa" associated with AN, BN, and ED-NOS. Future research would do well to continue clarifying the main effects of, and interactions between, mediators and moderators of these influences. Slater's (2007) model of "reinforcing spirals" (p. 281) of the reciprocal influence of media selection and media effects could prove useful in this effort. The example of social comparison processes points out the value for the EDs field of fundamental theory and research in the general field of social cognition (Forehand et al., 2011).

Third, the mass media's negative potential, which may well be magnified in as yet poorly understood ways by the power of the Internet, is particularly unhealthy for females who have internalized the beauty ideal of slenderness and youth, who tend to make upward social comparisons, and who already have high levels of weight and shape concerns. At present, these media interactions, as well as the moderating effects of gender, are not captured very well by current multifactor sociocultural theories (Stice, 1994; Thompson et al., 1999). Fourth, and along the same lines, the effects of media need to be understood in the context of other sociocultural factors, most notably family and peers (see Chapters 26 & 31). Finally, media literacy, as a multifaceted and interactive way of developing a critical social perspective, holds a great deal of promise for prevention. Media literacy research, including creation of new media for health promotion, is necessary to clarify the causal power of mass media in regard to EDs. This type of research also has the potential to illuminate what groups and individuals can do to limit negative media influences while improving use of media for promoting public health.

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Neurochemical Components of Undereating and Overeating

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Advances in physiological testing and neuroimaging techniques have allowed researchers to study various biological mechanisms that may be involved in the development and/or maintenance of physical and psychological disorders. While many biological substrates have been implicated in the pathology of eating disorders (EDs; see Chapter 17), serotonin, dopamine, and opioids have received a considerable amount of attention within the field, as alterations in each of these neurotransmitter systems have been reported among individuals with EDs. This chapter discusses many of the research findings that have been gathered by studying the potential role of these neurotransmitter systems in EDs that involve undereating or overeating.

Undereating

Undereating, or consuming fewer calories and nutrients than necessary to maintain homeostasis, is seen among individuals with anorexia nervosa (AN), although it is not limited to this population and can also be seen among individuals with bulimia nervosa (BN) as well as those diagnosed with eating disorder not otherwise specified (ED-NOS; see Chapters 2, 3, & 8–11). Although AN is reported to affect less than 1% of the population (Hudson, Hiripi, Pope, & Kessler, 2007; see also Chapter 5), this disorder has been associated with a number of psychological comorbidities, including depression and anxiety (Hudson et al., 2007; see also Chapters 15 & 54), even in those with subclinical AN symptoms (Touchette et al., 2011). In addition to psychological functioning, AN can pose serious health problems, including hormonal disturbances, osteoporosis, and cardiovascular abnormalities, such as bradycardia (Sharp & Freeman, 1993; see also Chapters 14 & 52). AN is also associated with a high mortality rate (Birmingham, Su, Hlynsky, Goldner,

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& Gao, 2005). There remains a need for the development of effective treatments for this disorder. This section will focus primarily on associations that have been found between AN and alterations in neurochemistry, a topic that may aid in informing our understanding of certain symptoms that are observed in patients with AN, as well as in guiding our inquiries regarding potential pharmacological treatment approaches for this disorder (see Chapter 59).

In addition to studying patients with AN, researchers have sought to understand the biological effects of food restriction using laboratory animal models. Restricting food intake can produce weight loss, thus allowing for features of AN to be modeled. In activity-based anorexia (ABA) model, rats are placed on a restricted diet and given access to a running wheel (Routtenberg & Kuznesof, 1967). Under these conditions, rats show a dramatic increase in running activity without sufficient compensatory increases in food intake, thus resulting in extreme weight loss. The effects of this model are so severe, in fact, that if experimenters do not intervene, ABA rats will die. Like AN, ABA is seen in both male and female rats, and female ABA rats show a disruption in their estrous cycles, similar to the former diagnostic criterion (American Psychiatric Association, 2000) of amenorrhea seen in females with AN.

Another characteristic seen in both ABA subjects and patients with AN is hypothermia, which has led to the theory that hyperactivity may serve as a mechanism to promote thermoregulation (Carrera et al., 2012). An alternative theory used to explain this behavior proposes that starvation and hyperactivity may activate brain reward systems, perhaps laying the foundation for an addiction to develop (Marrazzi & Luby, 1986), as discussed in greater detail below. Likewise, some investigators have hypothesized that increased physical activity may be an adaptive response to food restriction as, in the wild, hungry animals would most likely need to move in search of food (Pierce, 2001). These theories, along with others, have led to further research and discoveries regarding the ABA model of AN; however, ABA is still not fully understood. Additionally, it is important to note that these theories may not be entirely independent of one another. Findings from research using this animal model, as well as findings from numerous clinical studies, inform the following three sections, which discuss key neurochemical systems that have been studied in relation to undereating: the serotonergic, dopaminergic, and opioidergic systems.

Serotonin and AN

Serotonin (5-hydroxytryptamine; 5-HT) affects many physiological and behavioral factors, including mood regulation, appetite, and impulse control. Several lines of research have also provided evidence for a role for 5-HT in EDs. Based on findings of decreased cerebrospinal fluid (CSF) 5-hydroxyindoleacetic acid (5-HIAA), a major metabolite of 5-HT, in ill AN participants and significantly increased 5-HIAA levels in individuals who have recovered from AN (Kaye, 2008), researchers have hypothesized that increased 5-HT activity may predate the development of AN. However, it is difficult to determine whether certain characteristics precede or result from AN (see Chapter 67), as prospective studies of this disorder are challenging, particularly due to the low prevalence of this disorder in the general population (Kaye, Gwirtsman, George, & Ebert, 1991). Additional data come from Hassanyeh and Marshall (1991), who found blood 5-HT levels to be considerably lower among a subset of AN patients with the most weight loss but no other comorbidity. In contrast, individuals with both AN and an affective disorder were shown to have high blood 5-HT levels. It is important to note that in this particular study the participants' body weights varied, and many were higher than 85% normal body weight (Hassanyeh & Marshall, 1991). While mean values of

blood 5-HT were not statistically different between AN patients and controls in another study, AN patients were found to have greater variability in terms of 5-HT levels. Additionally, this study reported a positive correlation between anxiety and 5-HT levels among members of an AN subgroup characterized as more impulsive (Askenazy et al., 1998).

Use of neuroimaging techniques to study individuals with AN has also revealed alterations in the serotonergic system. When testing for differences between a population of interest, that is, patients with AN and normal controls, neuroimaging studies often measure binding potential at certain brain receptors. Binding potential is often assessed by using a chemical that competes with a neurochemical of interest, such as serotonin, to bind to its receptors. For example, [18F] altanserin has been used in neuroimaging studies because it specifically targets 5-HT2A receptors, a type of 5-HT receptor. Altered binding potential may suggest two possibilities. For example, decreased binding potential at 5-HT2A receptors may indicate that individuals with AN have more serotonin that may have bound to the receptors, blocking [18F]altanserin from binding. However, decreased binding potential might also occur if individuals with AN have fewer 5-HT2A receptors to which to bind. It has also been proposed that, over time, increased serotonin may result in a downregulation of such receptors. Thus, it is unclear whether such findings indicate an alteration at the neurochemical or receptor level; however, they do suggest a strong relationship between individuals with AN and the serotonergic system.

A number of studies have reported decreased binding potential at 5-HT2A receptors among ill and recovered individuals with AN, though this finding has not been universally supported (Kaye, Wierenga, Bailer, Simmons, & Bischoff-Grethe, 2013; Sigurdh, Allard, Spigset, & Hagglof, 2013). 5-HT2A binding potential has been positively associated with harm avoidance in ill AN as well as in ill and recovered AN patients with binge-purge subtype (Bailer et al., 2004, 2007). 5-HT2A binding potential has also been negatively associated with novelty seeking and drive for thinness among recovered AN patients with the binge-purge subtype (Bailer et al., 2004). In contrast to the findings for 5-HT2A, neuroimaging studies have revealed increased binding potential at 5-HT1A receptors in ill and recovered AN patients (Bailer et al., 2007).

The intake of certain foods, especially those containing tryptophan, a 5-HT precursor, may induce anxiety and dysphoric symptoms, as often more 5-HT receptors are produced in response to ingested 5-HT metabolites and precursors. It is possible that this results in hypersensitivity to even smaller amounts of 5-HT. Thus, upon a break in starvation and the ingestion of 5-HT metabolites, the increased number of receptor sites may become stimulated, leading to anxiety, further reinforcing food restriction. In individuals suffering with AN, as well as those who have recovered, reducing tryptophan has been shown to reduce anxiety (Kaye et al., 2003).

Preclinical research has also provided insight into the effects of food restriction, reduced body weight, and increased exercise on 5-HT. For example, whereas running has been shown to increase 5-HT and 5-HIAA in the medial basal hypothalamus of food-deprived underweight rats (Broocks, Schweiger, & Pirke, 1991), decreased levels of extracellular 5-HT were found in the nucleus accumbens (NAc) of ABA rats (Verhagen, Luijendijk, Korte-Bouws, Korte, & Adan, 2009). Such findings may be explained by differences in experimental protocol and/or highlight the importance of the brain region studied.

Evidence of alterations in 5-HT among individuals with AN has led researchers to explore the therapeutic effects of drugs known to target the serotonergic system. Atchley and Eckel (2006) have found that the typical increases in physical activity and weight loss observed in ABA rats are reduced when these rats are administered 8-OH-DPAT, which activates 5-HT1A autoreceptors and suppresses 5-HT activity. However, this treatment did not entirely abolish the effects of ABA and many of the treated rats reached the body weight loss criterion, though in a longer period of time. Klenotich et al. (2012) found that, while

it did not affect survival time in a modified version of ABA (with 6 h/day food access), treatment with fluoxetine, a selective serotonin reuptake inhibitor (SSRI), did increase food intake in ABA mice as well as reduce the physical activity often observed prior to food access, a phenomenon referred to as food anticipatory activity (FAA). This study also revealed that Balb/cJ mice demonstrate a greater susceptibility to ABA compared to A/J mice, which is of note since Balb/cJ mice also show reduced 5-HT and 5-HIAA compared to A/J mice. Researchers have also investigated the effects of the 5-HT agonist, fenfluramine, on the progression of ABA, but mixed findings preclude any certain conclusions. While one study did not find an effect on ABA rats (Hillebrand, Heinsbroek, Kas, & Adan, 2006), Atchley and Eckel (2005) found that rats treated with this agent lost weight faster than saline-treated and pair-fed rats, providing support for the theory that increased 5-HT activity may influence AN.

Dopamine and AN

Dopamine (DA) has also been implicated in the pathophysiology of AN. Similar to CSF 5-HT metabolite levels detected in ill AN participants, the DA metabolite, homovanillic acid (HVA) has been shown to be reduced in individuals with AN. However, unlike 5-HIAA, HVA appears to remain reduced in individuals with AN even following recovery (Kaye, 2008), suggesting that this may be indicative of a trait, rather than a state-related characteristic. As mentioned earlier, it is difficult to determine whether such findings suggest premorbid characteristics or demonstrate effects of the disorder. A neuroimaging study has also found increased binding potential at two types of dopamine receptors (D2 and D3) in the antero-ventral striatum, a brain region associated with reward, in those who have recovered from AN compared to controls (Frank et al., 2005).

Preclinical research using food-restricted animals has found levels of extracellular striatal DA to rise dramatically in response to food provided on an interval schedule (Church, Justice, & Neill, 1987). Verhagen, Luijendijk, Korte-Bouws, et al. (2009) also found increased extracellular DA in the NAc of ABA rats during feeding. Results from our laboratory have found increased NAc DA in ABA rats when running (Avena et al., 2013). However, this increase in DA seems to be regional, as Broocks, Liu, and Pirke (1990) reported decreased DA content in the medial basal hypothalamus. DA antagonists (pimozide and *cis*-flupenthixol) have been shown to decrease running activity in ABA rats (Lambert & Porter, 1992; Verhagen, Luijendijk, Hillebrand, & Adan, 2009).

Opioids and AN

Interest in better understanding how opioid systems may be involved in ED pathology was motivated by findings from animal studies showing opioid peptides to modulate feeding behavior (Baile, McLaughlin, & Della-Fera, 1986), and by the theory that EDs might have an addictive component driven by altered opioid activity (Marrazzi & Luby, 1986). While clinical research has revealed abnormalities in opioid systems among individuals with AN, the findings are mixed. Kaye, Pickar, Naber, and Ebert (1982) found CSF opioid activity to be significantly increased in underweight female patients with AN compared to women who had previously had AN but had been restored to 95% normal body weight, control women with no history of AN, as well as the same subjects when weight restored, indicating that this may be a state-related feature of AN. In light of such findings, Marrazzi and Luby (1986) proposed what is referred to as the auto-addiction opioid model of AN. This model states that higher levels of opioids are released by dieting, which may lead to an addiction perpetuating this behavior.

Further study of specific opioid peptides found normal CSF levels of the opioid peptide dynorphin in AN patients (Lesem, Berrettini, Kaye, & Jimerson, 1991). In contrast, significantly reduced CSF levels of beta-endorphin have been reported among underweight women with AN (Kaye et al., 1987). However, normal CSF levels of beta-endorphin immunoreactivity have also been found in AN women compared to controls (Gerner, Sharp, & Catlin, 1982). While not universally supported (Baranowska, 1990), a number of studies have reported increased plasma beta-endorphin levels in AN patients compared to controls (Brambilla et al., 1985; Melchior et al., 1990; Tepper, Weizman, Apter, Tyano, & Beyth, 1992). Furthermore, fasting has been associated with increased plasma beta-endorphin levels in a nonclinical sample (Komaki et al., 1990). It should be noted that levels of beta-endorphin in CSF and plasma may be unrelated. Treatment with the opioid antagonist naloxone or its longer-acting form, naltrexone, has shown promising results in AN patients (Marrazzi, Bacon, Kinzie, & Luby, 1995; Moore, Mills, & Forster, 1981), although such findings have been contradicted by a case report (Krahn, Dequardo, & Gosnell, 1990).

Preclinical research has found that in response to morphine activation of the opioid system, certain strains of mice decrease food intake and some also increase physical activity, contrasting with increased food intake seen in other species under these conditions. This finding prompted some researchers to view such mice as possible models of humans prone to AN (Marrazzi et al., 1990). ABA rats have been found to have elevated basal plasma beta-endorphin levels compared to weight-matched as well as exercise controls (Aravich, Rieg, Lauterio, & Doerries, 1993) and have been shown to exhibit withdrawal symptoms when administered naloxone (Kanarek, D'Anci, Jurdak, & Mathes, 2009).

Overeating

Overeating, defined here as repeatedly consuming excess calories, is seen in multiple clinical and subclinical populations. We will focus on overeating within the context of binge eating (which may or may not result in increased body weight). Recent cross-national data show average lifetime prevalence rates of 1% and 1.9% for BN and binge eating disorder (BED), respectively, and demonstrate the psychological and physical comorbidities associated with these two disorders (Kessler et al., 2013). Additionally, many participants with BN and BED report role impairment as a result of their disorder (Kessler et al., 2013; see also Chapter 62). Eating behaviors associated with BED have also been linked to obesity (Davis et al., 2009; Stunkard, 1959; see also Chapter 65). As a result, the neurobiological underpinnings of binge eating and pharmaceutical treatments for this behavior have been increasingly studied.

In addition to clinical studies, researchers have developed several animal models of this behavior. Through this work, a number of key factors related to the occurrence of binge eating have been identified, including food restriction, exposure to various stressors, and access to palatable food. For example, rats will binge eat when a stressor, such as space restriction or a cold swim test, is introduced following a period of food restriction (Inoue et al., 1998; Vaswani, Tejwani, & Mousa, 1983). Other investigators have observed binge eating when simply offering animals palatable food on a limited access schedule (e.g., 2 h daily or 2 h/day three times a week; Corwin et al., 1998). Along with identifying some of the variables that may influence this behavior, such models serve as an additional means to study the various brain mechanisms affected by or involved in binge eating behavior.

Serotonin and Binge Eating

Several studies have demonstrated a relationship between 5-HT and binge eating behavior. Increased binge frequency (more than two times per day) has been associated with reduced CSF levels of 5-HIAA in BN patients in some (Jimerson, Lesem, Kaye, & Brewerton, 1992) but not all studies (Kaye et al., 1990). In contrast, recovered BN patients have been reported to have increased CSF 5-HIAA levels compared to controls (Kaye et al., 1998), perhaps indicating a trait-related feature of BN. Binding potential at 5-HT2A receptors was found to be normal in acutely ill (Goethals et al., 2004) but altered in recovered BN individuals (Kaye et al., 2001). Kaye et al. (2001) proposed that this latter finding may be related to the increased CSF 5-HIAA levels also found among recovered BN patients, which may be evidence of increased 5-HT levels that may affect receptor density. Increased binding potential at the 5-HT1A receptors has been shown in both the ill and recovered states (Bailer et al., 2011; Tiihonen et al., 2004).

Reduced 5-HT transporter (5-HTT) availability in the thalamus and hypothalamus has been found among ill BN individuals, with individuals with longer illness durations showing less 5-HTT (Tauscher et al., 2001). Likewise, alterations in 5-HTT availability have been reported among individuals who have recovered from BN (Pichika et al., 2012). A reduction in 5-HTT binding has also been shown in the midbrain of obese individuals with BED compared to obese controls (Kuikka et al., 2001). Interestingly, a follow-up study by the same group found that 5-HT transporter binding was increased in the midbrain of these same participants following treatment with group psychotherapy and fluoxetine, when binge eating was reduced (Tammela et al., 2003). Further support for a relationship between 5-HT and binge eating comes from treatment studies. 5-Hydroxytryptophan (5-HTP), the intermediate metabolite for the amino acid necessary in 5-HT biosynthesis, reduces binge eating, causes a significant decrease in food intake, and a subsequent weight loss (Birdsall, 1998). In a review of pharmacologic treatments of binge EDs, Carter et al. (2003) provide evidence for the usefulness of SSRIs in the treatment of BED.

The relationship between binge eating and 5-HT has also been studied using animal models. One model of binge eating including a history of caloric restriction coupled with intermittent access to palatable food was associated with a 71% reduction of 5-HT in the rat medial prefrontal cortex (Chandler-Laney et al., 2007). Not only do these binge-promoting conditions reduce 5-HT levels, but they also appear to promote susceptibility to the anorectic effects of 5-HT (Corwin, Avena, & Boggiano, 2011). Fluoxetine seems to exert a stronger effect among rats with both a history of food restriction and sporadic extended access to palatable food relative to rats with a history of food restriction but access to palatable food every day (Chandler-Laney et al., 2007). The effect of serotonergic compounds on eating behavior may be also enhanced by stress, as studies have revealed that stress exacerbates sensitivity to the satiating effects of fluoxetine in rats with a history of dieting (Placidi et al., 2004).

Dopamine and Binge Eating

Evidence from clinical and preclinical studies suggests a role for DA in the binge eating of individuals with BN and BED (Bello & Hajnal, 2010). Lower CSF levels of HVA have been associated with increased binge frequency among individuals with BN (Kaye et al., 1990; Jimerson et al., 1992). Recovered individuals with BN, in contrast, have been found to have normal CSF levels of HVA (Kaye et al., 1998), perhaps indicating that the former finding of reduced HVA was state-related. DA transporter availability has also been found to be reduced

in the striatum of individuals with BN (Tauscher et al., 2001). Additionally, decreased DA receptor binding has been reported among BN patients. DA release has also been shown to be significantly decreased in the putamen of BN patients, and binge eating frequency has been related to striatal DA release. Specifically, greater binge frequency in the past month was associated with less striatal DA release (Broft et al., 2012). Interestingly, when administered methylphenidate, which blocks DA reuptake transporters, obese individuals with BED showed greater increases in DA in the caudate and putamen when seeing, smelling, and tasting food compared to obese individuals without BED. Further, higher scores of binge eating were associated with more pronounced increases in DA release in the caudate (Wang et al., 2011).

Animal models have provided further evidence of a relationship between binge eating and DA. Rats with a history of caloric restriction and intermittent access to palatable food were found to have a 58% decrease in DA in the medial prefrontal cortex. Interestingly, rats that did not have a history of food restriction showed a positive association between the turnover of DA and 5-HT in the NAc that was not seen in rats with a history of food restriction (Chandler-Laney et al., 2007). When given limited daily access to sucrose and rodent chow (12 h daily access followed by 12 h deprivation) we found that rats release DA in the NAc with repeated sugar binge episodes (Rada, Avena, & Hoebel, 2005); this effect is seen even when the animals only taste the sucrose via sham feeding (Avena, Rada, Moise, & Hoebel, 2006). This pattern of DA release in response to binge-like consumption of sucrose is unlike the normal waning of DA release in response to food that occurs when the food is no longer novel (Bassareo & Di Chiara, 1999). Instead this resembles the effect of drugs of abuse (Avena & Hoebel 2012; Avena, Rada, & Hoebel, 2008a). Fat also results in the release of DA, though it remains to be determined whether fat can elicit DA release with repeated exposure (Liang, Hajnal, & Norgren, 2006). Increases in D1 receptor binding in the accumbens core and shell and decreases in D2 receptor binding in the dorsal striatum have also been noted in response to binge consumption of glucose on this limited daily access schedule (Colantuoni et al., 2001). Interestingly, when rats maintained on this schedule are subsequently food restricted to reduce body weight, a heightened dopaminergic response to sucrose consumption is observed (Avena, Rada, & Hoebel, 2008b).

Corwin and Wojnicki (2009) found the D2 receptor antagonist, raclopride, to selectively attenuate binge consumption of fat precipitated by limited access, to have no effect on ad libitum intake of the same food, and to attenuate sucrose intake regardless of schedule of availability. Further, this D2 receptor antagonist differentially affects binge-like intake of varying sugar-fat mixtures, with maximum suppression of intake observed when a moderate amount of sucrose (10%), versus 3.2% or 32%, is added to vegetable shortening (Wong, Wojnicki, & Corwin, 2009). Such findings suggest that the effectiveness of a pharmacologic intervention targeting the DA system may, to some extent, be contingent upon the macronutrient composition of an individual's binge episodes.

Opioids and Binge Eating

There is considerable evidence implicating the opioid system in binge eating. For example, compared to controls, individuals with BN show reduced binding at mu-opioid receptors, a type of opioid receptor, in the left insular cortex, a brain region associated with taste and food reward processing as well as anxiety (Bencherif et al., 2005). Further, binding at mu-opioid

receptors was negatively correlated with frequency of fasting behavior in this population. These findings have been proposed to indicate a possible downregulation of opioid receptors due to increased endogenous opioid release following food restriction and binge eating (a "state-related" explanation of this observation). However, the authors of this study also raise the possibility that reduced mu-opioid receptor binding may be indicative of a "trait-related" characteristic in this clinical population (Bencherif et al., 2005). Further research is necessary to discriminate between these two possibilities.

Nonselective antagonism of opioid receptors reduces hedonic taste preferences and food intake, particularly of palatable foods, also causing short-term weight loss in humans (Nathan & Bullmore, 2009). The opioid antagonist naloxone leads to a general suppression of the hedonic response to foods but causes selective suppression of consumption of sweet and high-fat foods among binge eaters compared to non-binge-eating controls (Drewnowski, Krahn, Demitrack, Nairn, & Gosnell, 1995). Naltrexone has been shown, alone and with fluoxetine, to reduce the frequency of binge eating in case studies and open-label trials in patients with BED or BN (Alger, Schwalberg, Bigaouette, Michalek, & Howard, 1991; Jonas & Gold, 1986-1987; Marrazzi et al., 1995; Meyer, 2008; Neumeister, Winkler, & Wober-Bingol, 1999). The evidence is mixed regarding BN, however, with some (Marrazzi et al., 1995), but not all (Mitchell et al., 1989) studies supporting the efficacy of naltrexone in treating this population (see Chapter 59). Differences in dosage may explain negative findings, as higher dosages (200–300 mg) have been shown to be more efficacious in treating BN (Jonas & Gold, 1998).

Animal models have also served to elucidate how opioid systems may affect and be affected by various feeding behaviors. For example, injecting a mu-opioid agonist, DAMGO, in the NAc increases intake of palatable foods (Zhang, Balmadrid, & Kelley, 2003), while ingestion of palatable foods increases opioid receptor binding in the same area (Kelley et al., 2002; Kelley, Will, Steininger, Zhang, & Haber, 2003). After a history of binge consumption of a sugar solution, rats also show an increase in mu-opioid receptor binding in the accumbens shell (Colantuoni et al., 2001). Kelley and colleagues (2003) found that, compared to controls, rats with limited access to and binge-like consumption of a high-fat, high-sugar solution showed reduced expression of the opioid peptide enkephalin in the NAc. Such findings indicate that the consumption of palatable foods, particularly in a binge-like manner, is associated with perturbation of opioid systems.

Investigation of animal models of binge eating, including food deprivation and refeeding cycles, have shown that the kappa opioid agonist, butorphanol, enhances food intake in binge eating animals (Hagan & Moss, 1991). In rats with a history of these cycles, systemic administration of butorphanol has also been shown to increase binge eating induced by stress (Boggiano et al., 2005). Endogenous opioids also may be involved in binge eating, as noted in studies that observed signs of opiate-like withdrawal in rats with a history of intermittent food deprivation and binge eating sucrose following systemic administration of naloxone (Avena, Long, & Hoebel, 2005; Colantuoni et al., 2002).

Several studies in laboratory animals have also supported the efficacy of opioid antagonists in reducing bingelike food consumption. Local injection of naltrexone in reward-related brain areas leads to a decreased intake of a preferred fat or sucrose diet (Naleid, Grace, Chimukangara, Billington, & Levine, 2007). Further, in studies examining oil emulsions, sweet solutions, fats, and sweet-fat mixtures, naloxone decreased intake of the preferred food regardless of access pattern, though its effects were more pronounced in binge eating animals (Corwin & Wojnicki 2009; Rao, Wojnicki, Coupland, Ghosh, & Corwin, 2008; Wong et al., 2009). Finally,

nalmefene, a mu- and kappa-opioid antagonist, significantly attenuated binge eating in rats conditioned to binge eat a diet consisting of sweet chocolate (Cottone, Sabino, Steardo, & Zorrilla, 2008).

Conclusions and Future Directions

Understanding the neurochemical correlates of EDs can be a challenge for investigators. While several advances in the field have helped to elucidate some of the disturbances associated with under- and overeating, questions remain. Difficulties in conducting prospective studies limit our understanding of the neurochemical factors that may predate the onset of EDs, and studies of individuals while ill are limited by possible state-related confounds, such as malnutrition. Further, studies assessing the possible long-term effects of disordered eating and extreme weight loss rely on a well-constructed and consistent definition of recovery. Studies examining patients while ill may also be affected by when during treatment a patient is studied and by treatment progress. Despite these methodological issues, research findings such as those outlined in this chapter have informed our understanding of the various neural components that may contribute to, maintain, or result from EDs, providing greater insight into these complex disorders and guiding our pursuit of effective treatments.

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31

Peer Risk Factors and Eating Disorders

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Peer relations are fundamental to humans' social needs of acceptance and belonging. While these relations are important throughout the life span, they play a particularly salient role for well-being and adjustment in adolescence (Hartup, 1996). During this time in life, peers become important sounding boards for the formation of a more complex self-understanding and the adolescent psychological task of identity development. As will be argued in this chapter, peer relations further represent a sociocultural context that exerts a powerful influence over individuals' conceptions, beliefs, attitudes, and behaviors related to physical appearance.

Adolescence is also a critical developmental period for the rise of body dissatisfaction, dieting, and eating problems. Thus, these problems emerge at a time when peers have become increasingly important. Theoretical models of body image disturbances and eating pathology postulate that peers are important socialization agents for the formation of body image (Stice, 2001; van den Berg, Thompson, Obremski-Brandon & Coovert, 2002; see also Chapter 21). Sometimes, peers also provide a gateway into a culture of exacting appearance standards, and when they do, peers may be even more influential than parents (Salafia & Gondoli, 2010). Despite this, and in the existing research concerned with the development of disordered eating, only moderate attention has been given to the role of peers.

In this chapter the objective is to highlight the significance of the peer environment for body image concerns and eating problems. We will start with a brief outline of peer relations and peer influence, with a specific focus on adolescence, as this is a time of particular vulnerability for the development of body dissatisfaction and eating pathology. Thereafter, literature concerned with the links between different aspects of peer experiences, body dissatisfaction, and eating disorders (EDs) will be examined. A final objective of the chapter is to shed light on some questions that remain as yet insufficiently answered by the current state of research.

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Peer Relations and Peer Influence

By middle childhood, children can be said to participate in a social world truly their own: the world of peers (Gifford-Smith & Brownell, 2003). Children take part in this world as they attend school, during leisure time, and more recently, when they go online. By middle childhood, research indicates that more than 30% of children's time is spent with peers (Gifford-Smith & Brownell, 2003). In adolescence, and as peer relations grow further in importance, more and more time is shared with friends and in different peer group settings. The Internet and its opportunities for social encounters have also meant that the landscape of peer interaction has undergone dramatic change (see Chapters 29 & 46). Recent research indicates that physical appearance may be an extremely important component of online interaction (Tiggemann & Miller, 2010). This is, for example, represented by the widespread appearance commentary on social networking sites used by young people (e.g., Facebook and Instagram).

From research we also know that children's peer relations change in systematic ways as children age, and that they fulfill different needs at different developmental periods. In adolescence, intimacy, conformity, and closeness in relationships are especially valued, but there are also issues of acceptance, social status, and popularity. In their efforts to gain social approval, adolescents attribute major importance to the attitudes and behaviors of their peers. As a consequence, they seek to emulate what are considered important social norms in this world. Adhering to perceived peer group norms is associated with social rewards, for example an increase in popularity, while deviating from group norms may be associated with decreased popularity, or even peer rejection (Zalta & Keel, 2006). Importantly, these social norms may be perceived rather than real, yet they shape behavior (Jones, 2012). One example of such a norm is the belief that one needs to be skinny to gain approval among peers, a social norm that leads numerous adolescent girls to engage in restrictive eating and dieting. This belief is further complicated by the fact that girls who are indeed popular among peers, thus socially approved, may themselves engage in disordered eating at higher rates (Lieberman, Gauvin, Bukowski, & White, 2001).

Another reason why peers exert a powerful influence on the development and maintenance of body image concerns and disordered eating is because peer selection processes parallel those of peer socialization. More specifically, children and adolescents may become friends because they share similar beliefs and behaviors (peer selection). Over time, they become even more alike as a result of socialization processes occurring within friendships (Jones, 2012). In terms of the significance for body dissatisfaction and disordered eating, research indicates that socialization processes may result in a convergence of body image concerns and eating-related behaviors in close peer groups over time (Crandall, 1988; Hutchinson & Rapee, 2007; Zalta & Keel, 2006). Zalta and Keel (2006), for example, showed that blockmate college women shared similar personality traits in the beginning of the study, and that they developed bulimic symptoms in conjunction over time. Along with other similar studies, these findings suggest that selection and socialization processes are important to understand the development of body image pathologies. In order to understand the mechanisms underlying body dissatisfaction and EDs further, the current literature also highlights three aspects of the peer environment: adolescents' perceptions of their relationships with peers, aspects of the peer culture that more directly concern physical appearance (the peer appearance culture), and issues of peer victimization. Next, we will turn to a more detailed outline of these issues.

Perceptions of Peer Relationships

Perceptions of one's relationships with peers are important determinants of healthy social functioning. Therefore, some empirical work has focused on the role of psychosocial functioning and the quality of one's peer relationships in the development of body dissatisfaction and eating pathology. From the general literature on peer relations and well-being, feeling that one has social support in relationships has been identified as a key factor for healthy development. Perhaps not surprisingly, this extends to body-image-related issues as well. In a comprehensive meta-analysis of risk and maintenance factors for eating pathology (Stice, 2002), perceived social support emerged as a potent protective factor mitigating the effects of different risk factors for EDs. More recent research confirms this conclusion, as perceived social support, and the extent to which adolescents feel accepted by peers, have been linked to fewer body image concerns (Gerner & Wilson, 2005).

Conversely, perceived difficulties in one's peer relations may contribute to body dissatisfaction and eating pathology. Individuals who experience a lack of social support, higher levels of conflict, and dissatisfaction with peer relationships may engage in disordered eating. Schutz and Paxton (2007), for example, showed that negative friendship qualities (such as friend conflict and friend alienation) predicted adolescent girls' body dissatisfaction and disordered eating, and particularly so for bulimic symptoms. Positive friendship qualities, on the other hand, were unrelated to body dissatisfaction and disordered eating, which is in line with a broader literature showing that negative friendship qualities are a stronger predictor of adverse outcomes than are positive friendship qualities of positive ones.

Importantly, the causal chain between difficulties in peer relations, body dissatisfaction, and EDs remains unclear. Considering the potential mechanisms underlying these links, it has, for example, been proposed that individuals engaging in disordered eating perceive themselves as lacking in social competence, but it is not clear whether deficits in social skills constitute a risk factor or consequence of eating pathology (Ferriter, Eberhart, & Hammen, 2010). Potentially, individuals who feel they lack support in social relationships may engage in disordered eating to receive approval and validation from peers (Shutz & Paxton, 2007; Stice, 2002). Additional longitudinal research is clearly needed to further elucidate the effects of interpersonal variables (such as social support and quality of relationships) for ED symptomology.

Peer Appearance Culture

Until now, we have discussed the potential role in body image concerns and disordered eating of more general interpersonal influences and peer experiences, such as individuals' perceptions of social support and the quality of peer relationships. We will now turn to experiences with peers that more directly involve physical appearance, such as social pressure relating to appearance, modeling of eating behaviors, and appearance feedback and victimization. As was argued earlier, strict social norms regarding physical appearance may be conveyed in the peer environment. Seeking to examine how beliefs and behaviors related to the body and eating unfold, Jones, Vigfusdottir, and Lee (2004) focused on adolescents' "peer appearance cultures." More generally, the term "appearance culture" has been used to describe a culture that values, reinforces, and models strict cultural ideals of beauty and body shape (Thompson, Heinberg, Altabe, & Tantleff-Dunn, 1999; see also Chapters 21, 27, & 29), thus affecting body image and disordered eating on the individual level.

Jones and colleagues propose that adolescents engage in "appearance training" in their peer groups. Importantly, prior experiences relating to physical appearance are brought into the peer group and shape expectancies, for example, about the importance of physical appearance for social approval. In the peer group, adolescents learn further what looks are desirable and what looks are undesirable. They also learn to evaluate themselves and others on the basis of physical appearance. One specific example is so called "fat talk," which refers to conversations with self-disparaging comments made about one's own weight ("I am so fat"), eating behaviors ("I shouldn't be eating this"), or body shape ("My hips look big in this") in social interactions (Clarke, Murnen, & Smolak, 2010; see also Chapter 27). Although these conversations may fulfill psychosocial functions, such as bonding with friends through shared values, repeated negative remarks about one's body may have detrimental effects for body satisfaction. For example, in an experimental study exposing young women for fat talk, those who had heard a young woman complain about her weight reported elevated levels of body dissatisfaction at posttest (Stice, Maxfield, & Wells, 2003). Fat talk is commonplace among young women in a Western cultural context, and reflects one example of the *appearance pressures* that may operate within peer groups.

Appearance Pressure

Social pressure regarding standards for physical appearance is a well-documented peer-related risk factor for body dissatisfaction and disordered eating (Lieberman et al., 2001; Peterson, Paulson, & Williams, 2007; Shomaker & Furman, 2009; Stice, 2002), and it has been described as the primary mechanism for the transmission of peer group norms (Lieberman et al., 2001). More specifically, the presence of appearance pressure in one's social environment may lead to a desire to conform to cultural ideals of beauty, typically the thin ideal. Studies indicate that peer appearance pressure may be even more influential for disordered eating behaviors than pressure from alternate sources, such as parents or the media (Presnell, Bearman & Stice, 2004; Salafia & Gondoli, 2010).

Different forms of social appearance pressures operate among adolescents and their peers, some of which are direct (e.g., encouragement to diet) and some of which are more subtle (e.g., ostracism of someone who does not conform to group norms; see Chapters 21 & 26). Besides the kinds of verbalized appearance concerns in the form of fat talk as described earlier, other examples of social appearance pressure among peers may include preoccupation with physical appearance within the peer group and joint dieting or other behaviors aimed at altering body weight.

Most empirical work to date has examined the links between social appearance pressure, body dissatisfaction, and/or ED in female samples, with but a few exceptions of studies comprising both girls and boys (see Chapter 37). Peterson et al. (2007), for example, found that high-school girls and boys who perceived greater appearance pressure from peers also reported more ED symptoms. Similarly, a study by Helfert and Warschburger (2011) identified prospective links between peer pressure at the study outset, and weight (girls) and muscle concerns (boys) one year later. Shomaker and Furman (2009) found that adolescents' perceived pressure to be thin and friends' reports of the pressure to be thin both predicted concurrent increases in disordered eating. Along with results from a wide range of studies, mounting evidence implies that adolescents' experiences of a strongly appearance-focused peer environment is associated with an increased risk of developing ED symptomology (see Chapter 37).

Social Modeling

It has also been proposed that *social modeling* of attitudes and behaviors in one's proximal environment will increase the risk that young people engage in disordered eating (see Chapter 26). Social modeling occurs when individuals copy behavior they see others perform (Bandura, 1969). Importantly, *social reinforcement* processes precede and perpetuate modeling of eating behavior. One example of these processes may be how the thin ideal is maintained through constant positive attributions to people conforming to this ideal. Although peer modeling constitutes a core theoretical component for the development of body dissatisfaction and ED, direct modeling processes have been less researched than other factors (such as peer pressure).

Oliver and Thelen (1996) argued that peer modeling consists of two components, one based on the frequency of peer interactions about issues related to weight and body shape (e.g., fat talk and appearance commentary), and one based on direct modeling of maladaptive eating patterns. Peers could, for example, model weight loss behaviors, binge behavior, and self-induced vomiting. In a classic study by Crandall (1988), young women who belonged to the same sororities acquired the same binge eating behaviors over time. Crandall further demonstrated that not only was binge eating per se associated with greater popularity, it was also important for social recognition to engage in the right amount of bingeing (not too little nor too much). Importantly, these findings suggest that the impact of friends may become more powerful as friendships become more cohesive. Another extreme, and up-to-date, example of social modeling may be the acquisition of disturbed eating behaviors potentially resulting from exposure to so called pro-ED websites that promote ED as a lifestyle choice (Levine & Chapman, 2011; see also Chapter 29). While serious concern has been raised over the psychological and behavioral impact of these sites, it should be noted that we lack sufficient knowledge of whether pro-ED sites are in fact harmful. In particular, we need to learn more about how they affect individuals who already suffer from an eating disorder and repeatedly seek them out.

Because of its role as a risk factor for eating pathology, direct modeling of dieting has received the most attention in the literature. Friends' dieting behaviors have been identified as precursors for disturbed eating patterns among adolecents. Eisenberg and Neumark-Sztainer (2010), for example, found that friends' dieting frequency at baseline predicted disordered eating at follow-up for girls, but not for boys. It should, however, be noted that these studies build on adolescents' self-reported perceptions of their friends' dieting behaviors as predictors of eating pathology. To overcome this methodological limitation, Hutchinson and Rapee (2007) used social network analysis (and thus multiple sources of informants) to evaluate modeling processes within friendship cliques. Their analyses showed that an individual girl's dieting and extreme weight loss behaviors were predicted by her friends' respective reports of their own weight loss behaviors.

However, it should also be kept in mind that what can be interpreted as processes of social modeling may also account for selection processes, as discussed earlier in this chapter. According to selection processes, individuals may choose friends with whom they already share similar beliefs and behaviors (Hutchinson, Rapee, & Taylor, 2010), in this case saliency of appearance and eating behaviors. In one longitudinal study, Zalta and Keel (2006) showed that friends were similar in terms of the presence of bulimic symptoms at the study outset. The relationship between bulimic symptoms and verified friendships was strengthened with continued social contact, suggesting that social modeling might have occurred. Modeling may be especially

important for bulimic pathology. In his meta-analysis of risk factors for eating pathology, Stice (2002) showed that while social modeling failed to predict body dissatisfaction and dieting, it was significantly associated with the development of bulimic behaviors. For example, in one prospective study (Stice, 1998), peer modeling of abnormal eating behavior (e.g., dietary restraint, extreme weight loss behaviors and excessive exercise) at time 1 prospectively predicted the onset of binge eating and purging at follow-up.

Appearance Teasing and Bullying

Other experiences with peers that may impact body dissatisfaction and eating behaviors are stigmatization and marginalization by peers in the forms of appearance teasing and bullying (see Chapters 21, 27, & 43). While appearance teasing refers to comments about appearance that may, or may not, be intentionally hurtful, bullying is a wider concept. Bullying is used to describe intentional, aggressive acts that are directed toward a victim who cannot readily defend himself or herself, and that are repeated over time. Acts of bullying may be direct (e.g., use of violence, verbal threats) or indirect (e.g., rumor spreading and gossiping). Indeed, children's and adolescents' peers are the most common perpetrators of both appearance teasing and bullying. In terms of bullying, the number of individuals being the target of bullying varies widely across countries, but reviews of the literature indicate that about 10% are regular targets of this systematic abuse of power. Traditional, face-to-face bullying peaks in late childhood and in the early phases of adolescence. With the emergence of the Internet and mobile phone devices as important platforms for social interaction between peers, however, it appears as if online bullying (or cyberbullying) continues longer, well into the middle phases of adolescence (Tokunaga, 2010).

Appearance teasing experiences are commonplace. In a study of nearly 5,000 middle-school children, about one quarter of the participants reported having been teased about appearance several times within the past year (Neumark-Sztainer et al., 2002). While experiences of appearance teasing have been one of the most empirically established links to body dissatisfaction, somewhat fewer studies have sought to examine the associations between appearance teasing and EDs. In one prospective study that did focus on appearance teasing and eating pathology, adolescent boys and girls who reported being the target of weight-related teasing were more likely to engage in binge eating or unhealthy weight control (boys) or frequent dieting (girls) (Haines, Neumark-Sztainer, Eisenberg, & Hannan, 2006). These findings emerged after controlling for potentially confounding factors such as participants' age, race, socioeconomic status, and body mass index (BMI).

Research also indicates that bullying may place youth at risk for body image concerns and eating disturbances. Being the target of bullying in late childhood has been linked to concurrent body dissatisfaction (Lunde & Frisén, 2011; Lunde, Frisén, & Hwang, 2006, 2007), and later body shame (Lunde & Frisén, 2011), particularly among girls. Importantly, these findings stem from longitudinal work, suggesting that the impact of childhood bullying on body image concerns may be longstanding. In addition, bullying occurring on the Internet has recently been linked to higher levels of body dissatisfaction among adolescent girls and boys (Frisén, Berne, & Lunde, 2014). In terms of EDs, bullying has been linked to eating pathology in retrospective studies with clinical samples, and in some prospective work. Engström and Norring (2002), for example, found that young women at risk for EDs were more likely to report having been bullied in their past. Striegel-Moore et al. (2002) showed that bullying experiences were

related to binge eating, and Farrow and Fox (2011) showed that early adolescent girls' and boys' bullying experiences were related to body dissatisfaction and restrained eating.

The above findings raise the question *why* bullying may be linked to elevated body dissatisfaction and ED. Part of the explanation may be found in a study by Frisén, Holmqvist, and Oscarsson (2008), who found that 43% of 10-year-old children stated that bullied children are bullied because of their physical appearance. Similar patterns have been observed for online bullying. One study showed that a substantial number of the adolescent participants believe that cyberbullying is often or always directed at the victim's physical appearance, at least when girls cyberbully other girls (Frisén et al., 2014). Here it should also be noted that, although appearance teasing and bullying are directed toward individuals at all weight categories, overweight and obese youth are more likely to report victimization by peers (Frisén, Lunde, & Hwang, 2009; Thompson et al., 2007).

While the reasons why children are picked as targets for these systematic acts of aggression are much more complex than being solely a matter of physical appearance, adolescents' own views may mirror a reality wherein physical appearance is used as a potent way to put someone down, especially girls. Another explanation, not mutually exclusive, is that children who feel rejected by their peers may believe that they are rejected because of their physical appearance. While acts of bullying may or may not target physical appearance, there may still be adverse effects for the victim's body image. Given that many adolescents associate physical appearance with social approval, the conclusion victimized children may come to is that their physical persona is not good enough to be accepted by peers. Bullying may then give rise to body dissatisfaction and body shame, and with the common belief that thinness is associated with greater social approval, disordered eating may be engaged in as a means to gain acceptance by peers. Speculatively, these mechanisms may put individuals with past or present bullying experiences at greater risk for eating pathology.

Conclusions and Future Directions

A final objective of this chapter is to identify some unanswered questions relating to the role of the peer environment and its role in EDs. First, most studies and conceptualizations of peer risk factors derive from work with female samples. Much less is known about the role of the peer environment for the development of EDs in boys (see Chapter 37). Continued investigation of the role of peers with studies better designed to capture boys' experiences provides a fruitful avenue for future research. Second, although there is some longitudinal work, there still remain questions about the directionality of the associations between peer risk factors and EDs. For example, it remains unclear whether appearance teasing and bullying cause body dissatisfaction or disordered eating, or whether individuals struggling with body dissatisfaction/ED are more sensitive to appearance-related commentary. Similarly, it may be that adolescents who are occupied with physical appearance perceive that there is a strong appearance-pressure among their friends and peers. While there are some longitudinal and experimental data, we still have limited knowledge about the psychological mechanisms underlying the potential pathway between peer victimization and body dissatisfaction and EDs. Further prospective and/or longitudinal, as well as experimental work is clearly needed to further the understanding of the directionality of peer influences for ED symptomology. This would help us to elucidate and clarify which risk factors are most salient in the development of eating disturbances. Finally, our understanding of how the online peer environment may interplay with

body dissatisfaction and EDs is still limited. There are numerous, appearance-oriented Internet environments that are popular among young people, and that convey strict notions of physical beauty. The most extreme example is perhaps provided by so called pro-eating disorder websites (Levine & Chapman, 2011; see also Chapter 29). It should be noted that the knowledge about these kinds of novel peer influences for body image concerns and eating disorder pathologies is still in its infancy.

To conclude, the evidence to date lends weight to the notion that the peer subculture is an important one for the development of body dissatisfaction and disordered eating. The literature suggests that peer-related risk factors for eating pathology may involve perceived deficits in social relationships, being in an appearance-focused peer environment, and being exposed to negative appearance feedback or marginalization by peers. Although we still need to further the understanding about the mechanisms underlying the interplay between the peer environment and the development of ED in youth, the current literature highlights the need for ED prevention at the level of the peer group.

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32

Personality as a Risk Factor for Eating Disorders

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There are a number of ways in which personality factors and eating disorders (EDs) may be related. Personality factors may increase the risk of EDs, EDs may increase the risk of personality disorders, or certain third variables may increase the risk of disturbances in both areas. An overview of these and other models of the association between personality factors and EDs can be found in Lilenfeld, Wonderlich, Riso, Crosby, and Mitchell (2006). This chapter, however, will review personality and EDs from three different perspectives: (a) personality traits that may prospectively increase one's risk for the development of EDs and/or disordered eating behavior; (b) empirically derived personality subtypes of EDs; and (c) personality traits/personality disorder symptoms that commonly co-occur with EDs and may be related to ED course or outcome.

Models of Personality and Eating Disorder Associations

Consistent with prior studies addressing risk factors for ED symptoms (Stice, 2002), we defined the term "risk factor" as a variable that has been shown to prospectively predict some subsequent pathological outcome. Among the models explicating the association between personality factors and EDs, the *predispositional* model is the most consistent with this conceptualization of a risk factor. The predispositional model is a causal model positing that the presence of a personality trait may increase the likelihood that an individual will develop an ED at some later point in time (Lilenfeld et al., 2006). This type of model is best tested with a prospective design that follows a group of individuals for a period of time that encompasses the point at which EDs typically develop. However, given the relatively low base rate of EDs in community samples, these studies are often challenging and costly to conduct. Consequently, there are a limited number of prospective studies in the literature that have adequately tested the predispositional model of the role of personality factors in the etiology of EDs. The findings of these studies are presented below.

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Other models do not emphasize the prospective nature of the personality and ED association. Thus, while these models provide important theoretical explanations for the association between personality factors and EDs, personality in these models is not conceptualized as a true risk factor (i.e., it is not required to establish that personality factors were present before the development of the ED). Two such models are the *pathoplasticity* model, which posits a bidirectional causal influence between personality factors and ED psychopathology, and the *spectrum* model, in which personality and ED psychopathology are viewed as existing on a continuum, and the presence of one can serve as an indicator of the other (Lilenfeld et al., 2006). A number of different research designs can be used to examine particular aspects of these causal models. The remainder of this chapter highlights research findings that have relevance to understanding the association between personality and EDs as conceptualized in these various models.

Of course, personality and EDs may be related in other ways not directly addressed by these models. For instance, personality may be influenced by the symptomatology of the ED (i.e., *state effects* of the ED on other aspects of psychosocial functioning). When such effects persist following recovery or emerge thereafter, they are known as *scar effects* (i.e., the *complication* model; Lilenfeld et al., 2006). Further, both personality and EDs may be influenced by a third variable or may both be expressions of the same underlying vulnerability. Given the scope of the current chapter, findings pertaining to these types of relationships will not be discussed.

Personality as a Predispositional Risk Factor

Prospective studies investigating personality as a risk factor for ED psychopathology can be distinguished by whether they identified personality factors that predicted the onset of EDs—that is, full-threshold, clinically significant syndromes such as anorexia nervosa (AN) or bulimia nervosa (BN)—or personality factors that predicted the development of disordered eating, such as binge eating and purging. Overall, likely due in part to the low base rate of EDs in general, fewer studies have predicted the onset of diagnostic EDs than disordered eating behaviors.

Eating Disorders

Among the studies that have examined the development of diagnostic EDs, Ghaderi and Scott (2000) obtained 2-year follow-up data from 856 women ages 18 to 30 years out of a total of 1,157 women who were randomly selected from the general population of Sweden. Over this follow-up period, 33 women (3.8%) experienced the onset of an ED, whereas 636 women (74.3%) never reported ED symptoms. With regard to personality factors in this sample, compared to women who never reported ED symptoms, those women who developed EDs were found to exhibit significantly lower levels of agreeableness, less emotional stability (i.e., more neuroticism), and more openness to experience prior to the development of their ED. In another study, Cervera and colleagues (2003) reported findings from an 18-month follow-up of a representative sample of 2,862 women aged 12 to 21 years in Spain. Of the 2,509 (87.7%) women who did not have an ED at baseline and who provided follow-up data, 90 (3.6%) prospective ED cases were identified. Regarding the personality factors that predated the onset of the disorder, neuroticism was found to be elevated and self-esteem was lower at baseline in these 90 women relative to the rest of the sample.

In a study with a smaller sample but a longer follow-up period, Tyrka, Waldron, Graber, and Brooks-Gunn (2002) reported findings from 257 "predominantly White, middle- to

upper-middle-class" (p. 283) girls from private schools in New York City. The girls in the study were originally assessed between the ages of 12 and 16 years, and were reassessed at 2 years and 8 years after their baseline assessments. At the 2-year follow-up, 193 participants (75.1%) provided data, and at the 8-year follow-up, 134 participants (52.1%) provided data. Findings were reported with regard to the onset of an anorexic syndrome (i.e., either full-threshold AN or subthreshold symptoms consistent with an AN-like presentation) or bulimic syndrome (i.e., either full-threshold BN or subthreshold symptoms consistent with a BN presentation). The authors identified a total of 19 (7.4%) onset cases of an anorexic syndrome and 24 (9.3%) cases of a bulimic syndrome across the follow-ups. Tyrka et al. found that perfectionism prospectively predicted the onset of an anorexic syndrome during the follow-up period, while negative emotion prospectively predicted the onset of a bulimic syndrome. Finally, a study by Johnson, Cohen, Kasen, and Brook (2006) examined, at four occasions (from age 14 to 33), a sample of 570 individuals who initially did not have a history of an ED. Results indicated that borderline personality disorder (BPD; see Chapters 15, 54, & 57), histrionic personality disorder, and schizotypal personality disorder symptoms that occurred between the ages of 14 and 22 predicted the development of an ED by age 33.

Taken together, these studies indicate that a range of personality factors may function as prospective risk factors that predict later development of EDs, including low agreeableness, high neuroticism, high openness, and low self-esteem. Similarly, symptoms of several personality disorders (i.e., borderline, histrionic, and schizotypal personality disorders) were found to predict the onset of EDs, and in more specific predictions of ED diagnoses, perfectionism was found to predict an anorexic syndrome and negative emotion was found to predict a bulimic syndrome.

Disordered Eating

Among the prospective studies examining personality traits as predictors of the development of disordered eating behaviors, neuroticism was found to prospectively predict the onset of disordered eating behaviors (binge eating, purging, excessive dieting) in 580 twins followed over 7 years (Ferguson, Muñoz, Wineguard, & Wineguard, 2012). Further, negative affect, although broadly defined and not assessed consistently across studies, has been found to predict disordered eating symptoms in several studies, including one that followed 726 adolescent girls and 698 adolescent boys for 3–4 years (Leon, Fulkerson, Perry, Keel, & Klump, 1999), and one in which 2,109 adolescent Chinese girls and boys were followed over 12 months (Jackson & Chen, 2011). Additionally, the personality trait of ineffectiveness (i.e., feelings of inadequacy, worthlessness, insecurity) predicted the development of binge eating and purging in a sample of 394 middle-school girls followed prospectively and assessed in the fall of seventh, eighth, and ninth grades (Combs, Smith, Flory, Simmons, & Hill, 2010). Finally, a study by Leon, Fulkerson, Perry, and Early-Zald (1995) found that poor interoceptive awareness predicted the development of ED symptoms over 2 years in a sample of 852 adolescent girls, although no personality measures predicted ED symptom onset in the sample of 815 boys.

With regard to personality disorder symptoms as risk factors for disordered eating onset, Johnson and colleagues (2006) found that symptoms of antisocial, borderline, dependent, depressive, histrionic, passive-aggressive, and schizotypal personality disorders occurring between the ages of 14 and 22 predicted the development of recurrent binge eating episodes by age 33. In addition, borderline, histrionic, and schizotypal personality disorder symptoms were predictive of the development of recurrent purging, and depressive personality disorder

symptoms were predictive of recurrent dietary restriction. Further, in studies of three independent samples of adolescent women, Wonderlich, Connolly, and Stice (2004) found that objective measures of behavioral impulsivity (e.g., delinquency and substance use) predicted the onset of compensatory behaviors in two of the samples in which such a measure was available, and binge eating in one of the two samples in which it was measured. However, standard self-report trait measures of impulsivity failed to predict the onset of binge eating and compensatory behavior in all of the three samples.

In sum, similar to findings on personality constructs as risk factors for diagnostic EDs, several personality factors may function as risk factors for later development of subthreshold disordered eating symptoms. Specifically, studies have identified neuroticism, negative affect, ineffectiveness, poor interoceptive awareness, objectively measured behavioral forms of impulsivity, and symptoms of numerous personality disorders (e.g., borderline, depressive, histrionic, passive-aggressive, and schizotypal personality disorders) as prospective predictors of the development of varying types of disordered eating behaviors.

Personality-Based Eating Disorder Subtypes

Within-diagnosis heterogeneity in the EDs is a well-documented phenomenon (see Chapters 2–4, 8–11, & 15). For example, a group of individuals diagnosed with the same ED may display different symptom presentations or varying forms of co-occurring psychopathology, as well as in numerous other areas potentially related to the etiology or maintenance of the disorder. Within the diagnostic framework of the American Psychiatric Association's (2013) fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*-5), this within-diagnosis heterogeneity is in part addressed by diagnostic subtypes. For instance, AN includes two subtypes, that is, restricting versus binge eating/purging, that are applied based on symptom presentations (i.e., presence or absence or regular episodes of binge eating or purging), although these subtypes may have limited predictive validity (Wonderlich, Crosby, et al., 2007; Peat, Mitchell, Hoek, & Wonderlich, 2009). Further, the *DSM-5* does not include subtypes to account for heterogeneity within BN or binge eating disorder (BED), despite evidence that these disorders, similar to AN, are characterized by substantial heterogeneity (Wonderlich, Joiner, Keel, Williamson, & Crosby, 2007).

Of the characteristics that have been found to exhibit substantial within-diagnosis heterogeneity in the EDs, personality factors have been among the most widely researched (Cassin & von Ranson, 2005; Lilenfeld et al., 2006). In particular, a substantial body of research has examined personality subtypes within the EDs using empirical classification methods. These studies have used varying samples, including single diagnostic samples, as well as mixed ED samples. Although there have been mixed findings, the results of many of these studies support the presence of three personality-based subtypes. Although these studies have given differing names to the identified subtypes, they broadly correspond to:

1 An underregulated subtype characterized by patterns of impulsive behaviors and affective lability/instability.
2 An overregulated subtype characterized by inhibition and a restricted/constrained behavioral/affective presentation.
3 A normative subtype that is characterized by normative levels of personality functioning, despite the ED psychopathology (Wildes & Marcus, 2013).

Subgroups consistent with these three personality-based subtypes have been identified in adult BN samples (Wonderlich, Crosby, et al., 2007), adult AN samples (Lavender et al., 2013; Wildes et al., 2011), and adult mixed ED samples (Westen & Harnden-Fischer, 2001), indicating that the personality subtypes may be present across the range of ED psychopathology. Further, findings from other studies indicate that these subtypes are also present in adolescent AN samples (Gazzillo et al., 2013) and adolescent samples with mixed ED presentations (Thompson-Brenner, Eddy, Satir, Boisseau, & Westen, 2008), suggesting that they are present even earlier in the course of EDs.

These three subtypes have been validated using clinically important variables and have been found to differ in terms of co-occurring psychopathology, ED symptoms, and treatment response. For example, the underregulated personality subtype has been found to exhibit higher rates of substance abuse, childhood abuse history, severe co-occurring personality disorders, and the most severe ED symptoms (Claes et al., 2006; Gazzillo et al., 2013; Steiger et al., 2010). Additionally, with regard to treatment, individuals classified in the underregulated group are more likely to have a history of psychiatric hospitalization for an ED or other condition (Westen & Harnden-Fischer, 2001; Wildes et al., 2011) and are also at greater risk of discharge against medical advice and readmission following intensive ED treatment (Wildes et al., 2011).

In contrast, individuals who are classified in the overregulated subtype have been found to display a greater likelihood of a lifetime diagnosis of co-occurring obsessive-compulsive disorder, higher levels of perfectionism, and lower rates of binge eating behavior (Lavender et al., 2013; Steiger et al., 2009). Finally, consistent with the lower levels of personality pathology that characterize the normative subtype, studies have reported that individuals classified in this group tend to display less severe overall ED psychopathology (Espelage, Mazzeo, Sherman, & Thompson, 2002; Wildes et al., 2011), as well as the lowest rates of prior psychiatric hospitalizations (Westen & Harnden-Fischer, 2001; Wildes et al., 2011).

The existing evidence thus supports the presence of meaningful differences between these personality subtypes, which have been identified in a variety of ED samples. The goal of research examining personality-based subtypes within EDs has been to account for heterogeneity by identifying clinically useful and valid subgroups. Heterogeneity within the EDs is of concern given the potential negative impact on understanding both etiology/maintenance (e.g., problems identifying relevant variables if they vary across unidentified subgroups) and treatment (e.g., unidentified subgroups could potentially display a differing response to a given treatment). Thus, in light of the evidence suggesting the presence of differences across the empirically derived underregulated, overregulated, and normative subtypes, it may be that certain etiological/maintenance factors or mechanisms vary across the subtypes, suggesting the potential utility of considering these personality presentations in ED treatment.

Personality Disorders that Commonly Co-occur With Eating Disorders

Eating disorders are associated with high rates of comorbidity with other forms of psychopathology, particularly mood disorders and anxiety disorders (O'Brien & Vincent, 2003; see also Chapters 15 & 54). However, in addition to the common co-occurrence of these affective disorders, personality disorders are also commonly found to occur among those with EDs, although rates vary greatly across studies due to differences in methods of assessment (Cassin & von Ranson, 2005). Personality disorders are categorized within three clusters: Cluster A personality disorders are characterized by an odd/eccentric presentation; Cluster B

personality disorders are characterized by dramatic and emotional presentation; and Cluster C personality disorders are characterized by an anxious/fearful presentation (*DSM-5*, 2013). Within the ED literature, the majority of research has focused on disorders in Cluster B (particularly borderline personality disorder, or BPD) and Cluster C (particularly obsessive-compulsive personality disorder).

The co-occurrence of BPD with EDs, particularly BN, has long been noted (see Chapters 15, 54, & 59). In a review of the personality disorder prevalence rates from studies that used diagnostic interviews, an average of 21% of individuals with BN met criteria for co-occurring BPD, compared to 3% of individuals with restricting-type AN and 9% of individuals with BED (Cassin & von Ranson, 2005). Early studies comparing across individuals with EDs with and without BPD symptoms found that those with co-occurring BPD symptoms tended to have more severe problems on multiple indices of psychosocial functioning (Wonderlich & Swift, 1990). Other studies, however, suggest that the difference between these groups is largely due to differences in mood disturbance rather than BPD symptoms specifically (Steiger, Thibaudeau, Ghadirian, & Houle, 1992). Regardless of the specific reasons, however, it is likely that the presence of co-occurring BPD symptoms in an individual with an ED may be a marker for greater severity, and thus may potentially predict poorer outcome and/or treatment response (Steiger, Leung, Thibaudeau, & Houle, 1993).

Given that individuals with EDs commonly report elevated symptoms of anxiety, Cluster C personality disorders have also received attention in ED research. Obsessive-compulsive personality disorder (OCPD), particularly in AN, has been perhaps the most widely studied of the Cluster C disorders in ED samples. In their review of personality disorder prevalence rates based on diagnostic interviews, Cassin and von Ranson (2005) found average rates of co-occurring OCPD diagnoses to be 15% in restricting type AN, 9% for BN, and 10% for BED. These rates compare to OCPD co-occurrence rates of approximately 23% in those with obsessive-compulsive disorder, 17% in those with panic disorder, and 3% in healthy controls (Albert, Maina, Forner, & Bogetto, 2004). These findings thus support the suggestion that obsessive-compulsive presentations occur across the EDs (Altman & Shankman, 2009), versus the apparently more common co-occurrence of BPD symptoms in BN. This is likely due, in part, to elevations across the ED spectrum in particular personality traits that underlie OCPD-type symptoms (e.g., harm avoidance, as discussed below).

Personality Traits Commonly Studied in Eating Disorder Samples

Personality factors have long been the focus of research studies in the EDs literature. However, there are several specific traits that have received particularly extensive attention in the literature. Among the most widely studied of these are harm avoidance, sensation-seeking, and perfectionism. A brief review of major findings regarding the link between each of these traits and ED symptoms is provided below.

Harm Avoidance

Individuals who are harm avoidant are characterized by behavioral inhibition and a tendency toward worry, fear, and shyness (Cassin & von Ranson, 2005; Cloninger, 1987). Higher harm avoidance is common across ED types compared to healthy controls and community norms (Cassin & von Ranson, 2005) and is associated with serotonin (Kaye, Bailer, Frank, Wagner,

& Henry, 2005) and dopamine function in women with EDs but not in control women (Bailer et al., 2013; see also Chapter 30). Harm avoidance is also associated with self-injurious behavior and skin picking in the purging type of BN (Favaro et al., 2008) and suicide attempts in BN (Forcano et al., 2009). Men with EDs have been found to score lower on harm avoidance than women with EDs (Núñez-Navarro et al., 2012; Woodside et al., 2004).

There is also evidence suggesting that higher harm avoidance in ED populations persists following recovery (Klump et al., 2004). However, studies also have found that harm avoidance decreases after intensive treatment in a mixed sample of individuals with EDs (Bloks, Hoek, Callewaert, & van Furth, 2004), after a trial of cognitive-behavioral therapy (CBT; see Chapter 56) in 100 outpatients with the purging type of BN (Agüera et al., 2012), and after CBT in 149 inpatients with EDs (Dalle Grave et al., 2007). The latter study noted that this change was accounted for by changes in depression and ED psychopathology, which is an important caveat because harm avoidance is also related to depression (Mochcovitch, Nardi, & Cardoso, 2012) and elevated in many psychological disorders, some even more so than AN and BN (e.g., schizophrenia, major depressive disorder, obsessive compulsive disorder, panic disorder, and social phobia; Miettunen & Raevuori, 2012). However, other findings involving harm avoidance remain even after controlling for depression. For example, Peterson and colleagues (2010) found that individuals with BED scored higher on harm avoidance than normal-weight controls even after controlling for depression, indicating that harm avoidance may also have unique associations with ED psychopathology. At least one study has found that lower harm avoidance is predictive of rapid response to CBT for BN (Bulik, Sullivan, Carter, McIntosh, & Joyce, 1999), but another found that higher harm avoidance was associated with better treatment response in a mixed sample of 57 women with EDs treated with Brief Adlerian Psychodynamic Psychotherapy (Fassino et al., 2005).

Sensation-Seeking

Sensation-seeking, novelty-seeking, and impulsivity are three related constructs generally indicative of behavioral activation; each describes aspects of the tendency to explore in novel situations and to make impulsive decisions (Cassin & von Ranson, 2005; Cloninger, 1987). Evidence suggests that alterations in dopamine functioning are related to these constructs (Kaye, Frank, & McConaha, 1999). In contrast to harm avoidance, fairly consistent differences emerge between ED diagnostic groups in novelty-seeking. Individuals with AN tend to score lower than individuals with BN and controls on novelty-seeking, and individuals with BN tend to score higher than healthy controls and community norms (Cassin & von Ranson, 2005). Some research indicates that individuals with the AN restricting type, in particular, score low on novelty-seeking compared to those with AN binge eating-purging type, BN, eating disorder not otherwise specified (ED-NOS), and healthy controls (Bloks et al., 2004). Novelty-seeking may be particularly elevated in those with BN compared to a number of other major psychological disorders (e.g., schizophrenia, major depressive disorder, obsessive-compulsive disorder, and social phobia; Miettunen & Raevuori, 2012), which may be partly explained by the association of novelty-seeking with purging behavior within the ED samples (Dalle Grave, Calugi, & Marchesini, 2009; Reba et al., 2005) and in community samples (Wade, Treloar, & Martin, 2008).

Impulsivity has been found to be associated with poorer outcome (Fichter, Quadflieg, & Hedlund, 2006; Keel & Mitchell, 1997; Waxman, 2009) and treatment dropout (Fassino, Pierò, Tomba, & Abbate-Daga, 2009), at least in BN. In contrast to these findings, some

research on AN suggests a better prognosis (Fichter et al., 2006). In a group of 40 individuals with restricting type AN presenting for treatment, higher novelty-seeking was associated with improvement at 6 months (Fassino et al., 2001). Similarly, in a sample of 680 women with AN, recovery was associated with higher impulsivity, although the strength of this relationship decreased as the duration of illness increased (Zerwas et al., 2013). These findings reflect that individuals with AN who score higher on impulsivity are actually closer to normative levels. Providing some support for this notion is the finding that novelty-seeking tends to increase with recovery from EDs (Bloks et al., 2004). For a review of impulsivity in the EDs see Waxman (2009).

Perfectionism

Perfectionism has long been identified as a potential risk factor for EDs. For instance, it is included in the transdiagnostic model of the maintenance of EDs (i.e., "clinical perfectionism"; Fairburn, Cooper, & Shafran, 2003). In particular, perfectionism is conceptualized as driving attempts to control eating, shape, and weight, which in turn are viewed as representing the core psychopathology of EDs (see Chapters 22 & 56). Failure to achieve these high standards is theorized to motivate stronger control efforts, thus functioning to maintain the ED symptoms. In support of the potential role of perfectionism in the etiology or maintenance of various EDs, high levels of perfectionism have been found to be present across the spectrum of ED psychopathology, and it appears that these elevated levels tend to persist even following recovery from an ED (Cassin & von Ranson, 2005).

Of particular relevance to understanding the role of perfectionism in EDs is the conceptualization of perfectionism as a multidimensional construct. Specifically, there are dimensions of perfectionism that appear to have adaptive functions, while others appear to be associated with maladaptive outcomes (Frost, Marten, Lahart, & Rosenblate, 1990). For example, in a sample of 1,010 female twins, Bulik and colleagues (2003) found that elevated scores on the typically maladaptive dimension of concern over mistakes was associated with EDs, but not other forms of psychopathology. In contrast, scores on another maladaptive dimension, doubts about actions, were found to be elevated in both EDs and anxiety disorders. In contrast, Halmi et al. (2000) found that women with AN displayed elevated levels of all perfectionism dimensions compared to controls, regardless of the specific AN subtype (i.e., restricting, purging, or binge-eating purging). Finally, although the majority of studies have examined perfectionism cross-sectionally in currently ill samples, some studies have studied perfectionism prospectively with regard to its association with eating pathology. Although there have been mixed results, overall, findings suggest that perfectionism may function as both a maintenance factor and a prospective risk factor for the onset of eating pathology (Stice, 2002).

Conclusions and Future Directions

In sum, the existing empirical evidence regarding the role of personality factors in the EDs implicates numerous personality traits and symptoms of personality disorders as prospective risk factors, as well as potential maintenance factors and correlates of diagnostic EDs and subthreshold disordered eating. In particular, higher levels of traits such as negative affect/neuroticism, perfectionism, and ineffectiveness/low self-esteem, and symptoms of certain personality disorders have been linked to an elevated likelihood of developing EDs or disordered

eating over time. Further, evidence from nonprospective research suggests that personality disorders (particularly Cluster B and C disorders) commonly co-occur with EDs, and that there may be utility in considering subtypes of EDs derived from personality factors. Finally, elevations on certain personality traits (e.g., harm avoidance and perfectionism) are common across the range of ED presentations, while sensation-seeking/impulsivity may distinguish ED presentations with bulimic-type symptoms.

This chapter has focused on reviewing studies of the prospective prediction of EDs based on personality features, as well as on specific personality traits and disorders that have been widely studied within the EDs literature. However, there are numerous other personality factors that have not been addressed in this chapter, several of which have only recently begun to gain more attention in the literature. In particular, the construct of negative urgency, which refers to the tendency to act rashly in the face of intense negative affect (Cyders & Smith, 2008; see also Chapter 57), has received increasing attention in the literature. Negative urgency has been linked with binge eating in elementary school girls (Fischer, Settles, Collins, Gunn, & Smith, 2012) and has been found to prospectively increase the expectation of negative affect being relieved by eating (which is associated with binge eating; Pearson, Combs, Zapolski, & Smith 2012). Negative urgency also has been found to be prospectively associated with increased risk of binge eating in college women (Fischer, Peterson, & McCarthy, 2013).

Future research, particularly prospective studies and research with clinical samples, will help to further clarify the role of negative urgency as a risk and maintenance factor for ED psychopathology. Additionally, given evidence suggesting that certain personality traits may be state-dependent in EDs, while others may remit along with ED recovery, additional research will be necessary to further elaborate the complex interactions between personality variables and ED symptoms.

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Protective Factors

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> Much of the task of prevention in this new century will be to create a science of human strength whose mission will be to understand and learn about how to foster these virtues. (Seligman & Csikszentmihalyi, 2000, p. 7)

Like Seligman and Csikszentmihalyi (2000), scholars in the eating disorder (ED) field (Smolak, 2011; Striegel-Moore & Cachelin, 1999) have begun to acknowledge the value in studying variables that protect against eating- and body-related distress alongside factors that promote such distress. *Protective factors*, as defined by Rutter (1985, p. 600), "modify, ameliorate, or alter a person's response to some environmental hazard that predisposes to a maladaptive outcome." Environmental hazards specific to eating- and body-related distress include direct and indirect appearance-related pressures from peers, family, partners, society, and the media (Jones, 2011; Tiggemann, 2011; see also Chapters 26, 29, & 31), being sexually objectified (Calogero, Tantleff-Dunn, & Thompson, 2011; see also Chapters 19 & 27), and being told to ignore internal self-regulatory hunger and satiety cues (Cook-Cottone, Tribole, & Tylka, 2013). Maladaptive outcomes are ED symptoms as well as ED risk factors such as negative body image, internalization of societal appearance ideals (see Chapter 21), body surveillance, body comparison, and poor interoceptive awareness (Cook-Cottone et al., 2013). For simplicity, throughout this chapter we refer to these maladaptive outcomes as "ED-related outcomes."

Protective factors are not always the reverse of risk factors; they also include variables that do not lie on the same continuum as a risk factor (Gustafsson, Edlund, Kjellin, & Norring, 2009; Striegel-Moore & Cachelin, 1999; Tylka, 2011). Therefore, protective factors are viewed as distinct from the absence of risk factors. Protective factors can reduce the likelihood of ED-related outcomes in various ways: by decreasing these outcomes directly; by preventing the initial occurrence of a risk factor; by interacting with a risk factor to interrupt its deleterious effects; and by disrupting the mediational chain through which a risk factor operates (Crago, Shisslak, & Ruble, 2001). Prevention of EDs, then, hinges on accurately identifying and actively building protective factors within individuals, families, schools, communities, and culture (see Chapters 41–49).

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The Attuned Representation Model of Self: A Framework of Protective Factors

To integrate and organize research on ED protective factors, we chose to use the attuned representation model of self (ARMS; Cook-Cottone, 2006) for several reasons. First, the ARMS emphasizes wellness by highlighting a process, attunement, that is protective against EDs. Specifically, *attunement* is an ability to appropriately sense and respect the body by being aware of its needs and regularly engaging in adaptive behaviors to attend to these needs. If attuned, a person can implement health guidelines, preferences (e.g., eating locally grown food), and dietary guidelines due to medical conditions (e.g., food allergies, diabetes, celiac disease) while paying attention to hunger, fullness, and taste (Cook-Cottone et al., 2013). Thus, the person is connected to and honors her or his body, which guards against the self-regulatory deficits that are characteristic of those with EDs (Skårderud, 2009; see also Chapter 57). Second, the ARMS is a comprehensive model. It includes biological, psychological, and sociocultural factors grounded in extant ED research and theory and identifies ways these factors could either promote or disrupt attunement. Third, the ARMS focuses on the interplay among factors to prevent and remediate EDs, which can be used to generate ideas for future research on protective factors.

The ARMS contains the self-system and the cultural system. Wellness is represented by the two systems being interconnected by attunement. Attunement between the self- and cultural systems is facilitated by the *representational self*—a healthy way of interacting with families, peers, and communities. Specifically, if individuals are responded to and accepted within the cultural system, with their struggles and imperfections acknowledged, negotiated, and viewed as expected, then the self that they present to the cultural system develops as an accurate representation of their real self, promoting attunement (Cook-Cottone, 2006). Prevention efforts focus on supporting and constructing a representational self that facilitates attunement within the cultural system as well as a simultaneous and ongoing maintenance of self-awareness and self-care, including nutrition, hydration, exercise, self-soothing, rest, and medication if prescribed.

In the ARMS, ED symptoms emerge when influences (i.e., risk factors) from the self-system and cultural system individually or cumulatively create opportunities for misattunement. For example, a child may believe that she or he must be the way others want, expect, or need her or him to be in order to be accepted. The focus turns to external demands and expectations—the self is constructed to be in harmony with what the external system wants, expects, or needs (i.e., the self "I should be," "wish I could be," or "you need me to be"; Cook-Cottone, 2006, p. 227), and the child is left without an experience of attunement. It is this void that allows ED symptoms to emerge, to fill this space. ED behaviors could become important and functional in the regulation of thoughts, emotions, and physiology. The child becomes disproportionately attuned to these symptoms, creating a self-perpetuating and self-reinforcing disorder. The challenge for prevention, then, is to pinpoint and build the protective factors within the self- and cultural systems so that initial or prolonged misattunement does not occur.

Protective Factors of ED-Related Outcomes

Based on a thorough review of the literature, the following variables have been found to serve as protective factors for ED-related outcomes. We organized these protective factors within the ARMS's interlocking self- and cultural systems. Unless otherwise noted, the sample composition is predominantly White girls or women from the United States.

Protective Factors within the Self-System of the ARMS

The self-system consists of events occurring inside an individual, including cognitive, affective, and physiological factor subsystems. The self-system also has to process external events from the cultural system, such as societal pressures to be thin (see Chapters 31, & 35). Therefore, protective factors in the self-system help people negotiate their thoughts, emotions, and physical needs while successfully adapting to and functioning within their relationships, the community, and culture. Empirically supported ED protective factors (subsystem factors in parentheses) in the self-system include the following.

Body Appreciation (cognitive, affective) Body appreciation includes acceptance of, favorable opinions toward, and respect for the body (Avalos, Tylka, & Wood-Barcalow, 2005). Body acceptance inversely predicted subsequent ED attitudes and behaviors 4–5 years later for a large sample of early adolescent girls (Gustafsson et al., 2009), and 5 years later for an ethnically and socioeconomically diverse sample of overweight female and male adolescents (Neumark-Sztainer, Wall, Story, & Sherwood, 2009). Body appreciation negatively correlated with ED symptoms among cross-sectional samples of early adolescent girls (McVey, Pepler, Davis, Flett, & Abdolell, 2002), college women and men (Tylka & Kroon Van Diest, 2013), and adult community women (Augustus-Horvath & Tylka, 2011). Using an experimental design with college women from the United Kingdom, Halliwell (2013) found that high levels of body appreciation protected women from wanting to change their appearance when exposed to advertisements featuring thin female models, and this protective effect was present even if women had previously internalized the thin-ideal appearance standard. In qualitative research, adolescent girls and boys from Sweden (Frisén & Holmqvist, 2010; Holmqvist & Frisén, 2012) and a sample of African American and White college women (Wood-Barcalow, Tylka, & Augustus-Horvath, 2010) identified body appreciation as protective against ED-related outcomes.

Awareness of and Responsiveness to Internal Hunger and Satiety Cues (physiological, cognitive) The ability to be aware of internal hunger and satiety cues and respond by trusting and eating according to these cues (i.e., intuitive eating) has been found to be inversely related to ED symptoms, body dissatisfaction, and internalization of societal ideals among cross-sectional samples of college women (Tylka, 2006; Tylka & Kroon Van Diest, 2013). In a cross-sectional sample of early adolescent girls and boys, trust in and responsiveness to (but not awareness of) these cues were inversely associated with ED-related outcomes (Dockendorff, Petrie, Greenleaf, & Martin, 2012). A 24-week intervention designed to heighten participants' awareness of and responsiveness to these cues decreased many ED-related outcomes at a 2-year follow-up for a sample of middle-aged women (Bacon, Stern, Van Loan, & Keim, 2005).

Body Functionality Via (Certain Types and Levels of) Sport Participation (physiological, cognitive, affective) Considering the type and level of sports, Smolak, Murnen, and Ruble (2000) conducted a meta-analysis of 34 studies and found that participation in nonlean sports (i.e., sports that do not require a lean body to be competitive) at nonelite levels (e.g., high school) was linked to lower ED-related outcomes for women. Thus, nonelite and nonlean sports may provide girls and women with a sense of pride that is separate from appearance and help them focus on what their body can do rather than how it looks or being "the best" at a sport. As a cautionary note, participation in some sports that encourage thinness to improve performance

(e.g., ballet, aerobics, cheerleading) and elite sports were found to be risk factors for EDs (see Chapter 35). Smolak et al. (2000) observed significant heterogeneity within the analyses, indicating that other factors (e.g., personality, body acceptance, or thinness-related pressure by coaches and parents) rather than the sport itself, may contribute to ED protection or risk.

Constitutional Thinness (physiological) A lower body mass index (BMI) has been found to be protective against ED symptoms and related outcomes 4–5 years later for Swedish adolescent girls (Gustafsson et al., 2009), 7 years later for Swedish adolescent girls (Westerberg-Jacobson, Edlund, & Ghaderi, 2010; Westerberg-Jacobson, Ghaderi, & Edlund, 2012), and 1 year later for middle-school and high-school girls but not boys (Jones, 2004).

The protective effect of being naturally thin on girls' ED-related outcomes is likely due to Western culture aligning thinness with desirability, success, and attractiveness coupled with stigmatizing individuals who are heavy (Bacon, 2010). Specifically, those who are heavy are more susceptible to receiving weight loss messages targeted at them, which could create misattunement and place them at greater risk for EDs (Cook-Cottone, 2006). In contrast, thin individuals are more likely to experience body acceptance by others (or no commentary on their body) based on their natural conformity to societal appearance standards, which reinforces attunement (Augustus-Horvath & Tylka, 2011). Thus, rather than promoting weight loss for females who are not thin, professionals need to demand size diversity and acceptance in media and encourage these characteristics within interpersonal encounters.

Self-Esteem (affective, cognitive) High self-esteem was found to protect against ED-related outcomes for (a) adolescent girls from Sweden after a 4–5-year period (Gustafsson et al., 2009); (b) White, Hispanic, and Asian (but not Black or American Indian) adolescent females and White, Hispanic, Asian, and Black (but not American Indian) adolescent males in a large cross-sectional study (Croll, Neumark-Sztainer, Story, & Ireland, 2002); and (c) British women, where self-esteem was assessed during childhood and anorexia nervosa (AN) symptoms were assessed at age 30 (Nicholls & Viner, 2009). In addition, self-esteem acted as a protective factor by buffering the relationship between body dissatisfaction and ED symptoms among college women (Twamley & Davis, 1999).

Studies examining the effectiveness of interventions designed to improve self-esteem as a protective factor against EDs, however, have produced mixed results. An intervention program focused on building self-esteem was found to improve the body image of girls and boys, particularly among those at high risk for eating problems, as well as lower their drive for thinness, appearance concerns, dieting attempts, and unhealthy weight control behaviors after the intervention and at 1-year post-intervention (O'Dea & Abraham, 2000). However, the effectiveness of this program was not evidenced in another sample using this same program (Wade, Davidson, & O'Dea, 2004). Another self-esteem-based intervention program improved ED-related outcomes at post-intervention but the gains were not maintained a year after the study (McVey, Davis, Tweed, & Shaw, 2004).

Self-Compassion (affective, cognitive) Different conceptually than self-esteem, self-compassion involves being aware of painful emotions during challenges (e.g., hardship, failure, evaluative threats) while also generating kindness and understanding toward oneself by perceiving these challenges as part of the larger human experience (Neff, 2003). Activating and/or practicing self-compassion appears to reduce ED-related outcomes in both clinical samples of ED patients and nonclinical samples. More specifically, patients with EDs who had greater increases in

self-compassion early in ED treatment had faster decreases in ED-related outcomes over 12 weeks, even when pretreatment ED symptoms were controlled (Kelly, Carter, & Borairi, 2014). Community women who received an online 3-week self-compassion meditation training program experienced greater reductions in ED-related outcomes and greater body appreciation, and maintained these improvements at a 3-month follow-up relative to a wait-list control group (Albertson, Neff, & Dill-Shackleford, 2014). Among college women who restrict their eating, those who were induced to think self-compassionately after eating a doughnut as part of the experimental task (i.e., they were told that all people eat unhealthy foods at times and asked not to be hard on themselves because "this little amount of food doesn't matter anyway") were able to reduce their distress and disinhibited eating relative to a control group who did not receive the self-compassion induction (Adams & Leary, 2007, p. 1129). Following an experimental writing task in which college women discussed their perceived appearance-related flaws, higher state self-compassion predicted lower state body shame and lower anticipated ED behaviors for the following week (Breines, Toole, Tu, & Chen, 2014). Among community women, self-compassion buffered the relationships between perceived media pressures for thinness and ED-related outcomes (Tylka, Russell, & Neal, 2015).

Emotional Well-Being and the Ability to Regulate Emotions (affective, physiological, cognitive) Emotional well-being was found to be a protective factor for ED-related outcomes among White, Hispanic, Black, and Asian adolescent females and males as well as American Indian males (but not American Indian females; Croll et al., 2002). A combination of mindfulness-based interventions and skills for emotion regulation and distress tolerance, such as those found in dialectical behavioral therapy (Marcus & Levine, 2004; see also Chapter 57), were protective in the amelioration of binge eating disorder (BED), as these skills managed the urge to binge eat (Kristeller & Hallett, 1999; Leahey, Crowther, & Irwin, 2008). After an 8-week mindfulness-based ED treatment group, six college women with bulimia nervosa (BN) described their transformation from disembodiment, emotional distress, and self-loathing to cultivating an inner connection with themselves that resulted in greater self-awareness, acceptance, and compassion, and an improved ability to manage stress (Proulx, 2008). Further, dispositional mindfulness and thought suppression predicted fewer BN symptoms among college women and men (Lavender, Jardin, & Anderson, 2009), as well as lower body comparison and higher body satisfaction among Dutch women (Dijkstra & Barelds, 2011).

Yoga (physiological, affective) Yoga entails moving the body mindfully through a series of poses and stretching and balancing the body while in a single pose (Daubenmier, 2005). Also, by utilizing specific breathing techniques, yoga produces physical and mental relaxation as well as mind-body awareness and responsiveness (Scime, Cook-Cottone, Kane, & Watson, 2006). A 10-week ED primary prevention program for fifth-grade girls based on yoga and guided relaxation/visualization resulted in lower body dissatisfaction, drive for thinness, and media influence at posttest (Scime et al., 2006). The effects of this program were replicated in a larger sample of fifth-grade girls with similar effects—at posttest, girls experienced lower body dissatisfaction and fewer BN symptoms (Scime & Cook-Cottone, 2008). Adult women currently taking yoga classes reported lower self-objectification, body dissatisfaction, and ED symptoms than women taking aerobics classes (Daubenmier, 2005).

One study found that participation in yoga/Pilates (these were not measured separately) was protective for women's body dissatisfaction but not ED symptoms, and yoga/Pilates was a risk factor for men (Neumark-Sztainer, Eisenberg, Wall, & Loth, 2011). Cook-Cottone et al. (2013) asserted that yoga may be protective only under certain characteristics; for instance, the

exercise setting and instructor do not promote weight loss and instead emphasize the functionality of and appreciation for the body, and the participants' motive for engaging in yoga is not weight or appearance control. Thus, it is important that yoga prevention programs do not focus on weight/body size, but instead body function, body appreciation, self-compassion, how to extend yoga to assist with coping and life skills, and health (Cook-Cottone, 2013).

Neurobiological Functioning (physiological) Extensive research has indicated that there are brain pathways, neurocircuits, and chemical reactions that malfunction in persons with EDs (Hill et al., 2012; see also Chapters 17 & 30). For instance, those with AN and BN have a dulled sense of taste and hunger (Kaye, Fudge, & Paulus, 2009; Wagner et al., 2010). Hill et al. (2012) reported the following protective neurobiological process that individuals *without* EDs experience. When confronted with a tasty food, their anterior insula helps determine whether they are hungry. If they are and they take a bite, their anterior insula produces initial pleasure, the amygdala experiences calmness, the nucleus accumbens produces further pleasure, the orbitofrontal cortex determines that they like the food, the caudate nucleus articulates the pros and cons of taking another bite, the anterior cingulate cortex decides to take another bite, and the dorsolateral cortex anticipates that they can eat more. Their parietal cortex and lateral fusiform gyrus evaluate their external body accurately due to sufficient neural activity (i.e., blood oxygen level) in these regions (those with EDs have lower neural activity in these regions, which is believed to contribute to body distortion and a negative body evaluation; Uher et al., 2005). Also, experiencing a dopamine spike when making a decision (eating-related and non-eating-related) is likely protective, as it provides an emotional sense of pleasure and confirms that the decision was (or is going to be) good. In those with AN and BN, the dopamine spike appears weak or dulled after decision-making (Hill et al., 2012).

Self-Determination/Autonomy (cognitive) Individuals high in self-determination are motivated to regulate their behaviors through choice as an expression of themselves; that is, they pursue their own interests and not what others dictate for them (Deci & Ryan, 1985). Self-determination was related to fewer reports of pressures to be thin and BN symptoms; lower body dissatisfaction, endorsement of society's beliefs about thinness and obesity, and eating restriction; and higher autonomous regulation of eating behaviors (Pelletier & Dion, 2007; Pelletier, Dion, & Levesque, 2004). Women low in self-determination, after seeing a video emphasizing the thin ideal, perceived greater media pressure to be thin, and reported higher body dissatisfaction and concerns over quantity of food intake, whereas those who were high in self-determination valued food quality (Mask & Blanchard, 2011). Nonconformity, a construct similar to self-determination, buffered the link between exposure to thinness norms and internalization of the thin ideal for college women (Twamley & Davis, 1999).

Perceived Acceptance from a Higher Power (cognitive, affective) Having a secure attachment to God (i.e., viewing God as loving and accepting rather than judgmental, punitive, and rejecting) was linked to lower levels of pressure to be thin, thin-ideal internalization, body dissatisfaction, and dieting among college women (Homan & Boyatzis, 2010). Further, a secure attachment to God interacted with both pressure for thinness and internalization of the thin ideal to mitigate their connection to body dissatisfaction. Interviews with college women who endorsed a positive body image affirmed these findings (Wood-Barcalow et al., 2010). Feeling loved and accepted by a higher power, and believing that the higher power designed their bodies to be unique for a reason, created a sense of duty to take good care of and appreciate their bodies.

Persistence (physiological, cognitive) Persistence, a component of temperament, reflects the ability to maintain attention and persist with a task. In a large sample of children, parent-reported child persistence assessed at five time points was inversely related to girls' drive for thinness and BN symptoms in most concurrent and prospective analyses (Martin et al., 2000). Persistence at only one time point (7–8 years) was inversely related to boys' drive for thinness; other ED-related outcomes were not related to boys' persistence at any time point.

Protective Factors within the Cultural System of the ARMS

The cultural system includes family, community, and wider-cultural factor subsystems that interface with the self-system. Protective factors in the cultural system aid in developing and maintaining attunement within a person's thoughts, emotions, and physical needs. Empirically supported ED protective factors (subsystems in parentheses) in the cultural system include the following.

Positive Family Relationships (family) Several studies have explored family relationships as a protective factor against the development of ED symptoms. Specifically, positive parent-child relationships, a sense of connectedness among family members, and perceived parental support have been found to protect against ED symptom development among preadolescent and adolescent females and males (Croll et al., 2002; Fonseca, Ireland, & Resnick, 2002; McVey et al., 2002; Mellin, Neumark-Sztainer, Story, Ireland, & Resnick, 2002; Scoffier, Maiano, & d'Arripe-Longueville, 2010; Snapp, Hensley-Choate, & Ryu, 2012). Additionally, three longitudinal studies found that family connectedness, spending more time with parents, perceiving parents as friendly, and feeling closer to parents reduced the risk of ED-related outcomes among adolescents 1–5 years later (Byely, Archibald, Graber, & Brooks-Gunn, 2000; Neumark-Sztainer et al., 2009; Swarr & Richards, 1996).

Family meals (family) Regular family meals have been found to be protective against ED-related outcomes among adolescents cross-sectionally (Neumark-Sztainer, Wall, Story, & Fulkerson, 2004; White, Haycraft, & Meyer, 2014) and longitudinally over a 5-year period (Neumark-Sztainer, Eisenberg, Fulkerson, Story, & Larson, 2008; Neumark-Sztainer et al., 2009). In addition, frequent family meals have been associated with healthier eating behaviors and higher overall psychological well-being among adolescents (Neumark-Sztainer, Larson, Fulkerson, Eisenberg, & Story, 2010; White et al., 2014), lending further support to the positive benefits of eating meals as a family unit on a regular basis. In addition to the frequency of family meals, the atmosphere during meals is important to consider. Specifically, pleasant mealtimes were inversely associated with ED-related outcomes for adolescent girls, and anxiety and depression for adolescent girls and boys (White et al., 2014).

Media Literacy (community) Media literacy efforts, or education about the negative influence of the media-proposed ideal body, have been associated with fewer ED-related outcomes, including body dissatisfaction, thin-ideal internalization, and appearance comparison among adolescent females (McLean, Paxton, & Wertheim, 2013). Teaching media literacy skills to adolescent girls may help protect against their desire to appear like media-portrayed models who are dangerously thin and engaging in fad diets (Levine & Smolak, 2006) via encouraging girls to reject this thin-ideal body type (Smolak, 2011). Media literacy is often one of several components within ED prevention programs, and these programs have varied in their

ability to reduce ED-related outcomes (Stice, Shaw, & Marti, 2007). One ED prevention program that centralizes media literacy within its content has been effective in reducing ED-related outcomes for sixth- to eighth-grade girls and boys, even 2.5 years after completing the program (Wilksch & Wade, 2009). This program, Media Smart, contains eight sessions that teach students about advertisements, stereotypes (e.g., messages that media reveal about how we should structure our lives), idealized images presented for women and men (and of different races) in the media, and how to translate what they have learned into their daily lives (Wilksch & Wade, 2009).

Voicing Opposition Against the Thin Ideal (community) Programs such as the Body Project (Stice, Rohde, & Shaw, 2013) teach participants ways to oppose societal appearance norms through a series of verbal, written, and behavioral exercises where participants actively critique societal appearance ideals. Exercises include having participants engage in role plays in which they encourage others to resist societal appearance ideals and write counterattitudinal statements about costs associated with attaining societal appearance ideals. These exercises often result in cognitive dissonance, a state of psychological discomfort, for participants who equate beauty/attractiveness with thinness. Participants will be then be motivated to reduce this uncomfortable dissonance and will likely change their attitudes to fit their behaviors. Much empirical support upholds the ability of programs containing cognitive dissonance-producing exercises to lower ED-related outcomes in adolescent girls and college women and reduce their risk of developing future ED symptoms (Stice, Chase, Stormer, & Appel, 2001; Stice, Shaw, Burton, & Wade, 2006).

Body Acceptance by Others (family, community, culture) When women perceive that their body shape/size is accepted by others (peers, family, partners, society, and media), they experience higher body appreciation (Augustus-Horvath & Tylka, 2011; Avalos & Tylka, 2006), which, in turn, is protective against ED-related outcomes (Gustaffson et al., 2009; McVey et al., 2002). Body acceptance from family members buffered the relationship between exposure to thinness norms and internalization of these norms among women (Twamley & Davis, 1999).

Feminist Influences (community, family) Feminism encourages women to recognize that objectification, harassment, and pressures placed on them to adhere to the thin-ideal body type are forms of discrimination against women (Murnen & Smolak, 2009). Feminism also provides women with an option to define themselves based on nonphysical characteristics, such as intelligence, creativity, and resilience, as a form of empowerment. A meta-analysis demonstrated that women who identify as feminists are less likely to experience thin-ideal internalization, body shame, body dissatisfaction, and ED symptoms (Murnen & Smolak, 2009). Effect sizes for feminism as a protective factor increase in accordance with women's age, which may be due to their length of time exposed to feminism and adhering to these ideals (Murnen & Smolak, 2009). In addition, an advanced feminist identity (reflecting synthesis and an active commitment to feminism and societal change) was found to buffer the relationship between experiencing sexist events and ED symptoms among college women (Sabik & Tylka, 2006).

Although a feminist identity provides an alternative way to counter harmful cultural ideologies of women's bodies and specific strategies to resist these ideologies, a feminist identity alone is likely not enough to protect against ED-related outcomes (Rubin, Nemeroff, & Russo, 2004). During focus groups, feminist women revealed that they embraced a diversity

of body sizes and shapes and reframed deleterious cultural messages aimed at their bodies; however, these cognitive strategies were limited and insufficient to counter their emotional attachment to cultural messages about beauty (Rubin et al., 2004). This study provides evidence that the ED field must work toward cultural-level change to reduce objectification, harassment, and appearance-related pressures, not just individual-level change.

Future Research in Protective Factors

There is much clinical value in strengthening barriers that protect against ED-related outcomes. In order to develop comprehensive interventions to strengthen barriers, we need to know how protective variables influence one another to build these barriers. The ARMS (Cook-Cottone, 2006; Cook-Cottone et al., 2013) could be a useful guide for this research.

Because attunement is a dynamic process, reflected in the influence of protective factors between the self- and cultural systems as well as within each system, researchers need to study it as such. First, factors in the self-system can be strengthened by factors in the cultural system, thereby increasing attunement. For instance, body acceptance by others may strengthen body appreciation, positive family relationships may strengthen self-compassion, pleasant family meals may strengthen awareness and responsiveness to internal hunger and satiety cues, and media literacy may strengthen body appreciation. Second, the self-system could impact the cultural system, thus increasing attunement. For example, self-determination and autonomy could direct women to seek out feminist influences (e.g., taking a Women's Studies course as an elective) and participate in media literacy initiatives. Third, factors within each subsystem can strengthen each other, further enhancing attunement. Examples can include yoga and nonelite/nonlean sport participation strengthening body appreciation, body acceptance by family members enhancing family relationships, self-compassion strengthening body appreciation, and feminist influences contributing to media literacy and to opportunities to voice opposition against the thin ideal.

Whereas some of these associations have been investigated, more rigorous studies on how they mutually or unilaterally shape each other over time in the prediction of ED-related outcomes are needed. One particularly influential example of this type of research is that mindfulness meditation, including yoga and use of emotion regulation skills, is associated with neuroplastic changes in several brain regions implicated in EDs (e.g., the insula and anterior cingulate cortex, as evidenced in functional and structural neuroimages; Hölzel et al., 2011). These changes include enhanced self-regulation and interoceptive awareness.

To study attunement as a process, we could extend ED research efforts toward exploring "upward spirals," or combinations of protective factors that trigger self-perpetuating cycles and trajectories of positive psychological growth (Garland et al., 2010). Upward spirals have been studied in the field of positive affect, such as Fredrickson's (2001) broaden-and-build theory, where positive emotions broaden cognition, positive coping, and interpersonal trust (i.e., expanding people's mindsets), which then build behavioral flexibility and personal resources such as mindfulness, resilience, social closeness, and physical health. To study these upward spirals, Fredrickson used methods such as experimental designs (e.g., evoking positive emotion through mindfulness), prospective designs (e.g., investigating whether initial levels of positive affect build durable personal resources over time), mediation models (e.g., determining whether initial positive affect predicts subsequent positive affect through ongoing experiences of coping), and interventions (e.g., studying the effectiveness of compassion

meditation on positive affect). In ED research, Fitzsimmons and Bardone-Cone (2011) studied a "downward spiral," whereby initial weight concern predicted subsequent weight concern through body surveillance. ED researchers exploring protective factors could use this reciprocal-influence design (Burns et al., 2008) to study, for instance, whether initial levels of body appreciation predict subsequent body appreciation through other protective factors such as yoga, awareness and responsiveness to internal hunger and satiety cues, and mindfulness and/or self-compassion interventions.

Researchers have not yet explored whether protective factors disrupt the mediational chain in which a risk factor operates. Protective factors can be integrated as moderators within empirically supported models of ED-related outcomes, such as the Tripartite Influence model (Thompson, Coovert, & Stormer, 1999). Tests of moderated mediation can be used to investigate whether a protective factor disrupts mediational chains within the model. For example, would body appreciation disrupt the influence of media appearance-related pressures on body dissatisfaction through decreasing thin-ideal internalization?

We also encourage researchers to study protective factors in ways they have not yet been investigated. For instance, self-compassion may prevent the initial occurrence of ED-related outcomes. If supported, professionals may want to include self-compassion exercises within primary prevention programs. Also, additional research needs to explore self-compassion as a buffer between cultural risk factors, such as appearance-related pressures, and ED-related outcomes. Given that self-compassion is beneficial when experiencing perceived failures or shortcomings, self-compassion could facilitate how individuals respond to cultural risk factors, such as refusing to engage in body disparaging conversations with peers. This response could then temper their own distress, thereby preventing these cultural influences from creating ED risk.

Conclusions and Future Directions

Scholars are becoming increasingly involved in strength-focused and prevention-based research within the ED field. In this chapter, we have used the ARMS (Cook-Cottone, 2006), a biopsychosocial model grounded in attunement (mind-body awareness and responsiveness) and factor interconnectedness, to situate this research. Examining this research revealed protective factors that appear to:

1 **Decrease ED-related outcomes directly.** Interventions designed to help individuals become aware of and respond to internal hunger and satiety cues, evoke self-compassion during self-evaluative threats, engage in yoga, learn emotional regulation and mindfulness skills, become media literate, and voice opposition against the thin ideal appear to reduce ED-related outcomes.
2 **Prevent the initial occurrence of ED-related outcomes.** Appreciating the body, being aware of and responding to internal hunger and satiety cues, participating in nonlean/nonelite sports, being naturally thin, having emotional well-being and mindful awareness, having a brain neurocircuitry that functions well along with neurotransmitters in an appropriate balance, possessing self-determination, perceiving acceptance from a higher power, showing persistence with tasks, being media literate, receiving regular family meals in a pleasant environment, experiencing body acceptance by others, and having feminist influences appear to protect against the initial occurrence of ED-related outcomes.

3 **Interact with an ED risk factor to interrupt its deleterious effects.** Self-esteem, nonconformity, acceptance from a higher power, body appreciation, self-compassion, and body acceptance by family members appear to buffer the link between cultural risk factors and ED-related outcomes.

Importantly, most studies presented in this review were conducted with samples of predominantly White girls and women. Therefore, we strongly encourage researchers to begin such explorations and continue to research protective factors as illustrated in 1–3 above with diverse samples, so the ED field better understands not only *which* factors are protective, but also *when*, *for whom*, and *how* they are protective to better promote and maintain attunement and healthy eating, prevent EDs, and rebuild health while treating EDs.

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34

Stress, Trauma, and Adversity as Risk Factors in the Development of Eating Disorders

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This chapter will review current knowledge of the role of stress, trauma, and adversity as risk factors in the development of eating disorders (EDs). EDs do not occur in a vacuum, and stress, trauma, and violence are important contextual factors that shape the course of our lives and the course of psychiatric disorders, including EDs.

Definition of Trauma

There have been a number of differing definitions of trauma that have been proposed and/or endorsed by a variety of professional organizations. After an extensive review, the Substance Abuse and Mental Health Services Administration (2014) recently published a working definition of trauma: "Individual trauma results from an event, series of events, or set of circumstances that is experienced by an individual as physically or emotionally harmful or threatening and that has lasting adverse effects on the individual's functioning and physical, social, emotional, or spiritual well-being." This definition has the advantage of including not only traumas of commission, which include a variety of adverse events or acts done to or against people, but also traumas of omission, which include emotional and physical neglect, or acts not done for people.

Trauma in Women with Eating Disorders

Interest in and appreciation for child abuse and maltreatment was revived in the 1970s, and in 1980 the American Psychiatric Association (1980) independently defined two new disorders in the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-III*)—posttraumatic stress disorder (PTSD) and bulimia—although they were not known to be related at the time. While Gerald Russell (1979) is often given credit for first describing bulimia nervosa (BN), which he depicted as "an ominous variant of anorexia nervosa" (p. 429), it was Marlene Boskind-Lodahl (1976) and Boskind-Lodahl and White (1978) who first described binge eating and purging

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in normal weight college-age women, a phenomenon they termed *bulimarexia* given the mixture of both anorexic and bulimic symptoms (see Chapter 3). Bulimarexia was described as a Western culture-bound syndrome that developed from society's preoccupation with female thinness as well as its patriarchal oppressive restrictions of female gender roles. Their etiological conceptualization was based on a feminist perspective in which bulimarexia was viewed as a pathological outcome of a society dominated by men in which the self-esteem and social status of women was overly dependent on physical attractiveness to men.

Five years after bulimia and PTSD entered the psychiatric nomenclature Oppenheimer, Howells, Palmer, and Chaloner (1985) published the first report of childhood sexual abuse (CSA) in association with EDs in a group of 78 patients presenting to their clinic. One year later Sloan and Leichner (1986) reported five cases with EDs and histories of sexual abuse or incest. Nonetheless, as late as 1992, the role of sexual abuse in girls and women (predominantly) by male perpetrators (predominantly) in the etiology of BN remained controversial (Pope & Hudson, 1992).

Using the National Women's Study (NWS) as a database (n = 4,004), Dansky, Brewerton, O'Neil, and Kilpatrick (1997) obtained detailed histories of crime victimization experiences, including completed rape, contact molestation, attempted sexual assault, and aggravated assault, as well as lifetime and current prevalence rates of PTSD, major depressive disorder (MDD), BN, binge eating disorder (BED), and substance abuse/dependence using *DSM-III-R* and *DSM-IV* (BED) criteria. They found a significantly greater frequency of rape in this randomly selected, nonclinical sample of women (n = 3,006) meeting criteria for BN (27%) compared to those that did not (13%, $p \leq .001$, χ^2). In addition, those women with a history of BN also had significantly higher frequency of contact molestation (22% vs. 12%, $p < .05$), as well as aggravated assault (27% vs. 8%, $p < .001$), than control women without BN. Overall, 54% of women with a history of BN endorsed a major crime victimization experience, that is, rape, molestation, attempted sexual assault, or aggravated assault, as opposed to 31% of women without BN ($p < .001$). As will be discussed later, in the NWS Dansky and colleagues (1997) also found significantly higher lifetime and current rates of PTSD in women with BN, as well as higher lifetime PTSD in women with BED, as compared to women without ED. Importantly, in women with BN and a history of rape, the age of first rape occurred *before* the age of first eating binge in 84% of all cases, in 96% of rapes occurring during adolescence (12–17 years old), and in 100% of rapes occurring during childhood (≤11 years old) (Brewerton, 2004). These findings provided substantial validity for the notion that victimization, especially during childhood and adolescence, is indeed a causative risk factor for BN, albeit a nonspecific one. To date the NWS endures as the most wide-ranging study of the relationship of trauma history and PTSD to EDs and psychiatric comorbidity (see Chapter 15).

Throughout the late 1980s and the 1990s other investigators also reported higher than expected rates of CSA in association with EDs, particularly BN. Several major reviews looking at the available studies unanimously concluded that CSA is indeed a nonspecific risk factor for the development of EDs, particularly those with bulimic symptoms (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004; Jacobi, Morris, & de Zwaan, 2004; Molinari, 2001; Smolak & Murnen, 2002; Wonderlich, Brewerton, Jocic, Dansky, & Abbott, 1997). With the report by Dansky and colleagues (1997) of the link between BN and aggravated assault, more and more investigators also focused their attention on studying the role of other forms of trauma in EDs. Soon it became clear that the spectrum of traumatic experiences linked to EDs extended well beyond CSA to include a variety of other forms of abuse and neglect.

A major longitudinal, prospective study of a large community-based sample of mothers and their offspring (n = 782) followed over an 18-year period widened the scope of traumatic experiences that can pre-date eating-disordered behaviors (Johnson, Cohen, Kasen, & Brook, 2002). It was the most comprehensive longitudinal study on childhood adversities, including CSA, physical abuse (PA), and neglect, and the later development of eating- or weight-related problems, including EDs. It further established CSA as an important predictor of BN and bulimic disorders, and in addition it elucidated the role of physical neglect in forecasting disturbed eating behaviors.

Mitchell, Mazzeo, Schlesinger, Brewerton, and Smith (2012) reported on the types of trauma experienced by both women and men with and without EDs who took part in the National Comorbidity Replication Survey. This was a major national survey of a representative sample of women and men across the United States who were assessed using structured interviews. Of the women with a lifetime history of BN (n = 45), 100% reported any type of trauma and 78% reported any type of interpersonal trauma. The women with BN reported significantly more exposure to life-threatening automobile accidents, beating by parents or guardians, beating by romantic partners, rape, sexual assault other than rape, stalking, vicarious trauma, serious physical fights in the home during childhood, witnessing someone else being injured or killed, or to having purposefully injured, tortured, or killed someone else (all ps < .01). Of the women with lifetime binge eating disorder (BED) (n = 75), 90% reported any type of trauma and 64% reported any type of interpersonal trauma. Women with BED reported significantly more exposure to life-threatening automobile accidents, beating by parents or guardians, sexual assault other than rape, stalking, and serious physical fights in the home during childhood (all ps < .01). Of the women with lifetime AN (n = 18), 100% reported experiencing any form of trauma and 71% reported exposure to interpersonal trauma. However, none of the chi-squared values reached significance at p < .01, likely due to low power. Nevertheless, these data show inordinately high rates of traumatic experiences in women with EDs, particularly those with bulimic EDs. In fact, the bulk of the data indicates that traumatic experiences are significantly more commonly associated with bulimic EDs, such as BN, BED and AN-Binge-Purge type, than nonbulimic EDs, such as AN-Restricting type.

Trauma and Eating Disorders in Children and Adolescents

Once it became established in the scientific literature that traumatic experiences were causative risk factors for the development of EDs in adult women, other studies were published that demonstrated similar findings for children and adolescents with EDs (Brewerton, 2006). These included studies with large sample sizes of adolescents from both community samples (Ackard & Neumark-Sztainer, 2002; Fonseca, Ireland, & Resnick, 2002; Neumark-Sztainer, Story, Hannan, Beuhring, & Resnick, 2000; Perkins & Luster, 1999) and national samples (Ackard, Neumark-Sztainer, & Hannan, 2003; Ackard, Neumark-Sztainer, Hannan, French, & Story, 2001; Edgardh & Ormstad, 2000). In addition, clinical investigators were continuing to document the prolonged and delayed nature of the damage inflicted upon young victims of CSA that persists long past the actual period of abuse (Swanston, Tebbutt, O'Toole, & Oates, 1997).

Wonderlich and colleagues (2000) reported the results of a controlled comparison study that compared 20 sexually abused girls with 20 age-matched nonabused girls between 10 and 15 years old. All of the girls completed a series of psychometric instruments, including the

McKnight Risk Factor Survey, the Kids' Eating Disorders Survey, and the Body Rating Scale for Adolescents. The abused participants reported higher rates of weight dissatisfaction, more dieting and purging behaviors, less food intake when emotionally upset, and had a greater desire for thinner body types. They were also less likely to exhibit perfectionistic tendencies than the nonabused girls. These findings further supported the contention that sexual abuse predisposes female children and adolescents to ED-related symptoms.

Trauma and Eating Disorders in Boys and Men

The link between trauma and EDs is not just a female phenomenon. A wealth of published findings has also established a strong link between trauma and EDs in both boys and men (Brewerton, 2004, 2005, 2006, 2007). Kinzl and colleagues (1997) were among the first investigators to report that long-lasting adverse familial relationships, especially in association with PA, increased the risk for EDs in men. Grilo and Masheb (2001) discovered that BED patients endorsed a broad range of childhood traumas that did not differ by gender or weight status. Past emotional abuse was associated with greater body dissatisfaction and depression, and lower self-esteem in men and women, while sexual abuse was associated with greater body dissatisfaction in men. Mitchell and Mazzeo (2005) reported that physical neglect and PA were related to disordered eating in a sample of 168 undergraduate men.

A report by Lipschitz, Winegar, Hartnick, Foote, and Southwick (1999) indicated higher than expected rates of EDs in inpatient adolescent males with PTSD. In a study of 9,943 Connecticut students in grades 7, 9, and 11, Neumark-Sztainer et al. (2000) described higher rates of disordered eating among both boys and girls who reported physical or sexual abuse. In the abused group, there were also low levels of family communication, parental caring, and expectations. The odds ratio (OR) for the development of disordered eating following sexual abuse was 1.99 for girls and 4.88 for boys, and following physical abuse it was 2.0 for girls and 1.95 for boys. These associations between disordered eating and trauma persisted even after adjusting for differences in psychosocial and familial variables.

Fonseca et al. (2002) also examined the role of CSA in relationship to extreme weight control measures in a large group of adolescents (n = 9,042) in Connecticut. A history of CSA significantly predicted extreme weight control behaviors (use of diet pills, vomiting, laxatives, or diuretics to lose weight) in boys (OR = 2.8, $p < .001$). Ackard et al. (2003) assessed the rates of self-reported date rape and violence in a very large sample of high-school boys and girls (n = 81,247) and showed significant links between these violent events and eating disorder behaviors, especially purging behaviors, in both males and females.

Mitchell and colleagues (2012) reported on the types of trauma experienced by both women and men with and without EDs who took part in the National Comorbidity Replication Survey. Of the men with a lifetime history of BN (n = 7), 100% reported high rates of any type of trauma and 100% reported any form of interpersonal trauma. Men with BN were significantly more likely to report exposure to combat, terrorism of civilians, toxins, beating by romantic partners, mugging, rape, stalking, unexpected death of someone close, vicarious trauma, serious physical fights in the home during childhood, witnessing someone else being injured or killed, or having accidentally or purposefully injured, tortured, or killed someone else (all ps < .01). Of the men with lifetime BED (n = 30), 98% reported any type of trauma and 74% reported any form of interpersonal trauma. Men with BED reported significantly more exposure to toxins, man-made disasters, beating by parents, beating by romantic partners, mugging, rape, stalking,

unexpected death of someone close, vicarious trauma, witnessing someone else being injured or killed, or having purposefully injured, tortured, or killed someone else (all *p*s < .01). Of the men with lifetime histories of AN (n = 3), 100% reported exposure to any trauma and 68.2% (weighted %) reported any interpersonal trauma. Men with AN reported significantly more exposure to terrorism of civilians, beating by romantic partners, and sexual assault other than rape (all *p*s < .01).

Multiple Episodes or Forms of Trauma and Eating Disorders

A number of published reports suggest that multiple episodes or types of traumatic experiences are associated with EDs. Using an adult population-based sample (n = 1,987), Schoemaker, Smit, Bijl, and Vollebergh (2002) observed that a history of multiple abuses appeared to be a specific risk factor for BN as well as "dual diagnosis disorder" (comorbid psychiatric + substance use disorder).

Ackard and Neumark-Sztainer (2003) reported that girls with a history of multiple types of abuse had statistically significant ORs for vomiting (4.1), laxative abuse (5.1), diet pill abuse (4.3), binge eating (2.2), fasting (2.3), and thinking about or attempting suicide (6.1). Likewise, boys with multiple forms of abuse had statistically significant ORs for laxative abuse (29.2), vomiting (24.2), diet pill abuse (17.3), thinking about/attempting suicide (9.5), binge eating (5.6), and fasting (2.3).

Trauma and Multiple Types of Compensatory Behaviors

Multiple types of compensatory behaviors are associated with a history of trauma and/or PTSD. Tobin and Griffing (1996) stated "abused patients reported engaging in more types of compensatory behavior, with abused patients reporting an average of two compensatory behaviors and nonabused patients reporting only one (p < .001)" (p. 146). Brewerton, Dansky, O'Neil, and Kilpatrick (2015) reported results from the NWS showing that the number of purging strategies employed by women with BN was positively and significantly correlated with lifetime rates of victimization, PTSD, major depression, alcohol abuse, alcohol dependence, and total number of comorbid.

Trauma and Eating Disorder Severity

Although trauma is not necessarily associated with greater severity of the ED, it appears to be associated with certain psychopathological features. In the comprehensive review by Wonderlich and coworkers (1997), a history of CSA was not found to be associated with greater eating disorder severity. This finding has been replicated in some but not all recent studies of the role of trauma in EDs. For example, in a sample of 23 women with BN, Hartt and Waller (2002) discovered no dimensional relationship between the severity of four types of reported child maltreatment (CSA, childhood physical abuse, childhood emotional abuse, and neglect), and bulimic symptomatology. What they did find was a positive correlation between overall abuse severity and the levels of dissociation as measured by Dissociative Experiences Scale (DES-II) scores (r = .40, p < .05). Neglect and sexual abuse accounted for the majority of the variance.

Brewerton, Dansky, Kilpatrick, and O'Neil (1999) also reported higher rates of dissociative symptoms in women with BN. Specifically, subjects with BN had significantly more "forgetting" or psychogenic amnesia of traumatic events (27%) than subjects with BED (12%) or those without an ED (11%, $p < .001$). Those who endorsed "forgetting" reported that they had forgotten all or part of one or more previous traumatic events. Multiple linear regression using "forgetting" as the dependent variable identified a number of significant variables (in decreasing order of significance): lifetime PTSD, childhood rape, lifetime major depression, molestation, emotional problems in the family, laxative abuse, the total number of victimization experiences, age (younger), and vomiting. Based on these and other data, it was hypothesized that vomiting and laxative abuse, as opposed to binge eating per se, are maladaptive behaviors very closely linked to PTSD and MDD that facilitate avoiding, numbing, and forgetting traumatic memories. Other investigators have also reported that certain psychopathological features are higher in abused patients versus nonabused patients, such as higher scores on interpersonal distrust and interoceptive awareness subscale scores on the Eating Disorders Inventory (EDI; Matsunaga et al., 1999).

On the other hand, Groleau and co-investigators (2012) reported that childhood emotional abuse predicted the severity of eating pathology through the mediating effects of ineffectiveness and affective instability. They reasoned that childhood emotional abuse might influence the severity of eating disorder symptoms by impacting an individual's self-esteem and one's self-directedness and capacity for affect regulation.

Trauma, PTSD and Eating Disorder Comorbidity

A wealth of data indicates that trauma is associated with significantly higher amounts of psychiatric comorbidity, which is often mediated by the occurrence of lifetime PTSD (Brewerton 2004, 2007; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). In both the National Comorbidity Study and the NWS, psychiatric comorbidity was highly associated with a history of serious victimization and especially when it resulted in the emergence of PTSD. The ORs for major (Axis I) disorders ranged between 2.4 and 4.5 in the NWS, and similarly increased ORs were found in the National Comorbidity Study. Trauma and resultant PTSD offer a lot of explanatory power as to why comorbidity is the rule rather than the exception in patients with EDs.

This line of investigation leads to the question of whether it is abuse per se or resultant PTSD that incurs the risk of developing an ED. The data clearly indicate that it is the reaction to trauma, that is, PTSD, rather than the trauma alone, that predicts the development of an ED. For example, the age of first eating binge (in BN) was significantly earlier in cases of rape resulting in PTSD compared to those with rape without PTSD or no rape (Brewerton, 2004, 2007). Most convincingly, the prevalence rates for BN were found to be significantly higher in subjects with rape histories with PTSD (10.4%) when compared to those with rape histories without PTSD (2.0%) or those with no rape history at all (2.0%) (Brewerton, 2004, 2007). This strongly suggests that PTSD rather than prior abuse per se best predicts the development of BN as well as possibly other ED-related comorbid conditions.

Results from the National Comorbidity Survey Replication have confirmed the very high rates of PTSD and comorbidity in a random, representative sample of individuals with BN, BED, or clinical binge eating (Hudson, Hiripi, Pope, & Kessler, 2007; see also Chapter 15). Lifetime prevalence rates of PTSD (with OR) were as follows: BN: 45.4% (OR = 10.2*);

AN: 12.0% (OR = 1.6); BED: 26.3% (OR = 5.1*); any binge eating: 20.2% (OR = 4.0*). Only those with an asterisk (*) had an OR significantly different from non-eating-disordered controls ($p \leq .01$).

Other evidence supports PTSD as an important mediator between trauma and EDs. A study of 71 victims of sexual trauma and 25 control subjects reported significant association between a history of trauma and ED symptoms, as well as a history of trauma and PTSD symptoms (Holzer, Uppala, Wonderlich, Crosby, & Simonich, 2008). In addition, it was found that PTSD significantly mediated the relationship between trauma and EDs. Holzer et al. (2008) concluded that clinical interventions for individuals with trauma and EDs may benefit from a focus on resolving PTSD symptomatology.

Trauma and Partial or Subthreshold PTSD

Partial or subthreshold PTSD (pPTSD) can be defined in any number of ways but all share the presence of significant PTSD symptoms in one or more of the three clusters (re-experiencing, hyperarousal, and avoidance), but fall short of the full *DSM-IV* criteria (American Psychiatric Association, 2000). Several authors have argued that PTSD boundaries are too strict and that individuals with partial syndromes are significantly at risk for higher rates of a number of PTSD-related conditions or outcomes (Brewerton, 2007; Mitchell et al., 2012). These include higher rates of panic attacks, dissociative symptoms, alcohol and other substance abuse, suicidality, poorer outcomes, higher utilization of healthcare resources, less sense of well-being and satisfaction with life, and higher disability in comparison to those with no PTSD. Subthreshold PTSD, and hence the importance of traumatic events, is therefore often misdiagnosed and overlooked. Similar to both EDs and PTSD, pPTSD occurs more frequently but not exclusively in women. Partial or subthreshold PTSD may also be a risk factor for BN and bulimic symptoms (Brewerton, 2007; Mitchell et al., 2012).

Trauma and Body Dissatisfaction

Preti, Incani, Camboni, Petretto, and Masala (2006) investigated the role of body dissatisfaction as a mediator between sexual abuse and ED symptoms in a community sample of 126 young women. Those who reported CSA reported significantly more ED symptoms on a number of self-report measures than those who denied abuse. The experience of body dissatisfaction was found to be a significant mediating or intervening variable between sexual abuse and resultant eating disorder symptoms. The authors hypothesized that revulsion about the body stemming from CSA interacts with the development of body size and shape concerns.

Dunkley, Masheb, and Grilo (2010) examined the role of self-criticism and both symptoms of depression and body dissatisfaction in 170 patients with BED. Both emotional abuse and sexual abuse were specifically associated with body dissatisfaction, and statistical analyses demonstrated that self-criticism fully mediated the relationship between emotional abuse and both depression and body dissatisfaction. This study identifies self-criticism as a potential mechanism through which certain forms of child abuse may be associated with the later development of body dissatisfaction and depression in EDs.

Eubanks, Kenkel, and Gardner (2006) studied the relationships between physical, emotional, and sexual abuse up to adolescence and subsequent body size perception and body esteem in

38 college undergraduate women, one half of whom had experienced abuse. Those with abuse histories had significantly more distorted body size perceptions, worse body esteem, and more negative parenting experiences.

In another study, self-harming patients with a history of CSA scored significantly higher on measures of body dissatisfaction, EDs, suicidal ideation, physical abuse/neglect, and emotional abuse/neglect than self-harming patients without CSA (Murray, MacDonald, & Fox, 2008). The authors concluded that CSA is an extremely formidable trauma that often has harsh effects on an individual, not only in terms of self-harming behavior but also in terms of developing a wide range of maladaptive comorbid problems in conjunction with self-harm.

Muehlenkamp, Claes, Smits, Peat, and Vandereycken (2011) tested their conceptual model explaining the high co-occurrence of nonsuicidal self-injury (NSSI) in eating-disordered patients as resulting from a combination of childhood maltreatment, low self-esteem, psychopathology, dissociation, and body dissatisfaction in a sample of 422 young women consecutively admitted to an inpatient unit for EDs. Their results indicated that childhood maltreatment appeared to have a significant, indirect relationship to NSSI, and that dissociation and body dissatisfaction are especially relevant to understanding and treating NSSI in ED patients.

Eating Disorders and Sensitivity to Trauma

Eating disorder patients may be particularly sensitive or vulnerable to stress and its consequences. It is well known that individuals who develop EDs more often than not had preexisting or primary anxiety disorders, including PTSD (Bulik, Sullivan, Fear, & Joyce, 1997; Deep, Nagy, Weltzin, Rao, & Kaye, 1995; Godart, Flament, Lecrubier, & Jeammet, 2000; Kaye, Bulik, Thornton, Barbarich, & Masters, 2004). This predisposition to anxiety results in a heightened perception of and reaction to threat, as well as an increased sensitivity to stress, trauma, and adversity. This has been shown in a number of ways experimentally. Strober (2004) argued persuasively that patients with EDs have a propensity to extreme fear conditioning and greater than normal resistance to its extinction. McFillin and colleagues (2012) demonstrated that those with EDs are more likely to perceive hostile intent in human faces than matched controls. In addition, ED patients make more facial emotion recognition (FER) errors, often overinterpreting fear as anger (Ridout, Wallis, Autwal, & Sellis, 2012). They characteristically exhibit high levels of anxiety sensitivity characterized by fear of loss of control (Fulton et al., 2012). They are typically overconcerned with consequences and have exaggerated inhibition (Kaye, 2008) and exaggerated anticipatory anxiety (Oberndorfer, Kaye, Simmons, Strigo, & Matthews, 2011). They also manifest high punishment sensitivity (Harrison, O'Brien Lopez, & Treasure, 2010; Harrison, Treasure, & Smillie, 2011; Jappe et al., 2011). Neuropsychological studies have shown patients with AN to have impaired flexibility, decreased set-shifting (Roberts, Tchanturia, Stahl, Southgate, & Treasure, 2007; Roberts, Tchanturia, & Treasure, 2010), increased sensitivity to uncertainty (Frank et al., 2012), as well as weak central coherence (Lopez, Tchanturia, Stahl, Booth et al., 2008; Lopez, Tchanturia, Stahl, & Treasure, 2008, 2009). As a result they often miss seeing the "forest for the trees" or the "big picture" and get hung up or stuck in relative trivialities. As a result they are less adaptable to stressors, traumas, and adverse life circumstances.

The effects of a stressor or traumatic experience and whether it leads to or contributes to an eating or related disorder clearly depend on the individual's response and adaptation to that stressor or trauma. Avoidant coping and emotional coping are associated with intrapunitive

(self-punishing) cognitions and appear to increase the chances of developing an ED. In contrast, task or active coping does not increase risk and is associated with a better prognostic outcome (McFillin et al., 2012; Pamies & Quiles, 2012; Smith-Jackson, Reel, & Thackeray, 2011).

Trauma and Neurobiology

It has been reported that serotonergic brain systems are specifically involved in modulating adaptive responses to aversive events (Corchs, Nutt, Hood, & Bernik, 2009; Graeff, Guimaraes, De Andrade, & Deakin, 1996; Krystal & Neumeister, 2009). It is certainly conceivable, based on a wealth of available evidence, that early traumatic events interact in complex neurodevelopmental ways to produce long-term changes in affected brain systems, including the hypothalamic-pituitary-adrenal (HPA) axis, neuropeptides, and the monoamine neurotransmitters, including serotonin (see Chapter 67). There is evidence for an association between a history of sexual abuse, impulsivity, self-destructiveness, and reduced serotonin function in at least a subset of ED patients (Steiger, Gauvin et al., 2001; Steiger, Koerner et al., 2001; Steiger, Young et al., 2001).

Trauma, Eating Disorders, and Genetic Polymorphisms

Individuals with bulimia-spectrum disorders have been shown to exhibit heterogeneous profiles of comorbid psychiatric disorders that are not only in part due to varying degrees of environmental vulnerability but also to genetic vulnerability (see Chapter 28). Research exploring the interactional effects involving polymorphisms of specific neurotransmitter system genes, such as the serotonin transporter promoter polymorphism (*5-HTTLPR*), and selected forms of childhood abuse acting upon the severity of binge eating and related psychopathological systems has emerged over the last several years. A genetic polymorphism is the occurrence together in the same population of more than one allele or genetic marker at the same locus.

Steiger and colleagues (2008) at McGill University, where most of this type of work has taken place, reported the finding that bulimic *5-HTTLPR* S-allele carriers who had prior childhood sexual or physical maltreatment show elevations on personality measures of dissocial behavior, which is overrepresented in those with bulimia-spectrum disorders. This work validated previous observations concerning phenomenological correlates of traumatic stress in *5-HTTLPR* S-allele carriers. Richardson and coworkers (2008) reported that women with *DSM-IV* bulimia-spectrum disorders who had high degrees of comorbidity, including major depression, anxiety disorder, and substance use disorders, more conduct problems, and higher dieting preoccupations, showed greater likelihood of carrying the *5-HTTLPR* S allele and of childhood abuse than did the group of bulimic women with low degrees of comorbidity. These findings are consistent with prior findings characterizing a subgroup of women with bulimia-spectrum disorders with high degrees of psychiatric comorbidity, and suggest that the *5-HTTLPR* polymorphism and childhood trauma may both be pertinent to explaining the presence of greater psychopathology in bulimia-spectrum disorders.

Groleau and co-investigators (2012) explored the interactional effects involving several dopamine system gene polymorphisms, including that of the dopamine-2 receptor (*DRD2*), the dopamine transporter (*DAT1*), and catechol-*O*-methyltransferase (*COMT*). Sensation-seeking was elevated in carriers of the low-function allele of the *DRD2* Taq1A polymorphism

who also reported CSA, relative to that in individuals showing other combinations of alleles and abuse exposures. In addition, carriers of a low-function allele of *COMT* scored higher on compulsivity, lower on impulsivity, and marginally lower on frequency of binge eating than did individuals in whom the allele was absent. These findings indicate that genes acting within the dopamine system may contribute, either directly or indirectly (i.e., in interaction with traumatic childhood experiences), to variations in the presentation of comorbid traits and, possibly, of bulimic symptoms.

Steiger and associates (2011) also documented other gene–environment interactions between the glucocorticoid receptor polymorphism, *Bcl1*, and childhood maltreatment in women with BN. Compared to normal control women without BN, women with BN were significantly more likely to endorse histories of childhood maltreatment, to have the low-function *Bcl1* C allele (CG or CC genotypes), as well as to have both factors. Steiger et al. interpreted their results as suggesting that traumatic stress, when impacting persons predisposed to lower glucocorticoid receptor modulation, can be etiological for BN. In addition, Steiger's group (2012) has demonstrated that the *Bcl1* × child abuse interaction is decreased when levels of depression are accounted for, but is not affected by controlling for affective instability, sensation-seeking, or motoric impulsivity. In summary, their findings suggest that stress-induced alterations in glucocorticoid sensitivity contribute to both BN and depressive disturbances but not to the behavioral or affective dysregulation seen in many individuals with BN.

Trauma During Pregnancy and Birth

Traumatic experiences may even occur during prenatal and perinatal periods of life when vulnerabilities to stress are highest. It is certainly conceivable that such early traumas may be the very first psychobiological events that could trigger the development of subsequent EDs. Although one study did not find any association between prenatal and perinatal trauma and the subsequent development of EDs (Feingold, Sheir-Neiss, Melnychuk, Bachrach, & Paul, 2002), other studies have found significantly higher rates of such histories in individuals with EDs. In a large population-based study, Foley, Thacker, Aggen, Neale, and Kendler (2001) reported that prenatal or perinatal complications were associated with a significantly greater risk for developing BN later on in life. It was remarkable that these difficulties were specific for BN.

In one well-controlled study, Favaro, Tenconi, and Santonastaso (2006) revealed that patients with either AN or BN had significantly higher rates of labor and delivery complications than controls. It is noteworthy that all of the early insults reported involved the impairment of blood flow or oxygenation to the fetus.

Trauma, Course of Illness, and Treatment Outcome

Clinical experience and available scientific data strongly suggest that trauma and PTSD or its symptoms must be satisfactorily addressed and sufficiently resolved in order to facilitate *full* recovery from the ED and its associated comorbidity (see Chapter 57). Brewerton (2004) has hypothesized that trauma may serve as an "organizing principle" in terms of understanding causation from a developmental and biopsychosocial perspective. The greater the degree of psychiatric comorbidity that is manifested in any given patient, the greater the likelihood that prior serious trauma played a role in instigating and perpetuating the overall course of mental

illness. As advances in the neurobiology of mental illness have progressed (see Chapters 17 & 30), it has become clear that trauma-related disorders may share common underlying factors that account for their associations. These likely include: (a) common cognitive schemas involving issues of self-esteem, control, self-criticism, shame, and guilt; and (b) dysregulation in neuropsychobiological mechanisms, which are triggered by gene expression and may underlie affective dysregulation.

As described by Brewerton (2004), the body itself may become a trigger for trauma-related memories, thoughts/beliefs, affects, and behaviors. Thus, the body, or a certain body part, may become an object to be avoided, denied, forgotten, and/or abused. In particular, sexualized areas of the body, such as breasts, abdomen, thighs, and buttocks (which are higher in fat content than many other body parts) may become anxiety cues and are to be avoided or "gotten rid of." "I am fat" may translate to "I am anxious about (my) body/body parts/sexuality," or "My body is bad" or "I am bad."

PTSD tends to be a chronic disorder in that approximately one third of individuals with the condition still meet full criteria a decade later (Kessler et al., 1995). Its toll on human suffering is immense in all spheres measured: psychological, medical, economic, social, interpersonal, familial, spiritual, and so forth. PTSD has been reported to be an indicator of poor prognosis for mood disorders, substance use disorders, and other anxiety disorders, and it is likely to be indicative of treatment nonresponsiveness in ED as well (Agras et al, 2000; Brewerton 2004, 2007; Cachelin et al., 1999; Gleaves & Eberenz, 1994; Rodriguez, Perez, & Garcia, 2005; Sohlberg, Norring, Holmgren, & Rosmark, 1989; Vrabel, Hoffart, Ro, Martinsen, & Rosenvinge, 2010).

Conclusions and Future Directions

Taken together, it is clear that stress, trauma, and adverse life experiences are important risk factors for the development and maintenance of EDs, especially EDs with bulimic symptoms. However, there has been little or no recognition of this reality in prevention efforts against EDs. Future strategies to prevent EDs will do well to focus on the prevention of violence, especially interpersonal violence, including all forms of child maltreatment, as well as the teaching of active coping strategies to effectively manage life's potentially overwhelming challenges.

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Section VI
Special Groups

35

Athletes, Physical Activity, Dancers, and Eating Disorders

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Athletes generally are viewed as paragons of physical health, fitness, and well-being due to the general leanness and muscularity of their bodies, their levels of physical activity, and their abilities to accomplish incredible physical feats, singly or in combination with others. Despite the very real health benefits associated with being fit and active, athletes are not immune to the physical and psychological distress that sometimes results from intense physical training and participation in competitive environments. In fact, due to their involvement and training within sport, athletes may be at increased risk for experiencing physical injuries (acute and overuse), becoming psychologically "burned out" by their training, and developing an unhealthy relationship between their bodies, their weight, and how much (and what) they eat. As athletes work to find the right balance between the needed caloric intake for their energy output, and the ideal body size, shape, muscularity and weight for their sport, some may resort to pathogenic weight control behaviors, such as excessively exercising (aerobic and weight training), extreme dieting, and even vomiting, to manage this balance. And, for those athletes whose bodies are not naturally lean or muscular, or are far different from general societal ideals (e.g., women being thin but not overly muscular), the pressures to change physically may be even greater. Over time these pressures, in combination with the use of unhealthy weight-control behaviors, may lead to the development of subclinical or clinical eating disorders (EDs) that can disrupt physical performances, lead to psychological distress (e.g., depression, low self-esteem), and isolate athletes from their teammates and coaches.

In this chapter, we review the prevalence of EDs (and related conditions) among athletes, discuss unique factors within sport and performance environments that can contribute to athletes being body- and weight-focused, and identify the psychosocial variables that have been related to various ED indices through empirical research in sport environments. We conclude by offering suggestions for future research.

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Prevalence of Eating Disorders and Related Problems among Male and Female Athletes

Two meta-analyses have demonstrated that, in general, male and female athletes score higher on a range of ED indices, such as drive for thinness and bulimic symptomatology, than non-athletes (Hausenblas & Carron, 1999; Smolak, Murnen, & Ruble, 2000). The effect sizes from these analyses, however, have been small and marked by considerable variability, which suggests that prevalence may vary due to a range of factors, such as sport level, sport type, and how eating disorders are measured.

Eating Disorders

Based on self-report measures (Anderson & Petrie, 2012; Greenleaf, Petrie, Carter, & Reel, 2009; Johnson, Powers, & Dick, 1999) and, in some cases clinical interviews (Martinsen & Sundgot-Borgen, 2013; Sundgot-Borgen & Torsveit, 2004), prevalence rates for female athletes have ranged from 0% to 6.7% (anorexia nervosa; AN), 0% to 12.1% (bulimia nervosa; BN), and 2% to 13.4% (eating disorders not otherwise specified; ED-NOS) in samples drawn from the more advanced levels of sport competition (i.e., collegiate and national/international levels). Prevalence rates for male athletes at the same competitive levels have been slightly lower (Johnson et al., 1999; Martinsen & Sundgot-Borgen, 2013; Petrie, Greenleaf, Reel, & Carter, 2008; Sundgot-Borgen & Torsveit, 2004): AN (0%), BN (0% to 7.5%), and ED-NOS (0% to 9.7%). In a review of three studies that included samples of elite Norwegian female athletes from 1990/1991 to 2001/2002, Sundgot-Borgen and Torstveit (2010) reported a significant increase from 20% to 28% in clinical EDs across that timeframe, though the exact reason for the change was not known.

Overall, prevalence tends to be higher for elite as opposed to collegiate athletes and for female, in comparison to male, athletes (Anderson & Petrie, 2012; Greenleaf et al., 2009; Johnson et al., 1999; Petrie et al., 2008; Sundgot-Borgen & Torsveit, 2004). However, within samples of male and female collegiate athletes (Anderson & Petrie, 2012; Greenleaf et al., 2009; Petrie et al., 2008; Sanford-Martens et al., 2005) prevalence rates generally have not varied significantly as a result of year in school, age, or race/ethnicity, though Greenleaf et al. (2009) found that fewer minority female athletes (14%) were classified as symptomatic/eating disordered than nonminority female athletes (31.8%). Sport team classification (e.g., ballgame, power sports, aesthetic) may be related to ED prevalence for elite male and female athletes (Sundgot-Borgen & Torstveit, 2004), but not collegiate athletes (Greenleaf et al., 2009; Petrie et al., 2008).

Fewer studies have been conducted on adolescent athletes' prevalence, and recent research findings have been equivocal regarding athletes' risk in comparison to nonathletes (Martinsen, Bratland-Sanda, Eriksson, & Sundgot-Borgen, 2010; Martinsen & Sundgot-Borgen, 2013; Nichols, Rauh, Barrack, Barkai, & Pernick, 2007; Rosendahl, Bormann, Aschenbrenner, Aschenbrenner, & Strauss, 2009). For example, using the EAT-26 (score ≥10), Rosendahl et al. (2009) reported 26.7% of female athletes (vs. 36.1% nonathletes) and 10.4% of male athletes (vs. 12.3% nonathletes) were considered to be at-risk. However, Martinsen and Sundgot-Borgen (2013) found that, based on clinical interviews, male and female Norwegian adolescent elite athletes had higher prevalence rates of EDs than age-matched nonathlete controls.

The prevalence of clinical EDs does appear to be higher among more elite athletes (i.e., national/international, collegiate) in comparison to nonathletes, and higher in female

compared to male athletes, and may vary based on sport or weight classification, particularly at the elite level. At less elite levels, such as in high-school sports, athletes' prevalence may be lower than that found among nonathlete controls.

Subclinical Eating Disorders and Pathogenic Weight Control Behaviors

Prevalence rates for subclinical disorders and individual pathogenic weight control behaviors are far higher than clinical designations for male and female athletes. In independent mixed-sport samples, a substantial number of male (16.0% to 21.2%; Chatterton & Petrie, 2013; Petrie et al., 2008; Sanford-Martins et al., 2005) and female (14.5% to 25.5%; Greenleaf et al., 2009; Sanford-Martins et al., 2005) collegiate athletes were classified as subclinical (i.e., symptomatic). Subclinical prevalence rates have ranged from 20.9% to 28.9% in female collegiate swimmers/divers and gymnasts, respectively, as well (Anderson & Petrie, 2012). Similarly, among National Collegiate Athletic Association (NCAA) Division I, II, and III female athletes, 20.1% were classified as subclinical based on scores across several ED indices (Williams et al., 2003); rates, however, did not vary in relation to sport classification or Division level. At the national/international level, 1% of male and 4% of female athletes met the diagnostic criteria for anorexia athletica (AA), which is determined by low body weight, problematic eating, and excessive concern about body shape and weight (Sundgot-Borgen & Torstveit, 2004). Athletes also engage in binge eating, though no study has examined rates for binge eating disorder (BED). For example, male (6.8%) and female (7.8%) collegiate athletes indicated eating uncontrollably to the point of stuffing themselves at a frequency of two or more times per week (Chatterton & Petrie, 2013; Greenleaf et al., 2009). To date, no studies have examined, in depth, how athletes may progress from subclinical to clinical EDs.

Athletes use various behaviors to control their weight, particularly exercise and dieting. For example, across samples drawn from different collegiate sports (Chatterton & Petrie, 2013; Darcy, Hardy, Lock, Hill, & Peebles, 2013; Greenleaf et al., 2009; Petrie et al., 2008), athletes reported exercising specifically to burn calories (men 15.7% to 37%; women 20% to 25.5%) and dieting or fasting two or more times per year (men 10% to 14.2%; women 15.6%). Among German high-school athletes (elite and nonelite levels), more girls (33%) than boys (15.3%) had dieted (Rosendahl et al., 2009). Regarding extreme weight control methods (Chatterton & Petrie, 2013; Darcy, Hardy, Lock, et al., 2013; Greenleaf et al., 2009; Petrie et al., 2008), collegiate athletes indicated that they vomited one or more times per week (men 0.9% to 5%; women 2.5% to 3.7%), used diuretics two or more times per month (men 1.5% to 7%; women 1.5%), and took laxatives two or more times per week (men 0.45% to 7.9%; women 0.4% to 1%). The reliance on exercise and diet as athletes' primary forms of weight control is consistent with training and performance demands. These two behaviors can easily be "hidden" within sport environments or viewed as indicators of athletes' motivation. Athletes also may know that severe forms of weight control, such as vomiting and laxatives, can have serious side effects and interfere with their ability to train and ultimately perform at their best.

Other Eating-Related Syndromes

In addition to clinical and subclinical EDs, some athletes experience related psychological conditions, including female athlete triad (Triad), muscle dysmorphia (MD), and exercise dependence. The Triad occurs in women and involves the interrelation of low energy availability, menstrual function, and bone mineral density. Weight control efforts, such as reducing

caloric intake and increasing exercise, often result in reduced energy availability, which can disrupt menstrual functioning and lower bone mineral density (American College of Sports Medicine [ACSM], 2007; Rauh, Nichols, & Barrack, 2010). The Triad may lead to increased risk of injury (e.g., stress fractures), medical complications (e.g., infertility), and psychological distress (e.g., depression). Prevalence rates for the co-occurrence of all three conditions are generally low (Coelho et al., 2013; Nichols, Rauh, Lawson, Ji, & Barkai, 2006; Reel, SooHoo, Doetsch, Carter, & Petrie, 2007; Torstveit & Sundgot-Borgen, 2005), ranging from 1.2% (high school) to 2.7% (college) to 4.3% (elite). Rates are higher, though, when only two of the conditions are present, ranging from 5.9% to 27% amongst high school, college, and elite athletes. Rates are highest among athletes from lean-build, endurance, and aesthetic sports.

MD involves an extreme preoccupation with a perceived lack of muscularity and distorted body image (Olivardia, 2001; Pope, Gruber, Choi, Olivardia, & Phillips, 1997; see also Chapter 37) and is now a specifier within Body Dysmorphic Disorder (American Psychiatric Association, 2013). Individuals with MD perceive and fear that their bodies are small and weak, even though in reality they are of average or above average musculature (Grieve, Truba, & Bowersox, 2009; Leone, Sedory, & Gray, 2005). They often engage in excessive exercise and weight training, have highly restrictive eating routines, hide or cover up their physiques, and take nutritional supplements (and even steroids) to increase their musculature (Murray, Rieger, Touyz, & de la Garza Garcia, 2010; Pope et al., 2005). MD is associated with psychological distress, including depression, anxiety, and suicidal ideology (McFarland & Kaminski, 2009; Pope et al., 2005). MD is more prevalent among men than women and develops primarily during late adolescence (Murray et al., 2010) when boys may experience a great deal of social pressure to attain a muscular and powerful body. Reported prevalence rates for MD vary, ranging from 8% (Pope, Katz, & Hudson, 1993) to 25% (Maida & Armstrong, 2005) among body builders and weightlifters; risk appears to be greatest among body builders and weightlifters (Maida & Armstrong, 2005).

Exercise dependence is characterized by three or more of the following symptoms (Cook & Hausenblas, 2008; Hausenblas & Symons Downs, 2002):

1 exercise tolerance;
2 withdrawal symptoms, such as anxiety and depression, when unable to exercise;
3 intentions to limit exercise;
4 feeling out of control when unable to exercise;
5 spending considerable time exercising;
6 experiencing conflict with other social, work, and family obligations or activities because of time spent exercising; and
7 continuing to exercise even when ill or injured.

"Primary" exercise dependence occurs without the presence of disordered eating; when it co-occurs with pathological eating, it is considered "secondary" (Adams, 2009; Cook & Hausenblas, 2008). In male and female college students who are enrolled in fitness classes, 21% to 62% report some exercise dependence symptoms, but few are at risk (3.6% to 5%) and even fewer are actually dependent (Garman, Hayduk, Crider, & Hodel, 2004; Symons Downs, Hausenblas, & Nigg, 2004). Symptoms of exercise dependence may be more common among men than women (Symons Downs et al., 2004).

Sport and Performance Environments as Risk Factors

Sport and performance environments, with their unique characteristics, have been identified as the primary reason that certain athletes may be at increased risk of developing and experiencing disturbed eating and body-related attitudes and behaviors (Petrie & Greenleaf, 2012). Coach and teammate pressures, idealization of specific body types, body revealing uniforms, sport demands, competitive pressures, and personality characteristics associated with being a "good" athlete can make athletes more vulnerable.

Team Climate: Coach and Teammate Pressures About Weight, Body, and Muscularity

Within sport and performance settings, social influences and pressures can contribute to athletes' beliefs and behaviors about their body weight and physique. Coaches, because of their position of power and authority, play a particularly important role, directly and indirectly communicating messages regarding expectations about weight, body, and appearance. Coach behaviors, such as weighing athletes, assessing body composition, and/or making weight-related comments, communicate that weight is central and that a lean, toned, fat-free body is desired. Such pressures may influence athletes to internalize the idea that weight and body size are paramount and develop (or reinforce already existing) disturbed eating and weight attitudes and behaviors. Teammates' attitudes and behaviors related to weight and physique also are influential and may be adopted by athletes wishing to fit into the team culture or be like another athlete.Research confirms the uniqueness of the associations between coach and teammate pressures and disordered eating attitudes and behaviors (Stirling & Kerr, 2012). For example, after controlling for general sociocultural pressures about body size and weight, coach and teammate pressures were associated with higher levels of restrained eating and bulimic symptomatology among female collegiate athletes (Reel, Petrie, SooHoo, & Anderson, 2013) and more drive for muscularity and bulimic symptoms (Galli, Reel, Petrie, Greenleaf, & Carter, 2011) and greater dietary restraint and internalization (Galli, Petrie, Reel, Chatterton, & Baghurst, 2014) among male collegiate athletes. Qualitative studies have documented that coaches and teammates do influence athletes' eating, weight, and body attitudes (Arthur-Cameselle & Quatromoni, 2010; Kerr, Berman, & De Souza, 2006; Muscat & Long, 2008; Stirling & Kerr, 2012). For example, female athletes served as role models, even when it came to unhealthy eating and weight control behaviors (Arthur-Cameselle & Quatromoni, 2010). Pressures from teammates and coaches about appearance and weight can negatively affect athletes' body-and weight-related beliefs and behaviors, which can interfere with sport performances.

Sport Body Ideals

In some sports, a particular physique or body shape is thought to be "ideal" and to confer some performance advantage (Stirling & Kerr, 2012). For example, a thin, petite frame is thought to benefit figure skaters, particularly in jumps and spins. However, power lifters are expected to have a large, muscular body capable of quick, dynamic movements. Sport-specific body ideals can contribute to body dissatisfaction when athletes believe their bodies do not fit the idealized sport physique. Athletes in sports where a smaller, thinner body shape is considered ideal may engage in restrictive eating behaviors (e.g., limiting caloric intake) and excessive exercise to burn calories. For example, female college cheerleaders (Torres-McGehee, Monsma, Dompier, & Washburn, 2012) and female adolescent athletes (Rosendahl et al., 2009) wanted

to weigh 3.4 and 3.0 kg (7.5 and 6.6 lb), respectively, less than their current weights and engaged in self-induced vomiting, dieting, and excessive exercise, to control their weight.

Athletes, particularly males, in sports where a powerful body is considered advantageous may take dietary supplements and muscle building substances and engage in strenuous strength training to increase body mass and muscularity. In a study of college football, Steinfeldt, Gilchrist, Halterman, Gomory, and Steinfeldt (2011) found that players wanted to be more muscular to have a performance advantage and conform to social body ideals. Similarly, male college athletes reported desiring a more muscular physique, and more than 13% indicated using weight gain supplements (Raudenbush & Meyer, 2003). Athletes, because of their desire to succeed and excel, may be particularly susceptible to expectations about sport "ideals," and thus engage in unhealthy behaviors in order to reach the ideals.

Uniforms

Sport uniforms, particularly those that are revealing and/or form-fitting, often contribute to athletes' bodies being on display for evaluation and critique. Some uniforms and performance attire are designed to enhance or facilitate performance. Swimsuits, for example, although body-hugging, are designed to help athletes maximize their performances and thus have a functional purpose. On the other hand, the bikinis worn by female beach volleyball players (in comparison to the baggie trunks/shirts worn by men) and the tight spandex uniforms worn by female volleyball players seem to offer no real performance advantage and simply serve to display, and sexualize, female athletes.

Wearing tight and body-hugging uniforms has been reported as a factor contributing to female athletes' body dissatisfaction, which may in turn act as a risk factor for pathogenic weight control behaviors. Female collegiate cheerleaders, for example, reported more body dissatisfaction in relation to revealing uniforms compared to when they wore a more modest uniform (Torres-McGehee et al., 2012). Stirling and Kerr (2012) found that revealing attire was something female athletes believed increased their vulnerability to disordered eating. Similarly, female collegiate volleyball players felt self-conscious because their bodies were on display, which distracted them from their performance (Steinfeldt, Zakrajsek, Bodey, Middendorf, & Martin, 2013). Among male collegiate athletes, uniform pressures were highest for those athletes participating in endurance sport athletes, such as cross country and swimming (Galli et al., 2014). Uniforms not only heighten body consciousness, which can contribute to body dissatisfaction and pathogenic weight control behaviors, but they can negatively affect performance.

Sport Type

A number of different sport type categories may have heightened risk of EDs, including lean sports (i.e., sports in which a lean physique is considered advantageous, such as distance running and swimming), aesthetic sports (i.e., sports in which appearance and attractiveness are evaluated as part of the performance, such as figure skating and gymnastics), and weight-class sports (i.e., sports that require meeting weight criteria in order to be eligible to compete within a specified weight class or division, such as wrestling and judo). Many athletes and coaches believe that weight loss (and a low body-fat percentage) leads to improved performances, particularly for lean sports, even though there is limited empirical support (Bonci et al., 2008; Thompson & Sherman, 2010). Thus, coaches may pressure athletes to restrict their food intake and/or increase their levels of physical activity to create a caloric deficit and weight loss. Such pressures

as well as dietary restraint have been linked to EDs, including bulimic symptomatology, among female athletes (Anderson, Petrie, & Neumann, 2011; Stirling & Kerr, 2012).

Athletes in aesthetic sports also may be at increased risk of disordered eating because of pressures to have an attractive appearance that appeals to judges and fans. Sundgot-Borgen (1994) found that 40% of athletes in aesthetic sports had high scores on a measure of disordered eating attitudes. Among athletes in figure skating, gymnastics, ballet, roller skating, diving, and rhythmic gymnastics, wanting a thinner body in order to improve sport performance predicted disordered eating 1 year later (Krentz & Warshburger, 2013). Van Durme, Goossens, and Braet (2012) reported that female figure skaters and dancers had higher levels of eating pathology than adolescents from the general population. Finally, in a sample of female collegiate athletes primarily comprised of gymnasts, Anderson et al. (2011) found that the more dissatisfied the athletes felt about their body size and shape, which was associated with experiencing pressures about appearance from coaches and teammates, the more bulimic symptoms the athletes reported.

In their review, Sundgot-Borgen and Garthe (2011) provided evidence that dangerous dieting practices and disordered eating are part of the social norms for athletes in weight-class sports. Athletes in sports such as wrestling and judo have reported using saunas, rubber suits, spitting, and diuretics and laxatives to purposefully dehydrate, and extreme dieting, self-induced vomiting, and laxative use in order to lose weight (Alderman, Landers, Carlson, & Scott, 2004; Artioli et al., 2010). These types of weight control behaviors are dangerous and can have severe health consequences for athletes (Sundgot-Borgen & Garthe, 2011; see also Chapter 14).

Competitive Level

The association between competitive level and disturbed eating attitudes and behaviors is unclear, though highly competitive and elite athletes may be more likely to use pathogenic behaviors compared to athletes at lower competitive levels (Rosendahl et al., 2009). Elite performers, for example, are expected to be at the top of their game. Because their bodies are the vehicles through which performance is accomplished and because they may believe that a leaner body is more functional (and related to better performances), these athletes may engage in extreme behaviors to manage their weight and physique. Meta-analytic research supports this contention (Hausenblas & Carron, 1999; Smolak, et al., 2000).

Conversely, because highly competitive athletes and performers spend a great deal of time training and thus have high levels of fitness, they may be more satisfied with their bodies and thus be at lower risk than less competitive athletes and nonathletes (Hausenblas & Downs, 2001). Even so, recent research has shown that over the course of a competitive season, pressures from coaches and teammates about weight and body can lead to increases in body dissatisfaction among female collegiate athletes (Anderson, Petrie, & Neumann, 2012), which in turn has been related to higher levels of bulimic symptomatology (Anderson et al., 2011). The degree to which competitive level, and its resulting effects on body and physique, increases (or decreases) athletes' vulnerability to disordered eating attitudes and behaviors deserves more study.

Athletic Personality

Finally, some of the personality characteristics associated with athletes' success, such as being "coachable" (i.e., follows instructions without question), willing to excessively train or exercise, achievement-oriented, and perfectionistic, as well as having a strong work ethic, also may increase

athletes' risk of developing pathogenic eating and weight control behaviors (Thompson & Sherman, 1999). Female athletes have identified personality characteristics that they believed increased their susceptibility to developing an eating disorder, including (Stirling & Kerr, 2012): self-absorption (i.e., focus on training, diet, and appearance), achievement-orientation (i.e., disciplined, conscious of eating and weight), perfectionism (i.e., high expectations for appearance and weight loss and fear of weight gain), hypercompetitiveness (i.e., body image and weight management comparison), need for control (i.e., ability to ignore hunger), and enjoyment of pain (i.e., hunger as rewarding). Among female athletes, perfectionism (e.g., personal standards, concern about mistakes) has been found to be directly related to higher levels of bulimic symptomatology as well as to strengthen the relationship between body dissatisfaction and bulimic symptomatology (Brannan, Petrie, Greenleaf, Reel, & Carter, 2009).

Psychological Correlates of Eating Disorders and Disordered Eating

Broadly, the sport environment and the unique factors just discussed have been identified as reasons why athletes might become more focused on their bodies and engage in behaviors (sometimes unhealthy) that are designed to substantially change their leanness and/or muscularity. Petrie and Greenleaf (2012) embedded the potential influence of the sport environment into a larger socioculturally based model (see Chapter 21) to explain the development of disordered eating among athletes. They suggested that general sociocultural pressures along with body and weight pressures unique to the sport environment have independent effects on athletes' internalization of societal appearance ideals, body dissatisfaction, drive for muscularity, dietary intent, and/or negative affect. These psychosocial variables, in turn, were hypothesized to increase athletes' susceptibility to developing disordered eating attitudes and behaviors (see Figure 35.1).

This model, based on research with nonathletes (Stice, 2002), was developed to guide researchers in testing a range of psychosocial variables to determine their relative influence in increasing athletes' risk of developing disordered eating symptoms. A "risk" factor is defined as a psychosocial, environmental, and/or physical variable that increases the probability of an individual developing an ED (Stice, Ng, & Shaw, 2010), and can only be determined through either longitudinal or experimental studies where the variable is shown to precede (and contribute to) the onset of the disorder (Stice, 2002). Within athlete eating disorder research, unfortunately, the majority of studies have been cross-sectional, thus allowing primarily for a discussion of the psychosocial correlates of eating disorders.

As discussed earlier in the chapter, expectations from coaches about body size/shape and weight, messages from teammates about food and eating, judges' comments and messages about appearance, and revealing and/or tight-fitting uniforms, to name a few, may contribute to the development of body image concerns, unhealthy eating choices, and pathogenic weight control behaviors. Specifically, among male and female collegiate athletes, pressures from coaches, teammates, and from the sport environment in general about weight, appearance, and performance have been associated with higher levels of body dissatisfaction, dietary restraint, and bulimic symptomatology (de Bruin, Oudejans, & Bakker, 2007; Galli et al., 2014; Reel et al., 2013). In a longitudinal study examining the influence of the college sport environment, Anderson et al. (2012) found that pressures about weight, dieting, and appearance experienced at the beginning of the athletic season predicted increases in the female athletes' body dissatisfaction at the end of the season; the athletes' dietary intent was unaffected

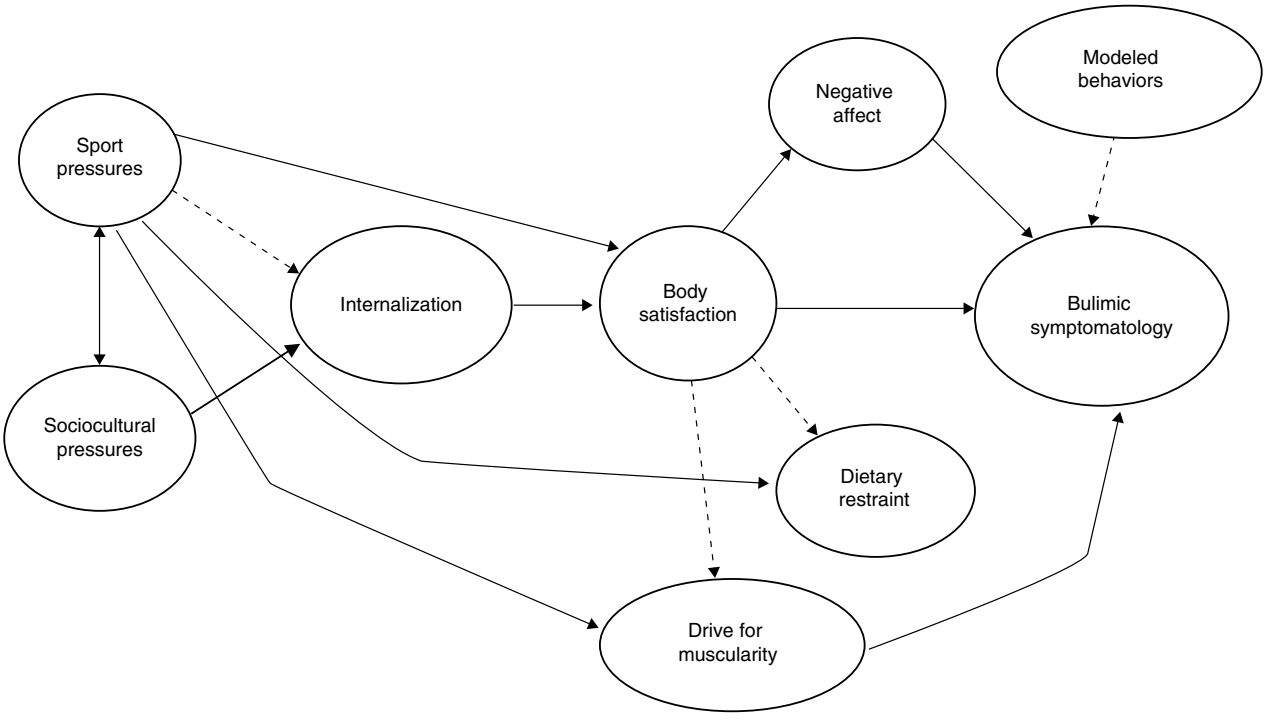

Figure 35.1 Sociocultural model of disordered eating for male and female athletes. Solid lines indicate pathways that have been supported in empirical studies; dashed lines suggest theoretically, but not yet empirically, supported relations amongst those constructs. From Petrie, T. A., & Greenleaf, C. (2012). Eating disorders in sport. In S. Murphy (Ed.), *The Oxford Handbook of Sport and Performance Psychology* (pp. 635–659). Oxford University Press. Reprinted with permission.

by these pressures. These studies have established that sport pressures exist for both male and female athletes, are unique from the general sociocultural pressures about appearance that athletes also experience, are correlated with different ED indices (e.g., dietary intent, bulimic symptomatology), and are a risk factor in the development of body dissatisfaction.

For female athletes, researchers have established empirically a number of psychological correlates of disordered eating. For example, general psychosocial variables, such as dietary intent/restraint (Anderson et al., 2011; Greenleaf et al., 2010; see also Chapter 24), drive for thinness (Hinton & Kubas, 2005; Krane, Stiles-Shipley, Waldron, & Michalenok, 2001), social physique anxiety (Hausenblas & Mack, 1999; Krane et al., 2001), negative affect (e.g., sadness; Anderson et al., 2011; Greenleaf et al., 2010; Petrie, Greenleaf, Reel, & Carter, 2009; see also Chapter 32), internalization of general societal appearance and body ideals (Petrie, 1993; Petrie et al., 2009; see also Chapters 21 & 29), overweight perceptions (Haase, 2011), body image concerns (e.g., body dissatisfaction, body esteem; Anderson et al., 2011; Brannan et al., 2009; de Bruin, Oudejans, Bakker, & Woertman, 2011; Ferrand, Champely, & Filaire, 2009; Greenleaf et al., 2010; Hinton & Kubas, 2005; Krane et al., 2001; Petrie et al., 2009; see also Chapter 22), perfectionism (Brannan et al., 2009), self-esteem (Berry & Howe, 2000; Brannan et al., 2009; Engel et al., 2003; Petrie et al., 2009), appearance orientation (i.e., how invested individuals are in improving their appearance; Petrie et al., 2009), and ego goal orientation (de Bruin, Bakker, & Oudejans, 2009), to name a few, have been related to ED indices including bulimic symptomatology and/or anorexic symptomatology. Sport- and exercise-specific variables, including sport anxiety (Holm-Denoma, Scaringi, Gordon, Van Orden, & Joiner, 2009), sport position (i.e., being a "flyer" in cheerleading; Torres-McGehee et al., 2012), exercising to improve appearance (Brannan et al., 2009; Petrie et al., 2009), and perceived athletic competence (Kipp & Weiss, 2013), also have been empirically supported as correlates of disordered eating. Consistent with nonathlete research, body image concerns, self-esteem, and dietary behaviors (and drive for thinness) have demonstrated the strongest and most stable associations with disordered-eating indices.

Although Petrie and Greenleaf's (2012) model was developed to apply to all athletes, few empirical studies have been conducted to examine the validity of these (and other) potential correlates in samples of male athletes, so much less is known about their experiences. For example, in one study that sampled male collegiate wrestlers, being "in" or "out" of season was found to be related to their drive for thinness scores, such that when wrestlers were in season and preparing for meets, they reported having a higher psychological desire to be thin and lose weight (Dale & Landers, 1999). In a mixed-sport sample of male collegiate athletes, scores on the Eating Disorder Inventory (EDI) bulimia subscale were related to higher levels of body dissatisfaction, ineffectiveness, interpersonal distrust, interoceptive awareness, and maturity fears (Petrie, 1996). Similarly, feelings of sadness and stress as well as a fear of becoming fat were associated with higher levels of bulimic symptomatology among male collegiate athletes (Petrie, Greenleaf, Carter, & Reel, 2007). A study by Terry and Waite (1996) reported significant correlations between Eating Attitudes Test (EAT) scores and body shape concerns among male lightweight and heavyweight rowers. Based on the existing research, it is unclear to what extent the variables identified in the Petrie and Greenleaf (2012; see Figure 35.1) model are valid for male athletes. Recent studies (Galli et al., 2011, 2014; Petrie et al., 2007), however, suggest that sport-specific pressures about appearance and weight and body image concerns may be important for explaining the disordered eating experiences of male athletes.

Conclusions and Directions for Future Research

Research on athletes, sport environments, disordered eating, and their correlates has lagged behind studies conducted in nonathlete populations, though in the last decade considerably more (and more theoretically based) studies have been undertaken. Although nonathlete investigations can provide direction for studies within sport environments, researchers will want to consider the unique factors discussed within this chapter when planning their projects and incorporate the following recommendations in their studies of athletes and EDs:

1 Early prevalence studies were hampered by the use of unvalidated measures and by invariance in measurement between athlete and nonathlete groups (Darcy, Hardy, Crosby, Lock, & Peebles, 2013). Although more recent studies (e.g., Petrie et al., 2009) have addressed that limitation by using self-report questionnaires designed to provide an ED diagnosis, these studies still are limited by generally small samples that are not population-based. Only a few studies (Martinsen & Sundgot-Borgen, 2013; Sundgot-Borgen & Torsveit, 2004) have incorporated clinical interviews in their protocol to validate diagnosis and provide a more accurate measure of prevalence. Further, recent research (Darcy, Hardy, Lock et al., 2013) suggests that prevalence rates may vary amongst athletes due to their additional involvement in recreational sport and physical activity. Thus, future prevalence studies would benefit, when feasible, by incorporating such interview procedures, by drawing larger, nationally based single-sport samples, and by determining the extent to which the athletes are exercising (or playing other sports recreationally) in addition to their involvement in their primary sport.
2 At present, no study with athletes has examined how they might progress, over the course of a season or their athletic careers, in developing an ED. As with nonathletes, researchers should examine how (and over what timeframe) asymptomatic athletes might become symptomatic, and the extent to which athletes who are symptomatic ultimately develop an ED (see Chapter 55).
3 Pressures within the sport environment, as communicated by teammates, coaches, fans, family members, and judges are related to a variety of disordered eating attitudes and behaviors for both male and female athletes. Given this connection, and the effectiveness of prevention programs among nonathletes (Stice, Shaw, & Marti, 2007; see also Chapters 42–47), researchers should examine the extent to which such interventions would work in reducing pressures within the sport environment. Initial studies in this area (Becker, McDaniel, Bull, Powell, & McIntyre, 2012; Elliot et al., 2004; Smith & Petrie, 2008) have demonstrated the potential efficacy of both peer- and professionally led programs that have been modified to address some of the unique needs of athletes (and the sport environment), but additional research is needed.
4 Research on psychological correlates of disordered eating among athletes has been sparse, particularly for boys and men. Further examination of variables that have been proposed theoretically and initially supported empirically, from general sociocultural (e.g., pressures, internalization, body image concerns) to personality factors (e.g., perfectionism, neuroticism), will be an important next step. Such studies should be multivariate so as to determine the variables' relative influence on (or connection with) the disordered eating indices. Furthermore, determining the potential moderating effects of personality variables on established relationships, such as between body dissatisfaction and bulimic symptomatology,

would be fruitful. Establishing which variables are related to disordered eating attitudes and behaviors is a necessary step in the development of effective intervention programs.

5 The vast majority of studies on athletes have been cross-sectional, which is appropriate when examining initial relationships amongst variables. However, as relationships are established, researchers may want to utilize longitudinal designs to test the temporal effects of the "risk" factors on the development of disordered eating.

6 Research on male athletes has lagged far behind what has been conducted with female athletes, so much less is known about their experiences with respect to body image concerns, eating behaviors, use of pathogenic weight control behaviors, and the development of disordered eating. As Petrie and Greenleaf (2012) have noted, there may be similarities between the experiences of male and female athletes in terms of how disordered eating develops, but empirical research is needed to test this assumption. Further, studies on male athletes should address the fact that muscularity is a primary part of men's identity (see Chapter 37) and a key part of their success in sport. Researchers may want to determine the relative influences of drive for thinness, drive for leanness, and drive for muscularity in male athletes' demonstration of pathogenic eating and weight control behaviors.

7 Finally, examining the extent to which involvement in sport may be protective and reduce behavioral and attitudinal problems related to weight, eating, and body will be important. For less elite athletes, sport participation appears to be associated primarily with positive outcomes, such as greater body satisfaction and lower scores on indices of disordered eating. Even for higher-level athletes, the exercise and physical activity associated with training can have positive physical and psychological effects. Following athletes over time as they move from less elite to higher levels of performance will be important for determining when sport involvement (and the pressures related to that) shifts from providing positive effects to becoming pathogenic for some.

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36

Eating Disorders Across the Lifespan

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There were numerous clues and warning signs. Researchers had long known that eating disorders (EDs) could be either recurring or unremitting, a fact that would mean that there would be illness and symptomatology well beyond the prototypical adolescent onset of the disorder. There was also the evidence that if mothers exhibited disturbed eating or body image, their daughters were at some increased risk of doing the same, perhaps even developing full-blown ED (Watkins, Cooper, & Lask, 2012; see also Chapters 26 & 28). And there were the pregnancy data, indicating an increase in both maternal and fetal risks, ranging from hyperemesis gravidarum to preterm delivery among women suffering from ED (Leddy, Jones, Morgan, & Schulkin, 2009; see also Chapter 34). Yet, aside from a small amount of demographic data indicating that there were fewer cases of ED with age, research concerning EDs in women after young adulthood was rare until about 2000. Indeed, the diagnostic criteria for anorexia nervosa (AN) included amenorrhea through the *DSM-IV-TR* (*Diagnostic and Statistical Manual of Mental Disorders*, 4th ed., text rev.; American Psychiatric Association, 2000), a phenomenon that will by definition limit the diagnosis to postpubertal but premenopausal women. Research concerning EDs in middle-aged and older men has been even more limited.

There is growing agreement that EDs are a problem at least through midlife among women and, perhaps, men. The purpose of this chapter is to explore EDs after early adulthood, after approximately age 30. The chapter considers the etiology of EDs and disordered eating as well as the long-term outcomes of these problems. Models and variables that have successfully explained onset and outcomes in adolescents and young adults will be examined, as will developmental tasks unique to middle and old age that might affect the trajectory of EDs.

Describing Eating Disorders in Middle and Old Age

There are at least four key questions to address in describing ED in older adults:

1 How common are the full-syndrome EDs as well as their major symptoms?
2 Do these appear to be the same disorders that occur among adolescents and young adults?

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3 Are there ethnic group and gender differences?
4 Do these issues or syndromes onset after young adulthood?

Prevalence

Table 36.1 summarizes the prevalence findings from several recent studies. The findings from these studies are not entirely consistent, likely due to differences in samples, patient status, and measures. However, they do establish that eating disorders exist among middle-aged American and Australian women. Research concerning men and older women (>65 years old) is very sparse. Data examining various ethnic and cultural groups are also very limited.

As is true with adolescents and young adults, community-based studies often find few cases of AN. In the Gagne et al. (2012) sample of women aged 50 years and older, about 3.5% reported binge eating while 0.2% reported binge eating and purging, and 0.1% had a body mass index (BMI) under 18.5. Marcus and her colleagues found that about 11% binged at least twice a week (Marcus, Bromberger, Wei, Brown, & Kravitz, 2007). There is evidence suggesting that bulimic symptoms such as these are fairly consistent over a 2.5-year period among middle-aged women (Procopio, Holm-Denoma, Gordon, & Joiner, 2006). Hudson, Hiripi, Pope, and Kessler (2007) and Preti et al. (2009) both found no new cases of AN after the mid-20s but did find new cases of binge-eating-related disorders.

These rates of ED are lower than among younger women (Forman & Davis, 2005; Gagne et al., 2012; Hudson et al., 2007; Preti et al., 2009; Wade, Bergin, Tiggemann, Bulik, & Fairburn, 2006; see also Chapter 5), but they are still sufficiently high to be concerning. Furthermore, some studies (Perez, Hernandez-Gomez, Clarke, & Joiner, 2007) find no age differences in symptoms. Indeed, body dissatisfaction, which is both a component and predictor of ED, tends not to show significant declines until women are solidly in the "old age" period (70 or later; Tiggemann, 2004).

Comorbidities

Adults who have eating disorders also tend to have other diagnosable psychiatric syndromes (see Chapters 15 & 54). Most commonly, they are suffering from either depression or an anxiety disorder (Hudson et al., 2007; Preti et al., 2009). For example, in their sample of 18–60+-year-olds, Hudson and his colleagues (2007) reported that most of the participants in a large community sample meeting the criteria for an ED also met the criteria for at least one other disorder. This was especially true of those with bulimia nervosa (BN), 94.5% of whom showed comorbidities. Of these people identified with BN, over 80% showed symptoms of an anxiety disorder, 70.7% of a mood disorder, 36.8% of a substance use disorder, and 63.8% of an impulse-control disorder. About 56% of AN sufferers and almost 79% of binge eating disorder (BED) sufferers meet the criteria for an additional disorder.

Studies of middle-aged women routinely find relationships between ED and depression or depressive affect (Hrabosky & Grilo, 2007; Marcus et al., 2007; Midlarsky & Nitzburg, 2008; Procopio et al., 2006). This relationship has been found in White, Black, and Hispanic American samples. Studies of clinical samples suggest that occurrence of depression is similar among middle-aged and young adult ED clients (Cumella & Kally, 2008a, 2008b; Forman & Davis, 2005). These studies are generally cross-sectional. In the 2.5-year longitudinal study by Procopio et al. (2006), depression predicted bulimic symptoms in bivariate correlations but not in multivariate regressions. Anxiety symptoms, however, did predict bulimic symptoms in

Table 36.1 Eating disorders among adults: studies from 2005 through 2012.

Author	*Date*	*Sample*	*Findings*
Cumella & Kally	2008a	604 inpatients; half 18–25 and half ≥40 years at admission. American	Compared to younger, older had more AN, less BN, greater severity, more mood disorders, greater sexual abuse history, better body image. Most adult cases were chronic
Cumella & Kally	2008b	50 ED American inpatient women with ED onset after 40	Less severe; almost 2/3 restricting AN or ED-NOS. More history of sexual abuse
Forman & Davis	2005	193 American inpatient women: ≥35 years, n = 43 (MA); ≤35 years, n = 150 (YA)	MA: 48.8% AN, 25.6% BN YA: 37.6% AN, 40.9% BN YA higher on body dissatisfaction and thin ideal internalization
Gagne et al.	2012	1,849 American women over 50; 92.1% White	Binge eating: 3.5%; BMI ≤18.5: 0.1%; Binge & purge:0.2%
Hay et al.	2008	Two Australian community samples; >6000 men and women ages 15 to >65	Purging and fasting were higher in middle age
Hudson et al.	2007	2,980 Americans >18 years	Lifetime risk for ED higher in younger cohorts for BN and BED but not AN or subthreshold
Keel et al.	2010	654 Americans, 20-year follow-up. M_{age} = 40; 81% White	Criteria still met by 14% of ED-NOS women and 14% BN women. All men recovered. 2% of women and 2% of men developed new ED
Marcus et al.	2007	589 American women 42–55 years old; 58% White, 27% Black, 15% Hispanic	11% binged ≥2×/week; 13.4% fasting
McKinley	2006	American women; 72 young adult and 74 middle aged; 11 year follow-up	Young but not middle-aged women showed decreased body shame, dieting, and restriction, and increased body esteem
Perez et al.	2007	1,807 women; 18–79 years, American	No age difference on EDI-Bulimia scale
Preti et al.	2009	1,741 males, 2,397 females; 18–65+ years old. Six European countries	Higher lifetime risk of AN, BN, BED in younger cohorts. No new cases of AN after 20; New cases of BED throughout
Procopio et al.	2006	150 American women; M_{age} = 45.19	Substantial stability in six bulimia symptoms (r = .67)
Wade et al.	2006	1,002 Australian twins, aged	7% of lifetime AN, 7% of lifetime BN, 10% of BED, 6% of ED-NOS continued to meet criteria

Note. AN = anorexia nervosa; BED = binge eating disorder; BMI = body mass index; BN = bulimia nervosa; ED = eating disorder; EDI = Eating Disorders Inventory; ED-NOS = eating disorder not otherwise specified; MA = middle-aged; YA = young adult.

the multivariate model (Procopio et al., 2007). In clinical samples, anxiety, like depression, appears to be about as common in midlife as in young adult ED clients (Cumella & Kally, 2008b; Forman & Davis, 2005).

Symptom Invariance Across Ages

Measures of EDs rely on symptom invariance across ages, gender, and ethnicities. If the pattern of symptom expression varies by age, then neither our assessment tools nor our diagnostic criteria will be applicable. Age comparisons will be even more difficult. Yet, we have few studies assessing the validity (via, e.g., confirmatory factor analysis) of measures across ages. The data on symptom similarity are also sparse.

In studies of inpatients, Cumella and Kally (2008a) reported that midlife women reporting a chronic course of ED showed greater severity of most symptoms but better body image than did young adult clients. On the other hand, when reporting on inpatients who reported midlife onset of ED, Cumella and Kally (2008b) found that symptoms were less severe than in young adults. It may be noteworthy that reports of sexual abuse (see Chapter 34) were higher in both samples of middle-aged women than is typically seen in young adult clients. Forman and Davis (2005) also found that inpatients who were 35 and older had lower scores on the Body Shape Questionnaire and the Sociocultural Attitudes Toward Appearance Questionnaire than those under 35. Hence, it appears that middle-aged ED inpatients may have better body image than those who are younger. Whether it is simply level of body satisfaction that differs or whether the actual nature of body image disturbances varies is unknown.

Ethnic Group and Gender Differences

As is true with adolescents and young adults, studies often focus on White women. However, when men are included, there is a pronounced gender difference indicating that eating symptoms and syndromes are generally considerably more common among women (see Chapters 5 & 27). Preti et al. (2009) found that AN, BN, and BED were three to eight times higher among women, while Hudson et al. (2007) reported that women suffered from these disorders 1.5 to 3 times more frequently. The samples in both of these studies included a wide range of ages but the authors opted not to do an age-by-gender analysis of the disorders. Although Keel, Gravener, Joiner, and Haedt (2010) reported that all of the men, but not all of the women, who had an ED were recovered 20 years later, about 2% of each gender met ED criteria for the first time at the 20-year follow-up. These data suggest that early adulthood may not be the peak onset age for ED for men and that, by midlife, the onset rates may be similar. This intriguing finding deserves much more research attention.

In one of the rare studies including people older than 70, women endorsed a thinner ideal than men did (Ferraro et al., 2008). In addition, the women thought more about their body shape and perceived themselves as "a little too big." Men, then, were generally more satisfied with their bodies.

In a study of women aged 42 through 55, Marcus and her colleagues (2007) reported that Black and White women both reported more binge eating than did Hispanics. Compared to Hispanics, White women were also more preoccupied with weight and shape. Black women also reported more purging behavior than did Hispanics or Whites. Hrabosky and Grilo (2007) surveyed a sample of 120 Hispanic and Black women ages 18 through 58. The two groups of women did not differ in their scores on measures of body image and disordered

eating. Interestingly, however, BMI was positively correlated with binge-eating and restraint scores among the Black but not the Hispanic women. This raises questions about at least the motivation for binge eating and restraint. This, in turn, might affect the likelihood of the development of full-syndrome disorders and is worthy of additional research.

Midlife Onset

When people report EDs or ED symptoms in midlife or after, it is possible that the actual onset of the problem was in adolescence or young adulthood. In some people, the disorder may be unremitting (Keel et al., 2010). In others, it may be a recurrence of an earlier disorder. As Cumella and Kally (2008a) reported, these individuals may suffer from particularly severe symptoms. They may also have high levels of mood disorders and suicidality.

It is clear, however, that these disorders can onset in middle age. Cumella and Kally (2008b) reported that, in their clinical sample, these were much more likely to be cases of restricting AN or eating disorder not otherwise specified (ED-NOS) involving restriction than cases of BN. These women reported unusually high levels of sexual abuse, with 64% having a history (see Chapter 34). Forman and Davis (2005) also reported more AN and less BN in their middle-aged sample than in the young adult sample, though they did not determine time of onset in the older sample. In community samples, however, it is BN and ED-NOS that appear more common, though the onset of either is more rare than in young adult women (Keel et al., 2010).

Influences on Eating Disorders in Midlife

Sociocultural approaches (see Chapter 21) have dominated the limited etiological research about EDs in midlife and beyond. However, some researchers have added developmental tasks to their models in recognition of the particular demands of middle age. Both types of models are considered here.

Sociocultural Factors

Probably the most frequently cited etiological model with adolescents and emerging adults is the Tripartite Model of Thompson, Heinberg, Altabe, and Tantleff-Dunn (1999; see also Chapter 21). This model emphasizes the thin-ideal cultural message that is conveyed by media, parents, and peers (see Chapters 19, 26, 27, 29, & 31). This message shapes or reinforces thin-ideal internalization and social comparison, which, in turn, leads to body dissatisfaction and disordered eating/eating disorders. The theory has received considerable support with children, adolescents, and emerging adults (see Chapter 21) though, admittedly, there are more studies with body dissatisfaction or disordered eating than EDs per se as the dependent variables. Is it reasonable to expect that this model is applicable to middle-aged women?

Media Middle-aged people watch more television than young adults do, with 35–49-year-olds watching an average of 34.2 hours per week while 50–64-year-olds watch 42.2 hours weekly ("Are young people watching less TV?," 2013). When older women watch U.S. television, they predominantly see the same thin, young women that girls and younger women see (Levine & Murnen, 2009; see also Chapter 29). The few middle-aged (40+) women they do

see are often unusually thin for this age group, as exemplified by actors such as Ellen Pompeo, Patricia Heaton, Dana Delany, and Paget Brewster. If they read entertainment magazines (like *People*) or fashion magazines (such as *Vogue*) they will see famous women over 40 who are quite thin, often for any age group, including the actors Gwyneth Paltrow, Sharon Stone, Diane Keaton, Jennifer Aniston, Helen Mirren, and Nicole Kidman. Thus, middle-aged and older women are indeed not only exposed to the thin-ideal message but are increasingly told it is attainable for them.

Slevec and Tiggemann (2011) surveyed 101 Australian women ages 35–55 years to test a sociocultural model emphasizing media. They found that exposure to both magazines and television, but especially the latter, was associated with a latent variable they titled "media processing." Media processing in this study included thin-ideal internalization, social comparison, appearance investment, and age anxiety. The first three of these are commonly treated as mediators in studies of the Tripartite Model (see Chapter 21). Age anxiety reflects both the media's preference for young women and normative adjustment to the aging process. Media processing, in turn, was associated with body dissatisfaction, as predicted by the Tripartite Model and as supported by substantial research (see Chapters 21 & 29). As expected body dissatisfaction was associated with disordered eating (EDI drive for thinness, bulimia symptoms, body dissatisfaction subscales). These findings are quite consistent with those for adolescents and young adults, except that age anxiety is a contributing factor and television exposure seems to be more important than magazine use.

The Slevec and Tiggemann (2011) study is probably the most sophisticated of the limited research examining sociocultural etiological factors. Unfortunately, this is a cross-sectional study that can identify correlates (albeit within a strong theoretical framework) but not risk factors. Other studies have, however, produced similar findings. For example, Midlarsky and Nitzburg (2008) found that sociocultural pressures to be thin (from media, peers, and family) and body dissatisfaction were related to disordered eating (measured using the EAT-26) in bivariate correlations and a multivariate model. Appearance concerns related to aging were significantly related to disordered eating in bivariate correlations but dropped out in the broader model. McLean, Paxton, and Wertheim (2010) found that importance of appearance was related to the EDE-Q subscales of weight concern, shape concern, eating concern, and restraint.

Peers There are numerous studies documenting various types of peer influence on disordered eating during adolescence and, to a lesser extent, during the college years (see Chapter 31). Gravener, Haedt, Heatherton, and Keel (2008) examined the relationship between peer dieting and drive for thinness in men and women in late adolescence (about 20 years old), adulthood (approximately age 30), and middle age (aged 40). Women's drive for thinness was significantly correlated with same-gender peers' dieting in each of the three age groups, although the strength of the relationship decreased (r values = .41 decreasing to .27). For men, the correlations were statistically significant and did not decline (r values = .39 to .37 at each age). Men's drive for thinness was also related to other-gender peers' dieting (r values = .22 to .17), whereas women's was not after late adolescence (r values = .11 to .06). These findings need to be replicated with an eye toward understanding why the relationship between peer dieting and drive for thinness might decline with age for women but not for men. It is also interesting to note that, across the entire sample, the men's and women's correlations were statistically identical, underscoring the frequent finding that when men are exposed to the same influences that women are, they behave the same (see Chapter 27).

In a 10-year longitudinal study, Keel, Forney, Brown, and Heatherton (2013) investigated the interesting relationship between college roommates' dieting and bulimic behaviors, purging, and drive for thinness among adults (M_{age} = 30 years). They also looked at parents' dieting as a predictor of disordered eating (see Chapter 26). Women's roommates were more likely to diet than were men's, and women had higher disordered eating scores. Men and women reported similar levels of dieting by their fathers and mothers, though mothers dieted more frequently than fathers did. Among women, adult drive for thinness was predicted not only by late adolescence (college) drive for thinness but also by BMI, college bulimic symptoms, maternal dieting and roommates' dieting (all measured in late adolescence). For bulimic behaviors in adulthood, women's late adolescent bulimic symptoms as well as BMI, drive for thinness, and roommate dieting were significant predictors. Late adolescent purging and roommates' dieting were predictors of adult purging.

Among the men, adult drive for thinness was predicted by only late adolescent drive for thinness, indicating stability in the behavior. Similarly, bulimic behaviors were predicted by earlier bulimic behaviors and drive for thinness. Roommate behavior was not an important contributor as it was in women. Men did not report adult purging and so no analysis could be performed.

Abuse

As we have seen, a particularly high percentage of middle-aged ED clients report abuse in their histories (Cumella & Kally, 2008b). Child physical and sexual abuse, as well as adult experiences of rape, have been associated with body image problems and ED in adolescent and young adult samples (Smolak, 2011; see also Chapter 34). It is noteworthy that, in addition to BN in particular, a history of sexual abuse is associated with borderline personality disorder, depression, and anxiety disorders (including posttraumatic stress disorder, PTSD), problems that are commonly comorbid with EDs (Maniglio, 2013; Smolak, 2011; Weiss, Longhurst, & Mazure, 1999; see also Chapters 15 & 54). Thus, abuse is a nonspecific risk factor for numerous forms of psychopathology at various stages of development.

Why then might the risk for ED subsequent to abuse seem higher in middle-aged adults than in adolescents or young adults? One possibility might be that as time goes on the comorbid disorders worsen sufficiently to trigger or intensify an ED as a coping mechanism. Or the ED might worsen, bringing the client to therapy either voluntarily or at the urging of a spouse or child. But what might create such intensification of symptoms that are at least partly rooted in abuse? Bulik (2013) offers some suggestions. It is possible that the traumatic memories that abuse survivors carry with them are triggered by events during midlife. These might include the aging, illness, or death of a family member who was the abuser. Or there may be additional abuse at the hands of a spouse or partner. The intensification of shame, anxiety, and depression, as well as perhaps a renewed sense of loss of control of one's body, might intensify or even ignite ED symptoms. This is an interesting issue for future research.

Midlife Transition

Development is a process marked by transitions. Although the adolescent transition and its pubertal changes have received the most attention in the ED literature, there are other similarly challenging periods in development. At each transition, we face a series of internal and external challenges that our extant coping structures cannot adequately address (Cytrynbaum

et al., 1980). Our psychological systems tend to resist change and seek stability. With our familiar strategies for regaining homeostasis rendered inefficient and perhaps inadequate, we may experience drops in self-esteem and self-confidence, rendering us vulnerable to stress and hence maladjustment (Pearlin, Lieberman, Menaghan,& Mullan, 1981). We must refine our self-systems and our coping mechanisms within the new context. Thus, any developmental transition presents a time of increased vulnerability. This includes the transitions to middle and old age.

Although we will focus on the challenges of midlife that require adaptation, there is substantial stability in personality throughout adulthood (Caspi, Roberts, & Shiner, 2005). Predilections for using emotional or instrumental, passive or active coping mechanisms may continue. But the types of stressors faced as well as the availability of social support influence which mechanisms are most effective. Aging tends to bring decreases in the controllability of stressors, perhaps necessitating a shift in one's "go to" coping choice, although individual differences will remain. Thus, some people continue to use EDs throughout adulthood as a way to establish control or relieve stress, others may have a recurrence as new and seemingly overwhelming stressors appear, and still others may find that for the first time in their lives they cannot control their life situations and so need an ED as a means to cope.

Tasks of Midlife A number of theorists, including Cytrynbaum et al. (1980), Havighurst (1972), Levinson (1986), and Valliant (1977), have outlined developmental tasks associated with the midlife transition. Commonly these tasks include adjustment to biological aging and death, appraisal of work goals, and reassessment of family and friendship relationships.

There are numerous biological changes in midlife, ranging from decreases in mobility due to arthritis, to increasing blood pressure, to greater risk for cancer and heart disease. For many people, even the more minor changes such as graying hair or facial wrinkles, are reminders of the aging process and that the end of that process will be death. Body fat distribution changes such that more fat is deposited around the midsection. Many of these changes are directly associated with the hormonal shifts in estrogen in women and testosterone in men.

For people who are invested in the thin ideal for women and the muscular ideal for men as well as the general cultural emphasis on youth, these changes may be unbearable. People may dye their hair, get botox injections or "instant" facelifts, or undergo cosmetic surgery to try to fight aging. It is evident that people do not simply graciously accept physical aging, given that there is not a decrease in body dissatisfaction during this time (Tiggemann, 2004). These changes and the difficulty in accepting them may trigger stress that contributes to emotional or binge eating. But they may also lead some women to diet extensively in order to maintain a thin shape, a goal that will likely require more and more extreme weight control methods because of age-related changes in metabolism and fat deposition. Men, too, will find it increasingly difficult to maintain a muscular or even lean shape and may resort to more extreme measures. Perhaps this is part of the reason that new cases of ED seem to occur at similar levels in both genders at midlife (Keel et al., 2010). These are all hypotheses, however. Research is desperately needed to understand the role of physical changes in EDs in middle age.

Stress may also be created by changes in family roles and relationships (Bulik, 2013). Children leave home. For women who were full-time homemakers, this may lead to questions about what to do next. Children moving out may also affect spousal relationships. For some couples, it may seem that they have nothing left in common once the children leave. For some parents, a child may be a "best friend" or a significant source of support, so their moving out may leave that parent with fewer resources. Similarly, marital relationships may change. There

may be infidelity. There may be differences in how partners want to approach aging and retirement, leading to tension. There are often decreases in sexual relationships, as physical illnesses, hormones, and time demands lower sex drives. Finally, parents and even friends may develop illnesses and die. The upshot of all of this is that it is possible that people lose social supports as they go through the transition. Those who do find their social support declining may be at greater risk for developing EDs. Again, these are dramatically under-researched questions.

Finally, there may be work-related changes. As workers age, they will frequently find that they have achieved as much of their career "dream" (Levinson, 1986) as they are likely to reach. There is typically reduced chance for advancement in work after age 50 or 60. And new jobs are difficult to come by as people age. So if someone wants to move up by changing jobs or loses her or his job, the loss of control, anxiety, and other forms of stress attendant to a long and perhaps unsuccessful employment search might contribute to the renewal or onset of ED symptoms (Bulik, 2013). It is interesting to speculate that the comparable rates of ED onset in middle-aged men and women (Keel et al., 2010) might reflect men's career investment creating a new form of stress.

The existence of ED in middle-aged women and men is unquestionable. But the causes of the problems are poorly understood and dramatically under-researched. As the next section indicates, the potential outcomes of ED in midlife and beyond demonstrate the importance of such work.

Outcomes

Developmental issues are very clearly exemplified in the consequences of ED in older adults. Physical changes associated with aging, due to genetics or lifestyle, or the aging process, likely intensify the negative effects of ED. Furthermore, parenting roles offer a new realm for negative effects of ED.

Of course, EDs always have the potential to ravage the physical well-being of anyone (see Chapters 14 & 52) and have the highest mortality rate of any psychiatric disorder (see Chapter 55). It is noteworthy that people whose ED is unremitting (or at least recurrent) into middle age may have particularly severe symptomatology (Cumella & Kally, 2008a; see also Chapter 12). Continued ED may be more common among women than men (Keel et al., 2010). On the other hand, women with onset of ED after age 40 may have less severe symptoms than younger clients do (Cumella & Kally, 2008b). So before all of the physical outcomes of ED in midlife and older sufferers can be delineated, we need to distinguish those who have early onset versus later onset. Unfortunately, such data are not available at this time.

Individual Outcomes

It is always difficult to get ED sufferers into treatment, particularly if their symptoms are not easily visible, as is often the case with binge eating or purging (see Chapters 3 & 9). However, treatment entry may be particularly complex for resistant adults with EDs (Bulik, 2013; see also Chapter 12). First, difficult as symptoms may be to recognize in a child or adolescent, it may be even trickier with adults. Adults can buy food for binges without others in the family knowing, for example. If they are restricting, they can easily claim they had a big lunch at work or ate earlier. If they are single parents or live by themselves, adults typically have complete control over the food in their homes. Children never do. All of this facilitates hiding symptoms.

Second, with a child or adolescent, parents can require the child to be hospitalized or treated. Most adults with an ED, other than perhaps those who are immediately suicidal, do not meet the legal definition of being a clear and present danger to self or others. Therefore, entry into treatment will be completely voluntary. Even if the person is losing weight at an alarming rate or is dramatically underweight, it is extremely difficult to have them involuntarily committed for treatment.

Bulik (2013) suggests that midlife sufferers may be particularly vulnerable to the physical effects of ED. First, those who have unremitting symptoms may suffer cumulative damage from, for example, laxatives or diet pills. Bone density will often be negatively affected by AN as well as by the aging process, especially among women. The risk of osteoporosis may become particularly high. Similarly, the gastrointestinal system may be damaged by various elements of EDs (e.g., use of purgatives). There are a variety of gastrointestinal illnesses, such as gastroesophageal reflux disease (GERD), that become more common and/or more severe with age (Lee et al., 2007). Again, a cumulative effect is possible.

Even if the ED onsets in midlife or later, the consequences of self-starvation, poor nutrition, and purging may complicate, or be complicated by, other medical problems. Gastrointestinal, cardiac, hormonal, joint, and bone problems are potential examples (see Chapter 14). Much more research is needed to clarify these concerns.

Parenting

As noted earlier, pregnancy and birth problems are more common among women with ED. In addition, child feeding and eating difficulties occur at higher rates. Mothers with ED report more restriction of child food intake (Reba-Harrelson et al., 2010). Such restriction and overcontrol by parents is associated with obesity and disordered eating among children (Fisher, Sinton, & Birch, 2009). Furthermore, both infants and children of mothers with EDs demonstrate more feeding problems, including small quantity feeds and poor sucking among infants, as well as toddlers' complaints about stomach aches (Micali, Simonoff, Stahl, & Treasure, 2011; Reba-Harrelson et al., 2010). Micali et al. (2011) reported that the relationship between maternal EDs and infant feeding problems was mediated by maternal distress in the form of depression and anxiety symptoms. This makes sense given that other studies (e.g., Dennis & McQueen, 2007) have found relationships between depression and anxiety and child feeding problems. In addition, Micali et al. also uncovered a direct pathway between maternal ED and child eating difficulties.

Watkins et al. (2012) reported that children with eating disturbances (see Chapter 13), including AN, food avoidance emotional disorder (FAED), or selective eating (SE), were more likely to have mothers with EDs than were the children in a community sample. Seventeen percent of the children with eating disturbances had mothers with EDs, while the rate in the community sample was 3–5%. Children suffering with FAED were particularly likely to have eating-disordered mothers.

One can only speculate on the reasons for higher rates of feeding and eating disorders among the children of ED sufferers. Genetically transmitted vulnerabilities are a possibility (see Chapter 28). Birth complications that contribute to infant fussiness may play a role; infant fussiness does increase distress among ED mothers, which in turn increases infant feeding problems (Micali et al., 2011). Food restriction by mothers may contribute to children's disordered eating (Fisher et al., 2009). In any case, there is cause for substantial

concern not only because of the immediate risks of poor nutrition but also because childhood eating and feeding problems do seem to be related to the later development of EDs (Marchi & Cohen, 1990).

It is noteworthy that paternal eating and feeding influences on children have been underinvestigated. Studying these relationships is important to help tease apart genetic and environmental influences on developing an early pathway to ED.

Conclusions and Future Directions

Research reviewed in this chapter clearly indicates that EDs continue to be an important health issue well into adulthood. There are both continuing and new-onset cases in both men and women. As might be expected, many of the continuing cases may be quite severe and unresponsive to most treatments (see Chapter 12). New-onset cases are probably less common in middle-aged and older adults than in adolescents or young adults, especially among women. New-onset cases may occur with comparable frequency among men and women but, across all cases, EDs are more common in women than men. There is no clear evidence suggesting that the symptoms of EDs are qualitatively different across the lifespan. But research does suggest at least quantitative differences, and much more work is needed to answer this crucial question. Very limited research indicates that there may be at least quantitative differences in specific symptoms (such as purging) among various ethnic groups. This, too, is a question that cannot be answered until more data are available.

All areas related to EDs in midlife and beyond are poorly researched. Information on risk factors for the onset and continuation of EDs is needed. There appears to be a complex constellation of risk factors including sociocultural factors such as peer and media influences as well as developmental task demands. Similarly, outcomes in adulthood are complicated by the possible chronicity of the disorders and by developmental tasks and changing roles. All of these issues need to be investigated across the lifespan in both women and men.

Prevention and treatment of ED in adulthood presents special problems. There is no adult equivalent of school or college as a convenient venue for large-scale prevention programs. Perhaps programs could be delivered through physicians, though such efforts are likely to be largely informational. Whether information-based programs would be more effective with adults than they are with younger people is unclear. However, medical personnel likely need more information about the frequency and symptoms of adult EDs. Training for health professionals working with adults, particularly in terms of early identification, might also help to reduce EDs in adults.

Fitness centers, whether housed in large companies or as stand-alone businesses, may also offer possibilities for prevention. Dissonance-based programs such as those developed by Stice and colleagues (see Chapter 44) could be instituted for clientele as well as for the trainers. Again, personal trainers could likely benefit from information about adult EDs.

Treatments for EDs in adults also present some challenges (see Chapters 50 & 56–64). On the one hand, adults can easily comprehend and participate in a variety of treatment programs. On the other hand, as noted earlier, there are few options to pressure them into treatment. ED treatment centers might offer groups for family members, including children and spouses, with information about how to recognize EDs in adults and how to discuss treatment with affected adults.

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Eating Disorders in Boys and Men

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Although eating disorders (EDs) have long been viewed to occur primarily in women, there is an increasing recognition that EDs affect a significant number of men (Darcy, 2011; Jones & Morgan, 2010). In studies conducted over 20 years ago it was estimated that men made up only 10% of the clinically diagnosed cases of EDs (Andersen, 1984; Carlat & Camargo, 1991; see also Chapter 5). More recent studies suggest that EDs among men are likely to be much higher and that they make up 20–25% of the cases of EDs, with the proportion of men varying by disorder (Darcy, 2011; Hudson, Hiripi, Pope, & Kessler, 2007). In addition, there is a growing body of research that has examined the nature of EDs in males, as well as associated attitudes and behaviors. A particular focus of this research is how EDs are expressed differently for males and females. This includes less extreme weight control methods, overeating, and behavioral problems associated with the pursuit of muscularity.

In this chapter we first consider how EDs have been ignored and misunderstood among males, and the bias in the diagnosis of EDs, given that definitions and criteria for diagnosing EDs have been primarily designed for women (see Chapters 8–11). We also examine disordered eating attitudes and behaviors associated with the pursuit of muscularity, given that men are often concerned with the attainment of muscular definition and strength rather than weight loss and a fear of fatness. We review the prevalence and presentation of EDs and associated behaviors among men, and compare these with those in women. Lastly, we examine the main risk factors that have been found to be associated with the development of disordered eating and body change strategies to achieve the muscular ideal.

Ignored and Misunderstood

There are several factors that help to explain why EDs among males have been largely ignored. Much of the research conducted over 20 years ago was embedded in feminist theory (see Chapter 19) and the focus was on women, so men were often excluded from studies (Darcy, 2011). In addition, assessment instruments designed to detect and evaluate EDs have been developed primarily for women and do not comprehensively assess EDs that are more specific

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to men (Stanford & Lemberg, 2012; see also Chapter 40). This focus has promoted the view that EDs are predominantly female diseases that do not occur among men, thus contributing to the reluctance of men and adolescent boys to seek treatment for their eating problems and the stigma attached to being a man with an ED (Darcy, 2011; Harvey & Robinson, 2003). However, given that EDs among males are poorly understood, men may also be less likely to recognize their eating problems and less concerned about their eating behaviors, and thus less likely to seek treatment (Darcy, 2011; Jones & Morgan, 2010).

Diagnosing Eating Disorders

It is more difficult to diagnose EDs in males, as men are less likely to use extreme weight loss methods, and many of the eating patterns such as overeating and binge eating that are seen as abnormal or inappropriate in women are socially sanctioned for men (Carlat & Camargo, 1991; Carlat, Camargo, & Herzog, 1997). Some of the behaviors indicative of EDs, such as excessive exercise and rigid eating patterns, are less likely to be viewed as problematic, as males are often heavily involved in sports, so these are accepted and not questioned (Muise, Stein, & Arbess, 2003). Moreover, given that EDs are poorly understood among males, when men present for treatment they are more likely to be misdiagnosed with other mental health problems such as depression, and they are less likely to be referred to specialist ED services (Jones & Morgan, 2010).

Definitions and criteria for diagnosing EDs have been primarily developed for women, so these are likely to underestimate and not accurately reflect the full spectrum of eating problems among males. Thus, it is important that we focus more on ED symptoms and partial syndrome eating disorders. ED symptoms include concerns about body weight and shape, extreme weight control methods such as fasting, purging, and excessive exercise, and binge eating. Partial syndrome EDs have also been referred to as subclinical levels of disordered eating, atypical EDs, and unspecified feeding or eating disorders according to the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*). Individuals with partial syndrome EDs usually experience considerable psychological disturbance and often engage in the same disturbed eating behaviors as those with full syndrome eating disorders, but at a somewhat lower level of frequency and severity (Shisslak, Crago, & Estes, 1995; see also Chapters 4 & 11).

Muscle Dysmorphia

During the last decade there has also been an increasing recognition that EDs may manifest rather differently among men, which is another reason why EDs among males have often been underestimated and misunderstood. Many men are less concerned with weight loss and fear of fatness, but are more concerned with muscular definition and strength, and are more likely than women to develop behavioral problems associated with the pursuit of muscularity. These include the use of anabolic steroids, extreme body-building behaviors, exercise dependence, and other attitudes and behaviors associated with muscle dysmorphia (MD; Pope, Phillips, & Olivardia, 2000). As in anorexia nervosa (AN), MD is characterized by an unrealistic perception of the body, but in contrast to AN, where there is a fear of being fat and a preoccupation that one's body is not sufficiently thin, the focus in MD is on not being sufficiently lean and muscular, and there is a fear of being too small or inadequately muscled (Pope et al., 2000). MD has been classified as a subtype of body dysmorphic disorder; however, there is increasing

evidence that it might be better classified as a type of ED (Darcy, 2011; Jones & Morgan, 2010; Murray et al., 2012).

Researchers have highlighted the similarities between MD and EDs, as both involve body image disturbance, excessive exercise, rigid diet patterns, compensation behaviors, appearance-enhancing substance use, elaborate body checking, and avoidance of bodily exposure (Darcy, 2011; Murray et al., 2012). In addition, several of the risk factors associated with the pursuit of muscularity, more broadly, include the same factors associated with disordered eating (Connan, 1998; Ricciardelli & McCabe, 2004; Rodgers, Ganchou, Franko, & Chabrol, 2012). For example, both disordered eating and the pursuit of muscularity develop during adolescence, which is a period of rapid change. Further, at this stage of development several biopsychosocial factors intensify the focus on physical appearance and promote body change strategies to cope with emotional and identity problems. In addition, extreme weight loss methods, associated with disordered eating, and the pursuit of muscularity are both strategies that may be used by adolescent boys to attain the muscular ideal that is endorsed by the broader society (Ricciardelli & McCabe, 2004).

Prevalence

In our 2004 review we found the estimates of the prevalence for the full syndrome of AN among adolescent boys and young adult men to range from 0% to 0.16%, as compared to 0% to 0.9% among adolescent girls and young adult women (Ricciardelli & McCabe, 2004). In a more recent review, the prevalence for AN has been found to be higher among both men and women, with estimates varying from 0% to 0.3% for men, and 0.3% to 2.2% for women (Smink, van Hoeken, & Hoek, 2012).

We found the estimates of the prevalence of the full syndrome of bulimia nervosa (BN) to range between 0% and 0.7% for adolescent boys and men, and between 0.34% and 1.5% for adolescent girls and women (Ricciardelli & McCabe, 2004). These levels are similar to those found in the more recent review, with the majority of studies reporting estimates between 0.1% and 0.5% for men, and between 0.5% and 1.6% for women (Smink et al., 2012). Estimates of the prevalence of binge eating disorder (BED) were also included in the recent review (Smink et al., 2012). These ranged between 0.3% and 2.0% for men, and between 1.9% and 3.5% for women.

Given that "men with eating disorders are currently underdiagnosed, undertreated, and misunderstood" (Strother, Lemberg, Stanford, & Turberville, 2012, p. 346), the prevalence rates reported for men are likely to be underestimates. Estimates that include partial syndrome EDs may be a more accurate indicator of the problem for men. The combined prevalence rate among men with full or partial syndrome of BN in a community sample has been found to be 1.08% as compared to 3.16% in women (2.9:1 female-male ratio; Woodside et al., 2001). Similarly, the combined prevalence rate among men with full or partial syndrome of AN has been found to be 0.92% as compared to 1.81% in women (2.0:1 female-male ratio; Woodside et al., 2001). In addition, the prevalence for subthreshold BED has been found to be higher among men (1.9%) than among women (0.6%; Hudson et al., 2007).

Several studies have also examined the prevalence of ED symptoms among men and adolescent boys, and compared these to women and adolescent girls. However, estimates vary widely, due to the different definitions and criteria used by researchers (Ackard, Fulkerson, & Neumark-Sztainer, 2007; Ricciardelli & McCabe, 2004). Moreover, it is important to note that effect sizes are small, thus suggesting that gender differences in disordered eating are less

pronounced than what is often assumed (Striegel-Moore et al., 2009). Given that most studies do not report effect sizes and place more emphasis on statistical significance, this has also contributed to an underappreciation of the extent and clinical significance of disordered eating among men (Striegel-Moore et al., 2009).

The overall prevalence of ED symptoms has been found to be higher among women than men in a community sample (Striegel-Moore et al., 2009). However, a substantial proportion of men report similar symptoms. In fact, overeating was found to be significantly higher among men (26% of men vs. 18% of women), while loss of control over eating (20% of men vs. 29.6% of women) and binge eating at least once per week (8.0% and 10.0% women) were found to be significantly higher among women. Also examined were the prevalence of vomiting, fasting, laxative use, and exercise for the purpose of avoiding weight gain after binge eating. Women were found to use vomiting (1.5% men vs. 3.7% women) and fasting (4.0% men vs. 6.3% women) significantly more frequently than men. However, there were no differences in the use of laxatives (3% men vs. 3.1% women) and exercise (5.6% men vs. 6% women).

In comparison to community samples, higher levels of ED symptoms have been found among college-age men (Cain, Epler, Steinley, & Sher, 2012). This includes overeating (47%), binge eating (13%), fasting (11%), self-induced vomiting (6%), and abuse of laxatives/diuretics (5%). The numbers of college students with an ED diagnosis also significantly increased between 1995 and 2008 for both men and women (White, Reynolds-Malear, & Cordero, 2011). In 1995, 7.9% of men and 23.4% of women were found to meet the criteria for an ED diagnosis, but this increased to 25% for men and 32.6% for women in 2008. The college years are seen as a particularly challenging time for both young women and men, in terms of the increased academic and social pressures, which are viewed as contributing to the development of disordered eating (Striegel-Moore, Silberstein, Frensch, & Rodin, 1989). Moreover, in the last decade young adults have been exposed to higher levels of sociocultural pressures that target the attainment of an increasingly lean and muscular body ideal in the case of men, and a thin body ideal in the case of women (White et al., 2011).

Levels of ED symptoms have also been found to be high among adolescent boys, and some of these are comparable to those found among girls (Ricciardelli & McCabe, 2004). For example, in one study, a similar number of boys and girls were found to endorse disordered eating behaviors (15.4% boys, 16.0% girls), which included binge eating, self-induced vomiting, laxative use, and/or excessive exercise (Ackard et al., 2007). A higher number of girls (36.4%) than boys (23.9%) endorsed a greater importance of weight and shape on self-evaluation or self-esteem. However, given that the focus was on weight and shape, boys' body image concerns are likely to have been underestimated.

Boys are primarily dissatisfied with and place more importance on their muscle size, height, strength, shoulders, biceps, and chests (Ricciardelli, McCabe, & Ridge, 2006). One of the potential problems associated with adolescent boys wanting to become more muscular, gain body size and weight, and increase body strength, is the increased likelihood of using anabolic steroids to achieve quick results. Estimates for the number of adolescent boys who have ever used steroids range from 1.2% to 12%, as compared to 0.2% to 9% for girls (Harmer, 2010; Ricciardelli & McCabe, 2004).

As there have been no formal epidemiological studies of MD in the general population, its prevalence is still unknown (Murray, Rieger, Touyz, & de la Garza García, 2010). Conservative estimates suggest that about 10% of body-builders and 9% of men with body dysmorphic disorder have MD (Olivardia, 2001). However, more liberal estimates suggest that MD among men may be as prevalent as AN is among women (Grieve, Truba, & Bowersox, 2009).

Presentation

Overall, men with EDs present with very similar profiles to women in terms of their clinical, demographic, and personal data (Gueguen et al., 2012; Muise et al., 2003; Oyebode, Boodhoo, & Schapira, 1988). For example, depressive and obsessional symptoms are common (see Chapters 15 & 54), and there is a strong family history of affective disorders and alcohol abuse (Sharp, Clark, Dunan, Blackwood, & Shapiro, 1994).

However, differences have also been noted. Some of these reflect behaviors that are more socially sanctioned for males in the general population such as exercise, overeating, and substance abuse (Ricciardelli & Williams, 2011). For example, men are less likely to use vomiting and laxatives as either weight loss strategies or as compensatory bulimic behaviors, but they are more likely to use excessive exercise (Jones & Morgan, 2010; Norris et al., 2012; Sharp et al., 1994; Strother et al., 2012). Men may use overeating in combination with weight loss strategies to improve their muscular build (Ricciardelli & McCabe, 2004). In addition, some studies have found higher levels of substance abuse among males with EDs (Muise et al., 2003; Strother et al., 2012).

The nature of binge eating is also different among men, and it may simply reflect overeating. Although a greater number of men report eating large quantities of food at times other than at meals, they are less likely to call this pattern "binge eating" (Katzman, Wolchik, & Braver, 1984). Men who have been diagnosed with BN are also less troubled by their binge eating, in that they are less likely to feel depressed and they report less guilt (Carlat & Carmargo, 1991). In addition, men are less likely to report a sense of being out of control during binge eating in comparison to women (Norris et al., 2012). In one study, Snow and Harris (1989) found that adolescent boys, in contrast to girls, thought that binge eating episodes were normal.

The age of onset for EDs among men has in some studies been found to be later than among women (Braun, Sunday, Huang, & Halmi, 1999; Gueguen et al., 2012; Keel, Gravener, Joiner, & Haedt, 2010; Muise et al., 2003; see also Chapter 36). For example, Braun et al. (1999) found that the average age for the onset of EDs in men is 20.56 years compared to 17.15 years for women. This age difference has also been specifically found for the development of bulimic symptoms (Abebe, Lien, & von Soest, 2012). For adolescent girls, bulimic symptoms have been found to increase between the ages of 14 and 16 years and then decline, while for adolescent boys bulimic symptoms have been found to decrease between the ages of 14 and 16 and then increase in their early 20s (Abebe et al., 2012). This later onset of EDs and disordered eating may be partly due to the later onset of puberty in males (Gueguen et al., 2012).

A delay between the onset of the disorder and the age of first treatment has also been found among men with BN, which is often attributed to men's reluctance to seek help for their eating problems (Muise et al., 2003). In addition, men are more likely to have a history of premorbid overweight, and a higher minimum, maximum, and desired body mass index (BMI; Gueguen et al., 2012; Strother et al., 2012). This is likely to be due to the fact that the ideal body for men involves both muscularity and leanness, and men often use both strategies to lose weight (e.g. fasting) and increase muscles (e.g. exercise) to achieve this ideal (Ricciardelli & McCabe, 2004).

Men with MD also display similar profiles to both men and women with EDs, particularly AN (Murray et al., 2012). This includes body image disturbance, dietary restriction, use of diuretics and other supplements, excessive exercise, use of baggy clothes to hide body parts, low self-esteem, high perfectionism, and a history of depressive disorders (Hale & Smith, 2012; Murray et al., 2012; Olivardia, Pope, & Hudson, 2000). For example, in Olivardia

et al.'s (2000) research, 50% of the men diagnosed with MD reported that they spent more than 3 hours per day thinking about their muscularity. Fifty-eight percent reported "moderate" or "severe" avoidance of activities, places, and people because of their perceived body defects (Olivardia et al., 2000). These men avoided beaches, swimming pools, locker rooms, and other places where their bodies might be seen, and they expressed marked levels of distress in these situations. In addition, 54% reported little or no control over their compulsive weight-lifting and dietary regimens.

Males at Higher Risk

Eating disorders and disordered eating have been found to be higher among particular populations of men. These include gay and bisexual men, athletes, and men from minority cultural groups and developing countries.

Gay Men

Between 14% and 42% of gay and bisexual men in both clinical and community samples have been found to have EDs (Feldman & Meyer, 2010; see also Chapter 23). In comparison to heterosexual men, gay and bisexual men report higher levels of body dissatisfaction, ED symptoms and EDs, and strategies to increase muscles (Bosley, 2011; Feldman & Meyer, 2010; Ricciardelli & McCabe, 2004). These higher levels have been attributed to the male gay subculture, which places greater emphasis on the lean and muscular body ideal, appearance, and fashion (Epel, Spanakos, Kasl-Godley, & Brownell, 1996). However, pressures to be lean and muscular from partners and friends are also important. Pressure from friends to be lean and muscular has been found to be related to gay men's disordered eating behavior, and pressure from partners related to muscularity enhancement behaviors (Tylka & Andorka, 2012). In addition, gay men's increased experiences of stigmatization and the resulting feelings of shame may be another factor that places them at a higher risk for the development of disordered eating (Ricciardelli & McCabe, 2004). Other factors that may mediate or moderate the relationship between sexual orientation and disordered eating include level of adherence to gay and lesbian subculture, coping skills, depression, anxiety, and gender role traits (Ricciardelli & McCabe, 2004).

Athletes

The sporting environment for male athletes has also been highlighted as a significant risk factor for the development of eating disorders and disordered eating, and the use of steroids (Glazer, 2008; Ricciardelli & McCabe, 2004; Strother et al., 2012; see also Chapter 35). Actual estimates of the prevalence of EDs and ED symptoms among male athletes vary from study to study due to different sampling procedures, sample sizes, and criteria to define EDs (Glazer, 2008; Ricciardelli & McCabe, 2004), but rates tend to be higher than those found in samples from the general population. Among male athletes estimates for the prevalence of AN range from 0% to 1.6% (2.85% to 4.2% for women), and for BN from 0.005% to 14.3% (1.1% to 39.2% for women; Ricciardelli & McCabe, 2004). In addition, subclinical levels of EDs among elite athletes have been found to be 10–50 times higher than those found in the general population (Glazer, 2008).

The sporting environment often involves a subculture that emphasizes attaining optimal weight for athletic performance, and promotes the use of unhealthy eating and extreme weight-management behaviors to achieve this ideal (Hausenblas & Carron, 1999; Wooldridge & Lytle, 2012). In addition, many of the individual characteristics that are advantageous for athletic performance are the same characteristics commonly found in individuals with EDs (Hausenblas & Carron, 1999). These include perfectionism, compulsiveness, self-motivation, and high achievement expectation. Athletes who are most at risk of developing disordered eating are those who take part in sports where (a) weight classifications apply (e.g., wrestlers, rowers), (b) weight or small body size is important for success (e.g., distance runners, cyclists), or (c) subjective evaluation and aesthetic ideals coexist (e.g., figure skaters, gymnasts, divers; Hausenblas & Carron, 1999). When different kinds of sports have been compared, participants most at risk of using steroids are adolescent boys who take part in power sports, such as field events and weight-lifting (Wichstrom & Pedersen, 2001), and bodybuilders (Blouin & Goldfield, 1995).

Minority Cultural Groups

Men from minority cultural groups, such as Blacks, Hispanics, Asians, and Native Americans in the United States, and men from developing countries with fast-changing social structures such as South Africa, Hong Kong, and Oman, also appear to be at greater risk of developing disordered eating. These cultural groups have been found to engage in more extreme weight loss strategies and binge eating than White men from Western countries (Ricciardelli, McCabe, Williams, & Thompson, 2007). The differences may in part reflect the changing status quo and power relations for males and/or the higher level of social isolation of men in minority groups when compared to the dominant cultural group(s) (see Chapter 25).

Sociocultural Pressures to Obtain a Muscular Ideal

A growing body of literature has found that males experience significant pressure from a range of sociocultural influences (such as messages from the family, peers, media, and the broader community) to achieve the lean mesomorphic muscular ideal (McCabe & Ricciardelli, 2005). These sociocultural pressures for males to obtain a body that is symbolic of masculinity and power have been shown to increase body image concerns among men who fail to achieve this body form (Smolak, 2009). Even at the age of 2 years, young boys are becoming increasingly aware of the importance of muscularity for their popularity, and consequently, their self-worth (McCabe et al., 2007; Thompson, Schaefer, & Menzel, 2012). This awareness of muscles and masculinity that begins early in life is reflected in the significant increase in the muscularity of male action figures over time (Pope, Olivardia, Gruber, & Borowiecki, 1999). Constant exposure to media depictions of powerful superheroes and muscular action figures, which are seen to reflect cultural ideals of the male physique, may distort young boys' earliest messages regarding the ideal male body, and can have a considerable negative impact on body image in later life (see Chapter 29).

McCabe and Ricciardelli (2004b) have previously demonstrated clear gender differences in the ideal physique in Western cultures: males focus on ideals of muscularity, power, and strength as opposed to females, for whom modelesque thinness is ideal. It is important to note that men, in comparison to women, tend to focus more on functionality rather than appearance (McCabe & Ricciardelli, 2004b; Phillips & de Man, 2010). For example, there is a strong

emphasis within Western society for males to excel in sports, and so the emphasis is on obtaining a (muscular) body that will allow males to achieve this goal (Ricciardelli et al., 2006). These pressures commence in late childhood and continue into adulthood (McCabe & Ricciardelli, 2004a). Deviation from these ideals is associated with eating and mental health problems (Smolak, 2011). Phillips and de Man (2010) found that male body image concerns and negative reactions to these concerns were often not detected by researchers because studies (mistakenly) focused on appearance.

The limited research that has examined the role of sociocultural factors on body image among male children suggests that parental, peer, and media influences are all associated with their body image concerns (Ricciardelli, McCabe, Mussap, & Holt, 2009) and that these concerns are linked to both weight loss and muscle-building strategies. Among adolescent boys, Ricciardelli, McCabe, and Banfield (2000) found that compared to parental influences, peers and the media had a larger effect on body dissatisfaction than parents. However, a study by McCabe et al. (2011) compared Australian adolescent boys of European heritage to Indo-Fijian, Indigenous Fijian, and Tongan boys and found, contrary to expected results, that very few Australian and Tongan boys received direct comments about weight or muscles from their parents and that the media did not present messages about the body for Australian, Fijian, or Tongan boys. In contrast, Indigenous Fijian and Tongan boys were encouraged to achieve a large muscular body by family and friends, but this body image was placed within the context of sporting achievement (i.e., increasing the size of the upper body to be stronger and not be so easily tackled in rugby). However, the authors also noted that Australian boys engaged in strategies to increase muscles if they were encouraged to do so, even in the absence of high levels of body dissatisfaction. Thus, it would appear that body dissatisfaction may not be a pathway for males to engage in health risk behaviors (e.g., steroids, exercise dependence, eating disorders) to achieve the muscular ideal. Rather the pathway may be through attempts to obtain a body that functions well in the sporting context. This is also consistent with research that shows that extreme weight-management behaviors, indicative of disordered eating, are frequent among athletes (Hausenblas & Carron, 1999), despite high levels of body satisfaction (Hausenblas & Symons-Downs, 2001; see also Chapter 35).

Adult men also receive messages from family, peers, and the media to achieve the muscular ideal (Gillett & White, 1992; McCabe & McGreevy, 2011; Ricciardelli & McCabe, 2001). A muscular physique is also seen to portray traits that include dominance, strength, sexual virility, and self-esteem (Pope et al., 2000). For adult men, the importance of appearance is related not only to what they look like, but also to their health, fitness, and general well-being (McCabe & McGreevy, 2011; McCabe & Ricciardelli, 2004a). Gillett and White (1992) claimed that failure to achieve the ideal body form leads to vulnerability and anxieties associated with this vulnerability, and results in high levels of body dissatisfaction. Halliwell and Harvey (2006) suggested that perceived pressure concerning appearance from the media, family, and peers leads to disordered eating though internalization of sociocultural ideals and body dissatisfaction. Whilst men across all age groups may report less body dissatisfaction and appear to be less affected by sociocultural pressures compared to women (McCabe & Ricciardelli, 2004a), there is evidence to suggest that men may internalize the lean but muscular body ideal featured in men's fitness magazines and other sources of media in a similar way to women (Labre, 2005). Further support for this view was provided by Barlett, Vowels, and Saucier (2008), who conducted a meta-analysis that showed that, in both correlational and experimental studies, high levels of perceived pressure from the mass media were related to high levels of body dissatisfaction, depression, and excessive exercise, as well as low levels of body esteem and self-esteem.

Other Risk Factors

A range of other biopsychosocial factors have been found to be associated with disordered eating and/or the pursuit of muscularity among boys and men. On the whole, these are similar to those found to be associated with disordered eating and body image concerns among girls and women. They include pubertal development, genetic factors, negative affect, perfectionism, family relationships, and physical and sexual abuse (Ricciardelli & McCabe, 2004; Strother et al., 2012; Wooldridge & Lytle, 2012; see also Chapters 26, 28, 32, & 34). However, in some cases there is either inconsistent or limited evidence for these relationships, and often the associations have not been verified with longitudinal research.

Pubertal development is usually considered a positive experience for boys, as boys add muscle and their shoulder width increases, which are physical changes that bring boys closer to the societal ideal shape for a man (Ricciardelli & McCabe, 2004). However, this does not appear to protect boys from weight loss strategies and disordered eating. One longitudinal study showed that early maturing boys, like early maturing girls, were more likely to adopt strategies to decrease weight than late maturing boys (McCabe & Ricciardelli, 2004b). Similarly, a more recent longitudinal study demonstrated that advanced pubertal development in early-to-mid adolescence was associated with greater disordered eating in late adolescence among both girls and boys (Baker, Thornton, Lichtenstein, & Bulik, 2012). In addition, while early maturing boys may be more at risk of disordered eating, later maturing boys appear to be more at risk of behavioral problems associated with the pursuit of muscularity. In support of this view, McCabe and Ricciardelli (2004b) found that late maturing boys were more likely to develop exercise dependence than early maturing boys. In addition, late maturing boys were more likely to use food supplements to build up their body than early maturing boys, and this moderately predicted increase in strategies to increase their muscle size. However, given that research examining the association between puberty and disordered eating is still limited, further studies are needed to verify these findings.

Genetic factors also appear to be influential in the development of disordered eating for males (Baker et al., 2009; Boraska et al., 2012; Suisman & Klump, 2011). However, studies that have examined heritability estimates for different aspects of disordered eating (e.g., body dissatisfaction, weight loss strategies, bulimic symptoms), have shown these estimates to be lower for males compared with females, suggesting that genetic influences are less important for males (Baker et al., 2009). Moreover, specific genes have been found to be related to disordered eating among females (Peňas-Lledó et al., 2012; Suisman & Klump, 2011), and while these also appear relevant to males, they have yet to be examined in samples that focus exclusively on males (Boraska et al., 2012).

Negative affect is an individual factor that has often been found to characterize disordered eating behavior among adolescent boys in both cross-sectional and longitudinal studies (Ricciardelli & McCabe, 2004). A higher prevalence of anxiety and depression has also been found among men with EDs in comparison to men without EDs (see Chapters 15 & 54). This finding is based on studies including ones that have examined community samples of adult men (Woodside et al., 2001), gay and bisexual men (Feldman & Meyer, 2010), and adolescent boys (Johnson, Cohen, Kotler, Kasen, & Brook, 2002). Studies have further shown that among adolescent boys negative affect is associated with eating and exercise patterns that are used to increase muscles (Ricciardelli & McCabe, 2004). Although there is ongoing debate about the directional and causal nature of this relationship, one of the main views is that disordered eating and strategies to increase muscles are used to regulate and/or alleviate negative affect (Ricciardelli & McCabe, 2004). The same view has recently been highlighted by Kaye,

Wierenga, Bailer, Simmons, and Bischoff-Grethe (2013), who argue that food restriction among individuals with AN may be used, in the short term, to regulate negative mood, and this involves the serotonin-based aversive or inhibitory and dopamine-based reward systems. This view is also consistent with the finding that anxiety and depressive disorders predate EDs (Feldman & Meyer, 2010).

Another individual factor shown to characterize both men and women with eating disorders is perfectionism (Wooldrigde & Lytle, 2012). Although studies with males are still sparse, higher levels of perfectionism have been found to be associated with greater disordered eating symptoms and the desire to become bigger among preadolescent boys (Saling, Ricciardelli, & McCabe, 2005), adolescent boys (Ricciardelli & McCabe, 2004), and adult males (Blouin & Goldfield, 1995). Given that perfectionists display extreme efforts to achieve often unattainable ideals, it is understandable that these often underlie their pursuit to achieve the lean mesomorphic muscular ideal.

In their review of AN in men, Wooldridge and Lytle (2012) also highlighted the importance of family factors. Boys and men with AN were more likely to live or have grown up in a single-parent home (Lindblad, Lindberg, & Hjern, 2006), and had absent fathers who were living away or who had died (Fichter & Daser, 1987). Other important family factors include the parenting styles of fathers and mothers. Fathers have been described as domineering, controlling, and rigid (Beumont, Bearwood, & Russell, 1972; Sterling & Segal, 1985), who place high pressures on their sons to excel in sports (Romero, 1994), while mothers have been described as overly controlling and protective (Romero, 1994).

Olivardia et al. (2000) found that a negative relationship with mother was a clinical feature associated with MD; however, the specific nature of these men's relationships with their mothers was not explained. It may be that these men, like women who display more eating-disordered behavior, feel less accepted, and feel that they are more criticized by their parents (Swarr & Richards, 1996). Moreover, some studies have shown that disordered-eating symptoms among adolescent boys are associated with more negative parental relationships in terms of lower perceived intimacy with both father and mother, poorer family communication, and low perceived parental caring (Ricciardelli & McCabe, 2004). However, these results have not been found consistently across all studies (Ricciardelli & McCabe, 2004).

Sexual and physical abuse is also frequently reported by eating-disordered patients, and EDs may develop as a way of coping with this abuse (Strother et al., 2012; Wooldridge & Lytle, 2012). In comparison to women, a smaller number of men with EDs report sexual abuse, but this may be due to the underreporting of abuse by men given that more shame and stigmatization accompanies this experience for men (Strother et al., 2012). Two studies were located that specifically examined disordered eating in relation to sexual and physical abuse during childhood, and both studies provided support for this relationship among adolescent girls and boys (Lock, Reisel, & Steiner, 2001; Neumark-Sztainer, Story, Hannan, Beuhring, & Resnick, 2000).

Conclusions and Future Directions

Although the prevalence of EDs, as evaluated by criteria that have been primarily designed for women, is lower in males, when we include partial syndrome EDs, eating patterns that are often dismissed (e.g., excessive exercise and overeating), and behavioral problems associated with the pursuit of muscularity, men's problems are as prevalent and significant as those found among women. In addition, it is important to note that effect sizes, when examined for the

gender differences in ED symptoms, have been found to be quite small, again highlighting that disordered eating attitudes and behaviors have been underestimated among men.

These findings demonstrate that there is a need to promote a greater awareness of the nature of disordered eating in males, and recognize that body dissatisfaction and disordered eating are not only "female" problems, so that prevention and intervention programs can be accessed by men without fear of stigma. Further, in order to more fully understand the development of disordered eating and associated behaviors from the perspective of boys and men, in-depth and less structured interview studies, using qualitative research designs are needed. These will allow issues that are specifically relevant for boys and men to emerge from the data and be used to develop new and more applicable models that address attitudes and behaviors related to both disordered eating and the pursuit of muscularity.

We still have only a limited understanding of the presentation and development of EDs and disordered eating among men. As concluded by Jones and Morgan (2010, p. 29): "we persist in attempting to fit men with eating disorders into a theoretical and clinical framework largely focused on the physical, psychological, and emotional development of women." Future research on EDs among men needs to broaden the diagnostic criteria and measures from those that traditionally apply to women to include those that represent male symptoms.

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Section VII
Assessment

38

Screening for Eating Disorders: An Updated Guide

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While base rates of eating disorders (EDs) in the general population are relatively low (see Chapters 5 & 37), early identification is strongly associated with better prognosis (Becker, Franko, Nussbaum, & Herzog, 2004). Furthermore, subthreshold disorders are associated with high levels of distress, and identifying individuals suffering from partial forms of EDs may provide a target for early intervention (Becker et al., 2004). While the literature is not fully convergent, it has been suggested that subthreshold forms of EDs may constitute precursors of full-syndrome EDs, at least among certain groups (Stice, Ng, & Shaw, 2010). Early referral may therefore assist with both improved prognosis and prevention of progression. In addition to early referral, screening for EDs may be conducted with the intention of better targeting an intervention, or establishing prevalence data. Screening tools therefore need to provide accurate information regarding ED status, but also be suitable for use with large groups such as school or university settings, to provide national prevalence data (e.g., Youth Behavior Risk Survey). Developing accurate screens may therefore contribute to better targeting of existing resources and improving the mental health of individuals suffering from or at risk for EDs.

Properties of Screening Instruments: How to Know When a Screening Tool is Useful

An important requirement for a screening instrument is validity, and in particular criterion validity, defined as how well the screen identifies real cases and non-cases. The criterion validity of a screening instrument can be determined by comparing the results obtained with the screen to the results obtained using a "gold standard" test in the same population, that is, the best available test at the time.

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Sensitivity and Specificity

Sensitivity and specificity are two indices of a scale's diagnostic performance, with high values indicating that the scale is highly accurate at classifying cases and non-cases in the correct category. Thus, an instrument with low sensitivity and specificity would both "miss" some of the individuals who actually should have the diagnosis, and misclassify healthy individuals (false positives). The sensitivity of a screening instrument refers to the proportion of cases accurately detected, that is, the proportion of true positives (i.e., cases) correctly identified by the screen. In other words, sensitivity tells us the proportion of people who should have been identified as having an ED who are identified as having an ED.

The specificity refers to the proportion of true negatives identified by the screen. Negatives are people who do not suffer from the disorder, in this case EDs. True negatives are those identified as not having the disorder who don't, while false negatives are those who actually have an ED but are identified as healthy. Thus it assesses the proportion of healthy people who do not have an ED who are classified as not having an ED. The specificity is equal to the number of non-cases correctly identified by the screen over the number of "true" non-cases as determined by the gold standard.

Importantly, there are no fixed guidelines for what constitutes acceptable sensitivity and specificity. Values around 80% are generally considered good; however, it has been suggested that among low-prevalence disorders such as EDs the sensitivity and specificity should be close to 90% (Williams, Hand, & Tarnopolsky, 1982).

Positive and Negative Predictive Value

The specificity and sensitivity of a test are useful parameters in that they respectively indicate how many individuals will be correctly classified as healthy or presenting the diagnosis. However, sometimes it may be more useful to know how many of the cases identified by a screening test are true cases. In other words, is the screening measure overinclusive in the cases it identifies, or on the contrary, too narrow? For example, are people who are healthy being mistakenly identified as having EDs? Positive and negative predictive values address such questions.

The positive predictive value refers to the proportion of individuals who are identified as cases by the screen who actually are cases. Thus it evaluates the capacity of the test to include only real cases as cases and not be overinclusive. The positive predictive value is highly dependent on the prevalence of the disorder. A correlate of the importance of prevalence in determining positive and negative predictive values is that for low-prevalence disorders, such as anorexia nervosa (AN), the positive predictive value can be very low even when sensitivity and specificity are high (Jacobi, Abascal, & Taylor, 2004). The negative predictive value correspondingly refers to the proportion of non-cases who are correctly identified as healthy by the screen. It evaluates the capacity of the measure to capture all of the cases.

Depending on the objective of the screening, it may be desirable to maximize different properties. In the case of screening for cases for treatment, for example, it is desirable to minimize the number of falsely identified negatives, thus increasing sensitivity, so as to be sure not to miss cases. In the context of wide screening for risk factors for EDs, however, such as might be conducted as an initial step on a multiphase screening program, a screening measure should minimize the proportion of false positives among those scoring above the cut-offs (i.e., the positive predictive value should be maximized) to reduce the number of interviews to be carried out in the second phase.

Caution in the Use of Screening Instruments for ED

In their review, Jacobi et al. (2004) called for caution in the use of screening instruments, identifying a number of limitations of the available instruments, including an overwhelming focus on diagnosis rather than on identifying high-risk individuals. Jacobi et al. also questioned the external validity of existing screens, in particular their usefulness among diverse populations. They suggested the use of a two-stage procedure that would begin with an initial broad screen with an instrument designed to assess risk status, such as the Weight Concerns Scale (Killen et al., 1993), followed by the administration of a more specific diagnostic instrument, such as the Eating Disorder Examination Questionnaire (Mond, Hay, Rodgers, Owen, & Beumont, 2004; see also Chapters 9 & 40), to those screening positive on the initial measure. Ideally, individuals identified as cases would then be referred for further evaluation and potential treatment. Although Jacobi et al.'s (2004) in-depth review was conducted over a decade ago, in many cases their criticisms are still valid.

Screening for Cases

Widely Used Screening Tools

Eating Attitudes Test (EAT) Both the original 40-item version of the EAT (Garner & Garfinkel, 1979), as well as the shorter 26-item version (Garner, Olmsted, Bohr, & Garfinkel, 1982), have been extensively used as screening instruments for EDs. Cut-off scores—defined as the score that identifies individuals warranting further evaluation for EDs or subclinical forms—of 30 and 20 have been established for the EAT-40 and EAT-26, respectively (Garner et al., 1982). Early studies of the EAT highlighted its low positive predictive power, particularly for classifying cases of AN (Mann et al., 1983). It was, however, suggested that this failing might stem from changes in diagnostic criteria, as the EAT was developed at a time when eating disorders were not included the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*), and the clinical population included in the initial validation met the Research and Diagnostic Criteria established by Feighner et al. (1972; cited in Spitzer, Endicott, & Robins, 1978), which were very different from those used in *DSM-III* (3rd ed.; American Psychiatric Association, 1980; Mintz & O'Halloran, 2000). In a later validation study of 136 college women using the *DSM-IV* (American Psychiatric Association, 1994) ED criteria, the EAT-40 and EAT-26 demonstrated a sensitivity of 77%, specificity of 95% and 94% respectively, positive predictive power of 82% and 79% respectively, and negative predictive power of 93% and 94% respectively (Mintz & O'Halloran, 2000). These findings suggested that the EAT may in fact be a useful instrument for differentiating between individuals with EDs, regardless of diagnosis, and those without. The EAT has been widely translated and is available in Arabic, Brazilian, Chinese, French, Greek, Italian, Japanese, Portuguese, Spanish, and Turkish.

Eating Disorder Examination Questionnaire (EDE-Q) The EDE-Q (Mond et al., 2004) is a 36-item self-report questionnaire derived from the *DSM-IV* (American Psychiatric Association, 1994) criteria for EDs, assessing eating behaviors over the last 28 days. As noted previously, the interview version of the EDE is one of the gold standard instruments often used in the validation of screening instruments (Cooper & Fairburn, 1987). A global score can be calculated as well as four subscales: Restraint, Weight Concerns, Shape Concerns, and Eating

Concerns. An initial analysis identified a cut-off score of 56 on the summed total score (2.3 on the global score). Using this cut-off among a community sample of 208 young women resulted in a sensitivity of 92%, specificity of 86%, and positive predictive value of 30% (Mond et al., 2004). Including two extra criterion items, namely the frequency of objective binge episodes and exercising for weight/shape reasons at least once a week, in addition to the cut-off score resulted in an increase in positive predictive value to 56%. In a later examination of the properties of the EDE-Q among 25 ED cases, analysis suggested 2.8 on the global scale as the optimum cut-off point, resulting in a sensitivity of 96%, specificity of 56%, and positive predictive value of 30% (Mond et al., 2008). Lowering the cut-off score to 1.61, so as to increase sensitivity, resulted in a value of 92% but did not affect the specificity (56%) and positive predictive value (30%).

The usefulness of the EDE-Q in relation to the proposed *DSM-5* (American Psychiatric Association, 2013) criteria (see Chapters 8–11) has also been explored. In a recent study validating the EDE-Q against the EDE-interview version among 217 treatment-seeking individuals, Berg et al. (2012) reported that when using the proposed *DSM-5* criteria, the EDE-Q demonstrated sensitivity ranging from 36.8% to 80.8% according to the diagnosis, specificity ranging from 77.3% to 98%, positive predictive values ranging from 41.2% to 81.5%, and negative predictive values ranging from 76.5% to 98.5%. The EDE-Q performed poorest for binge eating disorder (BED), but reasonably well for AN, bulimia nervosa (BN), and eating disorders not otherwise specified (ED-NOS).

Fairburn, Cooper, Doll, and Davies (2005) explored the longitudinal predictive validity of the EDE-Q among 2,992 young women who screened positive for dieting but did not have a present or past ED. The women were contacted every 6 months over a 2-year period to assess present ED symptomatology. Based on their findings, the authors developed an eight-item screening instrument, which included seven items from the EDE-Q, as well as body mass index (BMI). Using this screening instrument, sensitivity ranged from 69.5% to 79% and specificity from 52% to 76%, depending on the decision tree used, with the final survey therefore displaying good sensitivity and specificity of 79% and 76% respectively. This brief screening instrument therefore appears useful in detecting dieting young women at risk of developing a future ED. The EDE-Q has been translated into many languages including Spanish, Fijian, and Turkish.

Bulimia Test-Revised (BULIT-R) The BULIT-R (Thelen, Farmer, Wonderlich, & Smith, 1991) is a 28-item self-report measure designed to assess the presence and degree of bulimic symptomatology according to the *DSM-III-R* (American Psychiatric Association, 1987). Using a cut-off score of 104, initial examinations of the BULIT-R in a sample of 23 women with BN versus 153 controls, as well as a second sample of 53 subjects with scores above and below the cut-off, revealed rates of sensitivity of 83% and 62% respectively, and a specificity of 96% (Thelen et al., 1991). A second study explored the properties of the BULIT-R among 243 female college students (Welch, Thompson, & Hall, 1993). Using the original cut-off of 104, the sensitivity of the scale was 80%, the specificity was 99.5%, the positive predictive value was 80%, and the negative predictive value was 99.5%. In order to increase the positive predictive value, the authors proposed a new cut-off score of 98. Using this threshold, the measure's sensitivity was 100%, the positive predictive value was 71.3%, and the negative predictive value was 100%. A later validation study using *DSM-IV* diagnostic criteria for BN and the initial cut-off score of 104 in a sample of 147 women

revealed a sensitivity of 91%, a specificity of 96%, a positive predictive value of 81%, and a negative predictive value of 98% (Thelen, Mintz, & Vander Wal, 1996). Taken together, these data indicate that the BULIT is a useful and valid screen for identifying bulimic symptomatology. The BULIT has been translated into Korean and Spanish.

SCOFF The SCOFF (Morgan, Reid, & Lacey, 1999) is a five-item (Yes/No) screening instrument focusing on the core features of AN and BN:

- **S**ick (self-induced vomiting);
- **C**ontrol (loss of control);
- **O**ne stone (weight loss of 14 lb [6.35 kg]);
- **F**at (feelings of fatness);
- **F**ood (preoccupation with food).

In an initial examination among 116 patients with EDs and 96 controls, the threshold of two positive answers to SCOFF questions had 100% sensitivity for detecting AN or BN and an 87.5% specificity (Morgan et al., 1999). Confirmation of the two positives cut-off came from a study comparing the SCOFF and the EDE-Q in a sample of 257 primary care patients, which reported a sensitivity of 72% and specificity of 81% (Mond et al., 2008).

Additional studies have confirmed the high sensitivity and specificity of the SCOFF. An examination of the properties of the written and orally administered SCOFF in a sample of 178 college students revealed a high level of agreement between these two forms in the prediction of possible EDs using the two positives cut-off (Perry et al., 2002). Among 341 female primary care patients, the questionnaire demonstrated a sensitivity of 86.4% and a specificity of 89.6% (Luck et al., 2002). Using the cut-off of two positives, all women confirmed to be suffering from an ED were detected, in addition to 34 false positives.

Other authors, however, have reported lower levels of sensitivity for the SCOFF. In a sample of 305 graduate students, the SCOFF demonstrated a sensitivity of only 53.3% with a specificity of 90.3% (Parker, Lyons, & Bonner, 2005). The resulting positive predictive value was 66.7%, while the proportion of those who were correctly identified as not having an ED by the screen (NPV) was 88.7%. These findings suggested that the SCOFF may not be as sensitive as previously suggested among this population. Similarly, among a sample of 1,891 Finnish adolescents, the agreement between SCOFF ratings and nurse evaluations was low, with only a 20% agreement for students scoring above the two-positives cut-off (Hautala et al., 2009). The authors suggested that among adolescents, when it is preferable to be overinclusive rather than fail to detect an at-risk individual, a cut-off of one positive might be useful. Chinese, Finnish, French, Italian, Japanese, and Spanish versions of the SCOFF are available.

Less Widely Used Screening Measures

Bulimic Investigatory Test, Edinburgh (BITE) The BITE (Henderson & Freeman, 1987) is a 36-item self-report questionnaire that yields symptom (cut-off score = 20) and severity subscale scores (cut-off = 5), as well as a total score (cut-off = 25). The validity of the BITE was assessed in a sample of 55 ED patients and 27 control women (Waller, 1992). Findings revealed that the BITE performed well in identifying restricting AN cases as well as individuals suffering from BN with and without history of AN; however, the BITE performed poorly (false negative

rate = 60%) in the identification of AN/bulimic subtype. The author cautioned against the use of the BITE for screening, as it appeared to lack sensitivity, in particular regarding cases of normal-weight BN. A number of translations of the BITE exist, including Italian and a Portuguese version developed among a Brazilian sample.

Eating Disorder Diagnostic Scale (EDDS) The EDDS (Stice, Telch, & Rizvi, 2000) is a 22-item self-report measure designed to assess the presence of the *DSM-IV* (American Psychiatric Association, 1994) criteria for AN, BN, and BED. The EDDS was validated against the EDE in a sample of 367 women aged 13 to 61. For the three disorders, the EDDS demonstrated a sensitivity ranging from 77% to 93%, a specificity ranging from 96% to 100%, positive predictive values ranging from 80% to 93%, and negative predictive values ranging from 95% to 100%. Similarly, strong validity was found in a second study in which the EDDS was again examined against the EDE in a sample of 443 female adolescents and young adults, yielding a sensitivity of 88%, specificity of 98%, positive predictive value of 74%, and negative predictive value of 98% (Stice, Fisher, & Martinez, 2004). The validity of the Dutch version of the EDDS was also evaluated in a sample of 40 ED patients and 45 control undergraduate students (Krabbenborg et al., 2012). The EDE was again used as the gold standard. The EDDS demonstrated high sensitivity (>95%) and positive predictive value among patients suffering from AN and BN, but lower sensitivity (57%) and positive predictive value (80%) among patients suffering from BED. The authors further identified a cut-off point of 16.5 to distinguish patients from healthy controls. Together these findings suggest that the EDDS is a very useful screening tool. It is unclear why it is not widely used.

Questionnaire for Eating Disorders Diagnosis (Q-EDD) The Q-EDD (Mintz, O'Halloran, Mulholland, & Schneider, 1997) is a 50-item self-report questionnaire, based on the *DSM-IV* (American Psychiatric Association, 1994) diagnostic criteria, that aims to evaluate the presence of AN, BN, and four types of ED-NOS (subthreshold bulimia, menstruating anorexia, nonbingeing bulimia, and binge eating disorder). The Q-EDD was validated against a clinical interview and demonstrated a sensitivity of 97%, specificity of 98%, positive predictive value of 94%, and negative predictive value of 99% in distinguishing between eating-disordered individuals all diagnoses combined, and non-eating-disordered individuals. The Q-EDD has been used in large screening studies among young women (Franko et al., 2005).

Binge Eating Disorder: The New Addition to *DSM-5*

BULIT

The BULIT has been suggested to be well suited to the evaluation of binge eating as it contains items assessing compensatory behaviors (Vander Wal, Stein, & Blashill, 2011). In a sample of 15 individuals suffering from BED and 26 assessed as having neither full-syndrome nor subclinical BED, using a cut-off score of 80, the BULIT demonstrated a sensitivity of 100%, specificity of 96%, positive predictive value of 94%, and negative predictive value of 100%. Although provided by a single study, these findings confirm that the BULIT can successfully be used to classify individuals suffering from BED.

The Patient Health Questionnaire Module for Eating Disorders (PHQ-ED)

The PHQ-ED (Spitzer, Kroenke, & Williams, 1999) is composed of six binary (yes/no) items assessing binge eating and compensatory behaviors, and up to two optional follow-up questions that are asked only when binge eating or purging has been endorsed. A recent study examined the properties of the PHQ-ED as a screening instrument in a sample of 348 adults that included 259 cases of BED, BN, or Recurrent Binge Eating (Striegel-Moore et al., 2010). The PHQ-ED showed excellent sensitivity of 100% across all three diagnoses, and good specificity of 91.7% for BED and BN, and 92.4% for Recurrent Binge Eating. However, the positive predictive value was fairly low, with 10.4% for BED and BN, and 18.5% for Recurrent Binge Eating. The authors suggested that the low positive predictive value may have been due to the study sample, which was drawn from a community population with a low prevalence of BED. Clinicians should be cautious in inferring the likely presence of a disorder from a positive screen, particularly when using the PHQ-ED in community samples.

Bulimic Investigation Test, Edinburgh (BITE)

The BITE, as described above, has also been explored in the context of screening for BED. The BITE's psychometric properties were examined in a sample of 360 consecutive patients attending an outpatient clinic, using both a cut-off of 10 and a cut-off of 20 (Ricca et al., 2000). With the lower cut-off, the BITE displayed a sensitivity of 91%, specificity of 51.4%, positive predictive value of 71.8%, and negative predictive value of 98.2%. Using the higher cut-off of 20, the sensitivity decreased to 33.3%, while the specificity increased to 92.0%, with a positive predictive value of 30.5% and a negative predictive value of 92.9%.

Binge Eating Scale (BES)

The BES (Gormally, Black, Daston, & Rardin, 1982) is a 16-item self-report questionnaire, scored on a 0–3 scale. A cut-off score of 17 has been established as indicating the presence of binge eating. In a sample of 125 women attending a weight-loss program, only 51.8% of those identified as binge eaters using the BES cut-off score of 17 had a concordant diagnosis using the EDE (Greeno, Markus, & Wing, 1995). In a recent study among 473 consecutive patients presenting for psychological evaluation prior to gastric bypass surgery, using the conventional cut-off score of 17, the BES displayed a sensitivity of 94%, specificity of 76%, positive predictive value of 37%, and negative predictive value of 99% (Grupski et al., 2013). To increase the positive predictive value, the authors conducted the analysis using a higher cut-off score of 27. With this new threshold, the BES displayed a much lower sensitivity of 37%, a specificity of 96%, a positive predictive value of 56%, and a negative predictive value of 91%. An analysis confirmed 17 as the optimal cut-off score in order to maximize both sensitivity and specificity.

Questionnaire for Eating and Weight Patterns (QEWP)

The QEWP (Spitzer et al., 1992) is a 13-item self-report questionnaire assessing the presence of BED according to the *DSM-IV* (American Psychiatric Association, 1994) criteria. Data regarding the usefulness of the QEWP as a screening instrument have been mixed. In a sample of 157 adults screened for a BED treatment study, the QEWP demonstrated a sensitivity of 74% and a specificity of 35%. Using the Structured Clinical Interview for DSM-IV (SCID) as

a comparison (First, Spitzer, Williams, & Gibbon, 1995), the QEWP demonstrated a sensitivity of 55%, specificity of 80%, and positive predictive value of 79%. Further research is necessary to clarify the validity of the QEWP.

Overview of Tools for Screening for High-Risk Individuals

The McKnight Investigators

The McKnight Investigators (2003) conducted a 4-year longitudinal study of 1,358 high-school students. The McKnight Risk Factor Survey IV includes seven different factors: thin-body preoccupation and social pressure; substance use; parental influences; general psychological influences; social support; number of negative life-events; and school performance. The items from the thin body preoccupation and social pressure factors were examined for their usefulness as a potential screen for ED cases. With the aim of deriving the most parsimonious screening tool, three criteria were derived from four questions:

1 A score of 4 or 5 in response to "How often have you been on a diet to lose weight?"
2 A score of 5 in response to "How often does your weight make boys not like you?"
3 An average score ≥3.5 in response to the two questions: "How often do you change your eating around boys?" and "How often do you change your eating around girls?"

When considering participants who scored above the cut-off for any of these three questions, the screen demonstrated a sensitivity of 72% and a specificity of 80%. The predictive value of a positive test was 10% (of a base rate of cases of 2.9%), and the predictive value of a negative test was 99% (of a base rate of non-cases of 97.1%).

Weight Concerns Scale

Using data from the first wave of a large longitudinal study of 967 sixth- and seventh-grade girls, Killen et al. (1993) developed a five-item Weight Concerns measure assessing worry over weight and body shape, fears of gaining weight, importance of weight, perceived fatness, and diet history. Their initial analysis revealed that, compared to SCID diagnoses (First et al., 1995), a score of 57 on this scale yielded a sensitivity of 86% and specificity of 63%. Their measure demonstrated excellent 7-month stability and longitudinal validity, as scores were significantly associated with the onset of ED symptoms 3 years later (Killen et al., 1994). The Weight Concerns Scale is one of the few screening instruments available to identify high-risk individuals rather than cases and is supported by evidence for longitudinal validity.

Stanford-Washington Eating Disorder Screen (SWED)

The SWED (Wilfley, Agras, & Taylor, 2013) contains 11 items taken from the Weight Concerns Scale, the EDE-Q, the EDDS, and the Clinical Impairment Assessment (Bohn et al., 2008). The scale properties have been examined among college women compared to the SCID and the EDE-Q, and the SWED demonstrated high sensitivity and specificity (>90%) for AN and BN when using *DSM-IV-TR* (American Psychiatric Association, 2000) and *DSM-5* criteria (Wilfley et al., 2013). Further investigations of the criterion validity of the instrument using *DSM-5* (2013) criteria are underway, and the SWED appears to be a very promising instrument for screening for ED risk.

Specific Populations: Children, Adolescents, Athletes, and Men

Children's Eating Attitudes Test (ChEAT)

The ChEAT is a modified version of the EAT (see above) with language adapted for children (Vacc & Rhyne, 1987). However, data on the properties of the ChEAT as a screening instrument are somewhat lacking. One study conducted among 152 preadolescents aged from 8 to 12, testing a variety of potential cut-offs, suggested 25 as an optimal cut-off, but concluded that the sensitivity of the ChEAT was overall somewhat low and varied with age (Erickson & Gerstle, 2007). Using the threshold of 25, overall sensitivity was 36%; however, this increased to 53% among girls aged between 10 and 12 years. In a sample of 409 girls aged 9–13 years, compared to the child version of the EDE using the cut-off score of 20, the ChEAT demonstrated a sensitivity of 17%, specificity of 98%, positive predictive value of 63%, and negative predictive value of 87% (Colton, Olmsted, & Rodin, 2007). Decreasing the cut-off score improved the sensitivity, which reached 76% with a cut-off of 5; however, the specificity then fell to 59%. Taken together these findings suggest that the usefulness of the ChEAT as a screening instrument among children may be limited. Translations of the ChEAT exist in Croatian, Portuguese, and Spanish.

Children's Binge Eating Disorder Scale (C-BEDS)

The C-BEDS (Shapiro et al., 2007) is a seven-item self-report questionnaire designed to assess the core behaviors of BED as defined by Marcus and Kalarchian (2003), including eating in the absence of hunger, eating in response to strong emotions or external cues, and eating in secret. Among a sample of 55 children aged 5–13 years, using the SCID as a gold standard, the C-BEDS demonstrated a sensitivity of 71% and specificity of 89%. The authors concluded that the C-BEDS may be more useful than the SCID for screening for BED in children but additional evaluations in larger and more diverse samples are warranted.

Athletic Milieu Direct Questionnaire (AMDQ)

Athletes have been reported to be two to three times more at risk for EDs compared to non-athletes (Bratland-Sanda & Sundgot-Borgen, 2013; see also Chapter 35) and may have specific risk factors (Torstveit, Rosenvinge, & Sundgot-Borgen, 2008). The AMDQ, a screening instrument for EDs specifically tailored to athletes, was developed and tested among 149 college athletes (Nagel, Black, Leverenz, & Coster, 2000). The initial pool of 119 items was pared down to three statistical subsets including 35, 19, and 9 items, respectively. These three subsets yielded sensitivity values ranging from 80% to 82%, specificity values ranging from 75.27% to 79.57%, positive predictive values ranging from 63.49% to 68.33%, and negative predictive values ranging from 87.50% to 89.16%, indicating good psychometric properties. Thus, the AMDQ may prove a highly useful tool among athletes.

Eating Disorder Assessment for Men (EDAM)

EDs are disproportionately diagnosed among women, and the large majority of the assessment instruments were developed and normed among female populations (see Chapter 37). The EDAM (Stanford & Lemberg, 2012b), a new instrument for assessing EDs in men, includes 50 self-report items. The initial factor analysis revealed four underlying factors: BED, Muscle Dysmorphic Disorder, Body Dissatisfaction, and Disordered Eating. In a subsequent study, a

sample of 108 individuals (78% male) completed the EDAM and the Eating Disorder Inventory-3 (EDI-3; Stanford & Lemberg, 2012a). Criterion validity was not formally assessed; however, using logistic regression the authors concluded that the EDAM successfully predicted ED status above and beyond the EDI-3. Further research on the usefulness of the EDAM as a screening tool among men is warranted.

The Importance of Setting

Primary Care

General practitioners are in an ideal position to identify EDs that might otherwise go undetected (Mond et al., 2008). However, EDs often are overlooked and underdiagnosed in primary care practice, in part due to lack of time and the impact of nonclinical demographic variables such as ethnicity and gender on the likelihood of being diagnosed (Currin, Schmidt, & Waller, 2007; see also Chapter 25). One study found that screening was relatively acceptable, with approximately one half of the female primary-care patients aged 16–35 years agreeing to be screened (Johnston, Fornai, Cabrini, & Kendrick, 2007). However, general practitioners were found to be uncertain of the procedure to follow in the case of a positive screen, and referrals were rarely made. These data suggest that, while primary care settings provide a highly useful context for screening for EDs, there is a need for education surrounding EDs among general practitioners.

In a study exploring the comparative usefulness of the SCOFF versus the EDE-Q in general practice settings, Mond et al. (2008) concluded that the two instruments had relative merits according to the situation. In the context of routine screening, the brevity and ease of the SCOFF might make it more suitable; however, when seeking to confirm suspicion of an ED diagnosis, the EDE-Q could be preferable due to its higher specificity and positive predictive value, as well as the more detailed information provided, which may guide referral. Cotton, Ball, and Robinson (2003) compared the usefulness, in general practice, of the four-question Eating Disorder Screen for Primary Care (ESP) and the SCOFF. They found that, using a cut-off of two positive responses, the sensitivity and specificity of the SCOFF were 78% and 88% respectively, while those of the ESP were 100% and 71%. The authors concluded that, while both instruments were useful in detecting cases, the ESP performed somewhat better at ruling out potential cases.

Schools

Schools and college campuses provide an ideal setting for screening for EDs, due to the practical advantages of having youths concentrated in one place and the on-site presence of support staff (Austin et al., 2008). The first National Eating Disorders Screening Program (NEDSP) was launched on U.S. college campuses in 1996 as a confidential volunteer two-stage screening program for students (Becker, Franko, Speck, & Herzog, 2003). The first stage included a written self-report questionnaire, which was followed by a face-to-face interview with a counselor to review responses. In the 1996 wave of NEDSP, 9,069 students from 409 colleges returned questionnaires, and 5,787 of these (64%) met with a counselor to discuss their responses. Counselors were provided with a manual for identifying participants reporting potentially clinically significant symptoms (purging or binge eating at least once a month or significant distress), and were instructed to then make an appropriate referral for further evaluation and treatment, with three possible outcomes: no referral at this time, professional evaluation, and professional evaluation-urgent. Of the 5,787 program participants who met with a counselor,

4,398 (76%) met the criteria for clinically significant symptoms and were therefore referred for professional evaluation, including 71 (1.2%) who were referred for urgent evaluation.

Subsequently NEDSP was successfully extended to high schools, and in 2000 over 35,000 high-school students from 152 schools completed a NEDSP screening measure, including attitudinal and behavioral questions and the EAT-26 (Austin et al., 2008). The NEDSP screening measure was compared against the EAT within each gender separately (Haines et al., 2011). Among girls, the combination of the attitudinal and behavioral items performed well, with sensitivity ranging from 88% to 91%, specificity ranging from 73% to 82%, positive predictive values ranging from 36% to 45%, and negative predictive value = 98%. Among boys, the combination of attitudinal and behavioral items also performed well, although the positive predictive values were somewhat low: sensitivity ranging from 88% to 91%, specificity ranging from 87% to 94%, positive predictive values ranging from 22% to 36%, and negative predictive value = 99%. The authors reiterated the recommendations of Jacobi et al. (2004) that screening take place in two phases, with an initial screening (e.g., using the NEDSP screen) followed by a more specific instrument.

Cultural and Ethnic Considerations

Rates of EDs have been found to vary according to ethnicity and cultural context (Franko, Becker, Thomas, & Herzog, 2007; see also Chapters 23 & 25); however, to date, there has been little focus on the impact of culture or race and ethnicity in screening for EDs. As previously noted, the overwhelming majority of existing assessment instruments for EDs were developed among White English-speaking young women. While many of these scales have been translated and validated in different languages and different cultural contexts, it should not be assumed that they perform identically across cultural contexts (see Chapter 16). For example, Mumford, Whitehouse, and Choudry (1992) validated the EAT among a sample of 369 schoolgirls in Pakistan and reported that, despite a factor structure almost identical to that of the original instrument, the optimal cut-off score might be lower among this population. Similarly, a validation of the EAT among a sample of 67 patients suffering from BN and 109 patients suffering from AN in Hong Kong revealed that up to 41.5% of patients with typical AN scored below the traditional cut-off score on the EAT, suggesting that a much lower cut-off score would be appropriate among this population (Lee, Kwok, Liau, & Leung, 2002). Furthermore, the presentation of AN in this sample was somewhat different to that usually seen in Western countries, with a high proportion of patients described as non-fat phobic, who attributed their restriction to reasons other than fear of fatness. These considerations are not well assessed by the items of the EAT, which focus strongly on fear of fatness as a core dimension of AN. Taken together, these studies suggest that existing screening measures for EDs may indeed be ethnocentric, and should only be transposed to other cultural contexts with caution.

Regarding race and ethnicity, findings from the high-school NEDSP in 2000 documented few ethnic differences in the prevalence of EDs (Austin et al., 2008). The authors concluded that screening for EDs was necessary in schools with and without ethnic diversity and argued that targeting specific ethnic groups for screening would be inappropriate. The necessity of screening among all ethnic groups is particularly important in view of findings that Native Americans and Hispanic students were less likely to be referred than White students with comparable scores in the national college screening study (Becker et al., 2003).

Conclusions and Future Directions

Screening for EDs has a number of benefits for the mental health of individuals suffering from or at risk for EDs. While a large number of screening instruments have been developed to date, many of them still lack evidence of validity, both criterion and external. In addition, screening instruments tailored to particular groups such as athletes or males are lacking. Overall, the last decade has seen little effort to improve, refine, or seek to better establish the validity of existing screening instruments. In view of these shortcomings, the field would benefit from increased consensus regarding the use of screening instruments so as to facilitate the examination of psychometric properties as well as to conduct comparisons in terms of prevalence.

The success of programs such as NEDSP also highlights the usefulness of routine screening in school and college settings. However, widespread screening would give rise to a number of challenges, including the processing of the screening forms, and the allocation of necessary resources for the follow-up of potential cases for referral and treatment where necessary. The ethical implications of widespread screening in the absence of sufficient resources downstream are numerous.

In conclusion, screening offers a valuable opportunity for the early identification of EDs; however, efforts to further explore the psychometric properties of existing instruments and develop appropriate measures for specific vulnerable populations are warranted. In addition, greater efforts should be made to screen for EDs among youth in school and college settings.

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39

Clinical Interviews and Eating Disorders

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The clinical interview is an essential part of the evaluation and management of patients with eating disorders (EDs). This chapter provides an overview of what to cover when conducting clinical interviews for EDs, and discusses strategies for elucidating information on symptoms and the patient's general functioning. The information is primarily written to apply to adults with EDs. Many points will also apply to children and adolescents, but additional details on the management of these younger patients can be found in Chapter 13. The chapter does not review diagnostic criteria for EDs, as these are covered in detail in Chapters 8–11. All patients evaluated for an ED should have a thorough medical evaluation as part of the assessment process (see Chapter 14).

In most cases, a clinical interview will seek to achieve four key goals. First, the interview should establish the nature, duration, and severity of the patient's presenting problems. In the case of EDs, there will be a focus on problems relating to eating and weight, and on any comorbid psychiatric difficulties. Second, the interview needs to elicit general information about the patient's background, current circumstances, and general functioning. Third, a good clinical interview will help to engage the patient in the thought of treatment, including the idea of getting treatment at all, and then the prospect of changing behavior with treatment (see Chapter 63). It may provide the basis for an ongoing therapeutic relationship, if the assessing and treating clinician are the same. Fourth, the interview needs to inform decisions about treatment and management. To address all of these goals, the interview must be broad as well as detailed. This chapter addresses each goal in turn, but in almost all cases the interview will address different areas interchangeably, rather than moving through them in sequence.

Beginning the Clinical Interview

Some patients will be familiar with the structure and focus of clinical interviews, but others will be attending an evaluation for the first time. Similarly, some patients will have initiated the appointment themselves, but many will be attending at the insistence or encouragement of a partner, family member, friend, colleague, or other medical professional (Fairburn, Cooper, & Waller, 2008).

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Even if the patient did initiate the interview, they may be regretting their decision to do so now that the appointment has arrived. For these reasons, it can be helpful to spend a few minutes orienting the patient to the session and its focus. Explaining to the patient that you are interested in hearing about what is currently affecting them, including their perspective on any difficulties they are experiencing, can be a useful starting point. It is also worthwhile reminding the patient that the session is an opportunity for them to ask questions of you. From a practical perspective, letting them know how long you will be meeting and asking them how they would like to be addressed (*Do you prefer Amanda, or Ms Smith, or something else?*) can help to settle the patient into the session. With adults, it is common to meet with the patient individually first, before including any accompanying partners or family members, if indicated.

After establishing the focus of the interview, useful opening questions include asking the patient: *How did you come to be referred to the appointment? How do you feel about coming along to the appointment? What are you concerned about or what would you like help with?* and *What would you like to be different?*

Establishing the Nature, Duration, and Severity of the Patient's Presenting Problems

Some patients will volunteer considerable information if asked to describe their current difficulties. For others, specific questioning will be needed to ascertain the presence or absence of symptoms. When assessing for an ED, it is essential that current eating patterns and methods of weight control (or attempted weight control) are established, as well as the patient's beliefs and concerns about food, eating, weight, and shape. It is also important to establish a general timeline of when problems or concerns relating to eating, weight, and shape began. Achieving this requires systematic inquiry into the areas outlined in Table 39.1.

Formal assessment measures, covered in Chapter 40, may assist with collecting information on the nature and frequency of specific symptoms. They are also beneficial in providing a baseline against which changes in symptoms can be measured. Diagnostic interviews such as the Eating Disorder Examination (EDE; Cooper & Fairburn, 1987) and MINI International Neuropsychiatric Interview schedule (Sheehan et al., 1998) may be useful for diagnostic decision-making.

Current Eating Habits

Many patients will be self-conscious, defensive and/or anxious when asked about their eating habits. It can be helpful to set the patient at ease by acknowledging this, such as by saying:

> *I know many individuals referred for eating disorder assessment find it difficult to talk about their eating preferences and routines with a stranger. However, it is important for me to understand your eating patterns if I am to help you. Would you be able to summarize a typical eating day for me, in terms of how you plan to eat and what you actually eat? If you don't have a typical eating routine, perhaps you could provide me with some examples of how your eating has been over the past week?*

For patients who under-eat and are of low weight, it may be difficult to obtain accurate information about the amount of food eaten. This can stem from defensiveness, or a genuine misperception on the patient's part about what constitutes "normal" eating (Anderson,

Table 39.1 Areas to assess when establishing the nature, duration, and severity of eating disorder psychopathology.

Current eating habits:
- Structure/timing of meals and snacks
- Approximate amount eaten—desired amount and actual amount
- Range of foods eaten and avoidance of foods or food groups
- Specific dietary rules—rule content and how much of the time rules are followed
- Rituals or routines around food and eating
- Any binge eating—frequency, size of binges

Purging and other extreme efforts at weight control:
- Self-induced vomiting—frequency
- Laxative misuse—frequency, number of pills taken, type of laxative/s used
- Diuretic misuse—frequency, number of pills taken, type of diuretic/s used
- Diet pill misuse—frequency, number of pills taken, type of pill/s used
- Exercise—frequency, intensity, type of exercise
- Any other behaviors designed to control weight
- Whether purging and weight control behaviors are compensatory responses to binge eating or occur independently

Concerns and beliefs about eating, weight, and shape:
- Views on current eating habits and weight control behaviors
- Level of preoccupation with eating, weight, and shape and their control
- Avoidance of situations involving eating
- Avoidance of situations where weight or shape may be scrutinized

Trajectory of symptoms:
- Age and order of symptom onset
- Changes in severity over time
- Weight history relative to eating patterns

Past treatment:
- Type, acceptability, and success

Comorbid difficulties:
- Depressive symptoms
- Anxiety symptoms—generalized anxiety/worry, panic symptoms, social anxiety, obsessive-compulsive features
- Difficulties sleeping
- Self-harm
- Suicidal ideation, planning, and/or intent
- Personality features (impulsivity, fastidiousness, social avoidance)

Lundgren, Shapiro, & Paulosky, 2004). Asking the patient how their eating compares to that of their friends or family members may be of use, but if the patient insists that they eat three regular-sized meals and several snacks per day, despite being underweight, then this should simply be noted and the interview allowed to proceed.

Dietary rules and strict routines around eating are characteristic of anorexia nervosa (AN), bulimia nervosa (BN), and many unclassified EDs. These are often highly valued by patients, and patients may not be used to being asked about them. Inquiring about these areas can help to establish rapport and to reassure the patient that the interviewer is familiar with EDs and the nature of ED symptoms:

Most of us like to eat certain foods and dislike others. Sometimes people will also have certain routines around eating that they like to follow, such as eating at particular times, in particular ways, or in particular places. There are lots of different reasons that people develop such routines. I was wondering if you had any rules or routines around what you prefer to eat, or about when and how you eat?

When assessing binge eating, the use of exaggerated examples (Carlat, 2005) can also help reassure patients that they are not "abnormal" or "disgusting" in their binge eating behavior. For example:

Some people describe binge eating on, say, a few blocks of chocolate, while others will describe loading up a full shopping cart and then binge eating on the contents. What is it like for you?

The same technique can be used when inquiring about the frequency of binges, as well as other ED behaviors, such as dietary rules or purging behaviors.

When assessing binge eating, it is always worth taking the time to clarify what you mean by a binge, or if the patient uses the phrase, to check what they mean by it. "Objective binge eating" is the form of eating behavior referred to in diagnostic criteria for BN and BED, and this includes (a) a sense of loss of control over one's eating (being unable to stop eating or unable to control what or how much is eaten) and (b) the consumption of an "objectively large" amount of food (which may be defined as an amount more than three times greater than what others may be expected to eat in the same circumstances) (American Psychiatric Association, 2000; Fairburn & Cooper, 1993; see also Chapter 9). Experiencing a sense of loss of control over eating *without* consuming an objectively large amount of food is referred to as "subjective" binge eating, and is not uncommon in AN and eating disorders not otherwise specified (ED-NOS; Mond, Hay, Rodgers, Owen, et al., 2006). Both forms of binge eating tend to be accompanied by psychological distress and may trigger purging behavior, but they have different diagnostic implications and different implications for the patient's weight and overall caloric intake.

Purging and Extreme Efforts at Weight Control

It is advisable to ask directly about purging behavior, including self-induced vomiting, laxative misuse, and diuretic misuse, as these behaviors carry significant medical risks and if present need to be known about (see Chapters 9, 14, & 52). Moreover, shame or embarrassment may prevent patients reporting the behaviors independently. Normalizing questions can help to address these concerns and convey to the patient that you are familiar with EDs and their associated symptoms:

Many people who are highly concerned about food and weight will go to great lengths to try and manage their eating, weight, and shape. For some people, this includes making themselves throw up, or taking laxatives or other pills. Is this something you have experienced?

If someone has described binge eating, this can also be used as a lead-in to discussions about purging:

You have described feeling disgusted with yourself when you eat more than you had planned or go on a "binge". I wonder if that has ever prompted you to throw up after eating, or take laxatives, or do other things to try and manage your worries about your eating?

Establishing the frequency of purging is essential. Patients who purge frequently, and/or who have been purging regularly for extended periods of time, may require particularly careful medical management (Birmingham & Treasure, 2010; Fairburn & Harrison, 2003; see also Chapter 52). However, it is also important to assess the context of the behavior. Some patients will induce vomiting only after binge eating episodes, whereas others may induce vomiting after all episodes of eating, or after eating any quantity of foods perceived to be "bad" or "fattening." Similarly, some patients will take laxatives or other pills as a compensatory response to binge eating, whilst others will take them daily as a matter of routine. The context and nature of the purging provides insight into possible maintaining factors for the behavior, and may be important when planning the focus and form of treatment (Dalle Grave, Calugi, & Marchesini, 2009).

Exercise patterns should also be asked about directly. The interviewer should determine the frequency, duration, and intensity of exercise, and establish whether the activity is "driven" or "compulsive" in nature (see Chapter 35). It can also be useful to distinguish between formal exercise and "restless" behaviors. Formal exercise would include periods of walking, running, cycling, gym work, or weights work. Restless behavior includes standing instead of sitting when possible, walking up and down stairs repeatedly, pacing, cleaning, and consciously "jittering" or moving around when seated. This sort of activity can be particularly difficult to disrupt or change, and may be a marker of particularly intense preoccupation with eating, weight, and shape (Mond, Hay, Rodgers, & Owen, 2006). Few patients will volunteer information about restless behavior without specific questioning. If restless behavior is suspected, the interviewer may want to inquire about this in general terms, such as by asking:

> *Some people will say that they don't play sports or go running or go to the gym, but they will report doing other sorts of activity to try and use up calories or lose weight. For example, they may walk around a lot, or jump up and down in place, or keep very busy. Is that something you have experienced?*

Alternatively:

> *You have described exercising daily for at least one hour, by riding your bike or going to a gym class. Many people who exercise a lot will relax between their exercise sessions, but some people report wanting to be active for as much of the time as possible and finding it hard to sit still or "do nothing" between exercise sessions. What is it like for you?*

Driven or compulsive exercise can also be a marker of degree of psychological distress and preoccupation with eating, weight, and shape (Shroff et al., 2006). Driven exercise typically persists despite injury or inconvenience, is highly valued, is done largely or exclusively to control weight, shape, or negative emotions, and is accompanied by significant anxiety and guilt if the activity is blocked or otherwise cannot occur (Shroff et al., 2006; see also Chapter 35). Asking patients what it is like for them if they can't exercise will usually provide insight into whether exercise is driven or not. Patients who respond by saying they would always exercise, no matter what, could be asked what the *thought* of not exercising is like.

Other efforts at weight control may include diet pill use, chewing and spitting out food (without swallowing), excessive water consumption (with the goal of "flushing out" food or fat), and excessive consumption of foods designed to induce fullness or produce a laxative effect.

Concerns and Beliefs About Eating, Weight, and Shape

Many patients will find it distressing to verbalize their concerns and perceptions about their eating, weight, and shape. It is important to be sensitive to this. Inquiry in this area can focus on patients' perceptions of their eating patterns, weight, and shape (*Are you happy with your current pattern of eating? How do you feel about your weight and shape at present?*), as well as their level of preoccupation with these areas (*Would you say you spend much time thinking about eating, weight, or shape? Does thinking about eating or weight ever make it difficult to concentrate on other things?*).

Weight- and shape-related goals, such as fitting into a particular size or weighing a particular amount, can also be elucidated here. Frequency of weighing and other body-checking behaviors should be asked about, as well as any avoidance of seeing one's body or knowing one's weight.

The importance placed on eating, weight, and shape and their control should be evaluated. This can be achieved through questions like: *How much would it distress you if your weight changed by 5 pounds? Compared to other things in your life, like relationships or study, or work, how much do your eating, weight, and shape influence how you feel about yourself?* and *How important are your eating, weight, and shape to how you judge yourself as a person—to how you judge your self-worth?* The latter two questions address the over-evaluation of eating, weight, and shape, that is, the tendency to judge one's self-worth primarily or entirely in terms of eating, weight, and shape and their control (Fairburn, Cooper, & Shafran, 2003; see also Chapter 22). This is one of the diagnostic criteria for BN in the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*; American Psychiatric Association, 2000, 2013; see also Chapters 3 & 9) and should be addressed in all clinical interviews for eating disorders. Some patients may require more explanation of this construct, and creating a list of areas that have an impact on self-evaluation can be a useful lead-in if the patient is initially unclear on what is being asked.

CLINICIAN: *How important are your eating, weight, and shape to how you judge yourself as a person—to how you judge your self-worth?*

PATIENT: *I'm sorry?*

CLINICIAN: *Let me backtrack a little. I'd like you to think about all the things in your life that influence how you feel about yourself and judge yourself as a person—the things that impact on your sense of self-worth. Most of us have a number of different things that are important in this way. What would be important for you?*

PATIENT: *How I do at university. Getting high marks in exams and things. Also whether I have friends.*

CLINICIAN: *OK, great. So we have doing well at university, in exams, and other areas, and having friends—they would impact on how you judge yourself as a person. Compared to those things, how much would your eating, weight, or shape impact how you judge or evaluate yourself as a person?*

PATIENT: *Oh, I see. Well, quite a lot. My weight and shape would be as important as doing well at university. My eating would come after university, but above friends.*

Fear of gaining weight should also be assessed in all patients, and particularly thoroughly in low-weight patients, given that this feature is a diagnostic criterion for AN (*DSM-5*, 2013). Body image disturbance can be screened for by asking patients to indicate, as objectively as they can, whether they think they are underweight, at a healthy weight, or overweight according to medical cut-points.

Trajectory of Symptoms

Patients will differ in their ability to describe symptom onset and changes over time. However, most will be able to place symptoms in sequence, such as by recalling that they started dieting before they started binge eating, or that binge eating came before vomiting. They may also be able to describe changes in the intensity or severity of problems over time. This information is helpful in developing a sense of the patient and their presenting problems, and it can also give insight into factors that have triggered or maintained their eating disorder symptoms.

If patients struggle to describe their symptom history, useful discussion can be prompted by questions such as: *When was the last time you weren't binge eating or purging regularly? Are there things that tend to make your difficulties worse? Are there things that you have found helpful in managing your weight and shape concerns in the past?* and *How have your eating patterns changed over the last year?*

Past Treatment

It is important to ask about past treatment *referrals* as well as actual treatment attended. If a patient reports receiving a particular type of treatment, such as cognitive-behavioral therapy, it is also important to check whether your understanding of that treatment modality matches the patient's experience. "Cognitive-behavior therapy" (CBT) may refer to evidence-based CBT for EDs, to CBT for depression or another psychiatric disorder, or to a form of treatment that was labeled CBT but was in fact something quite different (Fairburn & Cooper, 2011; see also Chapter 56). Asking the patient how they found past treatment experiences validates the patient's perspective and may also provide clues as to what sorts of treatment will be acceptable to the patient now.

Comorbid Difficulties

In addition to ED symptoms, most patients will present with some degree of comorbid psychopathology (see Chapters 15 & 54). In some cases, these difficulties will have developed after the ED and may be seen as secondary to the ED (e.g., low mood secondary to starvation, social anxiety secondary to weight concern, obsessions or compulsions that relate exclusively to food). In other cases, comorbid difficulties will have developed before or in tandem with the ED. Inquiring about mood, anxiety, and self-harm should be standard in clinical interviews for adult eating disorders, and other difficulties should be assessed for if indicated. Sleep impairment is common in EDs as well as associated psychiatric disorders.

Suicidality should be screened for as per any clinical interview, and a decision made about the patient's level of suicide risk.

Personality features can be difficult to assess when someone is unwell with an ED, as starvation, binge eating, and purging all influence personality states. Obsessiveness, social withdrawal, impulsivity, irritability, and emotional lability are common consequences of EDs, and should not be viewed as characteristic of the patient unless the traits clearly preceded ED onset. For this reason, others have emphasized that personality disorder diagnoses should be made only with great caution when an ED is present (Fairburn et al., 2008). Despite these difficulties, an awareness of personality characteristics at the time of assessment can inform management in the short to medium term, and may identify areas (e.g., impulsivity) that will require attention if ED symptoms are to reduce.

As noted, all patients attending a clinical interview for an ED should have a comprehensive medical evaluation as part of their assessment. A general review of physical health can still occur within the clinical interview, particularly symptoms that may bother the patient and thus provide some examples of negatives associated with their situation. These include dizziness, weakness, sensitivity to cold, lanugo (hair growth), edema (fluid retention), and amenorrhea in females (see Chapters 14 & 52).

Assessing the Patient's Background, Current Circumstances, and General Functioning

This part of the interview shifts the focus away from the ED and to the person more generally. It is comparable to the general background component of any clinical interview (Carlat, 2005). The aim is to establish a sense of the person's history, and who they are outside of their presenting difficulties. This part of the interview can be quite brief, but should cover family background and early family life; school experiences and educational attainment; social and relationship history (including friendships, intimate relationships, and sexual history); work experiences; current social support; and interests, goals, and aspirations. The discussion should also establish how the patient is functioning now relative to in the past (in terms of work, study, and relationships) and how they are functioning now relative to their goals for the future.

Engaging the Patient

The entire clinical interview should focus on engaging the patient and developing therapeutic rapport (Fairburn et al., 2008). This can be difficult if the patient is reluctant to attend, particularly when coupled with the requirement to collect a large amount of information in a relatively short period of time (possibly from a patient who does not wish to provide it). However, if skillfully managed, the clinical interview can be highly effective at engaging even reluctant patients (see Chapter 63).

Starting the interview off well, by outlining the focus of the session and working to put the patient at ease, sets the scene for ongoing efforts at engagement. Being warm and courteous to the patient, genuinely interested in their functioning, curious about their perspective, and emotionally sensitive to their difficulties will go a long way in defusing resistance to the interview and anxiety over its possible outcomes. Conveying experience and competence in EDs is also essential, and this is most effectively achieved through questioning that normalizes the patient's experience and demonstrates familiarity with EDs (Fairburn et al., 2008; Peterson, 2005). The examples provided throughout this chapter are intended to model this manner of questioning.

Ideally, the interviewer will not just engage the patient in the interview, but also engage them in thoughts about treatment and change in the longer term. Achieving this requires careful attention to the patient's comments, particularly acknowledgements of frustration or distress over symptoms, ambivalence about ED treatment, or uncertainty about the future with an ED. Conversely, it is also important to attend to remarks that present the ED in a favorable light. Reflecting these to the patient can help to engage them in discussion of the advantages and disadvantages of their current difficulties, and move them toward acknowledgement of the need for change.

The following two sets of dialogue highlight ways that an interview can be shaped to elicit information whilst also striving to engage the patient in thoughts of change.

Case One

Ginny is a 20-year-old female referred for assessment by her family doctor. This occurred after Ginny acknowledged binge eating difficulties when seeking help to lose weight. She attended the appointment reluctantly, and only because her doctor would not prescribe weight loss medication. Ginny is in the healthy weight range and has tended to minimize her symptoms and downplay the severity of her difficulties in the interview thus far.

CLINICIAN: *You have described feeling disgusted with yourself when you eat more than you had planned or go on a "binge." I wonder if that has that ever prompted you to throw up after eating, or take laxatives, or do other things to try and manage your worries about your eating?* [Normalizing question]

PATIENT: *I guess I do sometimes throw up after bingeing. In fact, I would throw up every time I binge. I know, I know, I shouldn't do it. But I feel so much better after getting rid of the food. It is such a relief.* [Defensiveness; benefits of the eating disorder]

CLINICIAN: *It sounds like throwing up really helps you manage the guilt you feel after binge eating.* [Reflection and validation of eating disorder benefits]

PATIENT: *Absolutely! I can't imagine how awful I'd feel if I didn't throw up.*

CLINICIAN: *Do you ever vomit at other times, outside of binges?*

PATIENT: *No, never.*

CLINICIAN: *What about taking laxatives or doing anything else to try and counter a binge?*

CATIENT: *No, it's just the vomiting.*

CLINICIAN: *OK. So how long would you say the binge eating and vomiting have been happening for?*

PATIENT: *Oh, I don't know…not that long. It's really not that bad.* [Minimizing]

CLINICIAN: *Would you say a couple of years? Five? Ten?* [Exaggerated example]

PATIENT: *Oh, less than that. Probably about 2 years, but on and off. I don't do it all the time.*

CLINICIAN: *A lot of people tell me that their eating is influenced by other things happening in their life, or will go through stages over time. Would you say that is true for you?* [Normalizing question]

PATIENT: *Yes, absolutely. There are times when I don't binge or vomit for weeks, but then it might happen every day.*

CLINICIAN: *What do you think is happening at those times you binge and vomit every day?*

PATIENT: *I guess I'm usually a bit overwhelmed with things. Also, I seem to be able to control my eating for a while, but then the urge to eat just builds up and up and eventually I give in. I feel really weak, and I'll binge and purge for a while, and then I'll pull myself together again and eat healthy for a while. And then I'll binge again.*

CLINICIAN: *You sound exhausted when you describe that pattern. Does it feel exhausting?* [Reflection of eating disorder negatives]

PATIENT: *Yes. I hate it. I would like it to stop.*

CLINICIAN: *I'm really glad you were able to come along today, because part of this meeting is the two of us figuring out how to help you do that. Are there any other aspects of your eating that are exhausting sometimes?*

In the above example the dialogue is a fairly straightforward discussion regarding the presence, frequency, and onset of binge eating and vomiting behavior. However, the questions are designed to set the patient at ease, and to convey that the interviewer has seen these sorts of difficulties before, and can understand why the patient might engage in binge eating/vomiting even as they want to help the patient stop those behaviors.

Case Two

Ben is an 18-year-old male who has been brought to the assessment by his parents. He was a long-distance runner across high school and has always been slim. However, in the last year he has become concerned about his eating, reduced the range of foods he will eat, and lost about 15 lb (6.8 kg). He is now significantly underweight for his height and age, but perceives himself to be healthy.

CLINICIAN: *Ben, I know you have been brought in today by your parents. We might ask them to join us later, but I am most interested in your perspective on what is going on and why you were referred to see me. How do you feel about coming along today?* [Opening question]

PATIENT: *Pretty annoyed actually. It's a complete waste of time.*

CLINICIAN: *I'd be annoyed too if I was taken somewhere I didn't want to be. I am hoping I can make this session at least a little bit worth your time—if only to help you help your parents to worry less. Why do you think they insisted you come along?* [Validation, attempted engagement]

PATIENT: *They don't seem to want me to be healthy. They have this thing about me not eating enough, and losing weight and stuff. It's stupid.*

CLINICIAN: *How long have they been bothered about your eating and weight?*

PATIENT: *Maybe 4 months, a bit longer.*

CLINICIAN: *What has changed for you over the past 4 to 6 months?*

PATIENT: *I'm a runner, long-distance. I've just finished my last year in high school and I wanted to get really fit before college. I started eating healthier, getting fitter. My parents don't seem to understand that.*

CLINICIAN: *What sorts of distances do you like to run?* [Interest in the patient]

PATIENT: *The longer the better. I hold a couple of records at the 10 miles. I'd like to do a marathon one day.*

CLINICIAN: *Those are long distances! So what sorts of things have you been doing with trying to get fitter?*

PATIENT: *Just eating healthily.*

CLINICIAN: *Can you tell me more about that?*

PATIENT: *Well, you know, healthy eating. More fruit and vegetables. No junk food. My Dad seems to think you have to eat junk food or something is wrong with you.*

CLINICIAN: *That must have made things hard for you at home. Have you changed anything else over the last few months, like how often you run, or the size of your meals and snacks?*

PATIENT: *I run pretty much daily now. I'm really trying to get as good as I can. And I don't eat snacks; you don't need them.*

CLINICIAN: *It sounds like this is really important to you. Does training and healthy eating take up much of your time?*

PATIENT: *I guess, but not that much. I mean, it's probably the same as for other athletes.* [Defensiveness]

CLINICIAN: *OK. I wonder if you could talk me through a typical day for you at the moment? I know you've recently finished school—what would your training and eating look like with your focus on fitness?*

PATIENT: *Well, I run first thing. I don't eat before I run because then I feel sick. So I go about, maybe 9 a.m., run for an hour or so. I have a shower and stuff and then make breakfast. Do you want me to tell you what I eat?*

CLINICIAN: *If that's alright with you. It would help me get a sense of what things are like for you at the moment.*

PATIENT: *Right. Well, breakfast is usually some cereal with some fruit. Then I usually hang out at home for a while. We have a dog, I like spending time with him. Because I eat breakfast late, I'm not usually hungry again for a while. So I might have lunch about 3 p.m., but I usually try to keep it light because then I do weights work in the early evening. I might have a salad, with tuna for protein. Then I'll do some weights work—we have a home gym in the garage. Mom gets home from work about 4 p.m. so she usually arrives when I'm doing that. Dad gets home about 6 p.m. and they still make me eat dinner with them. I have told Mom I won't eat red meat or pasta or creamy dishes, so if she cooks something that doesn't have those things, I'll eat what she serves me. Otherwise I won't; it's not good for my training.*

CLINICIAN: *Thanks for taking me through that. How is your current routine different to, say, a year ago?*

PATIENT: *A year ago I just did running at school, a few afternoons a week and on Saturday mornings. I didn't care about my eating at all! I didn't know anything about nutrition. I ate out with my friends at least once a week and that was usually burgers and fries. I had cookies for snacks. I can't believe how I used to be.*

CLINICIAN: *What do you like about the changes that have happened over the year?* [Engagement; positive aspects of the eating disorder]

PATIENT: *I feel fitter, or at least kind of. I like having a focus and taking charge of my health. I think I'm a better person now, although I still have a way to go.*

CLINICIAN: *Sometimes people will tell me that when they make changes to their eating and exercise routines, they experience some benefits—like for you, you are feeling fitter and in charge of your health—but they also experience some things that are a bit hard. Is that true for you?* [Validation, normalizing question]

PATIENT: *I guess so. It's taken a lot of effort. I don't see my friends much these days, because they don't understand what I'm trying to do. My parents and I aren't getting along. Some days I do feel pretty tired.*

CLINICIAN: *Is there anything you'd like to be different?* [Engagement]

PATIENT: *I don't want to get lazy again, but it would be nice if I could think about food and exercise a bit less. It takes up a lot of my time. But everyone just seems to think I should stop caring about those things, and I don't want to do that!*

CLINICIAN: *That's OK. You mentioned that thinking about food and exercise takes up a lot of time. Do you set yourself particular goals, or rules, for food and exercise?*

PATIENT: *I suppose so, but I hadn't thought of it in those terms. I have to run at least 50 miles a week and do weights every day. I can't eat foods with more than 10 g sugar per 100 g, or more than 10 g fat per meal. I guess they're rules.*

CLINICIAN: *What is it like living to those rules?*

PATIENT: *I guess it's pretty hard.*

In this example the patient was attending the session reluctantly and initially denied any difficulties or problems. The discussion was designed to elicit information, but also to allow the patient to acknowledge difficulties associated with his situation, without requiring that he criticize the aspects of his eating and exercise behavior that he valued. Both of the above examples highlight the way that a clinical interview may move between different topics and symptom areas, combining specific questioning with more general questions and efforts at engagement.

Making Decisions About Treatment and Management

Outside of research settings, clinical interviews are conducted with the view to making decisions about treatment and management. This is an important end point for the interview, although the decisions may be made after the interview is over.

Covering the points outlined in this chapter, together with a medical evaluation, should allow informed decisions to be made about treatment and management options. Additional information that may inform this include the patient's willingness and capacity to attend outpatient appointments (in terms of other commitments, their geographic location, and their motivation for treatment), the intensity of their symptoms, their degree of comorbid psychopathology, their degree of functional impairment, their social support, and their degree of medical compromise. Asking the patient what they would like from treatment, in terms of frequency, style, and structure, as well as their goals for treatment, is a useful way of focusing the end of the interview and reaching collaborative decisions with the patient about their management.

Conclusions and Research Questions

The clinical interview is the first part of a patient's engagement with a service. Attention to this initial contact is imperative. A well-conducted interview collects information whilst engaging the patient in the interview process and with the idea of treatment. In addition to assessing for eating behavior and concerns about eating, weight, and shape, the interview should cover comorbid difficulties with mood or anxiety, general background information, and the patient's own goals for change.

Given the importance of the initial clinical interview, it has received surprisingly little attention in the research literature. Systematic evaluation of therapies and treatment approaches are accepted as necessary, indeed essential, for evidence-based treatment. Research on the aspects of the *clinical interview* that are effective in engaging patients in treatment would add to the literature (see Chapter 63). Research of this nature may also help to reduce the number of patients who attend an initial interview but do not return, a group that has tended to be neglected in treatment outcome research to date (Watson, Fursland, & Byrne, 2013).

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Research Tools for Assessing Eating Disorders

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Assessment in a research context can be challenging. Research studies, particularly treatment outcome studies, are costly and involve enormous amounts of time and effort, and decisions about how and when to conduct assessments can have a significant impact on the conclusions that can be drawn from a given research study. While there are numerous instruments available for assessing disordered eating, eating disorders (EDs), and associated psychopathology (Allison, 2009; Mitchell & Peterson, 2005), not all of these instruments are equally appropriate for assessment in a research context (Anderson & Murray, 2010). How then should researchers decide which measures to use in a research study? This chapter will provide some guidelines for those struggling with decisions about which measures to use in a research context. While we emphasize the use of assessment measures for treatment outcome research, the guidelines are applicable across research contexts.

Which Method of Assessment?

Researchers have many methods of assessment from which to choose, from interviews to self-report instruments to behavioral measures such as test meals. While it is beyond the reach of this chapter to discuss all aspects of choosing a method of assessment, we will review some issues particularly pertinent to the assessment of eating disorders.

Which Method of Assessment is Most Accurate?

It has been widely assumed that structured interviews represent the criterion method by which to assess ED symptomatology. In fact, one interview, the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993; Fairburn, Cooper, & O'Connor, 2008), has been frequently described as the gold standard measure of ED symptomatology (Wade, Tiggemann, Martin, & Heath, 1997; Wilson, 1993; see also Chapter 38). While a number of studies have found discrepancies between rates of endorsement of ED-related constructs on interview versus questionnaire methods of assessment, it has generally been assumed that an interview allows for more detailed

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questioning and thus more accurate rates of responding (Anderson & Murray, 2010). However, a growing number of studies suggest that some individuals are embarrassed by and ashamed of their eating-disordered behavior and thus minimize their symptoms to a greater degree when having to discuss them face to face as opposed to a questionnaire (Anderson & Murray, 2010). Thus, it is not clear which method of assessment is most accurate. Until this issue is resolved, we recommend the use of both interview and self-report instruments.

Behavioral Measures of Eating Behavior

Denial and minimization are common problems in the assessment of ED symptomatology (Anderson, Lavender, & De Young, 2010). Moreover, humans are notoriously inaccurate at reporting food intake (Forrestal, 2011; Rutishauser, 2005; see also Chapter 24). Thus, it can be extremely useful to get an accurate assessment of eating behavior by directly observing eating using test meals or other similar approaches. Test meals allow for the assessment of multiple aspects of eating behavior and can be an excellent way to verify the validity of self-report assessment tools.

Guidelines have been developed for the use of test meals as an assessment measure (Anderson & Paulosky, 2004; Williamson, 1990), although, as will be discussed later in this chapter, they do not appear to have been widely adopted. Nevertheless, we recommend the use of behavioral measures of eating behavior such as test meals whenever feasible.

How Often to Assess?

The simplest strategy for assessing change is to assess participants before and after an intervention. This strategy has the advantages of being time- and cost-effective. However, it does not allow for a more detailed examination of the treatment process. Treatment outcome research, both within the ED field and without, is increasingly moving away from investigating simpler questions such as "Does this therapy work?" to investigating more complex questions of mediation, essentially "*How* does this therapy work?" (Kazdin, 2009; Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001; Kraemer, Wilson, Fairburn, & Agras, 2002; Murphy, Cooper, Hollon, & Fairburn, 2009).

Answering such questions requires much more attention to the timing and frequency of assessments; in order to demonstrate mediation, change in the mediator needs to be shown to have occurred prior to change in the outcome of interest, and such changes often occur rapidly in the treatment process (Kazdin, 2009; Murphy et al., 2009). Thus, the best advice for assessment might be to assess early and often, perhaps even at every session.

How to Pick Specific Measures

Researchers can choose specific assessment measures for a multitude of reasons. In this section we will focus on two main justifications for choosing assessment measures and discuss the pros and cons of each.

Popularity

Sometimes researchers choose measures based on what others have used in previous studies or what measures are widely used in the literature. There are good reasons to do so; for example, using common measures allows for easy comparison across studies. This

practice can be problematic, however, when the commonly used measures do not accurately reflect the constructs of interest in a given research study. Also, the specific measures that are used commonly in the EDs literature have changed over time (aee Chapter 38).

Historical Patterns in the Use of Assessment Instruments for Treatment Outcome An early review of measures used in studies of bulimia nervosa (BN; Williamson, Anderson, & Gleaves, 1996) found considerable diversity in the type and number of measures used to evaluate treatment outcome. Most studies used multiple self-report questionnaires, and only one measure (the Eating Attitudes Test [EAT]; Garner & Garfinkel, 1979; Garner, Olmsted, Bohr, & Garfinkel, 1982) was used in 50% or more of the studies. Also, the EDE (Fairburn & Cooper, 1993; Fairburn et al., 2008) was used in fewer than 20% of studies.

A review in the mid-2000s (Anderson & Paulosky, 2004) found that patterns of assessment in treatment outcome research had changed somewhat. Although a large number of measures were still in use, three instruments had come to dominate the field. In studies of both anorexia nervosa (AN) and BN, two self-report measures were overwhelming favorites; the EAT and variations of the Eating Disorders Inventory (EDI; Garner, 1991; Garner, Olmstead, & Polivy, 1983). Use of the EDE remained steady. The majority (i.e., over 80%) of the reviewed studies of BN used self-report tools (often in the form of food records) to measure binging and purging.

These patterns have changed considerably in recent years. Tables 40.1–40.5 show the assessment measures used in recent treatment trials for EDs using cognitive-behavior therapy (CBT), family-based therapy (FBT), interpersonal therapy (IPT), dialectical behavior therapy (DBT), and mindfulness-based therapies (see Chapters 56, 57, 60, & 62). Table 40.6 provides more specific information about the various measures. As can be seen from these tables, while a wide variety of self-report measures are still being used, the questionnaire version of the EDE (EDE-Q; Fairburn & Beglin, 1994, 2008) has increased in popularity, particularly in trials of CBT. The EDE itself has also come to be used widely, again particularly among trials of CBT. This is in some ways not surprising, as the EDE and EDE-Q were designed by the primary developer of CBT-based treatments for EDs (Fairburn, 2008), and thus are a good theoretical match with CBT. As can be seen in the tables, however, it has also been used in studies of therapies other than CBT. Neither the EAT nor the EDI have been commonly used in more recent studies. Also in contrast to previous reviews, the use of self-monitoring as an assessment measure has decreased markedly. Conversely, the use of body mass index (BMI) as an outcome measure has increased markedly. This is to be expected; this review included trials of FBT for AN where previous reviews emphasized trials for BN, and more recent trials of CBT have included underweight individuals. In these cases change in BMI is a critical outcome measure. Finally, although they have been recommended (e.g., Anderson & Murray, 2010), behavioral measures of eating, such as test meals, remain rare.

As noted previously, the fact that a small number of assessment instruments have become popular in treatment outcome research (i.e., the EDE, EDE-Q, and BMI) is a positive development in that it allows for comparisons across studies. The EDE and EDE-Q also have very respectable psychometrics (Allison, 2009). Thus, although there have been some slight concerns about the EDE as an instrument (Anderson, De Young, & Walker, 2009), we encourage the use of these measures in research contexts whenever possible.

Table 40.1 Measures used in recent randomized controlled trials: cognitive-behavioral therapy for eating disorders.

	BASS[a]	*BIATQ*[b]	*BMI*[c]	*BSQ*[d]	*BAT*[e]	*BITE*[f]	*EAT-26*[g]	*EDE*[h]	*EDE-Q*[i]	*EDI*[j]	*Global AN*[k]	*Hunger Ratings (Visual Analogue Scale)*	*M-R*[l]	*SIAB-EX*[m]	*Self-monitoring*	*Test meal*	*SIBID*[n]	*TFEQ*[o]	*YBC-ED*[p]
Fairburn et al. (2009)			X					X	X										
Pike et al. (2003)			X										X						
McIntosh et al. (2005)			X					X		X	X								
Wilfley et al. (2002)			X					X											
Hilbert & Tuschen-Caffier (2004)				X				X								X			
Grilo et al. (2012)[1]			X						X						X			X	
Grilo et al. (2012)[2]			X					X											
Sanchez-Ortiz et al. (2011)								X											
Mitchell et al. (2011)			X					X											X
Byrne et al. (2011)			X					X	X	X									
Fairburn et al. (2013)			X					X	X										

Dalle Grave et al. (2013)				X							X											
Marco et al. (2013)	X	X				X	X	X												X		
Wagner et al. (2013)												X				X						
Munsch et al. (2012)				X								X										
Masheb et al. (2011)				X						X					X				X			
Grilo & Masheb (2005)				X						X	X								X			
Ruwaard et al. (2012)						X					X											
Wilson et al. (2010)										X												

Note. [a] Body Areas Satisfaction Scale, [b] Body Image Automatic Thoughts Questionnaire, [c] Body Mass Index, [d] Body Shapes Questionnaire, [e] Body Attitudes Test, [f] Bulimic Inventory Test, Edinburgh, [g] Eating Attitudes Test-26, [h] Eating Disorders Examination, [i] Eating Disorders Examination Questionnaire, [j] Eating Disorders Inventory-II, [k] Global Rating of Anorexia-Nervosa, [l] Morgan-Russell Outcome Criteria, [m] Structured Interview for BN/AN, [n] Situational Inventory of Body Image Dysmorphia, [o] Three Factor Eating Questionnaire, [p] Yale-Brown-Cornell Eating Disorders Scale.

[1] Grillo, White, Wilson, Gueorguieva, & Masheb (2012); [2] Grilo, Crosby, Wilson, & Masheb (2012).

Table 40.2 Measures used in recent randomized controlled trials: family-based therapy.

	BMI[a]	*ChEAT*[b]	*Course of ED*[c]	*DSM-IV*[d]	*EATATE*[e]	*EAT*[f]	*EDE-Q*[g]	*EDE*[h]	*EDI*[i]	*ICD-10*[j]	*M-R*[k]	*Relapse*[l]	*SEED*[m]	*YBC-ED*[n]
Dare et al. (2001)	X		X	X							X			
Eisler et al. (2000)	X		X			X			X	X	X	X		
Geist et al. (2000)	X								X					
LeGrange et al. (2007)	X						X	X						
Levine et al. (2001)	X	X												
Lock et al. (2005)	X		X	X				X						X
Lock et al. (2010)	X			X				X				X		X
Schmidt et al. (2007)	X				X								X	

Note. [a] Body Mass Index, [b] Children's Eating Attitudes Test, [c] Course of Disorder (i.e., duration previous treatment), [d] Criteria for ED—*Diagnostic and Statistical Manual for Mental Disorders* 4th ed., text revision, [e] EATATE semistructured interview (based on Longitudinal Interval Follow-Up Evaluation), [f] Eating Attitudes Test-40, [g] Eating Disorders Examination-Questionnaire, [h] Eating Disorders Examination, [i] Eating Disorders Inventory-II, [j] Criteria for ED—*International Classification of Diseases-10*, [k] Morgan-Russell Outcome Criteria, [l] Relapse/Remission Status, [m] Short Evaluation for Eating Disorders, [n] Yale-Brown-Cornell Eating Disorders Scale.

Table 40.3 Measures used in recent randomized controlled trials: interpersonal therapy.

	BMI[a]	*Course of ED*[b]	*EDE*[c]	*EDI-II*[d]	*Global Rating AN*[e]
Agras et al. (2000)			X		
McIntosh et al. (2005)	X	X	X	X	X
Wilfley et al. (2002)	X		X		
Wilson et al. (2010)			X		

Note. [a]Body Mass Index, [b]Course of Disorder (i.e. duration, length of treatment), [c]Eating Disorder Examination, [d]Eating Disorders Inventory-II, [e]Global Rating of Anorexia Nervosa.

Table 40.4 Measures used in recent randomized controlled trials: dialectical behavioral therapy.

	BES[a]	*BMI*[b]	*DEB-Q*[c]	*EDE*[d]	*EDE-Q*[e]	*EDI-II*[f]	*IA-E*[g]	*MAC-S*[h]	*PEWS*[i]	*QEWP*[j]	*Self-Monitoring*	*Test Meal*	*TFEQ*[k]
Chen et al. (2008)		X		X	X								
Hill et al. (2011)		X		X			X	X	X		X		
Kröger et al. (2010)		X				X					X	X	X
Robinson & Safer (2012)		X		X						X			
Roosen et al. (2012)		X	X			X							
Safer & Booil (2010)		X		X						X			
Safer & Joyce (2011)		X		X						X	X		X
Telch et al. (2001)	X	X		X									

Note. [a]Binge Eating Scale, [b]Body Mass Index, [c]Dutch Eating Behaviors Questionnaire, [d]Eating Disorders Examination, [e]Eating Disorders Examination-Questionnaire, [f]Eating Disorders Inventory-II, [g]Interoceptive Awareness Scale-Expanded, [h]Mizes Anorectic Cognitions Scale, [i]Preoccupation with Eating, Weight, and Shape Scale, [j]Questionnaire on Weight and Eating Patterns, [k]Three Factor Eating Questionnaire.

Table 40.5 Measures used in recent randomized control trials: mindfulness-based therapies.

	BES[a]	*Bio markers*[b]	*BMI*[c]	*BSQ*[d]	*DEB-Q*[e]	*Diet adhere*[f]	*EAT-26*[g]	*EDE-Q*[h]	*EDI-II*[i]	*FAAQ*[j]	*G-FCQ-T*[k]	*MAC-S*[l]	*PASTAS*[m]	*PEWS*[n]	*Self-monitoring*	*TFEQ*[o]
Alberts et al. (2012)			X	X	X						X					
Daubenmier et al. (2011)		X	X		X											
de Zwaan et al. (2009)			X		X			X							X	
Forman et al. (2009)			X		X					X					X	X
Pearson et al. (2012)			X				X		X			X	X	X	X	
Rain Carei et al. (2010)			X													
Tapper et al. (2009)	X		X		X	X										

Note. [a] Binge Eating Scale, [b] biomarkers (described in Methods), [c] Body Mass Index, [d] Body Shapes Questionnaire, [e] Dutch Eating Behaviors Questionnaire, [f] adherence to prescribed diet, [g] Eating Attitudes Questionnaire-26, [h] Eating Disorders Examination-Questionnaire, [i] Eating Disorders Inventory-II, [j] Food Related Acceptance and Action Questionnaire, [k] General Food Craving Questionnaire, [l] Mizes Anorectic Cognitions Scale, [m] Physical Appearance State and Trait Anxiety Inventory-Stats Version, [n] Preoccupation with Eating Weight and Shape Scale, [o] Three Factor Eating Questionnaire.

Table 40.6 Summary of recommended measures.

Measure	*Constructs assessed*	*Notes*
1. Body Areas Satisfaction Scale (BASS)	Body satisfaction/ dissatisfaction	Developed by Cash (2002); subscale of the Multidimensional Body Self-Relations Questionnaire
2. Body Image Automatic Thoughts Questionnaire (BIATQ)	Cognitive distortions related to appearance	Developed by Brown et al. (1990)
3. Body Shapes Questionnaire (BSQ)	Body satisfaction/ dissatisfaction	Developed by Cooper et al. (1986)
4. Body Attitudes Test (BAT)	Body satisfaction/ dissatisfaction	Developed by Probst et al. (1995); four subscales: negative appreciation of body size: lack of familiarity with one's own body: general body dissatisfaction: and a rest factor
5. Binge Eating Scale (BES)	Presence of binge eating behaviors, cognitions, and emotions typical of those who binge eat	Developed by Gormally et al. (1982); provides cut-offs for nonbingeing, moderate, and severe bingeing behavior
6. Bulimic Investigatory Test, Edinburgh (BITE)	Bulimic behaviors	Developed by Henderson & Freeman (1987); includes symptom and severity subscales; has only been validated in adult women and adolescents of both sexes
7. Children's Eating Attitudes Test (ChEAT)	Symptoms and concerns characteristic of eating disorders	Developed by Maloney et al. (1988); adapted version of the EAT-26 for children 8–13; may need additional adaptations for younger children (Smolak & Levine, 1994)
8. Dutch Eating Behaviors Questionnaire (DEB-Q)	General structure of eating behaviors	Developed by van Strien et al. (1986); Scales for emotional, external, and restrained eating
9. EATATE semistructured interview	Weight and ED history	Semistructured interview (Schmidt et al., 2007) based on the Longitudinal Interval Follow-up Evaluation (Keller et al., 1987); validation of the measure not yet published
10. Eating Attitudes Test-26/40 (EAT-26/ EAT-40)	Symptoms and concerns characteristic of eating disorders	Developed by Garner & Garfinkle (1979); cannot be used to make a diagnosis

(*Continued*)

Table 40.6 (*Continued*)

Measure	*Constructs assessed*	*Notes*
11. Eating Disorders Examination (EDE)	Behavior and attitudinal symptoms of disordered eating	Developed by Fairburn, Cooper, & O'Connor (2008)
12. Eating Disorders Examination-Questionnaire (EDE-Q)	Behavior and attitudinal symptoms of disordered eating	Developed by Fairburn & Beglin (2008)
13. Eating Disorders Inventory-II (EDI-II)	Behaviors and psychological features associated with ED	Developed by Garner (1991)
14. Food-related Acceptance & Action Questionnaire (FAAQ)	Psychological flexibility in a food-rich environment	Developed by Juarascio et al. (2011); subscales assessing acceptance of distressing food-related thoughts/cravings and willingness to engage in healthy eating despite these experiences; measure has only been validated in undergraduate/community samples
15. General Food Craving Questionnaire	Possible precipitants and consequences of general food cravings	Developed by Nijs, Franken, & Muris (2007); an adapted version of the Trait and State Food Cravings Questionnaire (Cepeda-Benito, Gleaves, Williams, & Erath, 2001)
16. Interoceptive Awareness Scale-Expanded (IAS-E)	Awareness of appetite signals and emotions	Developed by Craighead & Niemeier (2002); expanded version of the Interoceptive Awareness Scale of EDI-2 in order to include appetite awareness and emotional awareness
17. Mizes' Anorectic Cognitions Scale	Typical cognitions relevant to AN/BN	Developed by Mizes & Klesges (1989). Revised versions available (Mizes, 1994; Mizes et al., 2000)
18. Morgan Russell Outcome Criteria (M-R)	Recovery status in those with AN	Developed by Morgan & Hayward (1988); general criteria only speak to weight recovery and menstrual status and dichotomizes success into "good/intermediate/bad"
19. Physical Appearance State and Trait Anxiety Inventory-State Version (PASTA)	Current anxiety/nervousness about 16 specific body parts	Developed by Reed et al. (1991)
20. Preoccupation with Eating Weight and Shape Scale (PEWS)	Frequency and distress related to eating and weight/shape-related thoughts	Developed by Niemeier et al. (2002)

Table 40.6 (*Continued*)

Measure	*Constructs assessed*	*Notes*
21. Questionnaire on Weight and Eating Patterns (QWEP)	Assessment of eating patterns characteristic of BED	Developed by Spitzer et al. (1992); can classify individuals into no diagnosis, nonclinical levels of bingeing, and BED; adolescent and parent completion forms available
22. The Structured Interview for Anorexic and Bulimic Disorders for DSM-IV and ICD-10	Assessment and diagnostic categorization of ED	Newest version developed by Fichter & Quadflieg (2001); semistructured interview that shows correlations with the EDE
23. Situational Inventory of Body Image Dysmorphia	Frequency and intensity of negative appearance-related feelings in various situational contexts	Developed by Cash (1994); useful for determining situations that are distressing for persons with body-image difficulties
24. Short Evaluation for Eating Disorders	Key ED symptoms (3 for AN, 3 for BN)	Developed by Bauer, Winn, Schmidt, & Kordy (2005); allows user to calculate severity index for AN/BN
25. Three Factor Eating Questionnaire (TFEQ)	Three dimensions of human eating behavior—cognitive restraint, disinhibition, hunger	Developed by Stunkard & Messick (1985)
26. Yale-Brown Eating Disorders Scale (YB-ED)	Severity of illness in those with EDs	Developed by Sunday et al. (1995). Subscales that assess rituals/preoccupations and motivation for change

Patient Characteristics

Specific patient characteristics can be important when selecting assessment measures. On one hand, measures are often developed for or with specific populations (e.g., gender, diagnosis) and difficulties can arise when using measures on populations for which they were not designed (see Chapters 23 & 37). For example, many body image assessment instruments were developed on female samples and assess stereotypically female body image concerns (e.g., large hips and thighs), and some have argued that they may be inappropriate for use in males (Darcy & Lin, 2012; Thompson & Cafri, 2007). Also, with the advent of the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*; American Psychiatric Association, 2013), some older measures may not reflect current diagnostic criteria. Broadly speaking, researchers should select measures developed on their population of interest and for which there are appropriate norms.

On the other hand, researchers may have theoretical or practical reasons for wanting to use a specific measure, or a measure may not be available for their population of interest. Thankfully, norms have been developed for some measures for different populations than those for which

the measure was originally designed (see Allison, 2009, for extensive norms for eating disorders measures). It should be noted, however, that whenever a measure is used in a sample substantially different from the group on which it was developed, there exists a risk that the measure in question will exhibit differential item functioning (DIF), or item bias. DIF occurs when specific items within a measure exhibit different probabilities of item endorsement relative to group membership. This means that a member of one group would have a decreased probability of endorsing an item relative to a member of another group when the overall score on the measure is held constant. If DIF is present within a measure, one can assume reduced variability and subsequently misleading results regarding the latent trait. While very few eating-related measures have been subjected to DIF analysis, those that have been conducted have found substantial gender-specific item bias in measures of body checking (Alfano, Hildebrandt, Bannon, Walker, & Walton, 2011). Thus, even though norms may be available for a measure, a given score may not accurately reflect the underlying construct of interest. Until this issue is investigated more thoroughly in the EDs literature, researchers should interpret with some caution scores on measures developed under different populations than those they are studying.

Theory Testing

It has been argued that the theoretical model underlying treatment should play a critical role in the choice of assessment instruments (Anderson & Murray, 2010). CBT, DBT, FBT, and IPT all have some evidence for their efficacy in treating EDs (Hay, 2013; see also Chapters 56, 57, 60, & 62), but all are thought to achieve their effects through different mechanisms (Arcelus, Haslam, Farrow, & Meyer, 2013; Fairburn, Cooper, & Shafran, 2003; Lock, Le Grange, Agras, & Dare, 2001; MacPherson, Cheavens, & Fristad, 2013; Murphy et al., 2009; Murphy, Straebler, Basden, Cooper, & Fairburn, 2012). To test the possible mechanisms of these treatments, it is necessary to choose assessment instruments that measure the critical constructs and mechanisms of interest. As an example, CBT postulates that the core psychopathology of most eating disorders is the overvaluation of shape and weight and their control (Fairburn, 2008; see also Chapter 56). Accordingly, studies of CBT for EDs should include an assessment of this construct. DBT, however, suggests that failures in emotion regulation are a key feature in the maintenance of EDs (MacPherson et al., 2013; see also Chapter 57) and, as such, studies of DBT for EDs should assess emotion regulation where studies of CBT might not.

Historically, however, the EDs field has not done well in this regard. For example, an earlier review of CBT for BN found that researchers focused on the overt behavioral symptoms of BN more than the cognitive and other symptoms of the disorders, and almost no studies assessed all of the core domains hypothesized to play a role in the maintenance of the disorder (Anderson & Maloney, 2001). More recently, a review of DBT for EDs found that no studies to date have directly assessed a key component of the DBT model—that DBT improves emotion regulation skills and abilities (Bankoff, Karpel, Forbes, & Pantalone, 2012). Moreover, the IPT model for the development and maintenance of eating disorders (see Chapter 62) has not yet been directly tested (Murphy et al., 2012).

However, things are improving somewhat. As noted previously, researchers in the EDs field have begun to call for increased investigation of underlying mechanisms of change by investigating mediators of treatment (Kraemer et al., 2001, 2002; Murphy et al., 2009). This process requires careful thought about the instrument or instruments used to assess the proposed underlying mechanism as well as the timing of administration (Kazdin, 2009; Murphy et al., 2009). Researchers interested in investigating CBT have the advantage of having measures available that

specifically assess the underlying model of the treatment (i.e., the EDE and EDE-Q). Unfortunately, there are few options for researchers wishing to test other models of EDs.

Conclusions and Future Directions

The assessment of EDs and related symptoms has evolved over the past decades. To help advance the field, we offer the following suggestions:

1 Make sure to match assessment instruments to the underlying theory being tested. This will allow for better testing of these theories, as well as possible mediators of change.
2 Assess as often as is feasible. Because change often happens early in treatment, frequent assessment is necessary to capture this process.
3 Use popular measures, but not only popular measures. It is extremely helpful to be able to compare results across studies, and a common metric facilitates this process. But supplementary measures may be necessary to assess all aspects of the underlying theory. New measures may need to be developed to assist in this process.

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Section VIII
Prevention

41

Why Prevention? The Case for Upstream Strategies

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> I am standing by the shore of a swiftly flowing river and I hear the cry of a drowning man. So I jump into the river, put my arms around him, pull him to shore and apply artificial respiration. Just when he begins to breathe, there is another cry for help. So I jump into the river, reach him, pull him to shore, apply artificial respiration, and then just as he begins to breathe, another cry for help. So back in the river again, reaching, pulling, applying, breathing and then another yell. Again and again, without end, goes the sequence. You know, I am so busy jumping in, pulling them to shore, applying artificial respiration, that I have *no* time to see who the hell is upstream pushing them all in. (Irving Zola, as cited in McKinlay, 1979, p. 9)

Although the prevalence of eating disorders (EDs), as defined by the psychiatric criteria in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text revision; *DSM-IV-TR*; American Psychiatric Association, 2000), is relatively low—for example, the estimated lifetime prevalences for anorexia nervosa (AN) and bulimia nervosa (BN) among U.S. adults are 0.6% and 1.0%, respectively (Hudson, Hiripi, Pope, & Kesler, 2007; see also Chapter 5)—such disorders are characterized by severe adverse consequences to psychosocial and physical health (see Chapters 15 & 52). Harris and Barraclough (1998) report that across the spectrum of mental illnesses, EDs are associated with one of the highest risks of premature death. Relying on treatment to reduce the prevalence of EDs is undesirable, if not impossible, because most individuals with EDs do not seek treatment due to the various geographic, economic, and sociocultural (e.g., shame and stigma) obstacles (Cachelin & Striegel-Moore, 2006). In addition, available treatments are often expensive, complicated, and the prognosis of full recovery is relatively poor (Vitiello & Lederhendler, 2000).

Adverse health outcomes are also associated with less extreme forms of disordered eating, which are much more prevalent (Neumark-Sztainer, Story, Hannan, Perry, & Irving, 2002). Data from Project Eating Among Teens (EAT), a comprehensive study of eating patterns and weight concerns in a sample of 4,746 U.S. adolescents, revealed that 12% of adolescent girls and 5% of adolescent boys report engaging in at least one extreme disordered eating behavior (including vomiting or using diet pills, laxatives, or diuretics; Neumark-Sztainer et al., 2002). A much higher number of adolescents (57% of adolescent girls and 33% of adolescent boys)

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report engaging in at least one less extreme disordered eating behavior, such as fasting or eating very little food (Neumark-Sztainer et al., 2002). Although the youth engaging in these behaviors typically do not meet the diagnostic criteria for EDs, these behaviors can result in potentially serious adverse health outcomes, including poor dietary quality, unhealthy weight gain and obesity onset, and depressive symptoms, as well as the onset of EDs (Neumark-Sztainer, Eisenberg, Fulkerson, Story, & Larson, 2008).

Given that the consequences of EDs and disordered eating are serious and treatment is often expensive with limited effectiveness, efforts are needed to prevent the onset of EDs and disordered eating. In this chapter we will define prevention (including the different levels of prevention and how the level informs the type of intervention), compare and contrast the individual and population health approaches to prevention, and describe how the population health approach can inform etiologic research on EDs as well as prevention efforts. We will conclude the chapter by highlighting some key challenges and proposed next steps for the population health approach to the prevention of EDs.

Prevention Defined

Throughout the literature, many definitions and models have been presented to illustrate the concept of prevention. One such model, published by Gerald Caplan in 1964, is prominent not only in the EDs realm, but also in the broader domain of preventive medicine (Austin, 2000; Levine & Smolak, 2006; Offord, 2000). According to Caplan (1964), prevention can be divided into three levels: primary, secondary, and tertiary. Primary prevention is generally concerned with reducing the incidence (number of new cases) of a condition in a population, targeting those individuals who do not exhibit any signs or symptoms. Secondary prevention seeks to reduce the prevalence of a condition (or the proportion of the population affected by a condition) through targeting high-risk individuals who may be experiencing early signs and symptoms. Through early detection (see Chapter 38), these individuals with subclinical disease can receive appropriate medical interventions to either prevent or delay development of the full-fledged condition. The last level, tertiary prevention, is concerned with improving management of the condition and its potentially debilitating effects among individuals who have already been diagnosed (Austin, 2000; Caplan, 1964; Offord, 2000). It is important to note, however, that the distinction between primary and secondary prevention is not always black and white, as it can be difficult to differentiate healthy individuals from those who have a subclinical condition.

Using Caplan's triadic model as a basis, other researchers have proposed different frameworks for classifying prevention (Catalano & Dooley, 1980; Mrazek & Haggerty, 1994). For example, Catalano and Dooley (1980) argue that Caplan's notion of primary prevention can be further dichotomized into reactive and proactive approaches. Reactive primary prevention seeks to enhance individuals' resistance to potentially harmful stressors in the environment, equipping individuals with the necessary resources to effectively deal with a stressor. An example of reactive primary prevention in the context of EDs is implementing a school-based media literacy intervention (see Chapter 45) to teach young girls how to critically evaluate the portrayal of women in the media and the ideal that equates beauty with thinness. Proactive primary prevention, on the other hand, seeks to eliminate or avoid exposure to the stressor(s) altogether (Catalano & Dooley, 1980). Instead of using media literacy programs to reduce the adverse consequences of exposure to unhealthful media messages, those interested in proactive

primary prevention would try to get to the root of the problem by banning the use of extremely thin models in advertising (as was done in Israel in 2012; see Chapter 48), in an effort to eliminate exposure to unhealthful media messages altogether (British Broadcasting Corporation, 2012). Mrazek and Haggerty (1994) provide an alternative view to the categorization of primary prevention, defining the levels in their model by the target population such efforts are geared toward. In this sense, they conceptualize primary prevention as comprising both universal and selective approaches. Universal approaches to primary prevention target the population as a whole, in hopes of preventing the development of a condition in both healthy individuals and those at risk. Selective approaches focus on smaller subgroups of individuals within the general population who, though not exhibiting any signs or symptoms of the condition, are considered to be at high risk because, for example, they are avid dieters or have a family history of EDs (see Chapters 28 & 36). Given this requirement of having to be at high risk for developing a condition, selective approaches may be interpreted by some as more closely resembling Caplan's definition of secondary prevention (Levine & Smolak, 2006).

Caplan's notion of secondary prevention is also endorsed by Catalano and Dooley, who view secondary prevention as early intervention for the treatment of early signs and symptoms (Berry et al., 2008; Catalano & Dooley, 1980). Alternatively, in their scheme, Mrazek and Haggerty (1994) label this level of prevention as "indicated" or "targeted" prevention. This emphasizes that such prevention efforts are "indicated for" or "targeted toward" individuals who are noticeably demonstrating early signs and symptoms of the condition, in order to prevent their subclinical status from progressing to the fully developed condition. With respect to the third level of prevention in Caplan's model (tertiary prevention), Catalano and Dooley (1980) term this "tertiary *intervention*," implying that efforts within this level primarily involve intervening to manage chronic symptoms of the individual's condition, rather than taking measures to prevent it. Mrazek and Haggerty (1994) share a similar perspective, conceptualizing tertiary prevention as treatment rather than as a type of prevention.

To reduce the personal and societal burden of EDs, all three levels in Caplan's model of prevention are needed. Although individuals with a clinically diagnosed ED definitely deserve and require treatment or tertiary prevention to improve management of the disorder and its potentially debilitating effects, we also need to work to prevent the onset of new cases, through primary and secondary prevention efforts. However, while all three forms of prevention are needed, in the following section we will argue that universal population-based approaches will be paramount in reducing the incidence of EDs.

Individual Versus Population Approaches to Prevention

Geoffrey Rose, a prominent epidemiologist, identified two distinct strategies to address the prevention of both communicable and noncommunicable diseases: the "high-risk strategy" and the "population strategy" (Rose, 1985; Rose, Khaw, & Marmot, 2008). The high-risk strategy, or individual approach to prevention, focuses on the causes of disease in individuals. This strategy underlies the traditional medical approach to prevention, whereby healthcare professionals attempt to identify those individuals considered to be at high risk, and then offer them protection against disease. In this respect, the high-risk strategy closely resembles Caplan's notion of secondary prevention and Mrazek and Haggerty's (1994) concept of indicated or targeted prevention, both of which attempt to target high-risk individuals who may be experiencing early signs and symptoms of a disease (Austin, 2000; Caplan, 1964; Offord, 2000).

Conversely, Rose's population strategy underlies the public health or population health approach to disease prevention (Health Canada, 2001), focusing on identifying and controlling the determinants of disease in order to reduce the number of new cases (i.e., the incidence) in a population (Rose, 1985; Rose et al., 2008). In this respect, the population approach resembles Caplan's (1964) notion of primary prevention, while incorporating Mrazek and Haggerty's (1994) concepts of universal and selective prevention, in that the population approach attempts to reduce the incidence rates through targeting those individuals who do not yet exhibit any signs or symptoms (Austin, 2000; Offord, 2000).

High-Risk Strategy

The high-risk strategy provides potential advantages for individuals at risk and for the medical care system (Rose, 1985). One advantage is that interventions based on this strategy are specifically tailored to address the problem each individual patient is experiencing. For example, an adolescent female who demonstrates signs of disordered eating (in contrast to signs of panic disorder) may be referred by her family physician to a dietitian and psychiatrist for counseling. Working as an interdisciplinary team with expertise in EDs (see Chapter 50), the dietitian and psychiatrist can use aspects of cognitive-behavioral therapy (CBT; see Chapter 56) during counseling sessions in an attempt to change the client's maladaptive thinking around eating and body image and, subsequently, her or his disordered eating behavior (British Dietetic Association Mental Health Group, 2011).

When interventions are appropriate, they have the additional advantages of enhancing the motivation of both the person and the physician (Rose, 1985). Individuals will perceive such interventions as personally relevant and beneficial for their health, increasing their motivation to comply with recommendations. At the same time, increases in physician motivation will emerge from feeling justified that her or his medical intervention is necessary and welcomed by the patient. The final two potential advantages of the high-risk strategy relate to the favorable benefit/risk ratio and cost-effectiveness of this approach (Rose, 1985). If exposure to intervention carries some cost or risk to the individual, and if this risk is uniform across a population group, the benefit/risk ratio will be larger and more favorable for those individuals at highest risk, with the benefits outweighing the potential risks of intervening. Given the fiscal challenges facing today's medical system, it can be argued that it is more cost-effective to allocate limited funds and resources to those individuals who are most in need and most likely to benefit, rather than addressing the entire population that includes a sizable proportion of individuals who may not require intervention (Rose, 1985; Rose et al., 2008).

Although these potential advantages make the high-risk approach appealing to many individuals, healthcare professionals, and policy-makers, the ability of this strategy to effectively reduce the incidence of EDs and disordered eating is constrained due to some key limitations of the high-risk approach (Rose, 1985). First, the high costs and difficulties associated with disease screening (see Chapter 38) undermine the previously highlighted cost-effectiveness of the high-risk strategy (Rose, 1985). Since the risk for EDs starts relatively early in childhood and peaks in the late teenage years or early twenties, screening would need to be initiated early, universally, and repeated regularly in order to effectively detect those at risk (Mayo Clinic, 2012; Rose, 1985). Furthermore, developing an effective screening tool with an optimal balance of sensitivity and specificity is also a considerable challenge (see Chapter 38). *Sensitivity* refers to the proportion of individuals with an ED who are correctly identified as having an ED, while *specificity* refers to the proportion of individuals without an ED who are

correctly identified as not having an ED (Haines et al., 2011). In our study of school-based initiatives for the prevention of EDs among adolescent girls and boys, we assessed the effectiveness of attitudinal and behavioral items on a screening survey for identifying at-risk individuals in a sample of high-school students (Haines et al., 2011). Although the screening measure that combined attitudinal and behavioral questions was found to have a higher sensitivity than screening measures using only attitudinal items, this difference was moderate, and the balance between sensitivity and specificity was still not optimal (Haines et al., 2011).

A second reason the high-risk approach is limited is that a large number of individuals at low to moderate risk may actually lead to more cases of disease than a small number of individuals at high risk (Rose, 1985). In a groundbreaking paper on the prevention of EDs, Austin (2001) applied Rose's population-based prevention model to implicate dieting (see Chapter 24) as both a causal factor and a key point of intervention for the prevention of EDs and disordered eating. Despite the established fact that low-to-moderate dieters have a smaller risk of developing an ED (compared to extreme dieters), more ED cases will emerge from that group than from the high-risk dieters, as the former group of individuals comprises a much larger proportion of the population (Austin, 2001). To illustrate, consider a "population" of 1 million adolescent girls. Assume that 20% are high-risk dieters, and that of this group, 30% (n = 60,000) will develop an ED. Assume that for the remaining adolescent girls, 10% will develop an ED, because the high-risk group is at three times the risk. The "Rose paradox" here is 10% of 800,000 is 80,000, meaning that approximately 57% of the new ED cases come from the low-to-moderate risk group. Therefore, even if we were able to correctly identify those at high risk and effectively treat them, we would still observe a substantial number of cases emerging from the low-risk group.

Third, and most importantly, the high-risk strategy does not attempt to change factors within the individual's environment that perpetuate the development of EDs (Rose, 1985; see also Chapter 47). For example, although the dissonance-based intervention for the prevention of EDs developed by Stice, Chase, Stormer, and Appel (2001; see also Chapter 44) has proven to be successful because it addresses a key underlying risk factor for EDs (i.e., internalization of the thin ideal; see Chapter 21), such an approach does not attempt to eliminate the thin ideal mindset held within our society. As a result, this approach is both temporary and superficial, and will need to be sustained as long as susceptible individuals are present in the population (Rose, 1985; Rose et al., 2008). As highlighted by Irving Zola's river story, which introduces this chapter, if we do not identify and change the "upstream" causes of EDs, we will not be able to reduce the number of individuals who develop these disorders.

The last limitation of the high-risk strategy pertains to the fact that it is behaviorally inappropriate (Rose, 1985). Since many behaviors revolve around established social norms within a population, advising high-risk individuals to engage in behavior that violates these social norms will be difficult to sustain in the long term, because the environment is not conducive to supporting such changes (Rose, 1985). For example, "fat talk," defined by Nichter and Vuckovic (1994) as "a social phenomenon during which women speak negatively with each other about the size/shape of their bodies" (p. 636), is prevalent in Western society among women of all ages and is directly associated with body dissatisfaction and disordered eating behaviors (Salk & Engeln-Maddox, 2012; see also Chapters 27 & 31). Sadly, it is much more normative for women to be disparaging about their bodies than to accentuate aspects of their bodies that they like, let alone critically evaluate cultural forces that promote body dissatisfaction in tens of millions of women (Piran, 2010; Salk & Engeln-Maddox, 2012). To advise young women who are identified as high risk for developing EDs to avoid exposure to "fat talk"

or to engage only in positive body talk is unlikely to be effective since the social norms in our society do not support these recommendations. A preferred approach would be to involve all members of the population in working together to change the social environment, through campaigns such as "Fat Talk Free Week," which is a week dedicated to eliminating fat talk within our society (Smith, 2010). Through implementing such initiatives, the social norms guiding these behaviors can be changed to support our health messages.

Population Strategy

The population strategy is focused on identifying and either eliminating or modifying "upstream" determinants of a disease or condition. According to Rose (1985), adopting the population strategy for prevention offers three key advantages. First, this strategy attempts to understand *why* disease occurs through identifying and removing the underlying cause(s) of the disease/condition for the entire population, rather than waiting for individuals to succumb to the condition before intervening (Rose, 1985). This is in stark contrast to the high-risk strategy where intervention is postponed until one is considered to be at high risk of developing the disease, perhaps because one is showing early signs of the condition.

Second, the population strategy has potential to reduce the prevalence of adverse health outcomes across the population by shifting the distribution of risk in a favorable direction, as illustrated in Figure 41.1 (Rose, 1985, 1993). This favorable shift consequently leads to a reduction in incidence rates, since a greater proportion of the population now falls into the low-risk category. Revisiting Austin's (2001) example of dieting behavior, this favorable shift would cause a large proportion of individuals classified as moderate dieters to move into the low-risk dieting category, thereby reducing the number of new ED cases in a population (see Figure 41.1).

Lastly, the population approach to prevention creates changes in attitudes and behavior that are behaviorally appropriate, that is, are in accordance with the social norms of the population (Rose, 1985; Rose et al., 2008). Revisiting our previous example of the recent ban on using extremely thin models in advertising throughout Israel (see Chapter 48), changing how the female body is portrayed in the media could have a widespread impact on body image perceptions among females of all ages. The principal goal of this ban is to begin to change the public mindset in Israel around what is considered an ideal body, so that being extremely thin will no longer be identified as the ideal. By changing social norms around health beliefs and behaviors, population prevention strategies ensure that the social norms support healthful behaviors.

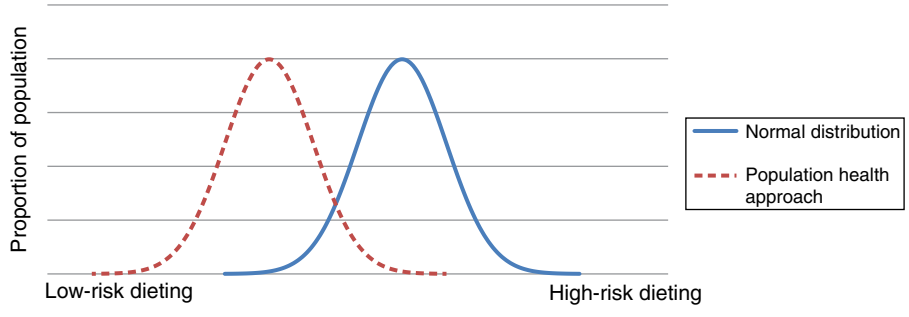

Figure 41.1 Shift of risk distribution observed in the population health approach (adapted from Rose, 1993).

Identifying Determinants of Eating Disorders

In his paper entitled "Sick individuals and sick populations," Rose (1985) described how, if a potential determinant of a condition or disease is ubiquitous throughout the population, its potential impact on disease risk could be missed or underestimated if we examine the impact of that risk factor *within* the population. To accurately identify the impact of a risk factor whose influence permeates the culture, Rose argues, comparisons *between* populations in which the level of exposure to the risk factor differs are needed. Cultural pressures to endorse and achieve the "thin ideal" or the "thin-muscular ideal" may be an important determinant of ED risk for females and males, respectively (see Chapters 21 & 27). These pressures are particularly prominent in Western cultures, where weight loss is heavily marketed and underweight models are portrayed as the ideal image of beauty. The media serve as a major outlet for delivering such messages, through channels including print advertising, television advertising, and the Internet (see Chapter 29). While small to moderate effects have been shown in studies examining the impact of media exposure on ED risk within the U.S. population, where media are nearly ubiquitous (Levine & Harrison, 2004), stronger evidence of the impact of media on risk of EDs comes from Anne Becker's research exploring the impact of television on disordered eating attitudes and behaviors in a population with no previous exposure to the media.

This research uncovered striking results about the effects of Western media on ethnic Fijian adolescent girls' body image and eating behavior (Becker, Burwell, Gilman, Herzog, & Hamburg, 2002; see also Chapter 23). Specifically, the introduction of television among this "media-naïve" population was associated with increases in two indicators of disordered eating over a 3-year period: a 17% increase in the proportion of adolescent girls with high EAT-26 scores, and an 11% increase in the proportion of girls who reported engaging in self-induced vomiting to lose weight. Since Fijians were previously known for their exceptionally low prevalence of eating pathology and had no previous exposure to Westernized media messages, this strongly suggests that television (and the media in general) could be responsible for the negative changes observed with respect to eating attitudes and behaviors in this group, among other potential economic and social changes accompanying the introduction of television (Becker et al., 2002).

In subsequent research in Fiji, Becker et al. (2011) found that having a social network of peers who are exposed to visual mass media (measured by asking participants how many of their closest friends and female peers at school have access to a television, video deck, or DVD player in their home) was modestly associated with disordered eating, independent of direct media exposure and other cultural exposures, such as lifetime personal overseas travel or family overseas travel to earn income over the past year. This indicates that diffusion of media messages through friends and peers has a substantial influence on adolescent girls' eating attitudes and behavior, over and above the impact of viewing such messages oneself.

Becker et al.'s (2002, 2011) Fijian studies illustrate the harmful effects that the media's portrayal of the female body has on women, especially those in the adolescent age group whose body image may already be fragile due to the pubertal changes they are experiencing at this age (Levine & Murnen, 2009). These studies also demonstrate that between-population comparisons of other potentially diffuse risk factors (see Chapter 23), such as dieting, sexual objectification, or social norms regarding fat-talk, may be needed to adequately estimate the impact of these population-level risk factors on ED risk.

Population Health Approaches to Preventing Eating Disorders: What Can We Do?

In this section, we identify and describe three potential avenues for the primary prevention of EDs using the population strategy:

1 Reduce unhealthful messages in the media.
2 Change social norms regarding body image.
3 Reduce the prevalence of weight bias.

Given the strong influence of media messages on body image concerns or eating attitudes and behavior, measures need to be taken at the population level to eliminate the unhealthy messages that are being portrayed to females (and society in general) regarding what constitutes beauty (Levine & Murnen, 2009; Smolak, Levine, & Schermer, 1998; Thompson & Heinberg, 1999). Levine, Piran, and Stoddard (1999) defined media activism as protesting (or conversely praising) media messages, advertisements, or products that are conveying (or contradicting) unhealthy messages. Thompson and Heinberg (1999) suggest that media activism has the potential to assist in reducing the high prevalence of body image concerns among individuals. Jasper (1993; cited in Thompson & Heinberg, 1999) presents the example of Hershey's discontinuing one of their chocolate bar advertisements that contained the slogan "you can never be too rich or too thin," after an EDs awareness group (National Association of Anorexia Nervosa and Associated Disorders, Inc.®; ANAD) wrote letters of complaint to the company.

This illustrates that we, as members of society and consumers of media messages, have the power to change the unrealistic "thin ideal" environment we live in to an environment that promotes health at every size. Generating support for a public backlash against unhealthy weight-related messages in the media could, in turn, influence the development of new policies designed to reduce exposure to unhealthful media messages. In an attempt to encourage the adoption of a healthier body image among its citizens at the population level, the state of Victoria in Australia has recently developed and implemented a voluntary media code of conduct (see Chapter 48). This code of conduct contains four clauses that address the negative portrayal of body image across the media, fashion, and advertising industries (Australia PolicyOnline, 2010; Paxton, 2011). For example, one of the clauses is concerned with discouraging the use of digitally altered or enhanced images, requiring companies to tag such images with the caption "this image has been digitally altered" to inform viewers that the image is not a realistic portrayal of the human body (Commonwealth of Australia, 2009). There is clearly a need for further research in this area, as initial findings regarding these disclaimers indicates they may actually be counterproductive (Tiggemann, Slater, Bury, Hawkins, & Firth, 2013; see also Chapter 29).

Along with changing weight-related messages that are disseminated to individuals through various media outlets, social norms around weight and body image need to be altered in order to create an environment that promotes and reinforces body satisfaction and healthful behaviors among individuals of all shapes and sizes (see Chapter 43). In her article advocating for a population-based approach to the prevention of EDs, Austin (2001) stresses that dieting not only has negative health consequences for the individual engaging in such behavior, but also may influence that person's peers to start dieting—especially when mass media and other prominent aspects of culture are promoting this to adolescent girls and young women—creating a "mass dieting" climate (see Chapter 31). Therefore, as noted previously, it is essential

to alter the public mindset around dieting so that it is no longer considered to be the norm (Austin, 2001). One example of an initiative addressing unhealthy social norms is "Fat Talk Free Week," which was discussed earlier. This national initiative emerged from a collaborative effort between Dr. Carolyn Becker (a psychology professor; see Chapter 44) and a sorority at Trinity University in San Antonio, Texas, and, with the help of numerous community partners, aims to raise awareness about the dangers of engaging in fat talk through advertising the motto "Friends don't let friends fat talk" (Smith, 2010). This initiative emphasizes that even seemingly harmless comments such as "You look so thin in that dress!" can have a negative impact on body image, reinforcing the idea that thinness is equated with beauty (see Chapters 27 & 31). Dedicating one week of the year to eliminating fat talk, however, is not sufficient to improve body satisfaction throughout a population; rather, such attitudes toward fat talk need to be maintained year-round through continued advocacy, in order to produce sustainable population-wide changes in both attitudes and behavior relating to body image.

Lastly, we also need to address the fat bias and weight stigma that is widespread not only within Western culture, but also within many other cultures across the globe. Over recent decades, research in the United States has shown that fat bias and weight stigma have become increasingly inherent in much of the television programming and advertising, especially with respect to the advertising of weight-loss products and programs. For example, Blaine and McElroy (2002) found that many messages present in weight-loss infomercials reflected common stereotypes, such as overweight individuals being gluttonous, lazy, and lacking in willpower. Furthermore, when normal weight, overweight, and obese individuals were used in weight-loss infomercials, heavier individuals were portrayed as more unhappy and unattractive than their thinner counterparts (Blaine & McElroy, 2002). Similar stereotypes are also promoted in news stories related to obesity and obesity research through the use of photos and video clips of overweight individuals consuming unhealthful foods or being sedentary. For example, in a recent content analysis, researchers found that 72% of images in online news stories relating to obesity were stigmatizing toward overweight and obese individuals (Rudd Center for Food Policy & Obesity, n.d.).

In a policy brief from Yale's Rudd Center for Food Policy & Obesity, Friedman and Puhl (2012) review a set of studies demonstrating that this negative, stigmatizing portrayal of obese individuals in the media reinforces and is reinforced by weight bias in other facets of life, including the workplace. In fact, one study found that 43% of overweight and obese individuals reported having experienced some form of weight bias by employers in the workplace (Puhl & Brownell, 2006). Even more disturbing are the negative consequences that overweight and obese employees face as a result of their weight. For example, it has been found that overweight and obese employees (especially females) earn less than normal weight employees in the same position and also receive fewer promotions.

The state of Michigan in the United States is currently the only area in North America where legislation exists to prohibit discrimination against overweight and obese individuals (Rudd Center for Food Policy & Obesity, n.d.). Specifically, under the Elliott-Larsen Civil Rights Act, overweight and obese individuals cannot be discriminated against on the basis of their weight, across a range of areas including employment, housing, public accommodations, public service, and educational facilities (Friedman & Puhl, 2012). When a sample of American adults was asked whether they would support legislation to limit weight discrimination in the workplace, 81% of females and 65% of males replied in the affirmative (Puhl & Heuer, 2010, cited in Friedman & Puhl, 2012). Collectively, these findings indicate a strong need and preliminary support for widespread legislation to prohibit forms of

weight-based discrimination, in order to maximize the health of overweight and obese individuals in various domains (i.e., social, economic, psychological, physical), as well as the opportunities available to these individuals (Friedman & Puhl, 2012).

Conclusions and Future Directions

The population approach to prevention is not without its limitations, many of which are the opposite of advantages observed in the high-risk strategy. At the forefront of these limitations is the fact that this strategy offers little benefit to individuals (Rose, 1985). This is because a sizable proportion of individuals in a population will never develop EDs or disordered eating behaviors, or be affected by observing a loved one who is suffering from an ED (Rose, 1985). The small benefit reaped by each individual necessarily decreases the benefit/risk ratio of the intervention strategies to the point where, in some cases, potential risk or costs (even if it is just time spent focused on a health message) may outweigh the benefit of intervention. When individuals perceive that prevention efforts are of little benefit or relevance to them, their motivation for healthy behavior change will likely decrease as a result (Rose, 1985).

This perception also presents a challenge when attempting to convince policy-makers to adopt a population health strategy for the prevention of EDs. Consequently, strong advocacy efforts are needed to convince policy-makers and health professionals that a population strategy is needed to reduce the incidence and ultimately the prevalence of EDs (see Chapters 48 & 66): if we can modify the cultural and social environments so as to remove the causes of EDs, there will be no susceptible individuals requiring intervention, and the high-risk strategy will therefore be irrelevant (Rose, 1985). If we were to adopt the high-risk strategy, however, such mass influences experienced by the population as a whole would not be addressed (Rose, 1985). Although the widespread response that is advocated by Rose et al. (2008) is not likely to deliver immediate, salient, and significant benefits to each individual, such benefits collectively have a large favorable impact on the population as a whole (Rose et al., 2008). Therefore, we need to move beyond individual-level approaches to addressing ED risk, and implement more widespread approaches while assessing the impact of these approaches at the population level (see Chapter 47). Through engaging in such efforts, we will be able to address the underlying factors contributing to the development of EDs by creating an environment that is supportive of a healthful body image and weight-related behaviors. By creating such an environment, we will be taking a promising step toward reducing the overall incidence and prevalence of EDs in our population.

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42

School-Based Prevention

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Schools offer a unique opportunity to disseminate programs to promote positive body image and prevent eating disorders (EDs) among an entire generation of young people in a convenient setting. Numerous school-based programs designed to prevent the development of EDs have been implemented since the 1980s (Neumark-Sztainer et al., 2006). Several lessons were learned from the first generation of ED prevention programs, and more recent school-based programs have generally been more successful in terms of improving body image, and disordered eating pathology.

However, although many exciting developments have been made in the area of school-based ED prevention programs, complete effectiveness and dissemination has not been achieved. The school environment is particularly challenging for prevention research due to issues of appropriateness and relevance of programs, teachers' adherence to content, and effectiveness of standardized measures in "normal" populations, or those not particularly at risk (see Chapter 41).

In this chapter we outline the current evidence in terms of approaches to promote body image and prevent EDs in schools. We also consider the practicalities of implementation that might result in a united approach to the broader dissemination of these activities.

Psychoeducation Approaches to Prevention in Schools

Psychoeducation seems to suffer from a problem of definition among various groups. We define psychoeducation as the provision of information about the nature of body dissatisfaction or EDs in order to facilitate attitudinal and behavioral change (Olmsted et al., 1991). However, others consider psychoeducation to be a "professionally delivered treatment modality that integrates and synergizes psychotherapeutic and educational interventions" (Lukens & McFarlane, 2004, p. 206). The majority of the time, users of this term do not indicate what they mean by it, causing confusion.

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Psychoeducation was widely used in the "first generation" of ED prevention programs (Stice & Shaw, 2004), where information about the disorders was generally provided in a didactic manner (Pratt & Woolfenden, 2004). These programs are said to have been based on the assumption that young people would avoid initiating dieting and disordered eating behaviors if they knew more about the negative consequences of EDs (Stice & Shaw, 2004). Several meta-analyses (Stice & Shaw, 2004; Stice, Shaw, & Marti, 2007) and our systematic review (Yager, Diedrichs, Ricciardelli, & Halliwell, 2013) have reported that this approach was not effective in preventing body dissatisfaction and eating problems when used among younger audiences aged 12 to 17 years. However, programs incorporating psychoeducation have had some success when they are used as targeted prevention among college students, or are incorporated into the regular coursework of undergraduates over an entire semester at university (Stice & Ragan, 2002).

There were early concerns about the suitability of the psychoeducation approach for prevention work with mixed-risk groups of primary and secondary school students due to the potential for unintended negative consequences, as the disordered eating behaviors were effectively being "taught" (O'Dea, 2000). Although meta-analyses and reviews have refuted the potential for iatrogenic effects (Stice & Shaw, 2004), most psychoeducation content now avoids explicit discussion of behaviors for this purpose.

Risk Factor Approaches to Prevention in Schools

The second generation of ED prevention programs has focused on targeting specific risk factors known to contribute to the development of body dissatisfaction and EDs (Stice & Shaw, 2004). Early developments in this area saw programs focusing on improvement of psychological or individual risk factors such as self-esteem and resilience. More recently, programs have focused on encouraging resistance to known sociocultural pressures to be thin or muscular, such as peers and the media (see Chapters 45 & 48). Other programs have utilized cognitive dissonance to reduce internalization of the thin ideal (see Chapter 44). We will now explore these approaches in detail.

Targeting Self-Esteem

Early work in this area assumed that poor self-esteem was a strong predictor of body dissatisfaction, dieting, and disordered eating behavior. However, recent longitudinal studies have led to the consensus that this relationship is actually not very well understood. Some prospective studies have found that low self-esteem predicted future body dissatisfaction (Wojtowicz & von Ranson, 2012), but others did not (Tiggemann, 2005).

The Non-Specific Vulnerability Stressor Model (Levine & Piran, 2001; Levine & Smolak, 2006; see also Chapter 47) provides a theoretical rationale for using a self-esteem approach. According to this model, targeting broader, more general risk factors, such as self-esteem, which might be common to the development of many psychological disorders and physical diseases, will also help to prevent the development of EDs. The application of this theoretical model is actually very possible in practice, as the school health curriculum is already likely to include lessons to improve self-esteem and life skills such as communication, decision making, and stress management.

The Everybody's Different Program (O'Dea & Abraham, 2000) was one of the first to demonstrate the success of an approach that focuses on self-esteem (see Chapter 43). Program activities included those targeting self-esteem, body image, relationship skills, and decreasing stress; and specifically avoided discussion of weight and EDs (O'Dea & Abraham, 2000). Evaluation of this program with 13-year-old boys and girls indicated a decrease in body dissatisfaction, and results were stronger among high-risk girls (O'Dea & Abraham, 2000). However, replications among a younger age group in Sweden (Ghaderi, Matrtensson, & Schwan, 2005) did not produce the same positive results, emphasizing the need to provide age-appropriate prevention materials.

A number of other programs have also used a self-esteem approach, and been effective in improving self-esteem and body image. Everybody is a Somebody (McVey, Lieberman, Voorberg, Wardrope, & Blackmore, 2003) and Beautiful from the Inside Out (Norwood, Murray, Nolan, & Bowker, 2011) had a positive impact among children under the age of 12 years. An evaluation of the Dove BodyThink Program (which includes activities focusing on self-esteem) also found that it improved body image among young boys and self-esteem among young girls aged 12–14 years (Richardson, Paxton, & Thomson, 2009). Self-esteem is a popular inclusion in many body image and ED prevention programs, and is largely considered to be safe for young people of all ages. These activities are also likely to be accepted by schools and teachers, as self-esteem is generally a focus of the mainstream health education curriculum in most schools.

Reducing the Negative Impact of Peers

The influence and acceptance of friends and peers have emerged as being strongly related to body image for girls and boys (Holsen, Jones, & Birkeland, 2012; see also Chapter 31). Prevention programs either include content that focuses on peer-specific risk factors, or the programs are taught by peers to enhance the relevance of the materials. Programs that target the impact of peers have focused on risk factors such as peer acceptance, appearance comparisons, and appearance conversations. Activities are generally interactive, and focus on changing the impact of these risk factors, as well as changing the peer culture in the classroom. Activities that prepare young people for dealing with appearance-related pressures from peers are more common in programs targeting adolescents aged 12–13 years. Happy Being Me (Richardson & Paxton, 2010), and Media Smart (Wilksch & Wade, 2009) were effective in improving body image and included content that focused on the impact of peers, as well as specific activities to discourage peer comparisons and "fat talk." Making Choices (Weiss & Wertheim, 2005) and Everybody's Different (O'Dea & Abraham, 2000) also improved disordered eating pathology using this approach. Happy Being Me has been the most effective school-based program with a content focus on peers among adolescent girls (Richardson & Paxton, 2010). This program has been replicated among a younger, mixed gender audience in the United Kingdom with promising results (Bird, Halliwell, Diedrichs, & Harcourt, 2013).

Other programs have used peers, and a peer support approach to deliver prevention materials. For example, in Healthy Buddies (Stock et al., 2007), students in older year levels (grades 4–7) received program content from their class teacher and they then taught this to their younger "buddies" in grades 1 through 3. These programs are based on the premise that young people will be more likely to listen to their peers, whose opinions they value more than their teachers, on topics relating to appearance. Although this program was thorough, the evaluation of *Healthy Buddies* did not indicate any improvement in body image or self-esteem,

and other programs using a peer support approach have had similarly modest effects (McVey et al., 2003; Thompson, Russell-Mayhew, & Saraceni, 2012). Programs that were conducted in peer support settings that have been successful in improving body image include an intervention in an elite ballet school (Piran, 1998; see also Chapter 43), Girl's Circle (Steese et al., 2006), and Girl Talk/Everybody is a Somebody (McVey, Lieberman, Voorberg, Wardrope, & Blackmore, 2003).

Targeting Media Literacy

Media literacy is defined as an approach to education that involves accessing, analyzing, evaluating, and creating media messages in a variety of forms (Center for Media Literacy, 2011). Health-promoting media literacy has been used to teach about a variety of topics (Bergsma & Carney, 2008). Although media literacy has been used in ED prevention for many years, new research has confirmed its importance as a risk factor for body image problems (McLean, Paxton, & Wertheim, 2013).

Media literacy activities in ED prevention programs generally contain three types of activities. First, programs generally aim to teach students about the gender stereotypes that are promoted through the media, and how these contribute to the development of the thin and muscular Westernized body ideals. Programs also commonly present information about the tricks and techniques that are used by the media to create idealized images, such as airbrushing and the digital manipulation. The third common activity found in media literacy programs is consumer activism, where students are encouraged to write to industry bodies and advertisers expressing their views regarding the images that are used.

Media literacy programs have been effective in improving body image and disordered eating. Programs such as Everybody's Different (O'Dea & Abraham, 2000), Dove Bodythink (Richardson et al., 2009), Happy Being Me (Richardson & Paxton, 2010), and Media Smart (Wilksch & Wade, 2009) have included activities that could be classified as media literacy, and been effective in improving body image and disordered eating outcomes with adolescents over 12 years of age. Developmentally appropriate media literacy activities have also successfully improved internalization of the thin ideal, body image, and disordered eating outcomes with younger children (9 to 11 years of age) in Everybody is a Somebody (McVey, Lieberman, Voorberg, Wardrope, & Blackmore, 2003), Healthy Schools Healthy Kids (McVey, Tweed, & Blackmore, 2007), Eating Smart Eating for Me (Smolak & Levine, 2001), and Beautiful From The Inside Out (Norwood et al., 2011). Furthermore, media literacy is suggested to be a particularly appropriate way of approaching body image programs among boys and young men, as there is the opportunity to examine media stereotypes among both genders (see Chapter 37). Improved body image levels in intervention boys have been reported for Media Smart (Wilksch & Wade, 2009) and Beautiful From The Inside Out (Norwood et al., 2011), both of which included media literacy content.

The media literacy program with the strongest evidence base to date is Media Smart, an Australian program that consists of eight, 50-minute classroom lessons. Activities focus on media literacy, activism, and advocacy, including an exploration of the media's stereotypical portrayal of women and men in advertising, learning about airbrushing, and writing protest letters to industry. Evaluation has found this program to be effective in reducing 13-year-old girls' and boys' concerns with, and overevaluation of, body weight and shape, dieting, body dissatisfaction, and depression (Wilksch & Wade, 2009). Results remained positive even after 30-month follow-up periods.

Cognitive Dissonance Approaches

This approach aims to reduce internalization of the thin ideal for females and muscular ideal for males through counterattitudinal activities that challenge these beliefs. There is a substantial amount of evidence supporting the success of this approach in reducing ED risk factors with high-risk adolescent girls and young women using The Body Project (Stice, Rohde, Gau, & Shaw, 2009). The Body Project was also adapted for, and implemented among, mixed-risk groups of women belonging to university sororities using Reflections (Becker, Smith, & Ciao, 2006). Cognitive dissonance approaches meet the American Psychological Association criteria for an *efficacious* intervention (Becker, Ciao, & Smith, 2008), and this program has also demonstrated *effectiveness* among high-risk women and sorority groups when delivered by peer-leaders, or school nurses, clinicians, and counselors (Becker et al., 2006; Stice, Butryn, Rohde, Shaw, & Marti, 2013).

Recently, the same cognitive dissonance program was adapted for the U.K. context, and conducted with mixed-risk groups of girls in secondary schools. The Succeed Body Image Program was successful in improving body dissatisfaction and internalization of the thin ideal among 12- and 13-year-old girls in the United Kingdom when delivered by the researchers (Halliwell & Diedrichs, 2014). This study demonstrates the potential for cognitive dissonance programs to be effective with younger, mixed-risk groups of girls. However, no evaluations of cognitive dissonance programs have utilized regular classroom teachers and mixed-risk groups.

Trends in School-Based Prevention

Some current trends in school-based promotion of positive body image, and ED prevention, include focusing on younger age groups, addressing the needs of males, the use of innovative new activities, integrated ED and obesity prevention, and ecological approaches.

Targeting Younger Age Groups

Initially, prevention programs tended to be implemented among 15–16-year-olds (mostly girls), as this was the age at which the recipients were considered to be most at risk of developing EDs. More recently, programs have been developed for younger cohorts, as researchers have realized the importance of implementing programs with preadolescents before body image problems have developed (Smolak & Levine, 2001). Many programs to improve body image have now been conducted in primary schools, but results have not been consistently favorable. The heterogeneity of program approaches and activities and varied use of measurement tools make comparisons of programs difficult with this age group.

Furthermore, some authors have suggested that there is a certain age at which body image program materials become particularly relevant to young people. In a trial of an online program for preadolescents, it was found that the program was much more effective among the students who reported that "puberty was definitely underway," indicating a need to direct program materials at a pubescent audience (Cousineau et al., 2010). Although it seems that 12–13 years is the age around which most programs seem to be able to reduce body dissatisfaction (Yager et al., 2013), meta-analyses indicate that ED prevention is more effective among older girls (Stice & Shaw, 2004; Stice et al., 2007). Further research is required in order to determine which activities are most effective when delivered at each stage of development.

Incorporating the Needs of Boys and Young Men

Most early ED interventions were designed for, and targeted toward, the needs of girls. More recently, there has been an increase in the number of programs that include males, and the development of some programs that target young men on their own (McCabe, Ricciardelli, & Karantzas, 2010; Stanford & McCabe, 2005). Meeting the needs of boys is difficult due to the comparatively lower levels of understanding of risk factors for body dissatisfaction and disordered eating among young men, and of theoretical pathways of intervention effectiveness. However, it is promising to see that there is a general trend toward gender-neutral programs. Interestingly, in an analysis of the body image programs that have been conducted in secondary schools since 2000, Yager et al. (2013) found that, when body image programs were conducted in coeducational groups, there were never improvements in body image at post-test among both genders. Coeducational programs resulted in pre- to post-test improvements in body dissatisfaction only among boys (Richardson et al., 2009; Wilksch & Wade, 2009). Body image programs that were designed and conducted for males only were not particularly successful in improving outcomes on primary measures (McCabe et al., 2010; Stanford & McCabe, 2005).

Among students aged under 12 years, boys and girls are more likely to be combined for ED prevention programs. Researchers seem to be less likely to conduct separate analyses by gender in children under 12 years of age, and improvements in body image and disordered eating were more likely to be noted for girls where separate analyses did occur (Smolak & Levine, 2001; Varnado-Sullivan et al., 2001). Further research is required in order to develop suitable coeducational programs that result in improved outcomes for both boys and girls.

Innovative Program Activities

Although the majority of school-based programs still revolve around traditional curricular approaches, newer programs have experimented with innovative activities in the hope that they will be more effective in improving body image and ED pathology. Programs aimed at young children have found success using innovative approaches. Reading the story *Shapesville* (Mills, Osborn, & Neitz, 2003) was found to improve appearance satisfaction and weight stereotyping among 5–9-year-old girls (Dohnt & Tiggemann, 2008), and this format represents a very approachable option for including prevention activities in the school curriculum. Other programs have utilized the performing arts, for example, a 30- minute puppetry performance, "It's What's Inside That Counts," was used for elementary school children, and a dramatic play, "Heavenly Bodies," involved junior high-school students as a part of a larger wellness-based program (Russell-Mayhew, Arthur, & Ewashen, 2007). Healthy Schools-Healthy Kids also incorporated the presentation of a play, performed by local high-school students (McVey et al., 2007), and in Very Important Kids, young boys and girls (mean age 10 years) worked with a local theatre company over 10 one-hour sessions to develop and perform a theatre production (Haines, Neumark-Sztainer, Perry, Hannan, & Levine, 2006; see also Chapter 43). These programs were successful in improving risk factors for body dissatisfaction and EDs, such as weight teasing and internalization of media ideals (Haines et al., 2006; McVey et al., 2007).

Many school-based programs have started to incorporate enjoyable movement and physical activity in attempts to improve body image and disordered eating. One program gave yoga lessons in conjunction with positive psychology content in an afterschool program conducted

with fifth-grade girls that reduced levels of body dissatisfaction at posttest (Scime, Cook-Cottone, Kane, & Watson, 2006; see also Chapter 33). The New Moves program offered an all-girls PE class that included fun activities such as dance, hip-hop, and kickboxing, in addition to other program components (Neumark-Sztainer et al., 2010). Evaluation indicated that there were improvements in body image and self-worth, as well as reductions in sedentary behavior and use of unhelpful weight control methods among intervention girls (Neumark-Sztainer et al., 2010). Another study compared the impact of a 6-week aerobic dance curriculum to the effects of regular physical education classes for 13–14-year-old girls in the United Kingdom and reported decreased body dissatisfaction among the dance group (Burgess, Grogan, & Burwitz, 2006). PE is typically already included as a part of the regular school curriculum, but the results of these studies indicate that the content of PE programming may need to be revisited to represent more enjoyable options, which do not focus on competitive sports, in order to meet the needs of young girls, encourage participation, and improve body image and disordered-eating attitudes and behaviors.

Programs conducted with adolescents over 12 years of age tend to have more consistent program content and have experimented with fewer innovative approaches. The main innovation is the use of technology. Computer-based programs and those delivered online (see Chapter 46) have demonstrated some success in the secondary school setting (ages 12–18), such as My Body My Life (Heinicke, Paxton, McLean, & Wertheim, 2007) and Student Bodies (Abascal, Bruning Brown, Winzelberg, Dev, & Taylor, 2004).

Integrated Prevention of Obesity and Eating Disorders

The trend of integrated approaches for the prevention of both EDs and obesity emerged in the early to mid-2000s (Austin, 2000; Irving & Neumark Sztainer, 2002; Neumark-Sztainer et al., 2006; see also Chapter 49). Risk factors for both obesity and EDs are similar, and pioneers of this approach indicated that improving body image could result in improved outcomes for weight disorders at each end of the spectrum (Austin et al., 2007; Neumark-Sztainer et al., 2010). The late 2000s saw the development and evaluation of several broader programs that targeted the combined risk factors for obesity and EDs and had an additional focus on the reduction of bullying and weight comparison. Successful examples of such programs include Healthy Schools-Healthy Kids (McVey et al., 2007) for children, and New Moves (Neumark-Sztainer et al., 2010), for adolescent girls in high school. The crowding of the school curriculum, as well as increased competition for implementation of interventions, make integrated programs with a broader health promotion focus more attractive (Neumark-Sztainer et al., 2006). These sorts of combined approaches can be implemented as a part of an ecological, or whole-school approach to health promotion.

Using an Ecological or Whole-School Approach

Ecological approaches to the prevention of body image and disordered eating problems go beyond the provision of a content-based curriculum to address the sociocultural environment in which these problems develop (Levine & Smolak, 2006). There may be some issues of terminology with this approach. Researchers might refer to "environmental" or "ecological" approaches. However, schools might be more familiar with the "whole-school approach," or regionally specific terms including "Healthy Schools" (United Kingdom), "Health Promoting Schools" (Australia, Germany), or the "Coordinated School Health Approach" (United States).

Broadly, all of these different approaches are based on the premise that making changes within the physical and social environment will lead to increased intervention effects. These programs usually incorporate some sort of formal curriculum, but then also include greater teacher and student participation as key stakeholders in the program. Some level of parental involvement, activities to change peer culture and ethos of the school, changes to the physical environment of a school, and modifications to school policy are also frequent features of these types of approaches. Several authors have specified the mechanisms required for effective application of this approach for ED prevention (Levine & Smolak, 2006). O'Dea and Maloney (2000) presented a suggested application of the Health Promoting Schools framework for ED prevention in Australia, and Evans and colleagues have done the same for the Coordinated School Health Program in the United States (Evans, Roy, Beiger, Werner, & Burnett, 2008).

ED prevention programs that have sought to take this broader approach have generally done so in primary or middle-school settings. For example Healthy Schools-Healthy Kids (McVey et al., 2007) and Very Important Kids (Haines et al., 2006) aimed to prevent EDs over an entire school year in school-wide interventions that included a broad range of activities. Evaluations of these programs revealed some reduced body dissatisfaction and use of weight loss strategies among high-risk girls (McVey et al., 2007), and reductions in problematic target behaviors such as level of teasing (Haines et al., 2006). However, results were generally disappointing given the extent of these programs. While whole-school approaches are feasible, they also require substantial time, resources, and commitment to implement, and further research is necessary to determine the factors that will ensure effectiveness.

Activities to be Avoided

There have been some concerns about iatrogenic effects of some programs and materials in the area of ED prevention (Levine & Smolak, 2006). Researchers and teachers were encouraged to "First, do no harm" when designing and implementing body image programs (O'Dea, 2000), as some early approaches to teaching about these topics were found to cause unintended negative effects. Since then, meta-analyses have refuted claims of unintended negative effects of ED prevention programs (Fingeret, Warren, Cepeda-Benito, & Gleaves, 2006). Nevertheless, there have not been any direct evaluations of potentially harmful activities, and so lessons learned in early ED prevention programs have led researchers to caution against the inclusion of some activities in school-based intervention programs, such as the following (O'Dea, 2000; Yager, 2007):

- Using images of "ideal bodies" from media sources without priming with media literacy, as laboratory studies indicate that viewing these images may contribute to immediate increases in body dissatisfaction (see Chapter 29).
- Using images of those suffering from EDs, as well as guest speakers, books, or other reports of individuals who have recovered from EDs. These approaches have not been found to improve body image, or "scare people off" developing an ED, but may normalize and glamorize disordered eating, and provide suggestive information to vulnerable young people.
- Research assignments focusing on EDs, as these are often chosen by young people who are susceptible and may facilitate access to information to fuel their disorder.
- Asking students to record food intake, as this is behavior associated with strict control of diet.

- Weighing students in class, weight loss contests, and conducting fitness testing in noncontrolled environments, as all of these may induce competition, social comparison, and embarrassment or prompt strict regimes to improve scores.

The use of guest speakers has been reported as one of the most common strategies used by schools when attempting to address the issue of body image and EDs (Ricciardelli et al., 2010). Schools typically report inviting someone who has recovered from an ED to come and speak to an entire grade, or to all female students. Although this represents a convenient solution, using stories of recovered peers is generally not recommended. While some argue that the recognition of ED symptoms among self and friends is critical to secondary prevention (Noordenbos & Van Duyn, 2009), these approaches have been found to be ineffective for primary prevention. Moreover, the normalization and glamorization of dieting and EDs may lead to adoption of potentially dangerous techniques by previously healthy individuals, as demonstrated in the study by Mann, Nolen-Hoeksema, and Huang (1997).

Who Should Deliver School-Based Programs?

If the promotion of positive body image and the prevention of the development of EDs are to be widely disseminated, schools need to be involved. The school curriculum in most countries has some sort of health education requirement, such that prevention content could be delivered by members of the teaching staff. This would reduce the need for costly interventionists, with whom the students have no ongoing relationship, to enter the school and deliver separate programs.

Although delivery of prevention programs by teachers is a feasible and sustainable option, there are some issues with the translation of research to practice that have so far limited the potential for prevention activities in the health curriculum (Yager, 2010). These issues include teacher training and personal experience, as well as deviation from intended program content. Most school-based prevention programs are developed and delivered by psychologists and research staff with no educational background, with the aim of being implemented by teachers who have no experience in the use of psychological techniques. In many evaluations of prevention programs that utilize classroom teachers for program delivery, teacher training is limited or not provided at all. Even those teachers specializing in health education receive very little initial training in body image and eating problems. Those at university who are training to specialize in health and physical education have been found to have increased body dissatisfaction, and disordered eating and exercise behaviors when compared to other university undergraduates (Yager & O'Dea, 2009). This limited training and potential for negative personal experience in the area may lead teachers to developing or selecting activities that are not recommended by the latest research in the area, so their efforts may be unhelpful in promoting body image.

When teachers have been used to deliver intervention programs, researchers frequently report that teachers often deviate from program content, even if the content was very specific (Smolak & Levine, 2001). This problematic reduction in "program fidelity" is not surprising, given that teachers are generally experts in delivery of education materials, whereas the researchers who developed the content are not. Those developing programs that are to be implemented by teachers should utilize their educational expertise and discuss the suitability and flexibility of activities, as well as program length.

In our experience as both education professionals and researchers, we have come to identify several interesting observations that might assist researchers in working with schools. Teachers and researchers share the same overall aim of improving well-being among young people. However, they have very different ways of achieving these aims, so it is important that researchers consider the ways that teachers and schools work when planning a trial of intervention activities in schools. Research-oriented program developers need to be flexible in terms of the content, time taken, and order in which activities should take place. Schools do not generally have the time to commit to an 8-week prevention program, but may be able to include some activities within a relevant section or two of the health curriculum. As researchers we need to ensure that the programs that we develop and test are relevant and feasible for eventual implementation and broader dissemination. We need to focus on utilizing our expertise in order to develop prevention activities that allow teachers to use their own educational expertise in the very important school setting. We therefore suggest that researchers develop and test the efficacy and effectiveness of individual activities in terms of improving body image, so that these can be recommended to teachers and schools for inclusion in the existing health curriculum.

Conclusions and Future Directions

Recent school-based programs to improve body image and prevent EDs have generally been successful in improving the well-being of young people. Approaches that target the influence of peers and the media and are aimed at younger adolescents (12–13 years) have been effective in improving body dissatisfaction and disordered eating. The inclusion of age-appropriate engaging activities such as musicals, puppetry, and storybooks are all exciting developments in school-based prevention programming. The fact that programs are becoming broader, combining with obesity prevention and healthy living interventions to target risk factors, and utilizing the powerful roles of parents, policy, and the school environment and ethos, is another major progression in this research.

Even though there has been significant progress, some limitations in research and program design need to be addressed in order to continue to improve body image in the school setting. We must consider the needs of students, teachers, and schools. We cannot expect them to prioritize this issue and implement long and complicated programs, given the crowded curriculum, and demands of other health issues of equal importance such as suicide prevention, depression, and substance use. We also need to use our expertise as researchers to develop evidence-based activities that are derived from the 30 years of research in this area, but allow teachers to use their expertise and do what they do best in delivering this material in the way that suits their teaching style, and their students' learning and behavioral needs. Collaboration in this manner could lead to effective widespread implementation of prevention activities that result in improved well-being for young people.

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43

A Feminist Perspective on the Prevention of Eating Disorders

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The chapter starts by discussing feminist political ideology and the relationship between values, ideologies, and health promotion interventions. Three sections follow this introduction: feminist-informed risk factor research and the shaping of eating disorder (ED) prevention; a feminist-informed frame to the prevention of EDs; and a review of feminist-informed preventative interventions and their outcome. The chapter concludes with recommendations for further developments in feminist prevention theory and research.

Ideology, Values, and Health Promotion

Health promotion activities and related research are inherently anchored in values and ideologies. However, these values are rarely explicated (Raphael, 2000; Tesh, 1990). For example, health promotion interventions that focus on individual lifestyle choices (e.g., healthy eating or activity patterns) differ in values and ideologies related to social equity and social justice from those that target environmental determinants of health (e.g., access to social and economic resources). These types of interventions also differ in that the latter have the potential to focus on social transformations anchored in the critical examination of social and political structures. Thus, programs that focus on individual-level lifestyle choices do not challenge social structures, whereas programs that focus on the environmental determinants of health aim at challenging social structures (McLaren & Piran, 2012). This chapter therefore starts by briefly explicating feminist values and ideologies as guidelines to feminist practice.

Overall, feminist research and practice are shaped by feminist political analysis and scholarship that examine the complex impact of structures of power and privilege related to gender, ethnocultural heritage, social class, and other social variables on individuals' opportunities to experience social power and worth and on their self and body experiences (Piran, 2010). In addition, feminist research and practice strive for social transformations toward enhanced equity in multiple facets of the social environment. Within the domain of mental health, multiple authors have described in detail the practice of feminist therapy (L. S. Brown, 1994; Wyche & Rice, 1997) and the application of feminist principles to the treatment of EDs

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(Fallon, Katzman, & Wooley, 1994; Piran, Jasper, & Pinhas, 2004; see also Chapter 55). In contrast, feminist principles in the practice of health promotion interventions have rarely been described in any detail, and only alluded to in relationship to critical psychology (Fox & Prilleltensky, 1997) and participatory practices (Denton, Hadjukowski-Ahmed, O'Connor, & Zeytinoglu, 2001). Piran (1999a) has described a feminist model of prevention of EDs. This model is described in a later section of this chapter.

Feminist-informed Risk Factor Research and the Shaping of Prevention Programs

In line with feminist ideologies and values, feminist-informed preventative interventions need to be anchored in etiological theories and risk factor research that examine the impact of power and privilege related to gender and other intersecting social variables on individuals' self and body experience, and, in particular, on negative body image and disordered eating patterns. However, to date, most prevention programs for EDs have not been anchored in feminist etiological theories (see Chapter 19) of EDs or in risk factor research that examines social privilege in relation to body and eating symptomatology (Piran, 1995, 1997, 1999b, 2001, 2010; see also Chapter 23).

One way to demonstrate the way different ideologies shape the risk factors that, in turn, determine the focus of prevention programs is to contrast the list of risk factors that have been addressed to date in most prevention programs for EDs with the list of risk factors that would be addressed by a feminist-informed prevention program (see Table 43.1). This comparison is modeled on the table in Raphael (2000), which contrasts "tips" regarding avoidance of risk factors for poor health that are shaped by what he terms "lifestyle ideology" and that therefore tend to be viewed as being under individuals' control, versus avoidance of risk factors shaped by environmental determinants that are beyond individuals' control and that imply the need for broader social transformations. The particular items selected for Table 43.1 are informed by risk factor research and risk factor reviews to date (Piran, 2002; Smolak & Murnen, 2004; Stice, 2001, 2002), as well as by feminist-informed risk factor research (Piran, 2010; Smolak & Piran, 2012; see also Chapters 19 & 27), and are presented as examples rather than as a comprehensive list. What is important to note is that the reduction in exposure to risk factors in the "feminist risk factor list" involves environmental changes that are guided by the study of the impact of gender and other social variables on the development of self and body experiences. Inevitably, as Table 43.1 demonstrates, a feminist-informed prevention program will be different in content and process from a prevention program that focuses on changing individuals' attitudes and behaviors and that does not involve a critical social analysis.

Piran (2010) addressed this distinction in more detail, articulating a feminist critique of the ED prevention field's heavy emphasis to date on individual-level risk factors, such as dieting, internalization of thinness, negative body image, poor self-esteem, or negative affectivity (see Chapters 42, 44, & 45), and the relative lack of emphasis on the way social variables such as gender, social class, or ethno-cultural group membership *shape the social environment* of youth. This is a common issue in health-related research and interventions (McKinlay & Marceau, 1999; McLaren & Piran, 2012), since most healthcare professionals are trained to work "personally" with individuals and thus focus on individual variables in relation to health. However, as Rose (1985) suggested, targeting individual-level variables results in prevention

Table 43.1 Comparison of tips for girls and women for avoiding risk factors for eating disorders: "Individual choice/lifestyle" and "Feminist/social determinants" ideologies.

Lifestyle ideology
• Don't diet
• Follow a balanced meal plan with plenty of fruits and vegetables
• Don't buy fashion magazines or watch (much) mainstream TV
• Avoid the use of mirrors because they may enhance your self-surveillance
• Find stores that carry your clothing size in fashionable styles
• Seek early intervention for body image difficulties
• Seek early intervention for challenges with emotion regulation
• Avoid comparing your body to other women
• Avoid social groups that engage in "fat talk"
• Develop your media literacy skills
Feminist lens ideology
• Live and work in a social environment where gender equity is practiced
• Live and work in a social environment where your social power will not relate to your appearance
• Attend schools or other organizations where women's contributions are honored similarly to those of men in relation to social/political events, written documents, or artistic endeavors
• Choose to visit, and encourage family/friends to visit, museums that respect and honor women's as well as men's contributions, or work to change those that do not
• Live in a social environment that enhances your opportunity to engage in physical activities
• Live and work in a social environment that encourages women's self-care, voice, and power
• Live and work in a social environment that encourages supportive, rather than competitive, relationships between women
• Live and work in a social environment that protects you from sexualization and objectification
• Live and work in a social environment that protects you from being sexually harassed or violated
• Live and work in a social environment that supports a healthy lifestyle (e.g., access to fresh produce, lack of fast food restaurants, safety in walking and engaging in physical activities outside)
• Live and work in a social environment where you feel accepted and valued

Note. This comparison is modeled on a table in Raphael (2000) that pertains to poor health in general.

effects that are palliative and temporary (see Chapter 41). Indeed, prevention programs for EDs that have aimed at individual change in children and younger adolescents, without corresponding changes to the social environment, have tended to have a small and fading effect size on body image and disordered eating patterns (Stice, Shaw, & Marti, 2007).

Further, the exclusion of higher-level social variables, such as gender, social class, and other social variables related to social power, in etiological models of EDs, precludes an understanding of the way these higher-level variables shape the daily lived experience of individuals, including social experiences related to their bodies (Katzman & Lee, 1997; Piran & Teall, 2012; Smolak & Murnen, 2004; Smolak & Piran, 2012; see also Chapters 23, 25, & 27). In his pioneering work about the study of the ecology of human development, Bronfenbrenner (1977) summarized succinctly the importance of examining the different layers of the social environment in studying the well-being of individuals throughout human development, which he described as:

> [the] progressive accommodation, throughout the life span, between the growing human organism and the changing environments in which it actually lives and grows. The latter include not only the

> immediate settings containing the developing person but also the larger social contexts, both formal and informal, in which these settings are embedded. (p. 513)

The importance placed upon higher-level social variables as risk factors in understanding individuals' lived experience, well-being, and coping in the world is shared by public health and feminist perspectives (Austin, 2000; Piran, 2010; see also Chapter 41).

Further theoretical and research work is needed to address the link between high-level social variables, such as gender, and the way they shape social institutions at different levels (such as the media, schools, peer systems, and the family environment), and, in turn, individuals' body experiences, including body image and EDs. Even more challenging, albeit no less necessary, will be investigations of the ways in which different higher-level social variables (such as gender, social class, and ethno-cultural group membership) intersect and impact the social environment as well as individuals' body experiences (Piran & Teall, 2012). Objectification theory (Fredrickson & Roberts, 1997; McKinley & Hyde, 1996; Smolak & Murnen, 2004; see also Chapters 19, 21, & 27) and the developmental theory of embodiment (DTE; Piran & Teall, 2012; see also Chapter 27) are examples of research-based theories that aim to address the link between higher-level social variables that are amenable to change (i.e., prevention efforts) and individuals' body and self experiences among girls and women. The DTE emphasizes the ways in which experiences of embodiment (e.g., control, self-care, physical freedom, joy) and disembodiment are shaped by and transact with experiences in three domains: the physical (e.g., violations of body ownership), social-stereotypes, and social-power (e.g., experiences of prejudice).

To date, these theories have relied predominantly on cross-sectional data (Moradi & Huang, 2008; Piran & Thompson, 2008), though McKinley (2006) examined the objectified body consciousness of college- and middle- aged women longitudinally, while the developmental theory of embodiment has been validated using a 5-year prospective qualitative design with girls (Piran & Teall, 2012). Prospective studies with youth of integrated theories that link gender and other high-level social variables with the lived body experience of individuals are needed to guide prevention programs further (Smolak & Piran, 2012).

Linking high-level social variables, such as gender, with the shaping of social institutions such as schools (Sigall & Pabst, 2005) that, in turn, impact individuals' self and body experiences, reconceptualizes gender from a "fixed" factor not amenable to preventative interventions to a variable that can and should be addressed in prevention efforts (Smolak & Piran, 2012; see also Chapter 27). Further, addressing the impact of high-level social factors such as gender and social class on social institutions can address simultaneously a number of low-level risk factors that are seen as separate and as requiring different preventative interventions (Piran, 2010). For example, addressing the multiple expressions of gender inequity in a school can simultaneously reduce peer norms of harsh social comparisons of appearance among girls and cross-gender peer norms of sexual harassment, as well as experiences of disempowerment, negative affectivity, and negative body image in girls (L.M. Brown, 2003; Piran, 2001, 2010).

Indeed, McKinlay and Marceau (1999), researchers in the field of public health, consider individual-level risk factors as epiphenomena of higher-level risk factors: "Risk factors and risky behaviors are obviously manifested in individuals, but they are generated and reinforced within an ecosocial context and they are strongly related to social position" (p. 297). Therefore, within the field of ED prevention, what may seem as disparate risk factors, such as dieting, negative body image, internalization of thinness, and negative affectivity can be seen as *epiphenomena of gender or other sources of inequity* (Piran, 2010). Further, addressing inequitable social environments has

the potential to prevent other expressions of disruptions in body-self connection, such as low participation in physical activities among girls postpuberty, sexual activity without desire, and plastic surgeries (Piran, 2010). Feminist theory, being focused on the broad impact of disempowerment (see Chapter 19), is in line with a focus on general risk factors, such as physical and sexual violations, that disrupt multiple domains of self and body experiences, rather than an exclusive emphasis on what is considered "disease specific" individual-level risk factors (Piran, 2010).

For example, sexual harassment and body-based teasing are central to feminist thinking regarding the safety and empowerment of children and women, and have been found to be associated with disordered eating patterns (Harned, 2000; Piran & Thompson, 2008; Smolak & Murnen, 2004; see also Chapters 19 & 27). Yet, very few prevention programs to date have addressed gender, sexual, and other body-based harassment and teasing (Piran, 2010). Further, negative social comparisons, body policing, and fat talk among girls, as well as similar risk factors for negative body image and disordered eating, are understood within feminist analysis as related to girls' and women's disempowerment and the need to rely on body appearance to access social resources (Piran, 2001, 2010; Smolak & Murnen, 2004). Yet, repairing unhealthy peer norms among girls has not been a common goal of prevention programs.

The emphasis on the social environment in feminist perspectives also includes a recognition that factors such as gender, social class, and ethno-cultural heritage may intersect in specific contexts or communities to give unique meanings to behaviors, such as food restriction. While self-starvation in countries that idealize thinness, particularly in women, may relate to the pursuit of such an ideal, in other countries self-starvation may have a very different meaning (see Chapters 6, 7, & 23). Katzman and Lee (1997) described women in Hong Kong presenting to therapy with anorexia nervosa (AN), which related not to the pursuit of thinness, but rather to issues of social power. The complexity of the social context therefore requires feminist interventions to follow dialogical formats that allow for specific "local" meanings to emerge.

A Feminist Frame for Prevention Programs

The framework presented in this section is an extension of my previous delineation of a feminist-informed prevention in the area of EDs (Piran, 1999b). The framework is grounded in the feminist ideology explicated earlier in the chapter, the feminist therapy literature (L. S. Brown, 1994; Wyche & Rice, 1997), and the application of principles of feminist therapy to the treatment of EDs (Piran et al., 2004). Similar to feminist therapy (L. S. Brown, 1994), a feminist framework for health promotion describes key principles, within which interventions may take different forms and emphases (Piran, 1999b). Four key principles pertain to: counteracting adverse social structures and values; empowerment; the relational context of interventions; and the need for systemic interventions.

Counteracting Social Structures, Values, and Prejudices that Adversely Affect Body and Self-Image

As emphasized previously, counteracting adverse social structures, values, and prejudices that adversely impact body and self-image is an overarching principle in feminist-informed prevention. This goal, related to the content, process, and target of prevention interventions, is challenging to achieve because dominant gender-related values and structures shape all aspects of the social environment without often being explicated.

In terms of content of intervention programs, counteracting social values, mores, and prejudices requires—indeed, begins with—a critical understanding of the ways in which gender and other social variables, such as social class and ethno-cultural group membership, shape individuals' experiences of social power and, in turn, self and body experiences. As noted (Table 43.1), such a critical understanding may challenge common content themes included in prevention programs for EDs. One such example (Piran, 1995, 2010) involves the common use of magazine advertisements of thin, sexy, often partially clad female models as part of prevention programs (e.g., in media literacy). Objectification Theory (Fredrickson & Roberts, 1997), the DTE (Piran & Teall, 2012), and other feminist analyses (see, e.g., Smolak & Murnen, 2004) name the common objectification of women's bodies as a process that disrupts girls' and women's connection to their bodies. Consequently, prevention work anchored in these feminist theories will likely not include program components that may, albeit unintentionally, enhance body objectification. In this regard, Tiggemann, Slater, Bury, Hawkins, and Firth (2013) found that adding warning labels to fashion magazine advertisements, an initiative advocated in different countries towards the prevention of EDs (see Chapters 41 & 48), resulted in *increased* body dissatisfaction in women with a personality characteristic that predisposed them to engage in appearance comparisons. It therefore seems that, despite the laudable goal of enhancing media literacy and a critical approach toward the printed image, exposure to the image itself and to the warning label may still mobilize the tendency toward negative social comparisons, at least among individuals with a specific vulnerability.

Another example of the challenges inherent in the goal of creating prevention content that does not collude with dominant social mores and prejudices includes the omission in prevention programs of domains of girls' experiences that are silenced by the culture at large. These include violations of the body territory at the onset of puberty, when girls start to inhabit women's body (Piran, 2001; Piran & Teall, 2012), and the challenges girls experience in managing puberty-related changes in their body, such as menstruation or their sexual desire (Piran & Teall, 2012; Tolman, 1994). Dichotomizing foods into "good" versus "bad," as part of healthy eating education, rather than constructing eating as a caring, attuned, joyful, and social experience comprises another content area commonly included in prevention programs with children that reflects larger social values that may have adverse impact on children (Piran, 1995).

Counteracting adverse social mores, values, and structures needs to occur also in the process domain. Given that feminist ideology construes negative body and self-experiences as understandable consequences of experiences of inequity, feminist-informed prevention programs aim at empowering participants and repairing relational connections disrupted by inequitable power distribution (Piran, 1999b). These process elements tend to shift the location of expertise and action from that of program designers to that of program participants (Piran, 1999b). For example, while engaging in a feminist-informed prevention program at a dance school that involved participants' own identification of social experiences that disrupted their body experiences, the author tried at one point to use a well-designed, short media literacy video, entitled "Behind Closed Doors" (Levine, Piran, & Stoddard, 1999), that demonstrated computer air-brushing techniques and physical manipulation of models' appearance. In the open discussion that followed the video presentation, the girls (ages 10 through 14) focused on the physical vulnerability and the compromised safety of, especially teen, models; on the harsh treatment and violation of the models' bodies; and on their own experiences of compromised safety when they walked near the school. In other words, the girls' own lived experience brought forward the issue of compromised safety as disruptive to their body experiences (see Chapter 27), rather than the issue of appearance-altering computer techniques in service of the slender beauty ideal (see Chapter 45). Following this group meeting, the girls

discussed the issue of safety with school staff. As a prevention facilitator I was keenly aware that, had I focused on what experts typically emphasize as the pernicious sociocultural factors, I would have unintentionally but actively silenced these girls' knowledge and voices about a poignantly experienced adverse factor in their lives. My willingness and ability to listen to their distress regarding safety and to support their plan to discuss it with school staff served to validate their rights to feel safe in their bodies and to demand that of others in their environment (Piran, 1995, 2001). Locating expertise, strength, and the will to action within youth may be at odds with many expert-designed prevention programs.

The feminist emphasis on identifying and counteracting unhealthy social mores, values, and practices also implies that prevention programs that do not seek to change certain aspects of children's social environment run a real risk of colluding with adverse social systems (see Chapters 41 & 47). For example, some prevention programs for EDs focus exclusively on children's self-acceptance or body practices such as eating but do not address body-based teasing and harassment. If, as is all too often the case, such threats and violations are active and disruptive in children's environments, this failure to contextualize the problems and to galvanize adults and peers in the process of change runs the risk of silencing what "everyone knows is going on" and thereby colluding with adverse social structures and values.

Empowerment

Within the social context of inequity, the body becomes a site of individual vulnerability and shame for girls and women (Piran, 2001; Piran & Teall, 2012; Smolak & Murnen, 2004; see also Chapters 19 & 27). Consequently, empowerment of participants is a key aspect of feminist-informed prevention interventions designed to instill and sustain positive body and self experiences. Such empowerment takes different forms. Feminist-informed programs validate and honor children's and adolescents' own lived knowledge and the contexts that shape their lives, and consider them as experts in reflecting on and making sense of their own life experiences. Such programs therefore invite participants' reflections about, and voicing of, their own experiences, so that participants engage actively, as individuals-in-relation-to-others, in knowledge production. The link between the location of knowledge production and the location and activation of power has been repeatedly emphasized within critical psychology (Brydon-Miller, 1997; Piran, 2001). This means that participants in feminist-informed prevention programs take an active role in generating strategies to address disruptive experiences that they identify, ranging from changes to their immediate social environment, such as quality of peer relations and peer norms, to larger changes in their social systems. Piran (1999b, 2001) has highlighted the process whereby, through the voicing of body experiences and their social context, what was initially perceived by girls as individual experiences of shame about the body was transformed through focus group discussions into meaningful social experiences, shared by participants and therefore requiring transformation in their social environment. Hence the body can become a source of knowledge and power that guides social changes.

Change Occurs in the Context of Relationships

Feminist practitioners consider the quality of the relationship with clients as central to processes of change, with a particular emphasis on empowerment and the creation of an egalitarian relationship to which all participants bring their own expertise and knowledge to form a collaborative process (L. S. Brown, 1994). This collaborative, relational perspective encourages feminist practitioners to draw on their experiences as women in order to validate, amplify, and

contextualize clients' voicing of their own experiences. In contrast to developmental theories that emphasize maturation as a process of autonomy and separation, relational cultural theory emphasizes growth within relational contexts and the role of mutually enhancing relationships in nurturing individuals' empowerment, self-worth, and well-being (Miller & Striver, 1997).

Within the context of a feminist-informed prevention program, relational contexts offer multiple opportunities for change (Piran, 1999b). As emphasized previously, relational forums in prevention programs that offer attuned validation of children's experiences allow for the emergence of body-anchored material that is often silenced. Relational forums also offer the opportunity to validate participants' strengths, courage, creative thoughts, and generative strategies to address adverse situations (Cook-Cottone, 2006; Piran, 1999b, 2001). Further, facilitators, anchored in their consciousness-raising, supportive relationships, and other life experiences, have the opportunity to offer guidance in areas rarely addressed with children that affect body experience, for example, girls' challenges with owning sexual desire in a male-dominated, objectifying environment, or with staying physically active when menstruating.

Facilitators' commitment to collaborative relationships with youth also involves supporting their efforts and successes in attempting to change adverse experiences in their social environment; in this regard, facilitators utilize their social power as adults to maximize the opportunity for children's and adolescents' initiatives to succeed. The establishment of constructive peer norms and peer relationships has been found to be an important component of prevention programs (McVey, Lieberman, Voorberg, Wardrop, & Blackmore, 2003; Piran, 1999b, 2001; see also Chapters 41 & 44). In particular, implementing more constructive changes in the peer group, such as not making body-based comments and evaluations, provides participants with greater safety in the body domain, and encourages them to pursue other constructive changes in their social environment (Piran, 1999b, 2001).

Systemic Interventions

Social transformation toward equitable social conditions for women and other oppressed groups is a stated goal of feminism. Further, in line with critical social theory and feminist theory (see Chapters 19 & 27), there is a strong connection between social structures of power and individuals' experiences of their body. Foucault (1979) in particular emphasized that individual citizens in a society learn, mostly unconsciously, about their social worth and rights, through the way their bodies are treated and disciplined. He further suggested that since the body is under direct social control, individuals engage in, mainly unconsciously, practices that comply with the status quo in making the body a "docile [compliant] site" (p. 136). Since body and culture are so inextricably connected, changes in the way individuals experience and live in their bodies require systemic changes.

In a feminist-informed prevention program, inviting children to reflect about experiences in their social environment that adversely impact their body experiences, inevitably brings forth a list of such experiences. For example, in the Very Important Kids (VIK) program, students identified body-based teasing as a disruptive experience to their body image, and participated in a campaign to alter the frequency of this adverse social experience (Haines, Neumark-Sztainer, Perry, Hannan, & Levine, 2006; see also Chapter 42). In Piran's Feminist Empowerment Relational-Dance School Prevention Program (FER-DSPP; Piran, 1999b, 2001) students highlighted a range of adverse social experiences, such as disruptive physical evaluations or challenges to body safety, that led to changes in multiple aspects of the school environment, including: curricula, school staff, physical environment, school policies regarding

harassment, school committees, and peer norms. In fact, expecting children to change without a corresponding change in their social environment raises ethical concerns (Austin, 2000; Piran, 1995). For example, is it ethical to provide children with information about natural weights and set-weight theory without addressing weight teasing by peers or a school district's body mass index (BMI) report card policy (Piran, 1999b)? Similarly, program facilitators need to use their power to enhance students' success in altering their social environment; this often requires educational programs for school staff and parents (Piran, 1999b).

Feminist-Aligned and Related Prevention Programs

Feminist-Aligned Programs

Feminist-aligned prevention programs are interventions explicitly designed on a foundation of feminist ideology. These programs examine and address the lived experience of girls and boys of diverse backgrounds with a critical lens that illuminates the impact of social structures of privilege and widely sanctioned values and mores. The feminist-aligned programs reviewed here empower participants by inviting their active reflections and initiatives in transforming aspects of their social environment.

The FER-DSPP (Piran, 1999b, 2001) has been implemented at an elite dance school and evaluated using repeated all-school surveys (Piran, 1999a) as well as through an intensive qualitative case study (Piran, 2001). The program centered on female and male students' own reflections in focus groups about social forces that adversely shaped their lived body experiences at the school, and on utilizing this knowledge to transform the school environment. In line with the feminist consciousness raising (MacKinnon, 1989) and with participatory (Freire, 1970) models, these reflections generated "local" knowledge about the shaping of individual and shared body experiences at the school, and, in turn, a critical understanding of the way in which gender and other social factors shaped the school environment. Piran (2001) described the emergent knowledge from the FER-DSPP program as highlighting the roles of physical experiences that compromised students' ownership of their bodies, of constraining gender stereotypes, and of experiences of prejudice and harassment in contributing to negative body image and disordered eating patterns (Piran & Teall, 2012). Changes in the school environment included: peer relations and norms; student-staff relations; the policies, norms, and procedures of the school; the training/curriculum; school committees; staffing; and the physical setting of the school. All these changes were made to create and sustain a school environment in which the body was experienced as a site of safety, agency, and knowledge. The relational component of the program was expressed through the enhancement of peer support by adopting constructive norms, and improvement of the ongoing relationships of students and staff with the program's facilitator (Piran, 1999b, 2001).

In addition to an application of the intensive case study approach to understanding the processes associated with the prevention program (Kraemer Tebes, Kaufman, & Connell, 2003), repeated school surveys with different school cohorts suggested the program was associated with significant and sustainable reductions of body weight and shape preoccupation and EDs (Piran, 1999a). Over the course of the study, the prevalence of AN dropped tenfold and there were no new cases of bulimia nervosa (BN)—and during all this there were no

changes (or possibly a worsening) of requirements regarding thinness in the ballet world. The FER-DSPP needs further evaluation in a general school setting, which will also make it possible to utilize a controlled design.

The Full of Ourselves: Advancing Girl Power, Health, and Leadership program created by Steiner-Adair and Sjostrom has been implemented with girls in general middle-school settings, and evaluated using a controlled experimental design (Steiner-Adair & Sjostrom, 2006; Steiner-Adair et al., 2002). This program addresses a range of gender-based risk factors through active engagement of participants in discussions, art projects, and role-plays. In particular, the program seeks to shift the normative disruption of connection to the body in girls by "dialoguing" with the body via discussions about the experience of body scanning and through writing a journal to the body. It further engages girls in activities that counteract the restrictions put on girls in acting in the world. These include "bio energetic" punching and other physical exercises, as well as assertiveness training. The program discusses weight prejudice as a social justice issue, and teaches strategies to resist adverse media messages. It trains participants to be activists at home and at school. Through discussions of gender-based experiences, the program also works to enhance supportive peer relations among girls. A quasi-experimental outcome evaluation revealed that Full of Ourselves produced positive changes in participants' body image, a result not often found in primary prevention programs with middle-school-aged children; moreover, this gain was maintained at the 6-month follow-up. Improvements in eating behaviors were marginally significant (Steiner-Adair et al., 2002). Consistent with feminist theory, Steiner-Adair et al. suggested that targeted environmental changes at the school and at home could have enhanced the maintenance of gains.

Feminist practitioners and activists in the field of EDs have developed and implemented other feminist programs; however, these programs have not been systematically evaluated. The GO GIRLS!™ feminist-informed program, developed and distributed by the National Eating Disorders Association (Levine et al., 1999), generates opportunities for adult mentors to help groups of girls to develop a critical perspective toward the media, and then to utilize these literacy skills to develop social activism and advocacy (see Chapters 29 & 45). This program was repeatedly and effectively implemented by Stinson in Red Wing, Minnesota (Levine & Kelly, 2012). Sigall and Pabst (2005) have engaged in prevention activities derived from their "'Gender Literacy' model." Their program emphasizes the disruptive effect of gender inequity in education and gender identity development on girls' self-concept, body dissatisfaction, and EDs. Their intervention aims to provide a corrective education model that supports girls in feeling more powerful through, for example, increased awareness of women's history and social contributions, attention to the social context of barriers women have faced, and exposure to women role models and mentors.

The Just for Girls program, developed by Friedman (1999), aims at addressing with girls their gender-based social experiences that leave them feeling out of place, powerless, and voiceless, which, according to Friedman, leads them to express these experiences through the "language of fat." The program also encourages and facilitates girls' connections with one another and with women mentors. Along the same lines, Larkin, Rice, and Russell (1999) describe activities such as: conducting consciousness-raising groups regarding body image and body-based harassment in schools; awareness raising among students, educators, and parents; and development and implementation of policies concerning harassment and students' empowerment.

Related Prevention Programs

Other primary prevention programs with schoolchildren, while not emphasizing gender as a risk factor, have included key elements of the feminist paradigm. The aforementioned VIK program (Haines et al., 2006) utilized a participatory process with students to identify the focus of the intervention. In focus groups, students identified teasing as a central factor adversely shaping body and self-image in their lives (see Chapters 21, 27, & 31). To address teasing, the program included both individual level and social-environmental level interventions that addressed students' involvement in and responses to teasing. For example, the program included a no-teasing campaign and a theatre production (with parental attendance) that focused on changing social norms regarding teasing. Compared to a control school, at postintervention the intervention school had lower rates of weight- and appearance-related teasing. The outcome evaluation study did not involve follow-up.

Another prevention program for girls that also emphasized participatory processes was developed by Cook-Cottone and collaborators in line with their Attuned Care Model (Cook-Cottone, 2006; see also Chapter 33). While the program has a general outline that includes yoga, self-care, and life skills, a centerpiece is the arrangement of groups in which students are guided to process their constructions of learned messages and the ways in which these constructions have shaped their thoughts, feelings, and body experiences. These discussions aim to increase attunement with internal processes, as well as encourage students' active engagement with health-promoting projects, such as the writing of journals. Outcome data available to date include pretest and posttest data from three groups. Among these 45 participants, there was a significant reduction on the Body Dissatisfaction and Drive for Thinness subscales of the EDI (Scime, Cook-Cottone, Kane, & Watson, 2006).

A number of prevention programs with children have emphasized the development, through interactive processes, of a critical perspective toward cultural pressures. Neumark-Sztainer, Sherwood, Coller, and Hannan (2000) developed and evaluated a media literacy program (see Chapter 45) implemented in troops of the Girl Scouts. The program engaged troop leaders and the scouts in a critical evaluation of real and virtual body types and advertisements, in generating skits for parents about this material, and in a campaign of letter-writing to corporations to advocate for healthful products or images. A controlled outcome evaluation revealed a significant change in the internalization of thinness, as well as in the frequency of reading *Seventeen* magazine, among the intervention group that was maintained at 3-month follow-up. However, there was no change in dieting behavior. Another program for youth that incorporated a critical social perspective was O'Dea and Abraham's (2000) preventive intervention to reduce EDs by enhancing self-esteem, as discussed in Chapter 42.

Only a few programs for the prevention of EDs among children have incorporated the goal of changing the school environment as a central target of the intervention (McVey, Tweed, & Blackmore, 2007; Piran, 1999a, 1999b; see also Chapter 47). McVey et al. (2007) developed a multicomponent, all-school intervention for middle school. While this program relied on a preconstructed curriculum and newsletter for parents, it also invited students' participation in small group discussions, and supported students' initiatives in changing aspects of their social environment, such as through public service announcements and publicly placed posters. The program was associated with significant positive effects among intervention schools compared with randomly assigned controlled schools.

Conclusions and Future Directions

Worldviews shape developments in research, theory, and practice. The integration of a feminist-informed perspective with ED prevention requires investigation of gender-related risk and protective factors (see Chapters 19, 27, & 33) in the development of body experiences in children. While certain segments of gender-related social experiences have been studied and incorporated into prevention programs, for example, heightened pressures for internalization of the slender beauty ideal, the complex and multidetermined effect of gender in shaping the social environment of children and their development has not been studied sufficiently and therefore it has not been effectively incorporated into prevention programs. Consequently, newer research-based theories, such as the developmental theory of embodiment (Piran & Teall, 2012) may be combined with aspects of well-established feminist theories such as objectification theory (Fredrickson & Roberts, 1997) in order to help inform prevention programs for EDs.

Overall, feminist practitioners and researchers have been active in developing programs for the prevention of EDs with children and adolescents, viewing a strong link between social structures constituting and supporting inequity and the development of EDs. Of those programs, the few that have been systematically evaluated have shown promising results and suggest the importance of further developments and systematic evaluation of programs that embrace a feminist frame to prevention. Additional support for further such developments can be derived from positive results from prevention programs for youth in schools that have included components emphasized by a feminist approach to prevention.

More studies of this approach to prevention are needed. The challenge will be to secure funding for large-scale evaluation of a feminist approach to prevention. It took over 30 years of practice of well-articulated feminist approaches to therapy to receive large-scale funding for an outcome evaluation study, leading to a related publication (Oakley et al., 2013). The challenge inheres not only in the lack of wide acceptance of feminist ideology within the field of mental health, but also in the reliance of feminist prevention on participatory processes, rather than on a fully manualized approach. Feminist ideology examines the way in which structures of privilege and oppression shape the experience of *all* members of society. For example, the developmental theory of embodiment, grounded initially in working with girls as well as boys, would suggest that boys who have endured physical violations, who have felt constrained by stereotyped gender roles, and who have experienced prejudicial treatment related to their body characteristics or practices are more likely to develop disrupted embodiment (Piran & Teall, 2012). Understanding the shaping of experiences at the individual level through a critical lens that looks at the effect of higher-level social variables, such as gender, social class, and ethno-cultural heritage, is likely to enrich and inform current developments in the field of prevention of a range of EDs.

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Cognitive and Behavioral Approaches to the Prevention of Eating Disorders

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Over the past decade, researchers have made substantial progress toward the goal of preventing eating disorders (EDs). Many of the most promising programs fall within the broadly defined area of cognitive and behavioral (CB) interventions. Despite progress, however, we must acknowledge that, to date, only two studies have yielded what can be referred to as true "prevention" effects. Here we define "prevention" as demonstrating that a given program reduces the onset of clinically significant EDs—for example, bulimia nervosa (BN) as defined by the criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (4th edition, text revision; *DSM-IV-TR*; American Psychiatric Association, 2000)—relative to a control condition. In the absence of true prevention effects, most studies seek to reduce empirically derived risk factors for EDs. Risk factor reduction is a common strategy in both physical and mental health disease/disorder prevention, and an important intermediary research step. Ultimately, however, researchers must demonstrate that risk factor reduction does in fact reduce the actual onset (i.e., the incidence; see Chapter 41) of EDs. In this chapter, we consider a reduction in continuous measures of disordered eating as risk factor reduction because to our knowledge no prevention study has demonstrated a reduction in the onset of clinical ED diagnoses via continuous measurement of disordered eating. In addition, nonclinical levels of disordered eating are considered a risk factor for development of clinically significant EDs.

Prevention programs are commonly categorically labeled to indicate the risk status of the population for which they were developed (e.g., universal, selective) in part because different populations have differing needs (see Chapter 41). In the existing CB literature, however, some programs are deployed to mixed-risk groups, that is, groups that include both high-risk and low-risk members. As such, rather than attempt to force categorical classification on what we view as a continuous construct (i.e., level of risk in a population), we note whether the populations studied are primarily high-risk (e.g., women with elevated body dissatisfaction) or

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mixed-risk (e.g., girls with both low and high body dissatisfaction or all children at a school). To our knowledge, no programs have been developed for purely low-risk individuals.

In this chapter, we first review programs that have been found to reduce ED risk factors; included is a discussion of the rationale for this research design. We then identify which programs also have been found to actually prevent ED onset and the research challenges associated with demonstrating this outcome. Finally, we discuss implications and future research directions.

Cognitive and Behavioral Intervention Approaches: Overview

The primary aim of CB prevention is to avoid the development of clinically significant EDs by (a) shaping and reinforcing attitudes and behaviors that support a positive relationship with eating and the body, and/or (b) reducing subclinical symptoms using standard CB strategies. This typically is achieved by attempting to modify specific CB risk factors and/or activating situations. As noted by Stice (2002), interventions that focus on decreasing specific malleable risk factors for eating pathology (e.g., body dissatisfaction, negative affect) are more likely to be effective in achieving behavioral change. Programs also may target factors likely to influence primary risk factors (e.g., perfectionism; Stice 2002).

Prevention Programs: Reduction of Eating Disorder Risk Factors

Thanks to advances in risk factor research, researchers can now systematically target empirically supported, potentially modifiable risk factors in their efforts to reduce the onset of EDs. The rationale for this approach rests on the assumption that if one can sufficiently reduce risk factors for a particular condition, then onset of that condition also should be reduced. For instance, smoking is a risk factor for development of lung cancer. Thus, lung cancer prevention approaches commonly attempt to prevent onset of smoking behavior or improve quitting. Similarly, prevention for cardiovascular disease often targets risk factors such as elevated cholesterol and blood pressure or behaviors associated with diet/exercise. To date, most CB ED prevention programs have targeted risk factors such as internalization of the thin-ideal standard of female beauty (see Chapter 21), body dissatisfaction (see Chapter 22), negative affect (see Chapter 32), eating and exercise behavior, and early stage ED behaviors.

We must note that efforts to target a particular CB risk factor do not imply that other factors are not also involved in the development of EDs. For instance, it is a mistake to interpret efforts to reduce body dissatisfaction, an empirically supported ED risk factor (Jacobi & Fittig, 2010; Stice, Marti, & Durant, 2011) as negating the role of other risk factors, including those rooted in biology (see Chapter 17) and genetics (see Chapter 28). Similarly, efforts to target a single risk factor do not mean that said risk factor is implicated in all cases of EDs. For example, lung cancer prevention that targets smoking does not negate the fact that some nonsmokers develop lung cancer. It is beyond the scope of this chapter to review the extensive ED risk factor literature. Readers are referred to Chapters 22 through 34 in this volume and to Jacobi and Fittig (2010). Because programs that produce transient changes in risk factors are unlikely to yield prevention effects, our review includes only programs that have successfully reduced risk factors out to 6 months following cessation of the program in at least one trial.

Cognitive Dissonance-Based Intervention: The "Body Project"

Originally developed by Stice and colleagues, the dissonance-based approach to prevention utilizes the well-known psychological phenomenon of cognitive dissonance (Festinger, 1957). Dissonance is an uncomfortable psychological tension that occurs when an individual's beliefs and actions are misaligned. To reduce dissonance, individuals typically alter beliefs to align with behavior. Applied to ED prevention, this approach encourages participants to voluntarily speak and act against the thin-ideal standard of female beauty. Theoretically, such actions are inconsistent with previously held pro-thin-ideal beliefs, and lead participants to reduce their internalization of the thin-ideal. According to Stice's (2001) dual pathway model of BN, reductions in thin-ideal internalization should produce a cascading effect of decreased body dissatisfaction and a reduction in other associated ED risk factors such as maladaptive dieting, negative affect, and early stage ED pathology (see Chapter 21). The dissonance-based intervention has been branded and studied under a variety of names (e.g., Sorority Body Image Program, Reflections: Body Image Program®; Succeed Body Image Program); however, since it was originally published as the Body Project (Stice & Presnell, 2007; Stice, Rohde, & Shaw, 2013), we use this title.

The primary goal of the Body Project is to have participants voluntarily produce as many anti-thin-ideal statements and behaviors as possible, so as to induce dissonance. The voluntary nature of participation is established by having each participant make a verbal commitment to "give the program a try and really participate" at the start of every session. Core activities of the Body Project include:

1 Collectively defining the thin-ideal.
2 Motivational discussion of ways in which the thin-ideal negatively impacts participants.
3 Generating a list of costs of pursuing the thin-ideal.
4 Role plays that encourage participants to argue against the thin-ideal and "fat talk" (i.e., speech that reinforces the thin-ideal; Nichter, 2000).
5 A behavioral challenge task in which participants expose themselves to situations previously avoided because of body image concerns.
6 A verbal challenge task in which participants report how they would now respond to previous pressures to pursue the thin-ideal.
7 A dissonance-based mirror exposure homework task.
8 Writing a letter to an adolescent girl explaining why she should not pursue the thin-ideal.
9 Strategizing how to respond to future pressures to pursue the thin-ideal.

Many strategies will be recognized as standard CB assignments, although the dissonance focus can subtly alter activities. For instance, dissonance mirror exposure asks participants to identify what they like about themselves (both emotional and physical characteristics), as opposed to typical mirror exposure, which usually rests on either a habituation or mindfulness-based paradigm (Hildebrandt, Loeb, Troupe, & Delinsky, 2012; Luethcke, McDaniel, & Becker, 2011). The Body Project typically is delivered in 3–4 one-hour sessions (Stice, Rohde, & Shaw, 2013).

The Body Project is supported by substantial research. Preliminary studies showed the intervention significantly reduced ED risk factors in self-selected, high-risk, body-dissatisfied college women (Stice, Mazotti, Weibel, & Agras, 2000; Stice, Trost, & Chase, 2003). A subsequent large-scale (defined here as over 400 participants) randomized controlled trial (RCT) found

the Body Project produced benefits for a high-risk, mixed college and high-school sample. Results showed significant reductions in thin-ideal internalization, body dissatisfaction, dieting, negative affect, and bulimic symptoms at postintervention and 6-month follow-up. Effects were maintained for many variables at 1-year follow-up, and at 3-year follow-up the effects were maintained for body dissatisfaction, negative affect, and psychosocial impairment (Stice, Marti, Spoor, Presnell, & Shaw, 2008; Stice, Shaw, Burton, & Wade, 2006). In addition, the Body Project has repeatedly outperformed active control interventions and assessment-only groups (Becker, Smith, & Ciao, 2006; Stice, Chase, Stormer, & Appel, 2001; Stice et al., 2003, 2006, 2008; Stice, Rohde, Gau, & Shaw, 2009).

The Body Project also decreases risk factors when delivered in varied formats (e.g., with variations in mode of delivery, group facilitators, and session number). For instance, an Internet version of the Body Project (eBody Project) produced similar effects to the group version in pre-to-post effects, though replication with a larger sample and longer follow-up is needed (Stice, Rohde, Durant, & Shaw, 2012). Stice et al. also found that high-school-based facilitators implementing the Body Project produced, relative to a brochure control condition, significantly greater decreases in (a) body dissatisfaction, dieting, and ED symptoms through 1-year follow-up (2009); (b) body dissatisfaction at 2-year follow-up; and (c) ED symptoms at 3 years (Stice, Rohde, Shaw, & Gau, 2011) in high-risk girls.

The Body Project also yields positive effects in college-age women when implemented by undergraduate peer-facilitators. Becker and colleagues conducted a series of trials aimed at studying the Body Project in mixed-risk, structured social systems, notably sororities and athletics, under increasingly real-world conditions. One feature of this line of research is that, with one exception (i.e., athlete study), the Body Project was run sustainably within the relevant communities without relying on research grants.

In their first study, Becker, Smith and Ciao (2005) randomly assigned sorority members to two 2-hour sessions of the Body Project, a less interactive media advocacy intervention, or waitlist control. Relative to waitlist, both interventions yielded significant changes in maladaptive dieting, body dissatisfaction, and bulimic pathology at 1-month follow-up, although only the Body Project reduced thin-ideal internalization. Results supported the benefits of the program for both lower- and higher-risk members.

In subsequent trials, Becker and colleagues moved to a community-partnership model (Becker, Stice, Shaw, & Woda, 2009), using peer-leaders to deliver the intervention, and investigating whether or not the Body Project yielded positive results when participants were required to attend the program. Becker et al. (2006) found that the Body Project generated significantly greater reductions in thin-ideal internalization, body dissatisfaction, and dieting compared to media advocacy at 8-month follow-up, although both programs decreased bulimic pathology. In a follow-up study, only the Body Project benefitted lower-risk participants (Becker, Bull, Schaumberg, Cauble, & Franco, 2008). Results from a more recent trial (Becker, Wilson, Williams, McDaniel, & Elmquist, 2010) indicated that a peer-led *Body Project* for sorority members yielded reductions in ED risk factors out to 14 months, with largely comparable effect sizes to those found at 12-month follow-up in Stice et al. (2006).

Support was also found for a peer-led version of the Body Project that was modified to meet the unique need of a mixed-risk cohort of female athletes, although, as discussed below, the athletes and athletic staff preferred an alternate approach (Becker, McDaniel, Bull, Powell, & McIntyre, 2012). Most recently, Stice, Rohde, Durant, Shaw, and Wade (2013) investigated the peer-led approach with high-risk college females outside a structured community such as sororities or athletics. Results supported the use of peer-leaders, as long

as the script was tailored to their needs (e.g., included additional detail) and they were provided with adequate training/support.

In summary, no other program has garnered more empirical support in reducing ED risk factors in both high- and mixed-risk adolescents and young adults than the Body Project. The Body Project also has been supported by research from other labs (Green, Scott, Diyankova, Gasser, & Pederson, 2005; Matusek, Wendt, & Wiseman, 2004; Roehrig, Thompson, Brannick, & van den Berg, 2006). Lastly, preliminary research supports the underlying theory of the Body Project (Stice, Becker, & Yokum, 2013).

Behavior Modification-Based Intervention: "Healthy Weight"

Initially developed by Stice and colleagues as a placebo control for the Body Project, the Healthy Weight program yielded such positive effects in high-risk females that Stice et al. (2001) moved to studying it as an active intervention (see Chapter 24). Using traditional behavior modification strategies, social psychology principles, and interactive psychoeducation, Healthy Weight encourages participants to develop and improve skills related to healthy weight management. Theoretically, increased use of healthy weight management techniques decreases risk factors such as dieting, inappropriate exercise, and other eating pathology (e.g., bingeing and purging), thus preventing future onset of ED development (Stice et al., 2001). An additional focus of the program includes enhancing motivation for change, which is accomplished through the use of motivational interviewing (see Chapter 63) and publicly stated commitment to lasting health.

Each session includes a variety of CB techniques (see Chapters 18 & 56) and interactive discussion to increase knowledge of nutrition, physical exercise, and body image concerns. Positive behaviors are shaped and reinforced to facilitate incremental, achievable behavioral modification with the aim of producing sustained behavioral change (Stice et al., 2006). The sessions also include:

1 Exploration of the advantages of pursuing the healthy-ideal.
2 Problem-solving regarding healthy diet and exercise.
3 Behavior modification.
4 Long-term goal setting.

Homework assignments include typical CB tasks such as self-monitoring of food intake and exercise, and implementation of individualized behavior change plans.

The Healthy Weight program has substantial support, albeit less in volume than that for the Body Project. For example, in one study, the intervention produced decreases in negative affect and bulimic pathology at 6-month follow-up in high-risk college women (Stice et al., 2003). Results from Stice et al.'s (2006, 2008) long-term Body Project trial, which included Healthy Weight, indicate that this intervention produces significant reductions in ED risk factors, bulimic symptoms, and mental healthcare utilization at 1-year follow-up, and that the positive effects are maintained for thin-ideal internalization, body dissatisfaction, negative affect, and bulimic symptoms through 3-year follow-up.

Becker et al. (2010) examined whether or not Healthy Weight could be modified to allow peer facilitation. The modified Healthy Weight program, consisting of two 2-hour sessions, was compared to peer-led Body Project with 14-month follow-up. Becker and colleagues increased script content, placed greater emphasis on defining and discussing the healthy-ideal

(to reduce confounding with the thin-ideal), and refocused portions of the dietary discussion on increasing consumption of nutrient-dense foods. Results were highly consistent with previous research by Stice and colleagues (2006). Although the Body Project significantly decreased ED risk factors relative to modified Healthy Weight at postintervention, both interventions similarly reduced thin-ideal internalization, body dissatisfaction, negative affect, dieting, and bulimic pathology at 14-months follow-up (Becker et al., 2010).

Becker et al. (2012) also investigated the peer-led Healthy Weight intervention with mixed-risk female athletes. In this study, the intervention was further modified to meet the unique needs of female athletes and to provide information regarding the female athlete triad (i.e., inadequate energy availability, menstrual disorders, and osteoporosis; see Chapter 35). Results indicated that the program effectively reduced all assessed ED risk factors at 6-week follow-up. Positive results were sustained for negative affect, bulimic pathology, and shape concern at 1-year follow-up. Qualitative data from athletes and athletic staff revealed that this modified Healthy Weight program was preferred to the athlete version of the Body Project (Becker et al., 2012).

In summary, several trials provide support for the Healthy Weight program. Although the volume of research support is lower than for the Body Project, existing studies indicate that Healthy Weight generally produces similar effects to the Body Project at 1-year follow-up and beyond (see Chapter 24).

Internet-Facilitated CBT: "Student Bodies"

Student Bodies is an interactive program that utilizes concepts and exercises typical for traditional face-to-face CB treatment/prevention, but delivers all content via the Internet (see Chapter 46). The program enhances self-help bibliotherapy by providing additional features, including interactive discussions and expanded access to prevention through the use of a novel format (Winzelberg et al., 2000; Zabinski, Wilfley, Calfas, Winzelberg, & Taylor, 2004). The primary aim of Student Bodies is to reduce shape/weight concerns, unhealthy weight-regulation behavior, and binge eating. Student Bodies is delivered through a combination of psychoeducation and either synchronous or asynchronous moderated group intervention (i.e., chat rooms/message boards), typically to body-dissatisfied, high-risk university women. Sessions include the exploration of body-image- and ED-related topics, including the cultural determinants of beauty, the role of the media in creating and reinforcing societal beauty standards, the consequences of EDs and restrained eating, and healthy nutrition and exercise (Celio et al., 2000; Winzelberg et al., 2000).

Student Bodies researchers have systematically evaluated this intervention for approximately 15 years (Sinton & Taylor, 2010). Early positive results for Student Bodies included significant improvements in such risk factors as weight/shape concerns, restraint, eating concerns, and drive for thinness, with results maintained out to 4 months in the study with the longest follow-up (Celio et al., 2000; Winzelberg et al., 2000; Zabinski et al., 2004). In a subsequent large-scale multisite trial, Taylor et al. (2006) randomized 480 participants to Student Bodies or waitlist control. Results indicated that Student Bodies reduced weight concerns, global scores on the Eating Disorder Examination-Questionnaire, and drive for thinness out to 12 months.

In summary, over a decade of research supports the efficacy of Student Bodies out to 1 year in reducing body dissatisfaction and ED pathology (see Chapter 46). In comparison to the Healthy Weight program and the Body Project, one weakness in the Student Bodies literature is the lack of true independent replication.

Internet-Facilitated CBT: "Set Your Body Free" and Extension Programs

Developed by Paxton and colleagues, Set Your Body Free combines psychoeducation with interactive activities to address body image (Gollings & Paxton, 2006) in high-risk, body-dissatisfied participants (see Chapter 46). The program, like Student Bodies, seeks to overcome barriers to access, such as lack of availability of programs, participant shame, geographic distance, and cost of treatment, via use of the Internet. Set Your Body Free integrates sociocultural components and CB activities, with the goal of enhancing body image, reducing ED symptoms, and normalizing eating. It also incorporates motivational interviewing techniques to improve motivation for change.

Set Your Body Free was initially developed to prevent ED development in young adult women (see Chapter 46). The program is implemented in eight weekly, online group sessions; each session is manualized and facilitated in real time by a therapist. Core activities include:

1 Psychoeducation regarding etiology of body dissatisfaction and EDs.
2 Motivational interviewing.
3 Self-monitoring.
4 Sociocultural appearance/body image topics.
5 Strategies to counteract pressures to conform to societal standards.
6 Problem-solving for weight, shape, and eating concerns.
7 Relapse prevention.

In a preliminary study, Gollings and Paxton (2006) compared two versions (Internet vs. face-to-face) of Set Your Body Free. Results indicated that young women (aged 18–30) experienced significant improvements in body dissatisfaction, eating, and psychological variables (e.g., depression, anxiety) with results maintained at 2-month follow-up. No differences emerged between groups. This result, combined with the lack of a no-intervention control, makes interpretation of results difficult. A subsequent trial included a control group and increased follow-up to 6 months (Paxton, McLean, Gollings, Faulkner, & Wertheim, 2007). Results indicated that, relative to the control, both versions of Set Your Body Free significantly reduced body dissatisfaction, disordered eating, and depression, with effects generally maintained at follow-up. Comparisons of the two formats suggested that the face-to-face version was superior at postintervention but not at follow-up, with participants in the Internet version catching up over time (Paxton et al., 2007).

The program also has been expanded to reach high-risk, body-dissatisfied women of other ages. Set Your Body Free Midlife uses the same framework to address ED risk factors in women ages 30 through 60 (McLean, Paxton, & Wertheim, 2011; see also Chapter 36). The primary goal is to restore protective factors against body dissatisfaction (see Chapter 33) through the use of cognitive reappraisal, with a general theme of acceptance. Specific session themes include acceptance of age-related appearance changes, increased engagement in self-care and body acceptance, and importance of appearance for self-worth, all in the context of midlife. Session activities include mirror exposure, movement scheduling, and recognizing/stopping negative self-talk. Participants in the program demonstrated significantly greater improvements than waitlist controls on measures of body dissatisfaction, body attitudes, eating, and physical self-care, and maintained most improvements at 6-month follow-up (McLean et al., 2011).

Finally, My Body My Life targets adolescent girls experiencing body image issues (Heinicke, Paxton, McLean, & Wertheim, 2007). Like the other two aforementioned programs,

My Body My Life combines psychoeducation and self-help intervention activities and is delivered in six online sessions with a follow-up session after 2 months. The format is postulated to be especially helpful for young girls, as they have a greater amount of Internet exposure and may be unable to seek treatment on their own (Heinicke et al., 2007). The program incorporates information and activities about social pressures, teasing, fat talk, and social comparison that may be more relevant to young girls. Each session works to address body image and eating issues, examine motivation for change, explore topics like low self-esteem, depression, and interpersonal relationships, and build awareness and teach coping skills (Heinicke et al., 2007). Girls participating in My Body My Life experienced significantly greater postprogram improvements (relative to delayed treatment controls) in measures of body dissatisfaction and disordered eating symptoms, with improvements in disordered eating symptoms maintained through 6-month follow up (Heinicke et al., 2007).

In summary, Set Your Body Free has been found to reduce body dissatisfaction and ED pathology at post-treatment with effects generally maintained at 6-month follow-up. A strength of this line of research is the wide range of ages addressed, although longer follow-ups are needed. As with Student Bodies, a weakness in this line of research is the lack of independent replication.

School-Based CBT: "New Moves"

New Moves was developed by Neumark-Sztainer and colleagues to address weight-related problems in high-school girls (Neumark-Sztainer, Story, Hannan, Stat, & Rex, 2003). The goals of New Moves are to produce changes in physical activity and eating behaviors that can lead to weight loss or maintenance, while helping heavier/obese girls avoid unhealthy weight control behaviors, function effectively in a thin-ideal focused society, and improve self-esteem. The program is based in social-cognitive theory, and targets socioenvironmental, personal, and behavioral factors to prompt behavioral change. New Moves also incorporates the Transtheoretical Model (Prochaska & DiClemente, 1984) to assess readiness for change, and motivational interviewing (see Chapter 63) to move girls forward through the motivational stages described by the model so as to promote change in physical activity and other behaviors. The program also uses the influence of peers as a platform for support/change. Classes are restricted to overweight adolescent girls or girls who are at risk for becoming overweight in an attempt to create a positive and comfortable physical activity environment.

New Moves targets eight specific behavioral objectives (Neumark Sztainer et al., 2003):

1 be more physically active;
2 limit sedentary time;
3 increase fruit/vegetable intake;
4 limit consumption of sugar-sweetened beverages;
5 eat breakfast every day;
6 pay attention to portion sizes, hunger, and satiety;
7 avoid unhealthy weight control behaviors; and
8 focus on positive self traits.

The feasibility study for New Moves revealed no statistically significant changes in target behaviors. However, qualitative data and a process evaluation supported the need for the program and provided evidence that changes in weight-related attitudes/behaviors were moving in the right direction (Neumark Sztainer et al., 2003).

Following the feasibility study, new aspects were incorporated into New Moves, including physical education classes, nutrition, social support, empowerment sessions, individual counseling sessions, lunch meetings, and minimal parental outreach (Neumark-Sztainer et al., 2010). The updated program lasts 16 weeks, with four physical education classes per week, other sessions one day a week, and approximately five to seven individual counseling sessions. Relative to controls, participants in the updated New Moves demonstrated significant improvements in physical activity, healthy eating, goal setting, portion control behaviors, unhealthy weight control behaviors, body satisfaction, perceived athletic competence, and self-worth, which were maintained at 9-month follow-up (Neumark Sztainer et al., 2010).

Programs: Prevention of the Onset of Eating Disorders

Although the reduction of risk factors is important, few studies have yielded true prevention effects, that is, reduction in onset of ED cases relative to a control condition. Many factors contribute to this phenomenon, but a few particularly stand out. First, it is challenging to develop relatively short, engaging interventions that can yield long-term effects. Relatively short interventions are needed in prevention because few real-world prevention settings (e.g., schools) will be able to implement very intensive and time-consuming programs (see Chapter 42). Second, studies must have a no-intervention control group so that there is a population that can demonstrate the natural rate of onset of the disorder to provide a comparison for the rate of onset in the intervention group. Third, follow-up needs to be long enough to give participants the time to develop EDs. Fourth, given the relatively low base rate of EDs in the general population (see Chapter 5), sample sizes must be large. Based on the two studies that have generated a true prevention effect (see below for discussion of the studies), it appears that studies need over 100 participants per condition. Fifth, given the difficulty of determining true case status using self-report measures, studies need to use interview methods to assess ED symptoms, and not rely solely on questionnaire measures (see Chapters 38 & 40). Finally, given points one through five, researchers need substantial grant support to conduct these studies and sufficient pilot data to warrant funding. To date, only three programs have successfully produced what could be called "true prevention" effects—the Body Project, Healthy Weight, and Student Bodies (Stice et al., 2008; Taylor et al., 2006).

In the study discussed above, Stice et al. (2008) found that the Body Project and Healthy Weight significantly lowered risk for onset of EDs in high-risk populations of women with body image concerns at 3-year follow-up. The Body Project produced a 60% reduction in the number of clinical cases compared to the assessment-only control group, and Healthy Weight produced a 61% reduction in number of cases. If results hold in general deployment of these interventions, the difference would mean a reduction in 9 cases per 100 high-risk participants who completed either intervention (Stice et al., 2008). Further analysis found that bulimic pathology was most affected by the interventions, with Body Project participants experiencing a 75% reduction in future cases.

Taylor and colleagues (2006) conducted the other study that yielded prevention effects (see Chapter 46). In the 480-participant study previously mentioned, Student Bodies also significantly reduced the onset of EDs in two high-risk subgroups of participants with body image concerns: participants with an elevated body mass index (BMI) at baseline, and participants with baseline compensatory behaviors (Taylor et al., 2006). Not a single intervention

participant with elevated BMI at baseline later developed an ED. Rates in the comparable control group were 4.7% at 1-year follow-up and 11.9% at 2-year follow-up. When examining participants with baseline compensatory behaviors, only 4% of the intervention group developed EDs at 1 year and only 14.4% developed EDs at 2 years. In contrast, 16% of individuals with similar baseline levels of compensatory behaviors group developed EDs at 1 year, and 30.4% developed EDs at 2-year follow-up (Taylor et al., 2006). It should be noted, however, that this study did not produce prevention effects when the entire sample was analyzed.

Conclusions and Future Directions

Results from CB prevention trials conducted over the last 13 years show significant promise for the reduction of ED risk factors via several programs (Body Project, Healthy Weight, Student Bodies, Set Your Body Free/My Body My Life) and the prevention of EDs via three programs (Body Project, Healthy Weight, Student Bodies). The existing research also indicates that it is feasible to deliver prevention programs via community providers (Body Project, Healthy Weight), and over the Internet (eBody Project, Student Bodies, Set Your Body Free/My Body My Life; see Chapter 46).

Despite these advances, numerous weaknesses in the existing literature remain. First, with the exception of studies conducted by Becker and colleagues with the Body Project and Healthy Weight, which supported the use of these programs in specific mixed-risk female samples, all studies have targeted high-risk samples—typically girls/women with elevated body dissatisfaction. Although high-risk samples may generate larger effect sizes and thus be appealing to researchers, communities often want to deliver programs to broader populations (e.g., all girls in an after-school program, all athletes). As such, we argue that further research is warranted using mixed-risk samples. It also is time for researchers to address the prevention of EDs in male samples, even though males are at lower risk for developing EDs than females (see Chapter 37).

A related weakness is the lack of research in younger girls and older women. With limited exceptions, the overwhelming majority of research has been conducted with high-school girls and college-aged women. Although late adolescence is a peak time for the development of EDs, they can onset in both younger and older populations (see Chapters 13 & 36). As such, these populations warrant further attention.

Additionally, although research design in CB prevention has significantly improved, room for further improvement remains. For instance, more RCTs need to test for prevention effects (vs. reduction of risk factors). In addition, to better understand the maintenance of effects of programs, longer follow-ups need to be included in trial design. Dismantling studies also are needed to determine which intervention elements yield greater effects. Identifying intervention components that have larger effects could impact the development of more effective programs, and/or allow for the development of briefer interventions by eliminating less effective components. In addition, the overwhelming majority of programs target body image, in large part because body dissatisfaction is a well-identified risk factor for the development of EDs (Stice, Marti, & Durant, 2011). Many community critics of the ED prevention field, however, have expressed understandable frustration that researchers have not directly targeted a broader array of risk factors (e.g., negative affect/anxiety; perfectionism; L. Collins, personal communication, 2012), although many studies do assess some non-body-image risk factors (e.g., negative affect) to determine if they decrease as a result of body dissatisfaction programs.

Regardless, it is time to design and test interventions aimed at risk factors other than body image. Lastly, we know too little about what factors mediate and moderate response to existing programs; more research is needed in this area.

Further research also is needed to address dissemination and to study programs under increasingly ecologically valid conditions. Preliminary research supports the viability of partnering with structured communities to deploy efficacious programs. To date, however, this research largely has been limited to sororities and, to a lesser degree, athletics. We need more research to identify successful strategies for partnering with other community groups to better understand factors that will be crucial to dissemination. It also will be important to investigate the inclusion of additional community providers to research designs (e.g., parents, teachers, school counselors, coaches) and to test the feasibility of implementing programs long term under sustainable conditions (i.e., without research grant money supporting implementation). Programs that can be run only with significant financial support or that require major incentives for participants are unlikely to be viable in most settings.

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Media Literacy in the Prevention of Eating Disorders

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Media literacy (ML) aims to empower participants to adopt a critical evaluation of media content so that they can identify, analyze, challenge, and propose alternatives to stereotypical and unhealthy messages presented in the mass media (Levine, Piran, & Stoddard, 1999). In brief, the approach involves observing stereotypical presentations of beauty in the media (typically thin-ideal for women and muscular ideal for men), encouraging participants to question the accuracy and fairness of such messages, and, using the related concepts of activism and advocacy, helping participants to generate strategies to protect themselves against such messages.

The theoretical foundation for ML lies in inoculation theory, which suggests that building skills to resist social persuasion will prevent the development of health-risk behaviors. Inoculation theory suggests that, rather than psychoeducation about eating disorders (EDs), young people need to be given the skills to be critical of connections in the media between thinness/muscularity and happiness, and to resist media images of specific body types (see Chapters 29 & 43). Thus ML is thought to work in a way similar to a medical inoculation, where a person is made resistant to an attacking virus by pre-exposure to a weakened dose of the virus (McGuire, 1964). Examples of ML activities in ED prevention programs include: examining media sources for stereotypical images of beauty in women and men; questioning the accuracy of these stereotypes; "sending-up" (parodying) these stereotypes; learning about the many techniques used to alter the appearance of models both before and after photos are taken; and writing to advertising companies requesting they convey more appropriate, positive body image messages in their advertisements (Wilksch & Wade, 2009a). This chapter provides an overview of the theoretical reasons supporting the ML approach, reviews studies that have investigated the efficacy of ML, and then closes with an outline of important areas still needing to be addressed if the use of ML in ED prevention is to be justified.

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Rationale for Use of Media Literacy in the Prevention of Eating Disorders

A range of reasons have been proposed for why ML might be a useful strategy in the prevention of EDs (Levine et al., 1999; Paxton, 1999; Wilksch & Wade, 2009a). The most compelling of these include the role of the media in ED risk factor research (see Chapter 29) and a range of proposed secondary benefits, including the interactive, skill-building learning style, appropriateness for young-adolescent audiences, and possible flow-on or spill-over benefits to broader issues such as peer relationships and self-worth.

ML seeks to target media internalization, which refers to strong investment in societal ideals of size and appearance to the point that they become rigid, guiding principles (e.g., the thin-ideal for females and muscular ideal for men; see Chapters 21, 27, 29, & 37). This construct, along with peer pressure (see Chapter 31), has emerged as an important prospectively identified ED risk factor that has been found to lead to eating pathology both directly (Field, Camargo, Taylor, Berkey, & Colditz, 1999; Stice & Agras, 1998) and via body dissatisfaction, which can then lead to bulimic symptoms through dietary restraint and/or negative affect, as specified in the dual pathway model of bulimic pathology (Stice, 2001; see also Chapter 21). This prospectively supported multivariate model of how risk factors operate in conjunction with one another is of particular value to prevention researchers, given that it provides theoretical targets for simultaneously reducing a range of ED risk factors.

Further, a recent meta-analysis of 33 experimental studies of the acute effects of exposure to media images of ideal body types revealed small to moderate effects on symptoms of eating pathology, with larger effect sizes found for those participants with higher baseline levels of ED risk due to depression and body dissatisfaction (Hausenblas et al., 2013). This was the sixth published meta-analysis of the effect of the media on body image concerns, which provides an indication of the level of interest in this topic (see Chapter 29). Finally, prospective research has also found media internalization in young-adolescent females to be a significant positive predictor of overevaluation of shape and weight 12 months later (Wilksch & Wade, 2010). Overevaluation of shape and weight refers to judging oneself largely, or exclusively, in terms of shape and weight and the ability to control them (Fairburn, 2008) and it is central to cognitive-behavioral formulations of EDs (Fairburn, Cooper, & Shafran, 2003; see also Chapters 18 & 56) as well as being a diagnostic feature of both anorexia nervosa (AN) and bulimia nervosa (BN) (American Psychiatric Association, 2000; see also Chapters 8 & 9). These respective strands of risk factor research have added scientific support to the use of ML to attempt to reduce media internalization as an ED prevention strategy.

An important point here, however (and one that is sometimes misunderstood in the wider ED field), is that media internalization is not viewed by prevention researchers investigating ML approaches as *the* cause of EDs or even necessarily as the most important risk factor for EDs. In the dual pathway model (Stice, 2001) it is conceptualized as a distal rather than proximal risk factor. EDs are well known for their range of multicausal pathways and of course it is rare in clinical settings for the media to be cited as a definitive trigger for an ED. Nevertheless, what is important here is the range of secondary reasons that support the use of ML.

First, the use of ML involves a student-centered, empowering teaching style. This type of interactive approach has been found to be associated with larger effect sizes in the ED prevention field than a didactic presentation style (Stice, Shaw, & Marti, 2007). Second, and related to the first, the rationale of skill-building rather than education about EDs is also supported from meta-analyses (Stice et al., 2007), and this very much fits with the ML approach.

Third, an important consideration in any prevention work is the target audience. When it comes to terminology and classification of audience types for prevention work there remains considerable inconsistency and confusion (see Chapter 41). Current conceptualizations include three categories. *Universal* prevention includes participants regardless of their level of risk of developing an ED, such as intact school classes of girls and boys. *Selective* prevention includes participants deemed to have one or more factors that might increase the likelihood of developing an ED, such as environments that might inadvertently promote a focus on shape and weight (e.g., ballet companies or modelling agencies). Finally, *targeted* prevention includes females screened to be at high risk of eating problems (see Chapter 38), such as university-aged women found to be significantly restricting food intake at baseline.

It has been proposed that ML might be particularly well suited to younger, universal-selective audiences because analysis and discussion of the media is generally regarded as an enjoyable learning activity by young adolescents (Wilksch, Tiggemann, & Wade, 2006). In addition, those students with significant shape and weight concerns can participate and benefit from a program without being stigmatized, while those without such concerns can still have a valuable educational experience and model alternative attitudes to their peers. Further, more general benefits (e.g., improved self-esteem, reduced ineffectiveness, and improved mood) might be experienced as a result of interactive group learning activities in which participants work together to share presentations and opinions with the class. This is in contrast to many other ED risk factors (e.g., gastrointestinal problems, adverse life experiences) whose relevance would be limited to few, if any, students in a class, or where the nature of the risk factor is not well suited to classroom discussion (e.g., parental psychopathology, childhood obesity).

Fourth, it has been proposed that ML might be a particularly good means of reducing both societal focus on the importance of appearance and adherence to stereotypes or pressure to conform more generally; of course, these factors are relevant to a range of ED risk factors, including overevaluation of shape and weight. Finally, it has been suggested that ML may convey a range of additional indirect benefits, such as improving social relationships through reductions in teasing and in unhelpful comments regarding weight and appearance.

Overall, the rationale for using a ML approach to reducing ED risk factors is compelling. However, the evidence supporting this approach is less compelling, and therefore we now turn our attention to a brief review of the evidence from efficacy studies for the ML approach to ED prevention.

Review of Prevention Programs Primarily Using a Media Literacy Approach

In order to identify quantitative evaluations of ML approaches with respect to modifying ED risk factors, a literature search was conducted by using the PsycINFO database, which covers the professional and academic literature in psychology and related disciplines, including medicine, psychiatry, nursing, sociology, pharmacology, physiology and linguistics. The keywords *media literacy* (Title), OR *media literacy* (Abstract) AND *eating disorders* (Abstract), were used to locate pertinent publications in all journals in a multifield search. In addition, this search was repeated replacing "eating disorders" with "body image." Forty-six studies were listed. Fourteen studies (30.4%) met the criteria for inclusion: appearing in an English language peer-reviewed journal, providing details of data and analysis, and utilizing

one condition that primarily focused on a ML approach. The 12 investigations of unique (i.e., nonoverlapping) populations are summarized in Table 45.1.

The quality of the methodology used in these studies was judged according to criteria for empirically supported psychological therapies (Chambless & Hollon, 1998), which include: (a) a comparison to another condition, including a placebo or control condition; (b) a randomized controlled design utilizing repeated measures; (c) sample size exceeding 25 in each cell; (d) follow-up assessment greater than 10 weeks after the cessation of the program; and (e) reliable and valid measures used. Quality ratings could range from 0 to 5, with a point assigned to each criterion. Seven investigations (58%) had quality of 4 or above. The criterion most commonly adhered to was the use of reliable and valid measures of risk factors, which typically included shape and weight concern, dieting, internalization of the thin ideal, and body dissatisfaction. Given that one might strongly predict that a ML intervention would reduce or prevent internalization of the thin ideal, it was of interest to note that only three of the seven investigations using the leading measure of media internalization (Sociocultural Attitudes Towards Appearance Questionnaire-3, or SATAQ-3; Thompson, van den Berg, Roehrig, Guarda, & Heinberg, 2004) found significant ML effects for decreases on that measure. Results suggest that ML approaches have a greater impact on the more proximal risk factors related to disordered eating in Stice's (2001) dual pathway model.

In addition to their criteria indicating quality, Chambless and Hollon (1998) specify that for an intervention to be classified as "efficacious" it needs be superior to a no-intervention control group, alternative intervention group, or a placebo control group in at least two independent research settings. One example of an efficacious intervention in prevention of EDs is the dissonance-based approach (covered in detail in Chapter 44), whose curriculum is contained in the book *The Body Project* (Stice, Rohde, & Shaw, 2013). While this program does not overtly cover topics of media literacy, it does encourage a broader "cultural literacy" (see Chapter 43) in questioning the fairness of the thin-ideal and the value of its pursuit, in samples of young women with high levels of baseline ED risk factors.

In comparison to the cognitive dissonance approach, and as can be seen in Table 45.1, ML has some way to go in order to achieve the status of an efficacious treatment. Of the seven intervention studies that showed acceptable quality, only four showed ML to be superior to the comparison condition, with one showing this superiority at postintervention but not follow-up (Wade, Davidson, & O'Dea, 2003), and another showing only one significant finding out of 10 different tests (Ridolfi & Vander Wal, 2008), suggesting the possibility of Type I error. Three of the five lower quality studies supported the superiority of a ML approach, with two showing a postintervention effect only (Irving, DuPen, & Berel, 1998; Wilksch et al., 2006) related to internalization of the thin ideal, and two showing negative as well as positive effects (Choma, Foster, & Radford, 2007; Coughlin & Kalodner, 2006).

The most impressive results come from two independent research settings (one in Spain and one in Australia) described in four published studies (Espinoza, Penelo, & Raich, 2013; González, Penelo, Gutiérrez, & Raich, 2011; Wilksch, 2010; Wilksch & Wade, 2009a). It is of interest to reflect on the similarities between these two sets of studies and any differences from the other studies, that is, unique qualities of these evaluations that may explain the positive findings. First, both programs were universal (having boys and girls at varying levels of risk in the intervention), whereas only four investigations out of the 12 (33%) were universal rather than selective or targeted interventions. Second, both included 13-year-old participants, and across the interventions generally it appears that younger adolescents get more benefit than older adolescents from a ML approach. Third, both had at least 300 minutes

Table 45.1 Quantitative evaluations of media literacy programs for eating disorder prevention.

	Participants					
Study	*n*	*Age*	*Design*[a]	*Main Outcome Measures*	*QR*	*Results*
Espinoza, Penelo, & Raich, 2013; Gonzalez, Penelo, Gutiérrez, & Raich, 2011	443 girls and boys	*M* = 13.4 SD = 0.4	Compared (1) ML (300 minutes), (2) ML + nutritional education (390 minutes), (3) control; randomization at the school level; 7- and 30-month follow-up; LMM, 2 (sex) × 3 (group) × 3 (time) covarying for baseline measures	Body Image Questionnaire (Penelo, Espinoza, Portell, & Raich, 2012) Eating Attitudes Test (EAT; Garner & Garfinkel, 1979)	4.5	(1) and (2) had significantly fewer body problems, more body satisfaction, and lower disordered eating than (3) at 30-month follow-up
Raich, Portell, & Pelaez-Fernandez, 2010	288 girls	*M* = 13 SD = 0.47	Compared (1) nutritional education + criticism of feminine aesthetic beauty model (ABM) + ML, (2) ABM + ML, (3) control; MANOVA, 3 (group) × 2 (risk: early menarche, overweight, restrictive dieting, distorted attitude toward food, high influence of aesthetic model)	Change in the EAT	3	No statistically significant interactions between intervention and risk
Yager & O'Dea, 2010	170; 65% female trainee health and physical education teachers	18–34 years	Compared 12 lessons (1) control (regular didactic curriculum), (2) self-esteem + ML, (3) self-esteem + ML + cognitive dissonance; 6-month follow-up; ANCOVA for each gender, 3 (group) × 2 (time, pre- to post-measure and post- to follow-up) covarying for age	Shape and weight concern—Eating Disorders Examination Questionnaire (SWC:EDE-Q; Fairburn & Beglin, 1994) Dutch Eating Behaviours Questionnaire (DEBQ; Van Strien, Frijters, Bergers, & Defares, 1986)	4	No statistically significant interactions between intervention and time for any dependent variables

Wilksch & Wade, 2009a; Wilksch, 2010	540 girls and boys	*M* = 13 SD = 0.37	Compared eight lessons (1) ML, (2) control; randomization at class level, 6- and 30-month follow-up; LMM, 2 (group) × 2 (sex) × 3 (time) covarying for baseline measures	SWC:EDE-Q DEBQ Sociocultural Attitudes Towards Appearance Questionnaire-3 (SATAQ: Thompson et al., 2004) Body Dissatisfaction scale from the Eating Disorders Inventory (BD-EDI; Garner, Olmstead & Polivy, 1983)	5	Main effects of group favoring ML for shape and weight concern, dieting, body dissatisfaction, ineffectiveness, and depression. High-risk ML participants did better than high-risk control (higher weight and shape concern) on overevaluation of weight and shape
Wilksch, Durbridge, & Wade, 2008	137 girls	*M* = 15 SD = 0.40	Compared eight lessons (1) perfectionism, (2) ML, and (3) control; randomization at class level; 3-month follow-up; LMM, 3 (group) × 2 (risk: weight and shape concern) × 3 (time) covarying for baseline measures	SWC:EDE-Q DEBQ SATAQ Concern over Mistakes–Frost Multidimensional Perfectionism Questionnaire (Frost, Marten, Lahart & Rosenblate, 1990)	5	An interaction favoring (1) at 3-month follow-up was found for concern over mistakes; no differences favoring (2)
Ridolfi & Vander Wal, 2008	81 college females	*M* = 19.16 SD = 1.46	Compared 1-hour presentations (1) ML, (2) stress management; random assignment; 4-week follow-up; ANOVA, 2 (group) × 3 (time)	SATAQ Appearance Schemas Inventory (ASI; Cash, Melnyk, & Hrabosky, 2004) Body Shape Questionnaire (BSQ; Mazzeo, 1999)	4	Out of 10 outcome measures, one interaction at 4-week follow-up, (1) showed greater improvement in BSQ scores than (2)
Choma, Foster, & Radford, 2007	366 college females	*M* = 18.65 SD = 0.88	Watching a video (1) "Slim Hopes," (2) control (National Geographic); random assignment; one-way ANCOVA covarying for BMI (post-test only)	State self-objectification (Fredrickson, Roberts, Noll, Quinn, & Twenge, 1998) SATAQ	3	Women in (1) had higher levels of self-objectification and awareness of media messages than (2)

(*Continued*)

Table 45.1 (*Continued*)

Study	*Participants*		*Design*[a]	*Main Outcome Measures*	*QR*	*Results*
	n	*Age*				
Wilksch, Tiggemann, & Wade, 2006	237 girls and boys	*M* = 13.79 SD = 0.42	Compared one lesson of ML; ANOVA, 2 (gender) × 2 (time)	SATAQ	2	At postintervention, boys had significantly lower SATAQ-3 scores on four of the five subscales, girls had significantly lower scores on one subscale
Coughlin & Kalodner, 2006	92 college females	*M* = 19.6	Compared two sessions (1) ML and (2) control; ANOVA, 2 (group) × 2 (risk: cognitions and behaviors associated with eating disorders) × 2 (time)	Subscales of the EDI SATAQ Physical Appearance Comparisons Scale (Thompson, Fabian, Moulton, Dunn, & Altabe, 1991)	2	Significant interaction favoring high-risk participants in ML for body dissatisfaction and drive for thinness, but also increases in perfectionism and physical appearance comparisons
Wade, Davidson, & O'Dea, 2003	86 girls and boys	*M* = 13.42 SD = 0.39	Compared five lessons (1) ML, (2) self-esteem, (3) control; randomization at class level; 3-month follow-up; ANOVA, 2 (time) × 3 (group) covarying for baseline measures	EDE-Q subscales (weight concern, shape concern, dietary restraint, eating concern)	5	At postintervention, ML had lower weight concern than control
McVey & Davis, 2002	282 girls	*M* = 10.88 SD = 0.43	Compared six consecutive weeks of lessons (1) ML + promoting self-esteem + nondieting approach to health + stress management, (2) control; randomization at the school level; 6- and 12-month follow-up; ANOVA, 2 (group) × 4 (time)	Body Image subscale–Self-image questionnaire (Petersen, Schulenberg, Abramowitz, Offer, & Jarcho, 1984) EAT, child version	5	No statistically significant interactions between group and time, significant main effect of group favoring ML group for body satisfaction
Irving, DuPen, & Berel, 1998	41 girls	*M* = 15.3 SD = 0.48	Compared (1) ML (one class session), (2) control; post-test only; independent *t*-tests	BD-EDI SATAQ	2	ML had lower levels of internalization of the thin ideal than control

Note. ML= media literacy; QR=quality rating; *M* = mean; SD = standard deviation; LMM=linear mixed modeling.
[a] Pretest and post-test assessments are assumed, only follow-up is specified.

(5 hours) of lessons over the course of at least 4 weeks, which was of a greater duration and intensity than less effective approaches with younger adolescents. Fourth, both incorporated the longest follow-up periods of any of the studies (i.e., 30 months), whereas the next longest follow-up with younger participants was 12 months (McVey & Davis, 2002). Fifth, both included an extra intervention component, with the Spanish study including a nutritional awareness component, and the Australian study including a module on standing up to pressure, which targets the "pressure from others" variable in the dual pathway model. Finally, these two programs were the only ones to use robust analytic techniques for repeated observations, allowing for missing observations (a common occurrence in such settings) and covarying for baseline observations, initially equating groups to better allow for direct comparisons between groups at final assessment.

How might these characteristics serve to moderate the efficacy of ML approaches? First, research supports the usefulness of peer support in mental health for adults (Walker & Bryant, 2013), as well as the value in adolescent prevention in general of training to resist peer social pressures (McAlister, Perry, Killen, Slinkard, & Maccoby, 1980), and there may be benefit from including peers from the wider environment that perpetuates weight-based teasing and endorses the thin ideal (Austin, 2000; Paxton, 1999; see also Chapters 21 & 31). One small finding that supports this suggestion is that, compared to other interventions, use of ML was associated with higher self-esteem relating to peer-friendships at 3-month follow-up (Wade et al., 2003). Second, it is possible that the ML approach is best suited to young adolescents in a universal setting, given that older adolescents may be immune to alternative messages about the media given a longer exposure to these messages. Third, it has previously been pointed out that it is unrealistic to expect changes in participants when a program is too brief and content is limited (Austin, 2000); a certain critical amount of content may be required to effect longer term change. Fourth, the changes initiated by such interventions may take time to express themselves as critical life events trigger adaptive or maladaptive responses. Fifth, ML alone may be insufficient to exert an effect, so an effective prevention program may require some component(s) that can be integrated with the ML content and that tackles important risk factors.

Conclusions and Future Directions

In regard to the methodological rigor of ML trials, the ML field within ED prevention has made real progress over its short lifespan of approximately two decades. However, in comparison to other ED prevention approaches, it is clear the field has some way to go before more meaningful conclusions can be drawn about the overall efficacy of the approach and before we can profess a better understanding of the moderators of outcome. We now have clear support for a range of programs with young-adult women at high risk for an ED (Stice, Marti, Spoor, Presnell, & Shaw, 2008; Stice, Rohde, Shaw, & Marti, 2012; Taylor et al., 2006) with no comparable extent of empirical support in regard to the universal-selective prevention of disordered eating for young adolescents. While ML shows promise with this age group, a meaningful increase in research output is required before confident conclusions can be drawn. Schools, parents, health groups, and other organizations are seeking evidence-based programs to reduce internalization of the slender ideal, improve body image, and prevent the onset of these dreadful problems in young people. The field has an obligation to conduct the studies to provide the science to find support for the ML approach or to find better alternatives.

Reviewing the efficacy evidence for ML in the prevention of EDs reveals two somewhat opposing themes. On the positive side and generally speaking, since the turn of the 21st century there has been an overall improvement in the methodological rigor of studies, and this is true not just of ML studies but in the wider ED prevention field (see Chapters 42 & 44). Here we are referring to larger sample sizes; longer follow-up durations; inclusion of control groups; completion of all measures at all data collection points; use of reliable and valid measures; and greater adherence to principles of efficacious delivery, including the use of an interactive teaching style and the avoidance of psychoeducation about EDs. These are all positive signs in our field and directions of which we can be proud.

However, a number of aspects in the use of ML as a prevention strategy and in the prevention field in general remain in need of improvement. Four of these are outlined below.

1 The ML field needs to emulate the cognitive dissonance field (see Chapter 44) in regard to the methodical and thorough evaluation of programs and, indeed, to attention to the overall body of evidence investigating the approach.

   As highlighted above, the selective-targeted approach of cognitive dissonance now has a strong body of empirical support and, importantly, replication by a range of independent research groups (see Chapter 44). ML is a long way from this stage at the current time, and there is a real need for an increase in the overall output of work in this area. A noteworthy feature here is that it is likely that more effort needs to go into repeated evaluations of existing ML programs rather than a strong focus on the development of new programs. One aspect of this direction would be reviewing the content of such interventions, which still differ in terms of the degree of empowerment and advocacy that is included, and focusing on a more consistent approach. It would be useful to agree upon a core set of ML activities that should be included in all such trials and could include: education about digital manipulation of images; critical examination of stereotypical presentations of beauty in the media; and advocacy activities such as emailing advertising companies to agitate for change to unhealthy media messages. More broadly, replication remains an important task for the ML field.

   Another issue with regard to the methodological quality of studies relates to the choice of outcome measures. While there has been improvement in this area generally over the last decade, it is important that ML prevention researchers are using the most valid and reliable outcome measures. Further, greater consistency in the choice of measures would increase comparability of findings across studies and research groups. Shape and weight concern remains one of the most proximal and important ED risk factors (Cooper & Goodyer, 1997; The McKnight Investigators, 2003), and this includes some overlap with the body dissatisfaction construct. The most robust measure of weight concern is the Eating Disorder Examination (Fairburn & Cooper, 1993; see also Chapters 9, 38, & 40), and therefore at a minimum the Questionnaire version should be used (Fairburn & Beglin, 1994). A range of measures of body dissatisfaction exist (Cash, Phillips, Santos, & Hrabosky, 2004), and this is reflected in the choice of instruments used in the efficacy studies. It would be helpful to select one valid measure of body dissatisfaction that can be used across studies. Given that the Eating Disorder Inventory (EDI-3; Clausen, Rosenvinge, Friborg, & Rokkedal, 2011) appears to be the most commonly used in the studies to date, we recommend this continue to be used, with the proviso that adaptations of the body dissatisfaction and drive for muscularity subscales of the EDI for boys are utilised (Hallsworth, Wade, & Tiggemann, 2005). Lastly, it is critical that media internalization be measured in all ML trials, because

this construct is the key theoretical target of ML. Current evidence (Wilksch & Wade, 2012) suggests the SATAQ-3 (Thompson et al., 2004) is the best option and, indeed, this measure has been used in 7 of the 12 ML interventions outlined in Table 45.1.

A final important direction for ML researchers is that as *efficacy* trials show promising findings, it is important that *effectiveness* trials then be conducted. While efficacy trials investigate an intervention's effect under well-controlled experimental conditions, effectiveness trials investigate how an intervention performs in applied, real-world settings where a particularly important question is how well the program performs when delivered by nonspecialist presenters (e.g., teachers). Such trials are endorsed by the Medical Research Council (2000) as the critical next step in disseminating research following efficacy randomized-controlled trials, and to date there is only one such study in the media literacy field (Wilksch, 2015). This is again an area where the field needs to emulate the work of cognitive dissonance and targeted prevention researchers.

2 We need to investigate moderators of outcome to establish which audiences will benefit most from the ML approach.

At this time we cannot confidently state which audience age groups (e.g., preadolescent vs. young adolescent vs. young adult), gender breakdown (e.g., girls-only vs. mixed-gender vs. boys-only), or level of ED risk (e.g., universal, selective, or targeted) are most suited to the ML approach. The most promising studies currently support the use of ML with universal audiences of young-adolescent age involving both girls and boys (González et al., 2011; Wilksch & Wade, 2009a). However, the absence of comparable high-quality studies with sufficient follow-up and sample size means we do not currently know if other audiences might also benefit from the ML approach. Again, we need more studies to be able to answer the question of moderators in more scientific manner.

As is the case for most ED risk factor research, we know far more about how media internalization operates in girls rather than boys. More investigations into whether the dual pathway runs in a similar manner for males as females would be of significant value to both the risk factor and prevention field (see Chapter 37).

3 ML prevention researchers need to decide whether their goal is to reduce ED risk factors or to also prevent onset of clinical and subclinical ED cases.

An uncomfortable truth for ML prevention researchers is that the actual impact of the approach on preventing clinical and subclinical cases of EDs has not been adequately investigated. Interestingly, this is a shortcoming that applied to the ED prevention field in general until recent years, although there has been a strong growth in the number of high-quality targeted prevention trials that have now investigated this (Stice et al., 2008; Stice, Rohde, Shaw & Gau, 2011; Taylor et al., 2006; see also Chapter 44). To date this has not extended to the ML approach because the higher quality ML studies have been conducted with young adolescent samples, in which there are more barriers to measurement of such behaviors and cognitions than there are in working with young adult samples. Younger participants need to be followed-up for longer periods of time to reveal actual effects on the true likelihood of developing an ED. In addition, some schools and parents have concerns about the possible iatrogenic effects (see Chapter 42) of the completion of such measures by students who were previously not displaying such behaviors, despite provision of evidence suggesting no such possible harmful effects have been found from completing such measures (Celio, Bryson, Killen, & Taylor, 2003).

Notwithstanding the above, it is also being increasingly recognized that, while preventing clinical and subclinical ED cases is critical, established ED risk factors such as body

dissatisfaction, overevaluation of shape and weight, negative affect, and dieting do convey their own level of suffering (Stice, Rohde, Gau, & Shaw, 2012; see also Chapter 41). Consequently, significant effects on these variables should also be valued as highly beneficial outcomes in their own right. Both the reduction of risk factors and the prevention of clinical cases are important goals, and as such, both need to be pursued in future studies.

4 ML researchers need to form greater collaborations with other relevant research groups and organizations.

It would be useful for existing ML researchers to collaborate with each other to directly compare existing programs in the same multisite research trial. This would increase both the internal and external validity of such studies and provide useful information regarding which program is best suited to particular audience demographics. ML researchers would also benefit greatly from forming research partnerships with other groups who currently focus on dissonance or other strategies and have not to date been involved in evaluations of ML approaches. This could benefit efforts to improve the methodological rigor of ML trials, which could have flow-on benefits that eventually strengthen the case for evidence-based ML programs to be adopted as part of health and education policies in schools.

Further, greater collaboration with obesity prevention experts is also likely to be useful (see Chapter 49). This is not a new suggestion (Austin, 2011; Neumark-Sztainer et al., 2006) and there are numerous reasons for taking a combined approach, including: obesity as a risk factor for disordered eating (Pearson, Goldklang, & Striegel-Moore, 2002); the finding that those with disordered eating are more likely to gain weight over time (Stice, Cameron, Killen, Hayward & Taylor, 1999); the risk of confusing messages to young people if they participate in separate ED and obesity prevention programs (Wilksch & Wade, 2009b; see also Chapter 47); a desire to avoid potential inadvertent harm to the other problem (Neumark-Sztainer et al., 2006); and limited space in school curricula for health promotion programs where increased efficiency of programs would be appealing (see Chapter 42). However, the most important reason is the evidence that there is overlap in the risk factors for both problems. Specifically, risk factors such as media usage, dieting, body dissatisfaction, depressive symptoms, perfectionism, shorter sleep duration, social problems, and difficulties with emotion regulation have been found to increase the risk of both disordered eating and weight gain (Allen, Byrne, Forbes, & Oddy, 2009; Haines, Neumark-Sztainer, Eisenberg, & Hannan, 2006; Haines, Neumark-Sztainer, Wall, & Story, 2007; Neumark-Sztainer et al., 2007; Stice, Presnell, Shaw, & Rohde, 2005). Given the evidence from some ML prevention trials that one or more of these risk factors can be significantly reduced, it would be valuable to conduct trials evaluating outcomes related to both EDs and obesity. Although some researchers have commenced doing this (Wilksch & Wade, 2013), this is again a field where cognitive dissonance researchers are leading the way (Stice et al., 2008; see also Chapters 24 & 44).

Finally, a recent development highlighting the urgent need to improve the scientific quality of ML research and universal-selective ED prevention more generally is the increase in corporate efforts to improve body image (see Chapter 48). For example, some large multinational companies have included messages promoting self-esteem as part of their advertising campaigns and have sponsored body image programs in schools and at other sites. While it is commendable that such organizations have taken an interest in this important area, and it represents a valuable opportunity given these organizations will likely have considerable resources to support wide-scale roll-out, a disquieting trend is for very wide-scale dissemination to occur prior to or without published evaluation

(Richardson, Paxton, & Thomson, 2009). ML prevention researchers should view this as a call-to-action to complete quality evaluations of such programs and thus assist corporations with either improving their current program or replacing with a more empirically supported option. This is a real opportunity for our field and needs to be addressed urgently.

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Computer-Assisted Approaches to Prevention

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In recent years the field of "e-mental health" ("e" for electronic) has expanded rapidly and the provision of mental health services via technology has become increasingly common. The questions surrounding the specific ways interventions based on information and communication technologies may advance mental health care have been discussed extensively in the literature (Kazdin & Blase, 2011; Simon & Ludman, 2009). In the field of eating disorders (EDs), a number of interventions have been introduced to this end. These include approaches for prevention, guided and unguided self-help, treatment support, and relapse prevention (Aardoom, Dingemanns, Spinhoven, & van Furth, 2013; Bauer & Moessner, 2013; see also Chapters 44 & 47).

In this chapter, after presenting the potential advantages of technology-based prevention, we then review the currently available literature on computer-assisted preventive approaches to EDs. In light of that evidence base, we then discuss directions for future development and research.

Advantages of Internet Technology in Prevention

Using technology, especially the Internet, to deliver preventive approaches appears promising for several reasons. These include the fact that such programs can easily be made available to large target populations and that they can be accessed at any time from anywhere with various devices (e.g., computers, laptops, smartphones). Internet technology allows service providers to deliver programs in a standardized way and to manage the delivery from a distance, that is, such technology does not require recruitment, training, and supervision of providers in each single location where the intervention is conducted (e.g., high schools or colleges).

Furthermore, technology-enhanced interventions allow for more flexibility compared to traditional approaches in which, typically, each participant would receive the same type, intensity, and duration of a prevention program at one specific point in time. In contrast, in Internet-based approaches, subgroups of participants may be offered specific modules of an intervention (e.g., based on their risk profiles, as determined by a valid online screening tool) and the content and dose of the prevention intervention may be adapted continuously based on individuals'

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needs throughout the course of participation. Such flexible approaches acknowledge that, despite all the advances in prevention and risk factor research over the past decades, our ability to reliably predict which individual will develop an ED at which point in time remains very limited. As long as the exact causes and risk factors of EDs and their interplay are unknown, the targeting and timing of preventive efforts will remain a challenge (Bulik, 2013). Technology-enhanced interventions may address this challenge by enabling us to better match programs to participants' individual needs over time rather than assigning the same fixed-intervention approach to all participants.

Another, yet related, advantage of Internet-based prevention programs is that they may facilitate the transition from prevention to early intervention and ensure continuity of care when a preventive program is not sufficient and individuals develop manifest EDs despite their participation in a prevention program. We know that only a minority of individuals who develop an ED seek and receive timely and adequate professional healthcare (Hart, Granillo, Jorm, & Paxton, 2011). At the same time, we know that early diagnosis and timely treatment are associated with better outcome and a better long-term prognosis (Keel, Mitchell, Miller, Davis, & Crow, 1999; Treasure & Russell, 2011; see also Chapter 55). When preventive efforts fail and an individual develops an ED, we should thus aim for early intervention that facilitates access to curative treatments (McGorry, 2013). Internet-based preventive approaches provide the opportunity to incorporate components that allow for the timely detection of illness onset and for the reduction of some of the barriers that impede and delay seeking professional help. These barriers include uncertainties, shame, stigmatization, and limited knowledge about ED symptoms and treatment options, that is, a lack of mental health literacy (Mond et al., 2010; Roehrig & McLean, 2010). Research has shown that many individuals consider EDs to be "bad habits" or "misguided choices" rather than severe conditions that require specialized treatment; in fact, compared to other illnesses, these stigmatizing attitudes are more prevalent with respect to EDs (Ebneter & Latner, 2013).

Technology-enhanced programs may enable us to facilitate the transition from prevention to early intervention by providing low-threshold, anonymous information and support tools and by applying automated screening and assessment procedures (see Chapter 38) that allow us to monitor ED-related risk factors, attitudes, and behaviors longitudinally. Based on each person's development over time, we may flexibly recommend specific levels of care to individual participants, for example, encouraging them to seek face-to-face professional support if needed (Bauer & Moessner, 2012).

In sum, it is assumed that technology may allow us to efficiently deliver ED prevention programs to large, diverse, and underserved populations, to overcome some of the limitations of traditional preventive approaches, and to better connect the care sectors of prevention and early intervention.

Computer-Assisted Approaches to Prevention of Eating Disorders

"Student Bodies"

Currently, the best-known and best-studied prevention program based on technology is Student Bodies, developed at Stanford University (California) by C. Barr Taylor and colleagues in the 1990s (Winzelberg et al., 1998; see also Chapter 44). Student Bodies is an Internet-based structured 8-week program based primarily on a cognitive-behavioral approach (see Chapters 18 & 56). The intervention consists of weekly psychoeducational readings on

various topics (e.g., body dissatisfaction, weight and shape concerns, excessive exercising, and nutrition), cognitive-behavioral exercises, weekly personal writings in an online body image journal, and participation in an asynchronous, moderated online discussion group. As part of the discussion group, participants are expected to post statements about their weekly readings and exercises to a message board and to regularly reply to other participants' postings. The target group of Student Bodies is college-age women identified as being at risk of developing EDs based on an initial assessment using the Weight Concerns Scale, which is a reliable and valid screening instrument (Killen et al., 1996).

Evaluations of Student Bodies in several studies in the United States and Germany have indicated that the intervention has significant positive short-term effects on body dissatisfaction, weight and shape concerns, and problematic eating behaviors compared with waitlist control groups (Jacobi et al., 2007; Winzelberg et al., 2000; Zabinski et al., 2001). A modified version of the program, including a synchronous, interactive, and moderated support group (based on Internet chat technology), generated somewhat more favorable effects at follow-up than the original version of the program. The authors suggest that more interactive, synchronous communication allows for better and more timely tailoring of responses to specific needs of participants, which might lead to improved outcome (Zabinski, Wilfley, Calfas, Winzelberg, & Taylor, 2004).

In order to evaluate the longer-term effects of Student Bodies on prevention of illness onset, Taylor et al. (2006) conducted a rigorous randomized controlled trial with 480 at-risk college students. Participants were randomized to the Internet-based prevention program or a control group that had the opportunity to participate in Student Bodies at the end of the follow-up period. Follow-up assessments were conducted over a period of up to 3 years. In the total sample, survival analyses indicated no significant difference in illness onset rates between the intervention group and the control group (with approximately 9% of study participants developing an ED over the course of follow-up). However, the results confirmed the previously reported significant reductions of weight and shape concerns in the intervention compared to the control group. Furthermore, effects of the program in terms of prevention of illness onset were identified in two subgroups. Specifically, participation in Student Bodies was associated with the onset of fewer clinical and subclinical EDs in participants who either showed an elevated body mass index (BMI) or profound compensatory behaviors at study entry (Taylor et al., 2006).

Recently, two additional versions of Student Bodies have been introduced by researchers in Dresden, Germany. "Student Bodies +" is an adapted version of the original program that aims at targeted prevention of EDs in women who experience manifest symptoms or even a subclinical version of the illness (Völker, Jacobi, & Taylor, 2012). In a randomized controlled trial with 126 female participants, this program proved to be superior at 6-month follow-up to a waitlist condition in terms of improvements of ED-related attitudes, reductions in binge-eating and purging episodes, and overall abstinence from ED behaviors (Jacobi, Völker, Trockel, & Taylor, 2012).

The second adaptation is "Student Bodies-AN." It specifically targets women who are considered at risk of developing anorexia nervosa (AN), that is, those who are underweight and report restrictive eating. In addition to adaptations in content to focus on those risk factors, the program was expanded from 8 to 10 weekly sessions for this target group. A recent pilot study showed that Student Bodies-AN is feasible and well accepted by women who experience weight and shape concerns and have a low BMI and/or restrictive eating behaviors (Ohlmer, Jacobi, & Taylor, 2013).

"Food, Mood, and Attitude"

Whereas Student Bodies was specifically developed for at-risk populations, another computer-based approach, Food, Mood, and Attitude (FMA), was designed to integrate universal and selective prevention (see Chapter 41) by targeting individuals at high risk for developing EDs as well as those at low risk (Franko et al., 2005). FMA, created in the United States (Boston), is a 2-hour CD-ROM-based, unguided (i.e., not moderated) program designed to educate participants and improve their skills with respect to different risk factor areas (e.g., pressure to be thin, thin-ideal internalization, body dissatisfaction; see Chapters 21 & 45). Ideas from multiple theoretical approaches and prevention theories are incorporated into the didactic information materials (presented in text, voiceover, or video), as well as the interactive tools and exercises. FMA is a structured program that requires participants to complete one module after the next without skipping any content.

The program was evaluated in a randomized controlled trial with 240 female students. Compared to a control group, high- and low-risk participants in the FMA program increased their knowledge of risk factors and EDs. Furthermore, in the at-risk group, participation in FMA resulted in improvements with respect to weight and shape concerns, as well as a decrease of the internalization of negative social and cultural attitudes toward appearances at 3-month follow-up (Franko et al., 2005).

"Set Your Body Free"

In contrast to Student Bodies and FMA, which were developed initially as computer-based interventions, other programs exist as both face-to-face and technology-enhanced versions of the same intervention. For example, the Australian (Melbourne) targeted prevention program Set Your Body Free (Gollings & Paxton, 2006; see also Chapter 44) was designed to address body dissatisfaction and problematic eating behaviors in a face-to-face setting. Set Your Body Free is a group-based, 8-week, therapist-led intervention. In the Internet-delivered version, participants meet with a clinician in a chat room for the weekly group sessions. In addition, they have the opportunity to post statements to a message board in between sessions; thus, the program combines synchronous and asynchronous ways of communication.

In a randomized controlled trial including a 6-month follow-up, the effects of the face-to-face version and the Internet-based version of Set Your Body Free were assessed relative to a control condition. The sample included 116 women aged 18 to 35 years with high levels of body dissatisfaction (assessed with the Body Shape Questionnaire: Cooper, Taylor, Cooper, & Fairburn, 1987; and the Bulimia Test-revised: Thelen, Farmer, Wonderlich, & Smith, 1991; see Chapter 38). The results showed that both versions of the intervention program were superior to the control condition, with slightly stronger effects in the face-to-face condition (Paxton, McLean, Gollings, Faulkner, & Wertheim, 2007). However, a larger sample is needed to investigate the equivalence of both delivery modes.

"Body Project"

Another face-to-face intervention for targeted prevention that has recently been adapted for delivery via the Internet is the dissonance-based prevention program called the Body Project. This program was created by Stice and colleagues in the United States (Oregon; Stice, Rohde, & Shaw, 2013). Research over the past decade has demonstrated that the 4-week face-to-face

version of Body Project is efficacious and effective in reducing ED risk factors and preventing illness onset in young females at risk for development of EDs (Stice, Marti, Spoor, Presnell, & Shaw, 2008; Stice, Rohde, Shaw, & Gau, 2011; see also Chapter 44).

The online version of the intervention (eBody Project) contains content similar to the face-to-face version and consists of six sessions. Stice, Rohde, Durant, and Shaw (2012) compared the effects of the online version with those of the face-to-face group version and two control conditions (educational video and educational brochure) in a randomized controlled trial with 107 college-aged women (M = 21.6 years, SD = 6.6) who reported body image concerns during a telephone interview. The results showed greater improvements (pre-post) with respect to ED risk factors and symptoms in the two experimental conditions (group condition and online condition) compared to the two control conditions. Similar effect sizes were obtained via online and face-to-face group delivery of the intervention. However, given the modest sample size in these two experimental conditions (n = 58), these findings should be considered preliminary, and more research is needed to determine the potential of eBody Project.

Individually Tailored Prevention and Early Intervention: The Heidelberg Approach

"Essprit"

In the approaches described above, all participants receive the same intervention package, that is, they are expected to complete the intervention in a predefined timeframe (e.g., 8 weeks) following a mostly standardized procedure. This is not the case for the program Essprit, whose title is an acronym for "**Ess**stoerungs**pr**aevention ueber das **In**t**e**rnet" (Eating disorder prevention via the Internet). The development in Germany (Heidelberg) of this program was based on the assumption that, because participants' needs with respect to intensity and duration of an intervention vary largely, a more flexible and individualized approach may better fit their needs. Therefore, Essprit does not require participants to use specific components or to complete specific modules. Moreover, it does not foresee a minimum or maximum intensity of use or a predefined frequency or duration of participation, but rather seeks to support more flexibility depending on participants' needs and preferences (Bauer, Moessner, Wolf, Haug, & Kordy, 2009). Essprit includes psychoeducational components as well as online screening, monitoring tools for tracking relevant factors (e.g., ED-related risk factors and behaviors), and provision of tailored feedback to participants. Additional support tools include online forums and chat modules. Two studies established the feasibility and acceptance of the program in college-age participants in Germany (Bauer et al., 2009) and Ireland (Lindenberg, Moessner, McLoughlin, Harney, & Bauer, 2011).

Based on this initial research, the program was adapted for younger age groups (YoungEssprit). In a recently completed randomized controlled trial, the developers investigated the efficacy of YoungEssprit in reducing the onset of self-reported ED symptoms in a sample of 1,667 German high school students (58% female; M = 13.78 years, SD = 1.20) who entered the study in two separate recruitment waves (n = 896 and n = 771). The findings were encouraging but to some extent inconclusive. Results from the first recruitment wave indicated a positive effect of the intervention. Within 12 months, the onset of ED-related symptoms was significantly lower in the intervention group (5.9%) than in the control group (9.6%). However, this difference could not be confirmed in the second subsample. Findings with respect to

uptake of the intervention were positive (98% of participants used the psychoeducational module and about 80% used at least one additional module), with approximately 75% reporting high satisfaction with the program (Lindenberg & Kordy, 2015).

"ProYouth"

The potential of an enhanced version of the programs Essprit and YoungEssprit is currently being investigated as part of a European Commission initiative entitled "ProYouth," which seeks to promote the mental health of young people by disseminating an Internet-based platform integrating prevention, early detection, and timely intervention in case of ED-related impairment. Building on Essprit and YoungEssprit, the ProYouth information and support platform offers young people easy and anonymous access to information materials and to online support tools in order to prevent development of EDs and to facilitate access to professional care if prevention is not enough. The platform is accessible through the website www.proyouth.eu. It was developed in six languages (German, Irish, Italian, Czech, Hungarian, Romanian) and can be accessed via various devices that allow users to connect to the Internet (e.g., computer, laptop, smartphone).

The ProYouth initiative has been cofinanced by the European Commission for a period of three years (2011–2014), which allowed the teams in the various countries to offer the program at no cost to participants. Recent analyses based on a German dataset show that the cost of providing the program amounts to approximately 15 euros (approximately 18 U.S. dollars in early 2015) per participant per year if 1,000 individuals are registered for participation (Minarik, Moessner, Oezer, & Bauer, 2013). This amount includes expenses for both technical maintenance (e.g., hardware maintenance, software updates) and provision of the online support (e.g., staff time to provide chat sessions) based on German salary levels. Most teams succeeded in implementing the program in a sustained way, indicating that the program will be available to participants at no cost beyond the initial funding period. This was achieved through (a) collaborations with various parties, such as student counseling centers or networks of school psychologists that provide the online support (i.e., they extend their regular face-to-face service by offering online services through ProYouth); and (b) utilization of one joint technological environment (i.e., server infrastructure) provided by the Heidelberg team, which limits the cost of the program in other countries.

As noted above, the main difference between this approach and other computer-assisted approaches to ED prevention is that it is not a fixed-intervention approach. Similar to the FMA program described above, ProYouth integrates universal and selective prevention and allows participants with and without an elevated risk for developing EDs to join. However, based on their specific risk profiles assessed during an initial screening procedure, and based on their development over time as assessed through the monitoring module, participants receive feedback on the level of support that might be most appropriate for them (see below).

One of the main objectives of the ProYouth initiative is implementation and dissemination of the online platform in various target regions. However, given that the program is freely available on the Internet, access is not limited to specific populations. Individuals who access the platform are informed about general issues related to data collection, data protection, and data security via the disclaimer on the website. As soon as they register for participation in the program they have to consent to the terms and conditions of use (by clicking a check box) informing them about issues related to anonymity and confidentiality. Those terms and conditions have been adapted based on national and legal regulations in the respective countries.

Participants are encouraged to choose a username that is different from their real name, and they are informed that no conclusions about their identity may be drawn unless they provide such information via their username, email address, or postings on the page. Furthermore, they are informed that any information they provide as part of their participation is kept confidential, and that, once again, data protection, data security, and emergency measures follow the latest recommendations and national guidelines (country-specific details are provided if available). Specifically, the ProYouth platform contains four modules:

Didactic information about mental health, eating disorders, and treatment. The platform provides comprehensive evidence-based information on the various EDs, typical symptoms, risk factors, and early signs of the illness. In addition, the platform informs participants about characteristics of the mental health care system in their respective country and about treatment options (including links to online search engines for clinical experts in the field of EDs, if available). Besides static information materials, moderators of the platform upload information (e.g., links to newspaper articles or online resources) to a "News" section on a regular basis. Overall, this module is designed to improve participants' psychoeducation, mental health literacy, and help-seeking skills, and to counteract stigma around EDs.

Screening and monitoring. Prior to registering for participation, individuals need to complete an online screening questionnaire including the Weight Concerns Scale (Killen et al., 1996; see also Chapter 38) and the Short Evaluation of Eating Disorders (Bauer, Winn, Schmidt, & Kordy, 2005) in order to assess ED-related risk factors, attitudes, and behaviors. Once they complete the screening questionnaire, participants automatically receive a feedback message. Depending on the entries, the feedback message directs individuals to register for participation in ProYouth and to use the available online tools, for example, in case they report an increased risk for EDs or slight symptoms of the illness. The feedback message in this case may read as follows:

> Thank you for completing the self-test. According to your entries you are more concerned about your weight and shape than other young people. In the longer term such concerns may have a negative impact on your eating behavior which might ultimately result in physical or mental problems or symptoms of an eating disorder. We recommend you to register for participation in ProYouth and take advantage of our various online modules. These include information materials, continuous monitoring of relevant attitudes and behaviors, peer discussions with other participants and student moderators, and chat consultations with experts. After registration, it is totally up to you to decide which modules of ProYouth you wish to use. Your participation is anonymous and free of cost. Please note: ProYouth provides information and allows you to talk to peers and online counselors via a secure online platform. However, participation in ProYouth cannot replace clinical diagnosis or treatment by healthcare professionals.

In addition to the feedback message, individuals receive instructions on how to interpret the message. They are informed that this is an automated message based on their entries in the screening questionnaire, so the message can provide only a rough evaluation of their attitudes and behaviors. Furthermore, they are informed that the screening results do not allow for predictions on an individual level (i.e., that the results do not allow for definite conclusions that an individual will develop an ED in the future), but that the probability of developing ED-related impairment may be increased. They are reminded that a self-test cannot be used for diagnosis, and, finally, they are encouraged to ask specific questions they may have on their feedback message via the contact form on the ProYouth website.

If severe impairment is identified in the screening questionnaire, the feedback message recommends participants to consider seeking face-to-face professional support. Furthermore, the message informs them that they may still consider registering on the platform, for example, in order to talk to experts online, to clarify questions or uncertainties, and/or to seek peer support. Participants whose screening questionnaire responses do not indicate any impairment related to risk factors or ED symptoms receive a feedback message stating that, based on their entries, there is no need for them to participate in such an intervention. However, they are still welcome to register if they wish to search for information or for support for themselves or others. Each feedback message contains an explicit statement that, of course, participation in ProYouth cannot replace clinical diagnosis or treatment.

Following completion of the screening, individuals can decide whether or not they wish to register for participation in the online program. Once they have activated their account, they receive access to all modules of the platform. As part of their participation, they are registered for the monitoring module, which is one of the key components of the platform. Throughout their participation they regularly receive emails, including a link to a brief questionnaire assessing attitudes, behaviors, and development over time. Following each completion of the monitoring questionnaire, participants receive a supportive feedback message commenting on their level of impairment and on positive and negative changes. The monitoring module aims to strengthen participants' self-management and self-care skills, for example, by encouraging them to seek more intense support in case of increasing symptoms and thus instructing them to take an active role in the management of their ED-related problems. In addition, this module supports the work of the online counselors by alerting them as soon as a participant's monitoring entries exceed certain cut-offs indicating significant impairment. In each of these cases, the online counselor would then contact the individual participant (see below). This combination of automated and personalized processes allows for inclusion of large samples of participants despite limited personal and professional resources.

Peer and professional support. Similar to the online prevention programs described previously, ProYouth provides online forums that allow participants to interact with each other, discuss various topics, ask questions, and provide support to each other. On a daily basis, moderators (e.g., trained psychology graduate students) read new postings, reply to questions, and delete inappropriate postings in order to maintain a positive communication atmosphere. Moderators may also initiate new topics to stimulate discussions among participants.

In addition to the forums, participants can communicate with each other in 60-minute chat sessions that are conducted at regular intervals in a group setting led by an online counselor. Participants who wish to talk to a counselor on a one-to-one basis may book an individual 30-minute chat session from a list of available appointments in order to discuss more personal questions or concerns. Moderators and counselors may also actively encourage participants to book such sessions if a participant reports severe symptoms in the forum, group chat, or monitoring module. In these cases, the online counselors contact the participants via a personal message explaining the reason why they would get in touch and why they think that it might be beneficial to have a chat meeting. From the counselor's perspective, a major purpose of the individual chat sessions is to discuss with participants the need for more intense (i.e., face-to-face) support and if necessary encourage them to utilize such support.

Early intervention and access to regular care. So far, there is no research in the field of EDs showing that technology-enhanced clinical interventions are as effective as face-to-face specialized treatment (Aardoom et al., 2013; Bauer & Moessner, 2013). Therefore, ProYouth counselors recommend that participants engage in regular mental health care as soon as they

experience substantial symptoms of an ED, such as severe underweight, frequent binge eating, or compensatory behaviors. The online counselors support participants in getting access to expert care by providing information on local or not too distant treatment providers and treatment modalities, and by answering questions and addressing concerns that may prevent participants from seeking face-to-face treatment.

It should be noted that the scientific evaluation of the ProYouth initiative is still underway and conclusions with respect to its potential cannot be drawn at this early stage. Two years after the launch of the online platform, more than 20,000 individuals have accessed the screening tool and over 8,000 have registered for participation in the program. Implementation and dissemination of ProYouth currently include activities via the Internet (e.g., postings to websites, forums), print media (e.g., newspapers, posters, flyers), and social media (e.g., Facebook, Twitter), as well as face-to-face activities involving young people (e.g., workshops in high schools) and stakeholders (e.g. student counseling services, authorities). Ongoing research will allow the research teams in the different European countries to address some of the research priorities in ED prevention science described below.

Conclusions and Future Directions

The objectives of this chapter were, first, to review the potential of information and communication technologies for advancing the field of ED prevention and, second, to outline implications for future development and research. Using technology in the context of preventive initiatives makes intuitive sense. The increasing availability of the Internet and mobile devices will continue to expand the reach of such interventions around the globe. The current state of the art indicates that computer-assisted approaches are promising tools that deserve further efforts in both prevention and treatment research. However, there is a limited number of high-quality studies to date and comprehensive research programs are needed in order to provide a sound evidence base concerning the efficacy, effectiveness, cost-effectiveness, and public health impact of technology-enhanced interventions.

Computer-assisted approaches to ED prevention are still a relatively new field and there are many unanswered questions at this stage. In the following, we discuss some of the most urgent topics for future development and research.

Evaluation of Existing Approaches and Development of New Approaches

The empirical evaluation of computer-assisted preventive approaches faces challenges on both the conceptual and methodological level. Some of these challenges are equivalent to the challenges inherent to noncomputerized preventive approaches; others are specific to technology-enhanced programs. Given that traditional prevention programs that are delivered in face-to-face settings are often unavailable, too complex, and too expensive for general distribution (van Vorhees et al., 2011), many researchers consider the use of information and communication technologies highly promising for the advancement of prevention science.

In the ED field, as in prevention of mental illness in general, there is a need for more systematic and rigorous studies evaluating the efficacy, effectiveness, and cost-effectiveness of such programs. In terms of efficacy, to date only two adequately powered studies have attempted to demonstrate the superiority of an online intervention (compared to a no-intervention control condition) in terms of the prevention of illness onset. Both studies reported mixed findings

concerning the overall efficacy of the respective intervention (Lindenberg & Kordy, 2015; Taylor et al., 2006). Most published studies report positive findings with respect to the feasibility and acceptability of computer-assisted approaches, and many report significant effects of such approaches on ED-related risk factors, attitudes, and/or behaviors. However, we would ultimately want to see these interventions reducing the incidence rate of EDs and/or reducing the duration of suffering for those who do fall ill. Research is challenged to use such strict outcome criteria in order to convincingly demonstrate the potential of a specific program (see Chapters 40, 44, & 45). In addition, the investigation of moderators, mediators, response patterns, and mechanisms of change may help to optimize existing computer-assisted approaches and/or may contribute to the development of new interventions.

In the context of computer-assisted approaches this research may be informed by information collected automatically via the software itself. For example, factors such as number of logins, number and type of websites visited, time spent on each page, and exercises or modules completed can be documented easily. This valuable source of information allows us to analyze patterns of use and to determine more or less important components of an intervention. So far, only one study (using the program Student Bodies) has addressed such questions in the field of computer-assisted ED prevention (Manwaring et al., 2008).

Furthermore, compared to traditional prevention programs, technology-enhanced interventions are easier to tailor to specific target groups. Researchers should take more advantage of this fact and tailor prevention programs to the risk profiles of their participants. Furthermore, they should consider providing adaptations of programs for subgroups of participants such as males, specific age groups, ethnic minorities, individuals with a migration background, or athletes. The current evidence base on computer-assisted approaches to prevention relies almost exclusively on studies conducted with young women in college or high-school settings. Even though this group is at particular risk of developing EDs, there is a clear need for research on other target groups and more diverse populations.

In addition to an intervention's efficacy, it is essential to determine its effectiveness in larger populations and in real-world settings (Becker, Ciao, & Smith, 2008; Marchand, Stice, Rohde, & Becker, 2011; see also Chapter 44), as is currently being done in the ProYouth initiative. Furthermore, research is needed on the cost and the cost-effectiveness of technology-enhanced approaches. The cost of a computer-assisted intervention is determined by the initial development of the intervention (e.g., programming, software), the technical infrastructure required (e.g., servers to host the program and to store data), and the cost for maintaining the program over time (e.g., software updates, back-up procedures). In addition, the extent to which a program is automated versus personalized has a major impact on the associated cost. Therefore, it is important to investigate how much involvement of online moderators, online counselors, or clinicians is necessary and to what degree a program may be automated while remaining acceptable and effective (Minarik et al., 2013).

Another important factor with respect to cost is staff time, and especially the requirements for expert staff. Fully automated interventions such as the FMA program described above do not require any direct contact between provider and participant. In contrast, programs including moderated synchronous or asynchronous discussion groups, personal emails to participants, and/or online counseling sessions require a specific amount of staff time. Some staff members need to be skilled in online counseling and all need to have expertise in the field of EDs. In the fixed-intervention approaches, it should be possible to provide quite exact estimates on the amount of staff time and hence the costs associated with delivery of the prevention program per individual participant. An estimation is more complex for more flexible programs such as ProYouth in which participants may display very heterogeneous patterns of

utilization, ranging from mere access of automated parts (e.g., the psychoeducation module or the monitoring module) to a very intense utilization of the individual chat module (which is the most costly module of the program). In any case, information on the cost related to the delivery of a prevention program should be assessed and reported as part of future research, because availability of such data is an important precondition when discussing the transfer of a program from the research setting into real-world settings (Minarik et al., 2013). Computer-assisted interventions share the challenges inherent to the research-practice gap with traditional prevention approaches (Becker, Stice, Shaw, & Woda, 2009).

Implementation, Dissemination, and Sustainability

Without any doubt, both the development of innovative technology-enhanced preventive approaches and research into the efficacy, effectiveness, and cost-effectiveness of new and existing approaches are of utmost importance. Yet, they are not sufficient. We also need to conduct studies related to the implementation, dissemination, and sustainability of such approaches in order to identify the most promising models of service delivery. Preventive approaches with a limited reach (due to complexity, required infrastructure, and lack of staff or financial resources) cannot contribute to a significant reduction of the burden of mental illness and may have only a limited public health impact (Muñoz, Beardslee, & Leykin, 2012).

Given the broad and increasing availability of information and communication technologies worldwide, computer-assisted preventive approaches (especially those using the Internet via mobile devices) may theoretically be implemented and disseminated broadly and thereby reach huge target populations. However, it is currently unknown how we should optimally address these populations via technology. The fact that a program is publicly available on the Internet does not automatically lead to huge numbers of individuals signing up for participation in the program. This is especially the case in prevention when we approach individuals who do not currently suffer from a full-blown ED but show a risk for development of the illness or experience initial symptoms. These individuals might not be aware of their risk and thus might not see a need to engage in an online prevention program. Therefore, as in traditional programs, active promotion of Internet-based preventive programs is required.

But what is the best strategy to do so? Of course, such programs may be promoted in high school, college, or community settings, similar to traditional preventive approaches. However, the reach of such face-to-face strategies is limited and requires substantial resources (e.g., for recruitment and training of facilitators). Other, less expensive options to promote an online program include informing the target population through various print, online, and social media campaigns or strategies. The efficacy of such strategies is largely unknown, as it is currently unclear which portion of a specific target population would take up a program via each of these avenues. Research into questions related to the implementation and dissemination of online preventive interventions is in a very early stage and to our knowledge nonexistent in the field of EDs. Clearly, work in this area is needed to guide future efforts related to the implementation and dissemination of computer-assisted preventive approaches.

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Developing an Ecological Approach to Eating Disorders Prevention: The Ontario Project

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Over the past 10–15 years the results of individual programs, literature reviews, and meta-analyses have converged to demonstrate that prevention of eating disorders (EDs) can work. For example, selective and indicated dissonance-based interventions (DBIs), focusing on certain types of risk factors (e.g., internalization of the thin ideal), consistently show sustained positive effects on mixed-risk groups of young adult females in college (e.g., in sororities and/or intercollegiate athletes) and on adolescent and young adult females already showing signs of ED symptoms (Becker, Stice, Shaw, & Woda, 2009; Stice, Shaw, & Marti, 2007). With the exception of Becker's DBI (see Chapter 44), the most promising programs, such as Stice's DBI, Stice's Healthy Weight intervention (see Chapters 24 & 44), and the Stanford-based Student Bodies online program (see Chapter 46), are designed for high-risk individuals. To help prevent the development of new cases (i.e., to reduce the incidence; see Chapters 5 & 41), we need to expand the scope of ED prevention to a broad, ecological perspective anchored in the health promotion and universal portions of the prevention spectrum (Levine & McVey, 2012; Levine & Smolak, 2008). This means that prevention necessarily involves efforts to reinforce health, resilience, and the ability to cope effectively with life's developmental challenges and hardships (Levine & Smolak, 2006; see also Chapter 33).

After briefly exploring the semantics of prevention, this chapter outlines the limitations of current efficacious and effective programs and then describes key features of an ecological model of prevention. The model is illustrated with McVey's program of research, advocacy, policy development, and professional training in order to explore lessons learned from and challenges in evaluating an ecological approach. We conclude with suggestions to guide future theory and research.

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The Semantics of Ecological Prevention

We prevent a condition when we understand the factors that significantly increase the probability of the condition and trigger its onset, and then we eliminate or moderate those factors in systematic ways to reduce the incidence of that condition or significantly delay its onset. In general, a program is successful when there are two principal outcomes (Institute of Medicine, 2012; Levine & Smolak, 2008). First, asymptomatic participants show a significantly lower rate—and, ideally, a low rate in absolute terms—of onset of disordered eating or EDs over time as compared to a group of nonparticipants with a comparable level of initial risk. In universal prevention, the expected outcome for the comparison group is the population incidence. Second, program benefits outweigh various costs, including the risk of unintended harm.

What "conditions" are being prevented? The ecological perspective is similar to the sociocultural paradigm in assuming that components of ED psychopathology occur on a continuum, are in and of themselves unhealthy, and some are well-established risk factors for EDs (see Chapters 1 & 67). Consequently, our ecological perspective proposes that prevention researchers conceptualize the EDs in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*; see also Chapters 2–4, 8–11, & 13) as extremes of six intertwined continua (Levine & Smolak, 2006):

1 Negative body image.
2 Unhealthy forms of weight management.
3 Overvaluing, in the definition and evaluation of self, perceived weight and shape in relation to unrealistic standards of beauty, fitness, and muscularity.
4 Irrational fear and loathing of body fat and fat people, feeding drives for thinness and leanness.
5 Harsh self-surveillance and self-criticism, in a reciprocal relationship with shame, anxiety, depression, and difficulties in self-regulation.
6 Binge eating.

People with eating attitudes and behaviors that generate mild-to-moderate problems (e.g., disability and distress) and who have moderate to high levels of continuum 1, plus 2 or 6, and at least one of 3 to 5, suffer from *disordered eating*. Both disordered eating (DE) and EDs are appropriate outcome measures for ecological prevention research, and the individual variables are reasonable candidates for mediation analysis in the prevention of EDs.

Ecological Models, Health Promotion, and Universal Prevention

There is no doubt that, over the past 15 years, evidence-based selective and targeted programs guided by social cognitive theory have elevated the scientific and sociological status of prevention in the ED field. Yet, at the same time there is increasing emphasis on prevention approaches guided by an ecological perspective with roots in public health and in the multidimensional, systemic approaches that have been effective in the substance abuse prevention field (Levine & Smolak, 2006). There are three major reasons for this development.

First, given the substantial evidence supporting the role of sociocultural risk factors in the development of EDs and DE (see Chapters 21 & 27), it stands to reason that prevention should focus on understanding and changing the multiple, interlocking systems that constitute

"sociocultural" environments. Various prevention-oriented theories in the ED field (Levine & Smolak, 2006; Piran, 2010; Smolak & Levine, 1996), in community psychology (e.g., community-based, nonclinical model; Institute of Medicine, 2012) and in developmental psychology (e.g., developmental contextualism; Lerner, Fisher, & Weinberg, 2000) emphasize sociocultural factors that can be organized within an ecological model. As a group, these approaches highlight four concentric arenas of risk (and potential resilience) that each individual exists within: *individual* (biological and personal history factors, e.g., temperament, level of physical maturation, age, history of abuse; see Chapter 34); *relationship* (peer, family, and intimate partner influences; see Chapters 26 & 31); *community* (neighborhood, school, athletic, or workplace influences; see Chapters 35 & 42); and *societal* (greater cultural and social policies and norms; see Chapters 23, 25, 29, 43, & 48). An ecological approach is entirely consistent with the fundamental definition of universal prevention as a health-promoting enterprise that works by improving *public* policies, groups, communities, and institutions such as school systems (Cowen, 1973; Levine & Smolak, 2006; Oesterle, Hawkins, Fagan, Abbott, & Catalano, 2010). The spirit of this perspective is captured by the Institute of Medicine's (2012) observation that "community-based prevention requires cultural, social, and environmental changes, much like the extensive changes in water, sanitation and housing, and nutrition that occurred in the first half of the 20th century" (p. 17). Trickett and Rowe (2012) provide an excellent review of the evolution of ecological thinking in prevention, health promotion, and public health.

The second pillar is Stice et al.'s (2007) meta-analytic finding that selective and, in particular, targeted programs for preventing EDs work best with those who are at high risk due to specific contextual and developmental variables (e.g., they are college students) or at very high risk due to, for example, a very negative body image. This is significant because, according to the Rose Paradox in public health, in a large population the clear majority of new cases of a disorder come from those at low-to-moderate risk because they far outnumber people at high risk (Austin, 2001; see also Chapter 41).

Finally, there has long been tension between meta-analytic findings that prevention effects are largest for females aged 15 and older versus a desire to develop and implement effective programs for children and younger adolescents well before they develop the components of DE (let alone ED) that produce so much suffering and loss of productivity and vitality (see Chapters 22 & 37). As yet there are no effective dissonance-based, healthy weight, online cognitive-behavioral programs for children and younger adolescents, although there have been notable successes applying media literacy (see Chapter 45). In addition, there have been a number of prevention programs for young participants that generated positive outcomes in the short term, only to see positive effects fade—without systemic support—over the subsequent 6 to 12 months (Levine & Smolak, 2006). This state of affairs reinforces the potential of an ecological perspective to add significantly to the overall landscape of prevention.

Key Features of an Ecological Model: The Ontario Project

How does one go about conceptualizing and implementing multilevel ecological/public health models for addressing prevention of the spectrum of DE and EDs? One effective way to answer this deceptively simple question is an extended example that illustrates various stages of policy and program development. Beginning in 1996, G. L. McVey has designed, implemented, and evaluated a series of prevention programs aimed at children, youth, and young adults, as well as adults who mentor them. Collectively, these programs and the policy

Table 47.1 Elements of an ecological approach to prevention demonstrated in the Toronto project (1996 through 2013).

	Phase			
Element	*1*	*2*	*3*	*4*
Focus				
Government and public policy (society)	Y	N	Y	Y
School, athletics, workplace (community)	Y	Y	Y	Y
Peers, family, adult role models (relationships)	Y	Y	Y	Y
Knowledge, values, and skills (individual)	Y	Y	Y	Y
Process involves:				
Partnership development with knowledge users from multiple sectors	Y	N	Y	Y
Training professionals from multiple disciplines	Y	Y	Y	Y
Knowledge gained is translated into policy development and sustainable programs	Y	Y	Y	N
Programs are acceptable and feasible	Y	Y	Y	Y
Evaluation is conducted at multiple levels including:				
Improvements at the individual level				
Reductions in disordered eating	Y	Y	Y	N
Reductions in eating disorders	N	N	N	N
Reductions in other psychological and/or physical problems	N	N	N	N
Increases in psychosocial strengths, resilience	Y	Y	Y	Y
Improvements in ecology				
Described and documented	Y	Y	Y	Y
Systematically assessed	N	Y	N	N

development, advocacy, organizing, and training that support and extend them are known as The Ontario Project because they have been carried out in the province of Ontario, Canada (Levine & McVey, 2012). In the following exposition special attention is given to the relevance, impact, and application of this work, and the ways in which the scope and innovative nature of The Ontario Project embody the key elements of an ecological approach to prevention. All the connections to be discussed between The Ontario Project and the foci, processes, and research-related elements of this ecological approach are summarized in Table 47.1.

Phase 1: Classroom-Based Prevention

The first phase of The Ontario Project encompassed the transition from experience with the limitations of traditional classroom-based prevention (see Chapter 42) to a more ecological approach. Thus, the initial studies were efficacy trials of classroom-based interventions aimed at promoting positive body image among females ages 11 and 12, that is, at the vulnerable developmental phase of early adolescence (Smolak & Levine, 1996). Drawing from social cognitive theory, the six-session prevention program incorporated media literacy about negative effects of the idealization of thinness. Guided by the nonspecific vulnerability-stressor model (Levine & Smolak, 2006), the program also promoted life skills, such as those for stress management and peer relations, including role-playing of social problem-solving techniques

to resist weight-based teasing/bullying and pressures to diet. A nondieting approach to healthy eating and physical activity was emphasized. Two randomized controlled trials (RCTs) produced promising but ambiguous and limited results (McVey & Davis, 2002; McVey, Davis, Tweed, & Shaw, 2004).

McVey subsequently incorporated all-girl groups facilitated by public health practitioners. This development reflected research illuminating peer pressure to diet, as well as Piran's (2001, 2010) school-based participatory action research linking creation of healthy peer norms (relationship level of the ecological model; Table 47.1) to sustained decreases in the incidence of EDs. Although the theoretical model, content, and goals of the intervention, including the classroom prevention curriculum, were an extension of the program implemented in the first two studies, other aspects of this second wave of studies also represented movement toward a more ecological, multisystems approach. In a very real sense, this development originated and blossomed at the community level. A mental health division of the government-mandated public health unit contacted McVey and asked that she apply her prevention and research expertise to the body image and eating issues that were highlighted by the unit's own assessment of the needs of young girls. Thus, the project's feasibility and potential effectiveness were increased by foundational buy-in and resource support from a mental health division of the government-mandated health unit. A potentially important collaboration was established between the researchers and the health unit management team.

An ecological approach was also present in the four ways selected to improve the program's sustainability and fidelity, that is, the probability of the program being implemented as intended. First, with respect to adult leaders and role models for youth (relationship level), facilitators for the all-girl peer groups were selected from a pool of public health nurses who were moderately knowledgeable about body image and had previous experience facilitating youth groups oriented toward mental health. Second, consistent with an ecological model's emphasis on collaboration with stakeholders (see process section of Table 47.1), the evidence-based, manualized prevention program (Every BODY Is A Somebody; Seaver, McVey, Fullerton, & Stratton, 1997) was coauthored by public health and school board colleagues in partnership with the research team, and matched to the provincial government's mandated objectives. Third, face-to-face workshops available through a Ministry-funded training grant provided detailed instructions for facilitating each lesson (process of multidisciplinary training). These workshops also provided background education in the prevention of EDs and DE and in the function of adult role models in promoting positive body image (relationship level). Finally, weekly group meetings, overseen periodically by the research team, enabled the peer-group facilitators and the lead public health nurse to discuss the previous week's session and prepare for the next week's session (relationship level).

This enhanced 12-session intervention was evaluated using original and replication RCTs (McVey, Lieberman, Voorberg, Wardrope, & Blackmore, 2003a, 2003b). The program was successful at 3-month follow-up in significantly improving body esteem and in reducing DE (McVey et al., 2003a). However, the replication study, in which the young adolescent females had significantly higher baseline levels of DE, was unsuccessful (McVey et al., 2003b). The implication that this type of more ecological intervention, anchored in the universal-selective portion of the prevention continuum (Levine & Smolak, 2006; Levine & McVey, 2012), may be more suitable for those at low to moderate risk was folded into subsequent training of other health units (process of knowledge translation to improve ecology; Table 47.1). It is noteworthy that open-ended feedback from participants following the intervention revealed that the group experience enhanced their sense of belonging and connectedness with peers and their school.

It appears that McVey's training of local staff to facilitate the interventions deepened the connection of the programs to the school as a potentially positive ecological influence. It also helped sustain programs beyond the study period; for example, the peer group program became embedded into the health unit's routine delivery of services to local schools (process of knowledge translation). McVey's partnership with the health unit also continued beyond the research design, allowing for periodic updates to programming, based on new findings from the EDs prevention literature. Supported by a publicly funded EDs training program, McVey conducted face-to-face workshops to disseminate the evidence-based intervention components to other public health units and school boards across the province of Ontario (McVey et al., 2005; focus on society level; processes of multidisciplinary training and knowledge translation). These workshops also alerted adult participants to how their own attitudes, comments, and behaviors can influence body image concerns, while highlighting how school personnel and public health staff can effectively participate in identification, referral, and support of youth showing signs of EDs (focus on relationship and community levels). Evaluations conducted with the professional participants revealed that the workshops were associated with significant increases in knowledge about EDs and in the confidence to facilitate body image and self-esteem promotion strategies with youth (McVey et al., 2005).

Phase 2: Toward a Whole-School Ecological Approach

As described in detail elsewhere, the next step in McVey's program was the Healthy Schools-Healthy Kids project (Levine & McVey, 2012; McVey, Tweed, & Blackmore, 2007). The ecological framework for prevention in early adolescence integrated the following: training of teachers and other staff regarding healthy influences on body image, nutrition, and physical activity (focus on relationship and community levels); curricula embedded into all grade levels and across multiple topics (community level); live theater highlighting the negative influences of media and appearance-based teasing (individual and relationship levels); after-school programs (community level); posters and public service announcements underscoring size acceptance, healthy eating, and active living (relationship and community levels); and inclusion to some extent of parents, teachers, and other school personnel in the intervention (relationship and community levels). The peer groups were also included, with graduates subsequently disseminating media literacy strategies to the rest of the student body (relationship level).

Healthy Schools-Healthy Kids, implemented over an 8-month period, was evaluated rigorously in a large-scale RCT with a 6-month follow-up assessment. One unique feature was an ecological assessment in which teachers completed, both before and immediately after the intervention, a measure reflecting how they perceived social, behavioral, and nutritional/physical aspects of the school "climate." Multivariate analyses revealed that, as predicted, program participation reduced internalization of the thin ideal among the male and female students in grades 6 through 8 and reduced DE among the female students ($d = 0.39$ at 8-month posttest and $d = 0.27$ at 6-month follow-up). Reductions in unhealthy weight-loss behaviors were significant for boys and girls at posttest, although this effect was lost by 6-month follow-up. There were no intervention effects on any of the measures completed by teachers, including perceptions of school climate.

Late Adolescence and Emerging Adulthood

The late adolescent transition is a high-risk period, so McVey and colleagues partnered with university-based practitioner-stakeholders to build the capacity of professionals to promote positive body image in male and female undergraduates ages 18 through 23. The six-session "Every BODY Is A Somebody" body image curriculum was integrated into the training program

for undergraduate peer health educators conducted annually by health promotion staff across three Ontario universities. Focusing on the individual, relationship, and community levels of the ecological model (Table 47.1), the purpose of this intervention was to influence the peer health educators themselves at the individual level in order to leverage their roles as mentors and leaders. The goal was to spread their newly gained knowledge and attitudinal shifts to the larger university community (rippling effect) through workshops they conducted with students and through role modeling of body positive, weight-bias-sensitive attitudes. To determine its feasibility in a collegiate setting, the program, cofacilitated by McVey and a health promotion specialist from student health services, was evaluated in an uncontrolled pilot study with a pre-post design (McVey et al., 2010). Participants were 30 undergraduate peer health educators. The standard training session format and location were modified based on stakeholder preferences expressed by both health promotion staff and students (process of acceptability and feasibility).

Quantitative analyses revealed that participation led to increased body satisfaction and decreased internalization of the thin ideal (McVey et al., 2010). Open-ended feedback revealed that participants particularly enjoyed the videos on set-point theory and on media influences, as well as information about life skills. An overwhelming majority said they also enjoyed the face-to-face format, the interactive activities, and the length of the program. The impact of this training program for peer influencers on the undergraduate community remains to be determined. Plans are in place to secure funding to conduct effectiveness trials on this collaborative model of prevention. Evaluations of Becker's Body Image Program for female undergraduates in sororities or in athletics (see Chapter 44) have demonstrated that peer health educators as trained agents of change can indeed affect the rest of the student population positively by actively engaging with the issues they believe in, by modeling body satisfaction, and by disseminating health-promoting knowledge and practices.

Phase 3: Toward a Societal-Ecological Approach

Educators and Public Health Practitioners Research confirms that teachers and public health practitioners in schools are important channels and mediators of community-based prevention (Institute of Medicine, 2012). McVey, Gusella, Tweed, and Ferrari (2009) created an online research-based training resource for teachers in grades 4 through 6 (focus on relationship and community levels). "The Student Body: Promoting Health at Every Size" uses classroom activities previously shown to improve body satisfaction and eating behaviors (McVey et al., 2007). Program content (www.aboutkidshealth.ca/thestudentbody) includes case studies, background information, classroom activities, and supplementary resources. There are six modules: media and peer pressure, healthy eating, active living, weight-based teasing, adult role models, and school climate. To maximize adoption by teachers and school-oriented public health practitioners, health-promoting activities were matched to three of the provincial government's mandated objectives (community and society levels), such as "Healthy eating: Outline the factors that influence body shape and size (e.g., heredity, diet, exercise)" (McVey & Gusella, n.d.).

The effectiveness of the online prevention program, supplemented by face-to-face tutorials conducted in small groups, was examined over a 60-day study period in an RCT with 78 elementary school teachers and 89 local public health practitioners. All participants completed (online) pre- and postprogram self-report assessments of knowledge about factors influencing body image in children and of self-efficacy in addressing weight bias in their school. Information was also solicited on program layout and content in relation to the feasibility and perceived benefits of this program as a knowledge translation tool.

The online training resource significantly improved teachers' knowledge about dieting, while increasing public health professionals' efficacy expectations for fighting weight bias. As

a group, program participants reported increased awareness about the presence and negative effects of potential weight bias in their work. In addition, the program's layout and content were well received, and the online program continues to be accessed by both new and returning visitors (process of knowledge translation).

Athletes In "BodySense: A Positive Body Image Initiative for Female Athletes" (www.bodysense.ca), McVey and colleagues collaborated with both a multidisciplinary steering committee of community-based health professionals and a national advisory group of relevant associations (e.g., Canadian Association for the Advancement for Women in Sport) to help reduce pressures to be thin in sport, and to promote positive body image and eating behaviors in young female athletes (Buchholz, Mack, McVey, Feder, & Barrowman, 2008; focus on societal level). The foundation of the 3-month BodySense program is 10 integrated "BodySense Basics" presented in workshops at gymnastic clubs: one workshop for parents and coaches and one for the gymnasts (individual and relationship levels). Gymnastics clubs, coaches, and parents were also given a BodySense Basics poster, a miniresource library of books, eight newsletters (on topics such as the Female Athlete Triad and sport nutrition), and a "fuel tank" box of low-cost, high-energy snacks that clubs could provide to athletes.

Participants in the initial outcome research were competitive female gymnasts (ages 11 to 18 years), parents, and coaches from seven gymnastic clubs across the province of Ontario. Four of the clubs were randomly designated to receive the 3-month BodySense program, whereas the remainder constituted a control group. Participation in the BodySense program resulted in athletes perceiving a reduction in pressure from their sports clubs to be thin, though no changes were found in body esteem, DE, or internalization of the media ideal. No significant change was observed over time on mothers' measures.

Phase 4: Capitalizing on Shifts in Ecology

Large-scale ecological programs such as The Ontario Project are necessarily connected to governmental policies and practices, which are subject to change, sometimes in response to public concerns. Even as Phases 2 and 3 were being enacted and evaluated in the mid-2000s, shifting government priorities over time and the ensuing mandate changes experienced at the local level by public health units began to offset the gains made in focused efforts to prevent EDs and DE. Time and resources devoted to these prevention activities were replaced by widespread promotion of healthy eating and healthy weights in an effort to reverse the rising trend of childhood obesity/overweight (see Chapter 65). Concerns for childhood obesity, although justified, overshadowed the serious public health issue of EDs and DE among Canadian children and youth (Public Health Agency of Canada, 2010). However, loss of resources devoted to ED prevention, coupled with a surge of educational programs designed to improve nutrition and physical activity, led to a new issue: inadvertent increases in anxieties about food and body weight.

As a result, in 2007 a planning meeting was organized and cohosted by Adair and McVey (Adair et al., 2008). Supported by the provinces of Ontario and Alberta, researchers, practitioners, and policy-makers at the federal, provincial, and territorial levels from the fields of obesity and EDs gathered to discuss areas of shared concern and potential conflict (focus on societal level; Table 47.1). Following recommendations generated at that conference, in 2008 McVey hosted in Ontario an international conference on the prevention of weight-related disorders. This conference linked internationally recognized academic researchers with knowledge

users engaged in educational, mental health, and other health-related policies and practices within Ontario (societal level; McVey, Levine, Piran, & Ferguson, 2012). In 2011 a Canadian Prevention Strategy Group, working across the fields of EDs and obesity prevention, was formed (process of partnership development) following a national planning meeting hosted by McVey. The guiding principles publicly endorsed at that 2011 meeting by professionals from multiple disciplines and various sectors of society reinforced The Ontario Project's emphasis on a social-ecological approach to prevention that, for greatest impact, reaches out to adult influencers of children and youth.

This shift in the ecology of public concerns and government priorities led McVey to expand her prevention efforts and research to align with government mandates that public health resources be devoted to preventing chronic disease via promotion of healthy weights (Ontario Ministry of Health Promotion, 2010). This population health approach (societal level) followed the principles endorsed by the Canadian Prevention Strategy Group and encouraged development, implementation, and evaluation of a training model that uses face-to-face workshops and case study coaching to enable "adult influencers" to deliver nonstigmatizing health promotion (focus on relationship level and process of multidisciplinary training; McVey et al., 2013). Specific components of the training ranged from how adults can be role models for healthy forms of self-care to nonstigmatizing and culturally sensitive ways they can apply key findings from the weight science literature (Neumark-Sztainer et al., 2007).

An evaluation (with 6-week follow-up) of the full-day, face-to-face workshop conducted with 342 Ontario public health promoters revealed that participation led to statistically significant decreases in antifat attitudes and internalization of media stereotypes, and to significant increases in self-efficacy to address weight bias (McVey et al., 2013). Participants indicated that this training heightened awareness of their own weight biases and of the need to broaden their perspective on healthy weight promotion to include mental health promotion. Future research will incorporate participant feedback that additional sessions are warranted to help translate the newly gained knowledge into daily practice, and that resource support at the organizational level will be pivotal (focus on process of acceptability and feasibility).

In response to the aforementioned national planning and dissemination meetings, which resulted in consensus to develop guiding principles for the prevention of the broad spectrum of weight related disorders, British Columbia (BC) rolled out a province-wide "Weight to Well-being" initiative that integrates ED and obesity prevention by including strategies to reduce weight stigma and bias in the BC healthcare system (societal, community, and relationship levels; Provincial Health Services Authority, 2013b). In addition to creating an innovative, interactive, online weight bias resource for healthcare practitioners (MacKean & GermAnn, 2013), BC has made important strides toward implementing a systems approach to prevention of the full spectrum of weight-related issues (Provincial Health Services Authority, 2013a).

The Ontario Project: Status Report 2013

Paxton (2012; see Chapter 48) has reviewed government public health initiatives to reduce body image concerns and EDs. In countries such as Argentina, Australia, Israel, Spain, and the United States, progress at the government level has helped establish regulations designed to discourage DE among children and youth and to limit or buffer widespread exposure of the public to media-based images of severely underweight fashion models. The Ontario Project represents Canada in this category.

The series of community-based prevention studies carried out as part of The Ontario Project spanned efficacy trials, effectiveness trials, implementation studies, and translational research. This research program took a lifespan developmental approach and, as shown in Table 47.1, was ecological in its insistence that (a) all phases throughout the research cycle be anchored in partnership development with knowledge users from multiple sectors and with trainees from different disciplines; and (b) knowledge gained be translated into policy development and sustainable programs—with implications for the societal, community, relationship, and individual levels of influence—not just limited projects, followed by papers and publications.

Specifically, as the prevention strategies were being integrated into the provincial curriculum so that all children living in Ontario could have access to them, workshop training on the research findings and the practical intervention strategies was conducted across the province with professionals from the sectors of education, public health, afterschool care, and parks and recreation/sport. This led to the uptake of evidence-based strategies among adult influencers constituting one of the natural support systems for children and youth. The resulting "buzz" created positive shifts at the institutional and community level in terms of attitudes and beliefs about food, weight and shape, and how to model these to children. At multiple, intersecting levels of the ecology, there was increased attention to and support for sensitivity about weight bias and weight-based bullying/teasing, adopting and promoting a nondieting approach to eating, and promoting a body positive environment.

Another ecological feature, and a unique strength, of The Ontario Project is the merging of prevention research and knowledge translation activities with established training and advocacy practices and with the building, at the societal level of ecological prevention, of a provincial network of specialized, ministry-funded ED treatment programs for clients across the age spectrum and their families (www.ocoped.ca). This network has enabled ED experts to conduct workshops and consultations with over 5,000 community-based health practitioners and health educators across Ontario, and to build a "community of practice" with a subgroup of them. Once a year this multidisciplinary network of approximately 120 health-care professionals now specializing in the treatment of EDs meet in person for training in up-to-date evidence-based assessment and treatment practices. These developments have greatly increased the integration and standardization of best practices in assessment, treatment, early identification, and multilevel prevention of EDs (McVey et al., 2005).

The Evidence Base

Outcome Evaluations The Society for Prevention Research (SPR; 2004) lists eight criteria for a successful prevention program:

1 The program is carefully derived from an explicit theoretical model.
2 Trained personnel have implemented it with high fidelity.
3 Outcome research uses samples permitting adequate statistical power and reasonable generalization.
4 Reliable and valid measures are used, and, if possible, administered by people blind to study conditions.
5 A pattern of predicted outcomes is seen in the target behaviors.
6 A pattern of predicted mediating effects is observed in the risk and protective factors derived from the model guiding program development.
7 Predicted outcomes are observed in the short term and over a meaningfully long follow-up period.

8 The first seven criteria have been met in at least two RCTs or reasonable substitutes, such as time-series designs with long baselines. Replication is most persuasive when accomplished by two or more independent sets of investigators.

To date, four EDs prevention programs meet or come very close to meeting these stringent criteria: the dissonance-based programs of Stice and C. B. Becker; Stice's Healthy Weight program; and C. B. Taylor's Student Bodies program. The Ontario Project meets SPR criteria 1 through 6 and a portion of criterion 7, but it does not meet criterion 8. Predicted outcomes were observed, albeit in the short term and not consistently in long-term follow-up. Original and replication RCTs were conducted, although not by independent sets of investigators.

Socio-Behavioral Evidence Ten years since the findings on the peer support group intervention were published, the health unit where the study took place still offers these groups as part of routine service delivery. Additional public health nurses at that study site have been trained internally to be group facilitators. Other health units across the province of Ontario have adopted this programming. Also, the Student Body online curriculum continues to be used by teachers and public health professionals in at least three Canadian provinces. Finally, face-to-face workshops continue to be offered free of charge throughout Ontario to disseminate up-to-date prevention strategies with stakeholders who work with children and youth.

Conclusions and Future Directions

The ecological paradigm focuses on helping youth cope with stressors, including developmental challenges (Smolak & Levine, 1996), in a healthy manner by improving various larger systems (e.g., school climate) surrounding them. The goals are to facilitate positive youth development (build their capacity to problem-solve, seek support, sustain healthy relationships) and prevent negative health outcomes such as DE and EDs. McVey et al.'s (2007) comprehensive school-based program, developed over time through the interplay of needs assessment and preliminary curriculum design and evaluation, became part of the Ontario Ministry of Education's curriculum, which ensured its dissemination in a timely way to teachers and public health professionals through province-wide professional development training (McVey et al., 2005, 2009, 2010).

This integrative prevention model, which has its ecological roots in a community-level approach called the Comprehensive School Health (CSH) model (Pan-Canadian Joint Consortium for School Health, n.d.), is itself already supported at the societal level by public health agencies and many other organizations throughout Ontario and Canada (e.g., the Canadian Association for School Health; the Ontario Healthy Schools Coalition; the Ontario Society of Nutrition Professionals in Public Health). Applying the CSH model to prevention of EDs and DE fits with emerging initiatives for the school setting, eliminating duplication and increasing the consistency of health promotion information (e.g., concerning sexual harassment, bullying and teasing, nutrition, active lifestyles) presented to educators, school support staff, students, and families. Coordination of prevention research and knowledge translation activities has been potentiated by McVey's active membership in various coalitions and through her delivery of many face-to-face, community-based prevention workshops across Ontario (McVey et al., 2005). In this way the province benefits from a geographical scaffolding (i.e., a network) of health, public health, and mental health professionals, nonprofit groups, and other concerned citizens who are united in efforts to promote

health and decrease disease related to the intersection of body image, obesity, and eating disorders (see Chapter 49). There is evidence that negative body image, eating pathology, EDs, substance use disorders, and nonsuicidal self-injury are part of a larger spectrum of significant problems in self-care (Lerner et al., 2000; Piran & Teall, 2012). Engaging youth and adults in creating positive and safe environments that foster a sense of inclusion, identity, and connectedness among children and youth has the potential to improve health outcomes and prevent a myriad of mental health concerns and risky behaviors.

Key Lessons from The Ontario Project

Partnership Development Community and institutional partnerships are key factors in ensuring that the development and implementation of an ecological prevention program is relevant, feasible, and sustainable (Levine & Smolak, 2006; Piran, 2010). It is important, right from the beginning, to reach out to established institutions that support children, youth, and young adults—and adult professionals—and invite them to be partners in programming and research. This helps to conceptualize the complex ecology of the target audiences, to generate creative programming ideas appropriate to their contexts, and to sustain programming beyond the scope of the research project. Collaborative, empowering relationships with local organizations provided end-users with a sense of ownership of the intervention, thereby increasing their motivation to support implementation of the intervention and sustain it over the long term.

Paying it Forward The ecological approach to prevention relies on building and maintaining relationships at the personal, professional, and political levels (Levine & Smolak, 2006; Piran, 2001, 2010). McVey's ongoing, active participation in working groups and community-based coalitions related to school health, body image, and healthy weights helped to build trust and sustain relationships between the stop-go research funding cycle, while optimizing the timing of the implementation and dissemination of intervention research.

Fostering Uptake An ecological approach is necessarily connected to the initial ("upstream") portions of the mental health intervention spectrum known as public health promotion and universal prevention (Levine & Smolak, 2006, 2008; Levine & McVey, 2012; see Chapter 41). This means that large-scale ecological prevention is eventually aligned with public policy. For McVey and colleagues, integrating prevention strategies with policy by matching them to the Ministry of Education's learning objectives was critically important to foster dissemination and uptake of the evidence-based strategies. In turn, McVey was asked to be part of government "think tanks," and to contribute further to curriculum and policy-related development. As noted previously, sustainability was extended and reinforced through integration of evidence-based interventions into routine public health service delivery in the province of Ontario by practitioners whom McVey consulted with and trained in her collaborative studies.

Challenges and Future Directions

Large-scale, multidimensional, and ecological prevention programs such as The Ontario Project face many challenges, some of which are similar to the obstacles encountered in ED advocacy work in general (see Chapter 66). For example, McVey and colleagues found that the

greatest barrier was the ongoing struggle and all too frequent failure to secure ongoing administration and financial support for the program of prevention research and its roll-out in the community.

Full adoption of an ecological perspective will require exploration of the long, rich, and complex history of community-based prevention and health promotion (Institute of Medicine, 2012). This would help tremendously in developing research-oriented criteria for effective ecological programs. We need to clarify (a) what we mean by, and how we measure, various forms of social, institutional, and cultural change; (b) the processes by which differences between and within those ecological segments affect individuals and groups; and (c) the multiple effects of social change on the intended health outcomes and on other important realms, such as community well-being (Institute of Medicine, 2012; Piran, 2001; Trickett, 2009). Experience with The Ontario Project is consistent with previous work in the EDs field (Levine & Smolak, 2006; Piran, 2001, 2010) in emphasizing that such ecological constructs include healthy social norms, effective social policies, solid relationships between children and adults (including parents), and opportunities for children and adolescents to be coached and mentored by adults who embody constructive goals, values, and behavioral repertoires.

Conceptualizing, implementing, and measuring environmental changes that affect health promotion for the public will benefit from expanded and strengthened connections to the fields of public health and public policy (Austin, 2012; McVey et al., 2009, 2013). In general, as has been the case in the development and dissemination of other empirically supported EDs prevention programs to date (Becker et al., 2009), this important work will require collaboration between various stakeholders, including professionals from the sectors of public policy, health, education, and athletics (Institute of Medicine, 2012) and from the fields of (in alphabetical order) anthropology, human development, medicine (e.g., pediatrics, nursing), psychology, psychiatry, public health, public policy, sociology and social justice, and social work. As seen in The Ontario Project, integrating prevention work across the public health issues of EDs and obesity will allow researchers, practitioners, and activists to capitalize on shared risk factors (Neumark-Sztainer et al., 2007) and to leverage, instead of compete for, limited resources.

A related challenge is supplementing sophisticated outcome evaluations with equally useful analyses of the processes involved in promoting the uptake and feasibility of the intervention and the research. Trickett and Rowe (2012) comment that the steps taken to conduct an integrated, systemic health intervention at the level of the schools—such as school readiness, administrative support, teacher buy-in, and collaboration with local champions—are not typically conceptualized up front as part of the intervention model. This was done in The Ontario Project, but future research needs to pay careful attention, not only to the process of preparation, but also to documenting processes in ways that are useful to readers of publications and to local and ongoing knowledge consumers and translators.

In conclusion, while we applaud the tremendous recent strides made in evidence-based prevention in the selective and targeted portions of the mental health intervention spectrum (Becker et al., 2009), we believe that theory, previous research, and the findings of The Ontario Project warrant renewed attention to public health promotion and universal-selective prevention using an ecological perspective (Wilksch, 2014). We hope that this work, informed by a variety of research methods and by work in a variety of fields, will make prevention and the meanings of success more broadly and deeply "sociocultural."

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Social Policy and Prevention

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Social policy relates to societies' responses to social needs and to policies that support the well-being of its members (Dean, 2012). Social policy, therefore, includes policy in areas such as welfare, family services, community services, health, education, and immigration, to name but a few. Societies respond to social needs in different ways, including through informal institutions (e.g., family groups) and private organizations, but the focus of social policy has typically been on government actions in relation to maintaining the well-being of the community. This chapter describes social policy activities that aim to contribute to the prevention of body image problems and eating disorders (EDs), and focuses on government policy rather than the independent actions of privately supported organizations (see Chapter 66). Most of the policy work has focused on body image problems, as these are widespread and linked to many different psychological and health issues (Cash & Smolak, 2011), and thus more likely than EDs to attract attention. Of course, body image problems are also a precursor and/or symptom of EDs (see Chapters 1–3, 8, 9, & 22).

To highlight the diversity in social policy approaches that have been implemented, I am using illustrations from around the world that have come to my attention. Collecting this information has been largely an informal process, as a central database of social policy activities related to prevention of body image problems and EDs is not available. In addition, some new government activities in their early stages may be in place but not currently publicized and therefore overlooked. Consequently, it is likely there are important initiatives that I have omitted, and I welcome any new information that can be made available to me.[1]

Governments at all levels (national, state or province, and local) may implement social policy in a range of ways, including legislation, regulations, guidelines, public comment, and persuasion. However, as Dean (2012) notes, "the most conspicuous evidence of the importance of social policies is 'social spending'" (p. 2). That is, governments implement most social policy by allocation of taxpayers' funds without resort to legislative action. For example, they may fund services that deliver prevention interventions or that develop and disseminate

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resources. To justify commitment to this expenditure both governments and their constituents need to appreciate the need to do so. In addition, governments need to have an opinion as to what may be a worthwhile course of preventive action, plus community and business support for that action. These factors have been very important in relation to social policy for prevention of body image and eating problems, as achieving each of these has often been challenging (Paxton, 2012).

Only relatively recently have governments become aware of the seriousness of body image and EDs. Heightened awareness has in part been the result of tireless work by advocacy organizations such as the National Eating Disorder Association (NEDA, United States), Beating Eating Disorders (Beat, United Kingdom) and the Butterfly Foundation (Australia). In addition, the quality and breadth of research in this field has been crucial, in that research findings have often been used to underpin the work of advocacy groups and to highlight areas that need to be tackled to facilitate effective prevention efforts. In Australia, additional support for the case for the seriousness of body image problems has been derived from Mission Australia's (2012) widely publicized findings from its annual survey of youth concerns. Mission Australia has consistently found that over one third of youth identify body image amongst their top three concerns. In the 2012 survey, 20.4% and 7.3% of 15–19-year-old females and males, respectively, described themselves as *extremely* concerned, while 22.6% and 11.3% of females and males reported being *very* concerned about body image.

A crucial way to influence governments is to raise awareness not only of the prevalence and personal costs of a disorder but also of the financial costs, especially directly to the government. To this end, for example, the Butterfly Foundation (2012) commissioned a report to identify the economic and social costs of EDs. It was found that in 2012 the health system costs were approximately 99 million Australian dollars and the productivity impact was estimated at 15.1 billion Australian dollars, similar to productivity impacts of anxiety and depression. A report highlighting similar issues was published by Beat (Beating Eating Disorders, 2012) in the United Kingdom. Data of this kind have been very important in moving governments to action in different parts of the world.

Although the hard facts are important, frequently body image and eating problems become government priorities only after intense personal advocacy by a committed individual, a personal experience of an ED by a government official, or the death from an ED of a high-profile figure. It is also extremely valuable to have a champion within government to strongly present the need for spending on prevention in the face of many competing interests. It is notable that behind much of the social policy that has been enacted around the world there has been a strong female advocate within the government who has been able to provide effective leadership. Examples include members of parliament in Australia (Jacinta Allan and Kate Ellis), Quebec (Christine St-Pierre), and the United Kingdom (Lynn Featherstone and Jo Swinson) (Paxton, 2012).

Policies related to prevention of body dissatisfaction and EDs can be implemented at any stage along the universal-selective-indicated prevention spectrum (National Research Council and Institute of Medicine, 2009; see also Chapters 41 & 47). However, governments have seldom considered how an intervention along this spectrum could have maximum impact. Although over recent years the prevention research literature has generally used a risk factor model to guide prevention strategies in a systematic way (Richardson & Paxton, 2010; Wilksch & Wade, 2009a), government interventions have tended to be unsystematic, with selection of strategies that may address a risk factor but not necessarily in a manner that will have maximum impact. This is attributable primarily to the desire to use very low-cost interventions that are

not likely to be sufficient to address such a pervasive problem as the continuum of body image-related issues. Unfortunately, therefore, policies selected are frequently piecemeal and not ones that are likely to have the greatest impact, as will be demonstrated in a number of cases in this chapter.

There is a range of other political factors that may constrain government action. In some countries health and mental health are regarded as personal responsibilities rather than community responsibilities. This is particularly an issue in relation to body image and EDs, as there is still a widely held view in the Western world that these problems are the result of personal failings, such as vanity and attention seeking, and are largely under individual control (Mond, Robertson-Smith, & Vetere, 2006). In this situation, social policy in this field is unlikely to be enacted.

Finally, there are many competing financial and political pressures on governments that restrain action, in addition to competing interests within government for limited resources. Most notably, governments typically have a very strong commitment, if not an ongoing debt, to business and industry. Consequently, for example, should a government desire to restrict the sale of potentially harmful weight-loss products (Pomeranz, Taylor, & Austin, 2012), it is very likely that it would meet extraordinary opposition from not only the weight-loss industries, but also other business interests worried about government intrusion into business practices. As we have seen with the tobacco industry, significant government action of this kind could be taken, but the fight would be long and hard, requiring conviction and strong social support. Further, governments are at the mercy of election cycles, with the consequence that policies are frequently short-term and political leaders who strongly support prevention and public health can lose their influence.

Despite these difficulties a wide variety of social policy actions have been taken around the world. To demonstrate this diversity, illustrative activities will be presented in three broad areas: government-supported interventions to reach the broad population; interventions within educational settings especially designed to reach adolescents; and interventions that aim to modify the potentially negative influence of fashion media and advertising. Finally, research possibilities and future directions for social policy action will be considered.

Population Interventions

Broadly, population interventions (see Chapter 41) have taken two forms, a social marketing approach designed to communicate information to the whole community, and the creation of Internet resources to support prevention across the universal-selective-indicated dimensions that are available for the whole community. Interesting examples of government actions using both these approaches have been implemented as described below.

Social marketing has recently been defined as "the adaptation and adoption of commercial marketing activities, institutions and processes as a means to induce behavioral change in a targeted audience on a temporary or permanent basis to achieve a social goal" (Dann, 2010, p. 151). Social marketing in relation to health has frequently taken the form of media advertising campaigns that aim to raise knowledge or change attitudes and thereby provide impetus for behavioral individual change. An early example of this approach in the field of EDs was the advertising campaign, conducted in 2007 by the Victorian Government (Australia), entitled "Fad Diets Won't Work" (Paxton, 2012). As dieting has been found to be a risk factor for the development of EDs (Neumark-Sztainer, Wall, Story, & Sherwood, 2009; Stice, Marti, &

Durant, 2011; see also Chapter 24), the aim of the advertisements was to discourage fad dieting in young women by raising awareness of the fact that fad diets are typically ineffective and frequently have undesirable effects such as weight gain. A series of prominently displayed posters and billboards featured young women with a voice caption such as "Fad dieting helped me go from a size 14 to a size 12, and back to a size 16" (a dress size of 14 being the average for young Australian women) and the tag line "Fad diets won't work." Although the advertisements did receive wide exposure at the time, the campaign was short-lived and not thoroughly evaluated. Despite the relatively high costs of intensive social marketing campaigns, in light of the extremely entrenched misperceptions about the usefulness of fad diets there would certainly be value in more extensive use of this approach to dispel widely held destructive myths.

A second social marketing strategy conducted by the Victorian Government in 2009 was designed to raise levels of media literacy among adolescent girls and boys regarding the digital manipulation of media images of young people, thereby challenging internalization of media body ideals (Paxton, 2011; see also Chapters 21 & 29). This advertising campaign, titled "Real Life Doesn't Need Retouching," constructed a series of visual images of idealized and digitally altered young people contrasted with unretouched images of young people in order to highlight the unreality of glamorized advertising images (Paxton, 2012). The campaign was built on research that supported the benefit of media literacy (Espinoza, Penelo, & Raich, 2013; Neumark-Sztainer, Sherwood, Coller, & Hannan, 2000; see also Chapters 29 & 45) and was conducted on a social media site that was popular at the time with adolescents, thus potentially reaching a large audience. Focus group research indicated that the campaign communicated its message well to young people. However, the campaign was short-lived and its impact was not assessed.

In this digital age, it is now possible for governments to make available interactive prevention or early intervention resources using the Internet to reach whole populations and to cross national boundaries. One innovative approach of this kind is the ProYouth (www.pro-youth.eu) initiative financed by the European Commission with the involvement of German, Czech Republic, Romanian, Hungarian, Italian, Irish, and Dutch governments (Bauer & Moessner, 2013; see also Chapter 46). ProYouth is an Internet program that takes into account the differing needs of young people in relation to prevention, early intervention, or treatment and is tailored accordingly by using a stepped care approach. A person interested in gaining support for body image and EDs can log into the website and complete a validated online screening tool. On the basis of his or her results, the visitor to the site receives different intensities of tailored feedback and the opportunity for ongoing monitoring. The approach has received some positive support in a study of an earlier related program, YoungEssprit, in which ED symptoms were reduced in high-school students (Lindenberg & Kordy, 2015; see also Chapter 46). Evaluation of ProYouth is in its early stages, with acceptability, reach, and effectiveness of the Internet-based platform being evaluated (Bauer & Moessner, 2013). As more and more people are exposed to the Internet in a range of different ways, this avenue for reaching the wider community is likely to be extended.

School and Education Settings

In many countries, government social policies are implemented through the education systems over which the government has control, and this is certainly the case in the prevention of body image and EDs. An important reason for this is that young people who are particularly

vulnerable to body image and eating problems are gathered conveniently in schools (Wilksch & Wade, 2009b; see also Chapter 42). In addition, significant sources of sociocultural influence on young people can be reached, in particular peers, teachers, and parents (Paxton, 1999; Rodgers & Chabrol, 2009; Yager, Diedrichs, Ricciardelli, & Halliwell, 2013). To date, a number of strategies have been used within education settings, including: media literacy programs (see Chapter 45); awareness-raising resources designed to aid prevention; parent information; and screening for early intervention (see Chapter 38). Examples of government initiatives in each of these areas will be described.

Media Literacy Programs

The British Government has been an international leader in raising awareness of problems associated with body dissatisfaction and it has taken positive steps in relation to prevention, especially through the Body Confidence Campaign initiated by Jo Swinson MP (Member of Parliament) (Department for Culture, Media and Sport, & Government Equalities Office, 2013). In particular, the British Government has supported a number of nonlegislative approaches to promote positive body image and thereby reduce rates of EDs in the United Kingdom. One of these has been support for the development and dissemination of a free media literacy lesson plan in partnership with Media Smart, a nonprofit media literacy organization funded by advertising businesses in the United Kingdom (Diedrichs, Yager, Paraskeva, & Halliwell, 2012). The 1-hour lesson plan was designed for 10–11-year-old primary school children, and accompanying teacher packs supporting the lesson are also available (Media Smart, 2012). The teacher pack provides a lesson plan that guides teachers to explain the purpose of advertising, the aspirational nature of the images, the unreal nature of the images, digital manipulation, and finally the importance of personal attributes over physical beauty. In addition, a media literacy pack for parents of 6–11-year-old children is also available on the site, but it is not designed specifically to be used in conjunction with the lesson.

Although reportedly downloaded over 30,000 times (Department for Culture, Media and Sport, & Government Equalities Office, 2013), the Media Smart lesson plan was not evaluated prior to release. This led Diedrichs et al. (2012) to conduct an evaluation in which 204 children in grade 6 classes were randomly assigned either to receive the lesson or to be on a waitlist (i.e., in a control condition). Assessments of body image and media influence were conducted prior to, immediately after, and 3 months after the lesson. However, no differences on these measures were observed between the groups. Possibly students had insufficient opportunity to challenge the many highly idealized advertising images presented in the lesson. In addition, approximately one third of students felt uncomfortable in the lesson, primarily due to the images of half-naked models used in the teaching materials. Diedrichs et al. (2012) concluded that dissemination and government endorsement of an ineffective intervention may be a missed opportunity and highlight the importance of formative and pilot evaluations to maximize the value of limited resources.

The SeeMe Media Literacy Project (SeeMe) is a further example of social policy being implemented by the Victorian Government, Australia, through spending on education (Queen Victoria Women's Centre, 2012). In 2010, the Victorian Government provided funding to the Queen Victoria Women's Centre Trust (QVWCT) to develop a media literacy program for grade 8 girls and boys (ages 13 through 14). It was based on the rationale that media exert pressure on young people to conform to unrealistic body ideals and narrow gender stereotypes (see Chapters 21, 27, 29, 42, 44, & 45). These influences may be

internalized and have a negative impact on the way that young people think and feel about their bodies, their self-confidence, and their career and life aspirations. It was proposed that greater knowledge about media manipulation, as one important aspect of media literacy, would provide the skills needed to "unpack" media messages and resist internalization of body image ideals, thereby reducing body dissatisfaction. Consistent with this rationale, media literacy has been shown to be inversely associated with internalization of media body ideals, body comparison, and body dissatisfaction (McLean, Paxton, & Wertheim, 2013), and body dissatisfaction prevention programs for early high-school students that contain media literacy components have had promising results (Richardson & Paxton, 2010; Wilksch & Wade, 2009b).

Unique aspects of SeeMe include the fact that it is an online media literacy resource for both students and teachers and, importantly, it was specifically designed to link to Australian English curriculum requirements for grade 8 students, a subject all students are required to do. This latter aspect ensured that SeeMe was easy to incorporate into the school curriculum at an appropriate age. The curriculum has six modules in which students learn to critique texts and develop skills in critical media analysis through analysis and discussion of online material. A qualitative evaluation indicated improvements in media literacy, body image, and awareness of gender stereotypes in the media (Foundation for Youth, 2012).

Educational Resources

Social policy actions with the goal of preventing body dissatisfaction have also been implemented through government funding of resources for schools and teachers (see Chapter 47). I am aware of a number of these developed in Australia and disseminated widely in the state education system in response to recommendations made by the National Advisory Group on Body Image (2009). The Advisory Group developed a checklist for "body image friendly schools" to guide them in providing a positive body image environment for their students. To support this initiative, the National Government's Department of Youth funded the development of a poster and a series of "conversation starters" for student leaders, school leaders and teachers, and parents. These and many other resources are available at the youth.gov.au website (http://www.youth.gov.au/sites/Youth/bodyImage/Resources). Each of these conversation starters provides information about risk factors for body dissatisfaction and ways to address these risk factors within the school environment.

The Department of Youth also funded the development of classroom activities, from primary through to high school, to promote positive body image. This initiative resulted in *Free to BE* (Butterfly Foundation, 2011), an educational resource for teachers that provided lesson plans for all grades, based on current prevention research evidence. It was extensively piloted in classrooms and the activities were well received by students. In a similar way, the Government of South Australia funded the Centre for Health Promotion (2010) to produce a teacher's guide to achieving a body-image-friendly school environment that provides useful information and specific actions that teachers can take. For example, these include actions related to the school culture, such as including physical appearance comments in school antibullying and antidiscrimination policies, and ensuring school uniforms suit a diverse range of sizes and shapes. In relation to physical activity classes, it is recommended that activities are promoted for fun and the experience of movement rather than health, and that praise is provided that is not appearance or weight related. Initiatives of this kind are likely to raise awareness of strategies that can be adopted in educational environments to enhance body image, but their reach and impact have not been thoroughly evaluated.

Parent Information and Screening for Early Intervention

Another social policy approach to prevention (indicated prevention) and case identification (National Research Council and Institute of Medicine, 2009) is government support for the early identification of ED symptoms, accompanied by referral to prevent the establishment of long-term clinical EDs. Improving early identification is potentially an important step in indicated prevention (see Chapters 38 & 46), as knowledge about symptoms of EDs in the community is low and accurate information is likely to improve detection by parents in particular. Further, a low level of ED mental health literacy is one reason why treatment seeking for EDs, especially bulimic disorders, is low (Hart, Granillo, Jorm, & Paxton, 2011). In addition, fear of stigma also contributes to low treatment seeking, and it has recently been shown that greater knowledge is associated with lower stigma related to EDs (Hepworth & Paxton, 2007).

Early identification may be facilitated by a number of means, including screening young people (see Chapter 38) and teaching parents to recognize early signs of EDs. Both approaches were adopted in March 2013 by the Commonwealth of Virginia (Virginia State Government, United States), which passed a law effective July 1st, 2013 (HB 1406; Virginia State Government, 2013), following advocacy by NEDA. This law requires all public schools in Virginia to provide parents of students in grades 5–12 with information about EDs annually to facilitate early identification. In addition, this law requires the Virginia Department of Education and Health to develop guidelines for a voluntary ED screening program most likely based on use of the SCOFF screening instrument (Morgan, Reid, & Lacey, 2000; see also Chapter 38). This information will be available to schools so that all school boards that wish to implement an ED screening program will be empowered to do so. This resource is scheduled to be available for the 2013–14 school year. This is a novel government resource and, if implemented in a way that has safeguards against possible stigmatization of students with symptoms, could be of enormous value. Evaluation of this social policy intervention is needed and will be of great interest.

Fashion and Advertising

Legislation and Regulation

Although governments have legislation and regulation at their disposal to make certain activities illegal, this approach has not been widely used as a means to enact social policy to prevent body image and eating problems. However, in 2012 Israel passed a law, effective at the beginning of 2013, to make it illegal for the fashion or advertising industries to use a model with a body mass index (BMI) of less than 18.5. For example, it would be illegal to use a model who is 5′10″ (1.88 m) and weighs l28 lb (58.1 kg) or less. This law also requires that advertisements that depict a person's image that has been graphically manipulated for the purpose of narrowing body measurements should include a clearly recognizable clarification regarding the use of such graphic manipulation. Although initial findings regarding disclaimers about graphic manipulation suggest that they are not helpful (Tiggemann, Slater, Bury, Hawkins, & Firth, 2013; see also Chapter 29), it has been argued that fashion models frequently become role models, especially for young people, and thus, if they are very thin, they promote an unhealthy and unrealistic portrayal of beauty that young people may seek to emulate with negative consequences. In particular, young people who believe they fail to

achieve the thin ideal are vulnerable to developing body dissatisfaction and related problems. Consequently, this legislation is designed to minimize the negative impact on body image and self-esteem of exposure to advertisements depicting models as extremely thin, leading to a reduced risk in the Israeli population for development of EDs (Levush, 2012).

Another piece of legislation that has been enforced in the interests of reducing pressure on women to achieve an unrealistic body ideal is the U.K. Code of Non-Broadcast Advertising, Sales Promotion and Direct Marketing. Of particular relevance, this law insists that marketing communications must not be materially misleading or be likely to mislead consumers by exaggerating the performance of a product (Committee of Advertising Practice, 2010). This is especially relevant in relation to postproduction techniques such as retouching photographic images, where retouching is likely to mislead. In the United Kingdom in 2012 an advertisement for an "age defying" coverage make-up was forced to be withdrawn because it used an image of a celebrity that had clearly been retouched to remove facial lines (wrinkles). Although it has not been used widely, consumer protection of this kind can protect the public against the worst excesses of the advertising industry.

Voluntary Codes and Positive Recognition through Awards

Governments can also have an impact by taking an opinion leadership role, and this approach has been used in a number of countries around the world to try to modify actions of the fashion and advertising industries. One strategy to achieve this has been the promulgation by governments of self-regulation codes and charters, as seen in Italy (2006) and France (2008). Another important example was the launching in 2009 of the Voluntary Québec Charter for a Healthy and Diverse Body Image by the Ministry of Culture, Communications and the Status of Women of the Québec Government, Canada. The goal was for the Charter to act as a health promotion tool that would actively engage industry and industry leaders by outlining positive actions and principles that could be endorsed by organizations and individuals. The Charter briefly describes the need for change and the leadership that fashion, advertising, and media industries could provide. It then requests individuals and businesses to pledge support for seven statements, including that they will:

- "Promote a diversity of body shapes"
- "Encourage healthy eating and weight control habits"
- "Discourage excessive weight control practices for appearance modification"
- "Refuse to subscribe to aesthetic ideals based on slimness"
- "Act as agents of change in order to promote healthy and realistic practices and images regarding the body" (Ministère de la Culture, des Communications et de la Condition Féminin, Gouvernement du Québec, 2012).

By engaging the community in this positive way the Québec Government received endorsement from over 15,000 individuals and organizations. Importantly, the Charter was followed with an ongoing implementation phase during 2010–13 in which there was to be a focus on working with media, fashion, and other industries.

The success of the Charter in reaching a wide audience has been demonstrated by a unique study of its population reach, acceptability, and perceived potential among Québec adults (Gauvin & Steiger, 2012). In this research, 1,003 residents of Québec over 18 years old were randomly contacted and asked to complete a phone survey 6 months after the launch of the

Charter. More than 35% of the sample recognized the Charter, 33.7% were very favorable toward personally adhering to the Charter, and 32.7% perceived the Charter as having the potential to sensitize people to the negative consequences of disordered eating. Women were 77% more likely than men to recognize the Charter (Gauvin & Steiger, 2012). Overall, these results are very encouraging in light of the fact that there was not mass advertising of the Charter but rather only reporting of a number of launch and media events. Thus, the Charter appears to have raised awareness of body image problems. However, the extent to which it brought about positive change has yet to be determined.

A related approach has been adopted in Australia, first by the Victorian State Government in 2008 and then by the National Government in 2009, which released the *Voluntary Industry Code of Conduct on Body Image* (Australian Government, 2009). The Code was recommended by a group established by the Australian Government: the National Advisory Group on Body Image (2009). The Advisory Group was made up of representatives of magazine, fashion, and advertising industries, clinicians and advocates for people with body image and EDs, and researchers with expertise in body image. The Code outlined principles to guide these industries in order build on and further encourage the positive steps already being taken within the fashion, media, and advertising industries to promote positive body image. The Code articulated principles to guide these industries in creating advertising content and messaging that support the following:

- development of positive body image and of realistic and healthy physical goals among consumers;
- selection of diverse and healthy models;
- availability of a wider range of clothing sizes in retail fashion;
- use of realistic and natural images of people; and
- disclosure when images have been digitally manipulated (Australian Government, 2009).

The Code appears to have been particularly valuable in encouraging teen magazines in Australia to take responsible actions to promote positive body image. Both *Dolly* and *Girlfriend*, magazines with a very large reach into the teen market, have consistently implemented actions that support the principles outlined in the Code, using diverse and healthy models and not digitally manipulating images. In addition, a number of fashion houses have extended the sizes in which their fashion garments are available and use diverse models in advertising.

To support the Code, the National Advisory Group on Body Image (2009) recommended the establishment of the Positive Body Image Awards to recognize the constructive, healthy steps taken by the fashion, media, entertainment, and advertising industries to adopt the principles outlined in the Code and thus encourage change in these industries. These awards were offered for the first time in 2012 and held again in 2013, for which a health and well-being industry category was also added. The awards are presented amidst publicity and the winners of awards are able to display a Positive Body Image Award Symbol for advertising purposes. In 2012, *Dolly* magazine received the award in recognition, as noted above, of its strong commitment to promoting positive body image messages to young people through business policies and practices in line with the Code. In particular, it has a policy of non-manipulation of images and identification of those images as nonmanipulated, and aims to present healthy images of young women by including nonmodels and plus-size girls in photo shoots. In 2013 the overall winner of the awards was the fashion retailer, Sportsgirl, in recognition of a range of policies supporting positive body image, including their body image

training of staff and their sale of fashion clothing in a diverse range of sizes. In addition, awards were made to *Girlfriend Magazine* for policies restricting manipulation of images, and to the ISIS Eating Issues Centre for their body image prevention intervention. An increase in the number of applications suggests increased awareness in fashion and media industries of the need for them to take greater responsibility for their role in the development of body dissatisfaction and EDs.

In 2012, the British Government, again through the Campaign for Body Confidence, also presented Body Confidence Awards to recognize and honor positive body image initiatives by sectors of the advertising, beauty, media, retail, and education industries, as well as advocates and celebrities. The awards were presented in a high-profile setting and served to raise awareness of the potential for both positive and negative influence of industries and celebrities, as well as to reward industries and individuals that have promoted diversity and self-esteem (Campaign for Body Confidence, n.d.). Winners included Dove for communicating a positive body image message to girls, a cosmetics company (Boots) for their decision to eschew retouching and for celebrating the ideal of real women, and a retailer (Debenhams) for an inclusivity campaign featuring imagery that is inspirational and realistic in its employment of models that are older, curvier, and physically disabled. The All Walks Beyond the Catwalk Fashion Award went to Mark Fast, a designer at London Fashion Week for introducing the mix of realistically proportioned models into the show.

Although there has been little research into the impact and reach of these social policy actions, with the exception of the Québec study, it is likely that these charters and high-profile events are at a minimum raising awareness of the potentially positive roles that the media and fashion industries could be playing. It is hoped that these social policy actions will pave the way for greater change in these influential industries.

Research Questions

One of the positive features of the recent social policy activities described in this chapter is that they have implicitly or explicitly endeavored to have an impact on risk factors for the development of body image or EDs. For example, internalization of unrealistic body ideals promoted in media have been shown to play some part in the development of body dissatisfaction (see Chapters 21, 29, & 44), so trying either to bring about change in media images (e.g., Israeli legislation, Codes of Conduct) or to build media literacy (see Chapter 45) to enable young people to challenge the images (e.g., Media Smart UK) are worthy approaches to trial. But do they work and have the most effective approaches been selected? Although there are notable exceptions, as described above, most social policy initiatives have not been extensively evaluated.

One reason for the lack of evaluation of social policy actions is that most researchers in the body image and ED fields are psychologists; although they have skills in examining the impact of programs on an individual level, they are less skilled at population approaches required to evaluate policy actions. As argued by Austin (2012), the skills of public health practitioners are needed to inform the population research required to move this area forward (see Chapters 41 & 47). Further exploration of the reach and acceptability of interventions is required, similar to that conducted by Gauvin and Steiger (2012). This is especially relevant in evaluation of social marketing campaigns.

In relation to education resources it is clearly essential to establish their usefulness on a small scale (see Chapter 42). However, the next step is to examine the extent of uptake and more

importantly the frequency with which programs are faithfully implemented. Similarly, when an educational program is made available, how many people actually implement it and when implemented how often is it conducted in the manner intended?

Although it is clear that innovative evaluation techniques need to be employed, in many cases failure to evaluate an intervention has been more an issue of neglect and lack of funding. For example, it would not be intrinsically difficult to identify what proportion of a random selection of Australian government schools had attempted to make school policy changes in line with the checklist for body-image-friendly schools and to examine body image in those schools. However, it would require commitment and funding. The recent introduction of ED mental health literacy for parents and screening in Virginia offers a clear and exciting opportunity for evaluation of early referral rates for EDs in that state. It is to be hoped that funding will be made available for this.

Finally, it is essential for research to keep up with our rapidly changing world and to provide tools for social policy that are contemporary. In this respect, it is essential for us to develop a greater understanding of the effects of social networking on body image (see Chapter 31) and to develop and evaluate prevention strategies that can be translated into effective social policy.

Conclusions and Future Directions

When stepping back and taking a bird's eye view of social policy and prevention of body image problems, including EDs, around the globe, such as interventions at the population level, interventions in school and educational settings, educational resources, parent education, screening, and policies for fashion and advertising, a number of factors stand out. It is remarkable that all the activities that I have identified have taken place within the last 10 years, and the great majority have occurred during the last 5 years. Thus, social policy in relation to body image and EDs is an extremely new field but one that is expanding. Being in its infancy, the field is still at a tentative and experimental phase, with many diverse options being explored. These are important first steps, but at this stage there is very little evidence that the policies that have been implemented are having a meaningful impact on body image problems, let alone EDs. Not only is this disappointing, it poses difficulties in further engaging governments that are keen to see tangible results. Recognizing that we are in the early stages of development, rather than being discouraged, researchers and activists in our field need to continue to explore creative social policies to ensure our field matures (see Chapter 66).

Another aspect of social policy for body image and the prevention and treatment of EDs that stands out when surveying the whole field is the piecemeal nature of activities, so it is important to consider what might reasonably be expected of any one social policy action. Government actions tend to be sporadic across time when it is likely that consistent action is required. For example, social marketing to increase media literacy has been explored in Australia but over such a short time that no impact could be expected. However, the piecemeal nature of activities is especially notable within nations. Although many governments are taking an action in one or two areas, I am aware of none that are taking meaningful action across the range of possible social policy spheres. In such a complex field as body image, it is highly unlikely that anything other than a comprehensive and concerted approach to social marketing will be effective. Whilst reducing media pressure to conform to an ideal is certainly crucial to reducing these problems, so too is bringing about change in industry, family, and peer environments. To achieve this, social policy interventions will need to be implemented simultaneously in many different settings and across childhood, adolescence, and adult life.

Having said this, it is also clear that governments vary enormously in their commitment to prevention of body image problems and EDs. Consequently, our field still has a great deal of work to do in gaining recognition of the serious consequences of body dissatisfaction and the consequent need for social policy action and government investment. Thus, we must continue to work closely with governments to raise their awareness of body image issues and to ensure they gain the recognition they deserve (see Chapters 47 & 66). The recent period of development of social policy for body image and EDs has also coincided with a period of financial difficulties, austerity, and instability around the world. In such a climate, governments tend to focus less on mental health issues in general. However, there are signs that economic improvements are underway, and consequently it will be very important to continue pressure on governments to implement effective policies over the coming years.

Exciting steps in social policy for body image and EDs have been taken by many governments around the world. It is now time for governments to learn from each other and identify and implement ways in which sweeping rather than piecemeal changes can be made, then to evaluate the outcomes with the wide range of tools available to us.

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Integration of Obesity and Eating Disorders Prevention: A Holistic Approach to Wellness in Elementary Schools

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This chapter focuses on theory-based and evidence-based recommendations for policies and programs providing integrated primary and secondary prevention (see Chapter 41) for the continuum of eating problems. We focus on the elementary-school age population, given our expertise with this age group, and because this developmental stage is critical in establishing life-long eating and exercise habits, and body esteem. Moreover, due to the significant public health concern about eating and exercise, schools and parents appear more receptive to prevention efforts addressing these concerns within this age group than ever before. Further, our professional and anecdotal experiences suggest that obesity prevention (in some form) is a reality in many schools. Thus, it is imperative for eating disorder (ED) researchers to be at the forefront of these efforts in order to minimize their potential for iatrogenic effects, and to maximize their beneficial impact on the continuum of eating habits and related behaviors and attitudes.

Rationale for an Integrated Approach

Several authors have advocated for the integration of obesity and ED prevention (Neumark-Sztainer, 2003; Sánchez-Carracedo, Neumark-Sztainer, & Lopez-Guimera, 2012). Simultaneously addressing both conditions (and subthreshold manifestations) is supported by empirical evidence suggesting that eating- and weight-related problems share several risk factors, frequently co-occur, and often become more severe over time (Neumark-Sztainer, Story, Hannan, Perry, & Irving, 2002; Neumark-Sztainer, Wall, Larson, Eisenberg, & Loth, 2011). For example, compared with normal weight adolescents, overweight and obese

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adolescents in Project EAT reported more unhealthy weight control behaviors, and more binge eating. They were also more likely to continue these behaviors into adulthood (Neumark-Sztainer et al., 2002; Neumark-Sztainer, Wall, Story, & Standish, 2012). Other studies have highlighted the increased risk that overweight adolescents have for both binge eating disorder (BED; Fairburn et al., 1998) and bulimia nervosa (BN; Fairburn, Welch, Doll, Davies, & O'Connor, 1997) in adulthood.

Although these data make a strong case for simultaneously addressing the risk factors these conditions share, the ED and obesity fields have historically targeted different prevention objectives (Neumark-Sztainer, 2012). ED prevention typically focuses on specific risk factors and symptomatology such as body dissatisfaction, thin-ideal internalization, and unhealthy weight control behaviors (see Chapters 42, 44, & 45). In contrast, obesity prevention usually focuses on healthy lifestyle behaviors such as nutrition and physical activity, and pays less attention to body image, size acceptance, media literacy, and the dangers of unhealthy weight control behaviors (Sánchez-Carracedo et al., 2012). Inattention to shared risk factors could lead to conflicting messages and inadvertently promote unhealthy behaviors, particularly in vulnerable individuals (Carter & Bulik, 2008).

Supporters of an integrated approach to obesity and ED prevention recommend that programs promote healthy, sustainable eating and activity behaviors and positive body image, while avoiding a focus on weight (Sánchez-Carracedo et al., 2012; see Chapter 47, but cf. Chapter 24). Integrating prevention efforts for the range of eating- and weight-related conditions has the practical benefit of maximizing resources. Moreover, in the last few years, concerns about the increases in the prevalence of obesity among children have led to national calls for action (Koplan, Liverman, & Kraak, 2005). Thus, although few empirically supported approaches to obesity prevention in elementary-age children are available, efforts to improve students' nutrition and exercise habits are on the rise (Carter & Bulik, 2008; Justus, Ryan, Rockenbach, Katterapalli, & Card-Higginson, 2007). It is therefore vital that ED prevention advocates work directly with groups interested in obesity prevention, to ensure that these efforts are implemented in a manner that does not inadvertently lead to the encouragement of overly restrictive eating behaviors or weight discrimination (O'Dea, 2000).

Why the Concern about Obesity, Nutrition, and Related Attitudes in Children?

Nearly one third of children ages 6 through 11 are overweight or obese, and these proportions are even higher among African American and Hispanic youth (Ogden, Carroll, Curtin, Lamb, & Flegal, 2010). Obesity and overweight status are defined using an individual's body mass index (BMI). Children with BMIs between the 85th and the 95th percentiles for their age and sex are considered overweight, while children with BMIs at or above the 95th percentile are considered obese (Kuczmarski et al., 2002). Overweight children are over 20 times more likely to become obese adults compared with peers (Whitaker, Wright, Pepe, Seidel, & Deitz, 1997). Moreover, poor dietary habits and low activity levels are negatively correlated with academic performance, as well as physical and emotional well-being (Kiess et al., 2001; Schwimmer, Burwinkle, & Varni, 2003). Further, poor dietary habits and low activity levels increase risk for many chronic diseases, even among individuals who are not overweight (Nicklas, Baranowski, Cullen, & Berenson, 2001), suggesting that a focus on improving children's eating and exercise behaviors is far more important than targeting weight.

However, traditional obesity prevention programs are time- and resource-intensive, and not universally available, limiting their impact on some of the groups most vulnerable to obesity, including lower-income families (Ammerman, Leung, & Cavallo, 2006). Thus, many have recommended that obesity prevention programs be implemented in schools, where they can reach a large number of children from all demographic backgrounds (Story, Nanney, & Schwartz, 2009).

Schools as Prevention Settings

Children spend more than half their waking hours at school and consume at least one meal per day there. School-based approaches to wellness can be provided at little to no cost to families, and integrated into the existing school environment in which children make dietary choices (Story et al., 2009; see also Chapter 42). However, schools are also environments in which students are faced with unhealthy nutritional choices multiple times per day. Unhealthy foods are sold in vending machines and school stores, and are promoted in fundraisers. Classroom parties regularly include unhealthy food choices, and many schools continue to use unhealthy foods to reward good behavior, as well as restrict recess as a form of discipline (Finkelstein, Hill, & Whitaker, 2008; Story et al., 2009). Given these issues, students likely receive conflicting messages about making healthy dietary choices, the role of food as nutrient (vs. reward or comfort), and the importance of activity.

Policies Targeting Elementary-Age Children

A range of interventions, including new or more strongly enforced school policies, have been proposed to improve children's health, including enhancing the school food environment, encouragement of fruit and vegetable (F&V) consumption in school cafeterias, notifying parents of their children's body mass indices (BMIs), and nutrition education (Brownell, Schwartz, Puhl, Henderson, & Harris, 2009; French & Stables, 2003; Koplan et al., 2005). Although these policies are likely promoted by those interested in obesity prevention, it is essential to consider their potential impact on the full range of eating problems, including the possibility of iatrogenic effects. In the following sections, we review evidence for a small number of the least intensive, yet promising, of these policies that are the most likely to be disseminated, given their feasibility and sustainability in this era of fiscal austerity. Please note that this chapter focuses specifically on school-based, policy-related interventions targeting young, elementary-age students. There are several promising programs targeting older students, such as Planet Health (Austin, Field, Wiecha, Peterson, & Gortmaker, 2005; Gortmaker et al., 1999), which might be translatable to younger students; however, a review of these interventions is beyond the scope of this chapter.

Improving School Food Environments

Improving the quality of food available at schools remains a vital component of health promotion efforts (Briefel, Crepinsek, Cabili, Wilson, & Gleason, 2009). Experts have argued that changing the school food environment is a cost-efficient and effective method for improving dietary intake compared with resource-intensive nutrition education (Brownell et al., 2009;

Gearhardt et al., 2012). Recent research suggests that enhancing the quality of foods provided in elementary school settings is associated with both a reduction in the incidence of obesity, and decreases in sedentary activity (Foster et al., 2008).

One particularly important area of focus is the sale of competitive foods, that is, "foods that are available in schools but are not part of US Department of Agriculture (USDA) school meals" (Briefel et al., 2009, p. S92). These foods compete with those included in the National School Lunch Program (NSLP) and are sold à la carte in the cafeteria, in vending machines, and in school stores (Wharton, Long, & Schwartz, 2008). Competitive foods are typically highly processed, high in artificial coloring, fat, sugar, and carbohydrates, and low in vitamins and minerals (Kramer-Atwood et al., 2002). However, these foods often generate revenue for schools (Briefel et al., 2009). Desserts and snacks are the most common competitive foods consumed daily in elementary schools; almost one third (29%) of students consume these foods during an average elementary school day (Fox, Gordon, Nogales, & Wilson, 2009). The availability of competitive foods in schools increased greatly in the last several decades (Wharton et al., 2008), mirroring the increase in obesity rates (Ogden et al., 2010). Data from younger students are limited, but research has consistently found that the availability of competitive foods is associated with lower F&V consumption and higher fat intake among middle-school students (Cullen et al., 2007; Kubik, Lytle, & Story, 2005).

Turner and Chaloupka (2012) assessed the availability of competitive foods in a nationally representative sample of elementary schools as part of their investigation of the effects of the 2006 federal law requiring school districts to develop wellness policies (Serrano et al., 2007). Data indicated that in 2009–10 (compared with 2006–07), fewer public schools had a "pouring contract" with an outside beverage vendor; more schools offered wholegrains and low-fat milk; and more public schools placed restrictions on food-related fundraising. However, 58.4% of schools surveyed at the second data collection point had *no* food-related fundraising restrictions. Finally, many policies and practices did not change significantly between the two time periods. For example, only one third (33.5%) of schools in 2009–10 indicated that they did not "use food as a reward for academics or behavior" (vs. 29.1% in 2006–07; Turner & Chaloupka, 2012, p. 1382). Turner and Chaloupka concluded that there has been "minimal progress" (p. 1386) in improving the school food environment, despite the federal mandate.

One factor that has likely limited progress in removing competitive foods from schools is the often-cited argument that sales of these items contribute to school revenues, a particularly salient concern in the current financial climate (Story et al., 2009). However, recent data suggest that removal of competitive foods does not negatively impact school revenues (Wharton et al., 2008). Thus, it seems appropriate for professionals, parents, and concerned citizens to advocate for the removal of competitive foods from schools, as they appear to offer little, if any, financial benefit to districts and, due to the poor nutritional value of competitive foods described above, have the potential to negatively impact all students' health.

Low-Intensity Cafeteria-Based Interventions

In addition to removing unhealthy foods from the school environment, it is important to increase the availability and consumption of healthy foods within this setting. The majority of research on school food environments has involved older students (middle- and high-school age youth; Kubik et al., 2005; Schwartz, Novak, & Fiore, 2009). However, a few studies have suggested that relatively low-intensity interventions implemented in the cafeteria setting could yield positive effects for younger children (Hendy, Williams, & Camise, 2005, 2011). For

example, Perry and colleagues (2004) investigated the effectiveness of a program (Cafeteria Power Plus) designed to increase F&V availability in the cafeteria through various methods, such as food presentation, verbal prompts, and tastings. They found that verbal encouragement positively influenced children's fruit consumption.

To address whether a lower-intensity intervention could positively influence fruit selection and consumption in elementary school students, Schwartz (2007) investigated the effects of cafeteria staff members' verbal prompts on dietary outcomes. This study included one intervention and one control elementary school. The same foods and beverage options were available in both schools. In the intervention school, cafeteria staff asked students, "'Would you like fruit or juice?'…if a child indicated that he or she did not…the food service worker did not prompt further" (p. 2). Results indicated that students at the intervention school were significantly more likely to both select and consume fruit (and juice) compared with students in the control school. Moreover, students ate nearly all of the fruit they selected. These are very encouraging results given the low-intensity, low-cost (and thus, high feasibility) of this intervention. Future research should include follow-up assessments to determine whether prompts continue to be sufficient to maintain high levels of fruit consumption over the longer term, and whether prompts could be extended to vegetables.

One challenge in improving F&V intake is that children often report not liking these foods (Satter, 1987). To address this issue, Lakkakula et al. (2011) implemented a cafeteria-based F&V tasting intervention. First- and third-grade students were offered tastings of four fruits and four vegetables, twice per week over an 8-week period (and during a 2-week follow-up period). Results indicated that the intervention enhanced preferences for these foods among students who initially reported disliking these items (Lakkakula et al., 2011). Moreover, these preferences were maintained at 4- and 10-month follow-up. This intervention is more intensive than that of Perry and colleagues (2004) or Schwartz (2007), but is low-intensity compared with many multicomponent school-based programs that have yielded modest effects (French & Stables, 2003). Moreover, unlike some other low-intensity cafeteria-based programs, this intervention also increased vegetable consumption, which appears to be less amenable to change.

Kids Choice (Hendy et al., 2005) is another cafeteria-based program targeting F&V intake among elementary school students. This program is more complex than the interventions reviewed above, and involved "small and delayed reinforcement, food choice, and conditions that encourage peer participation and modeling" (p. 252). Students were assigned to either a standard lunch, or to the intervention, in which they received a token reinforcement (a hole punch in a card) if one eighth of a cup of the targeted food was consumed. After three such reinforcements (i.e., after three hole punches) the card could be traded for a small prize. Admired peers explained the study to participants to enhance modeling effects. Two fruits and two vegetables were offered at each lunch. Students could select any of the targeted food items, although this was not required.

Results identified significant increases in F&V consumption among intervention children in all three grades assessed (first, second, and fourth). These changes persisted 2 weeks after the intervention but were not evident at 7-month follow-up; however, preferences did not drop below baseline levels even at 7-month follow-up (Hendy et al., 2005). A follow-up analysis of the Kids Choice program (Hendy et al., 2007) found that both overweight and normal weight children were equally responsive to this intervention. This finding is important, as it is vital to identify programs that benefit a broad range of children. Such programs are not only cost- and resource-efficient, but also avoid stigmatizing overweight children, as they place an emphasis on health rather than appearance.

A more recent iteration of Kids Choice (Hendy et al., 2011) targeted a larger number of health behaviors, including consumption of F&Vs, low-fat foods, and healthy drinks, and physical activity. In addition, the program's duration was increased from 1 to 3 months. An optional parent component was also included in this revision. Results indicated that all of the targeted behaviors significantly improved over the course of the intervention. Program participation was also associated with reductions in BMI percentile among overweight children after 3 months, yet overweight children regained weight 6 months later, again suggesting the need for ongoing intervention. A cost analysis indicated that the program is inexpensive to implement (< US$2/child per month), and can be successfully delivered by volunteers, making it feasible for many school districts. Parents, children, and school staff reported high levels of satisfaction with this intervention, although only a minority of parents completed the parent component, suggesting that intervention efforts might be most efficient when focused within the school setting. Future research could attempt to dismantle the intervention, perhaps focusing on the cafeteria component, as this is the most feasible and sustainable in most school settings, given its lack of interference with instructional time.

BMI Notification

In addition to making changes within the school setting, many policy-makers have advocated initiatives that reach beyond the classroom, such as informing parents about their children's weight status. Although there were no data supporting their initiation, BMI notification letters have become one of the most frequently highlighted universal policy initiatives in the fight to address childhood obesity. BMI notification is designed to inform and alert parents about their children's weight status (Kaczmarski, DeBate, Marhefka, & Daley, 2011). In 2005 the Institute of Medicine recommended that schools calculate children's BMI and report the information to parents as a strategy for preventing or slowing the rate of excessive weight gain in children (Koplan et al., 2005).

However, concerns persist about the potential of these notifications to cause peer stigmatization and shaming by parents. Moreover, providing parents with this information without also giving them strategies for discussing the issue with their children or for how to improve their family's lifestyle habits might contribute to use of inappropriate and harmful approaches to weight management (e.g., shaming, severe calorie restriction, punishment; Kaczmarksi et al., 2011). Parents and children need education to understand the importance of healthy eating and exercise. However, this policy is often being implemented without appropriate educational supports. For example, Madsen and Linchey (2012) found that in California school districts that have implemented BMI letter notification, 79% of letters reviewed provided students' BMI but only 12% provided an explanation of this value. Moreover, only about half of the letters provided guidelines for parents. These data might explain why, although the number of school districts using BMI notification has increased, there has been no effect on pediatric obesity (Madsen, 2011). Moreover, BMI notification does nothing to address the pernicious problem of the food environment or the dearth of community resources that could aid overweight and obese children and their families. It seems that both parents and children would be better served by initiatives that make it easier to make healthy choices, rather than focusing on weight.

Focus on Holistic Wellness

Although obesity prevention programs obviously place a significant emphasis on nutrition, exercise, and weight loss and BMI reduction as a primary outcome, a concern of ED advocates is that such programming can overemphasize rigid approaches to eating (e.g., rigid calorie-counting;

severe dietary restraint; dichotomizing of "good" and "bad" foods), promote unhealthy levels of exercise, and inadvertently encourage weight discrimination (Carter & Bulik, 2008; Neumark-Sztainer, 2012). Therefore, many health professionals call for attention to a broader picture of health that goes beyond nutrition and exercise and includes multiple components of wellness. Specific non-eating-related behaviors play a vital role in health, including metabolic functioning. In particular, sleep and emotional regulation have repeatedly been shown to influence students' mental and physical health, as well as their cognitive performance (Fallone, Acebo, Seifer, & Carskadon, 2005; Katz, McHorney, & Atkinson, 2000). The following sections review the importance of sleep and emotion regulation skills as components of wellness programming.

Emotion Regulation and Mindfulness Emotion regulation involves adjusting behavior in response to a feeling state (both positive and negative), physiological experience of an emotion, or an emotion-related goal (Eisenberg & Spinrad, 2004). The ability to regulate emotions is related to children's overall adjustment (Greenberg, 2006). Indeed, children who have difficulty with self-regulation of emotions, cognitions, and behaviors are at increased risk for social, behavioral, and emotional problems (Guerra & Bradshaw, 2008; see also Chapter 17). Thus, a broad range of prevention efforts have focused on enhancing children's emotional regulatory capabilities (Domitrovich et al., 2010).

Mindfulness (see Chapter 33), a technique that aims to enhance awareness of one's moment-to-moment experience, has been found to enhance self-regulatory skills (Katz et al., 2000). Ludwig and Kabat-Zinn (2008) have suggested that practicing mindfulness might influence health by reducing anxiety, enhancing motivation for lifestyle changes such as diet and physical activity, or altering biological pathways (e.g., neuroendocrine function, immune system, autonomic nervous system).

School-based mindfulness interventions targeting elementary students have yielded positive results. For example, Mendelson et al. (2010) evaluated a 12-week school-based mindfulness intervention with fourth- and fifth-grade students, and reported improved social stress responses including reduced rumination, intrusive thoughts, and emotional arousal. However, there are few randomized clinical trials evaluating mindfulness in younger children.

Mindfulness has gained attention in the obesity field in recent years, given the accumulating evidence linking stress and weight gain (Mechanick & Apovian, 2009). Chronic exposure to stress hormones is associated with changes in metabolism, appetite, and fat storage (Holmes, Ekkekakis, & Eisenmann, 2010). Further, emerging evidence supports the use of mindfulness-based techniques in ED treatment (Wanden-Berghe, Sanz-Valero, & Wanden-Berghe, 2011), and yoga practitioners report reductions in body objectification (Impett, Daubenmier, & Hirschman, 2006). Further, mindfulness interventions might be particularly effective in treating binge and loss of control (LOC) eating among children, as children with LOC eating use more maladaptive emotion regulation strategies in response to negative emotions than those without LOC (Czaja, Rief, & Hilbert, 2009).

Incorporating mindfulness into prevention efforts targeting children (see Chapter 33) offers opportunities to promote skills relevant to overall wellness, including behavior and academic performance. The broad, positive effects of this type of intervention could enhance schools' willingness to support this type of programming as part of a holistic wellness approach.

Sleep Empirical support for the importance of sleep to emotional and physiological functioning continues to accumulate (Gregory & Sadeh, 2012). However, the amount of sleep children

engage in has decreased significantly over time (Matricciani, Olds, & Petkov, 2012). This is concerning, as associations between shorter sleep duration and overweight have been reported in numerous studies (Gupta, Mueller, Chan, & Meininger, 2002; Nixon et al., 2008), and longitudinal research suggests that early-age shorter sleep duration is associated with the development of overweight and obesity (Reilly et al., 2005; Sugimori et al., 2004). In young children, inadequate sleep duration is also linked to decreased academic performance, daytime sleepiness, attention problems, poor impulse control, and impaired memory and executive functioning (Durmer & Dinges, 2005; Fallone et al., 2005).

Consequently, it is vital that healthcare professionals, parents, and schools attend to the sleep patterns and habits of young children. Interventions addressing sleep are not common in the elementary school setting. However, health and educational professionals interacting with families should promote positive sleep hygiene practices such as regular bedtimes and removing screens (televisions, cell phones, tablets, etc.) from the bedroom to promote quality sleep. Parental education is a feasible and highly effective intervention strategy for promoting proper sleep habits (Mindell, 1999). These interventions could focus on behavioral strategies such as self-monitoring, positive reinforcement, and limit setting. Schools can also use sleep and screen time logs to raise students' and parents' awareness of their sleep hygiene and related behaviors. These low-intensity interventions could further reinforce the message that holistic wellness is not just about weight. Rather, a broad range of factors, including emotional self-regulation, sleep, and eating and exercise behaviors, are relevant to lifelong well-being.

Conclusions and Future Directions

Although historically considered separate health issues, EDs and obesity share many risk factors, and the integration of efforts to prevent these problems makes practical and scientific sense. The promotion of universal, holistic wellness will help ensure that *all* children receive education about better overall health (see Chapters 33 & 42). These efforts can facilitate the creation and maintenance of daily environments in which it is easy for both children and their caregivers to make healthy lifestyle choices (see Chapters 37, 43, & 47). Programs do not have to be extremely time- or resource-intensive to yield beneficial results, as demonstrated by the research on cafeteria-based programming reviewed in this chapter. Professionals interested in preventing EDs and obesity should jointly advocate for enhancing children's access to high-quality food. Schools are an ideal place to begin this advocacy. In addition, including education on mindfulness and sleep hygiene in schools is important to optimize children's quality of life and long-term health.

In sum, school-based obesity prevention is happening. Advocates for ED prevention (Neumark-Sztainer, 2012) and promoters of health at every size (Bacon, 2010) should continue to be involved in these efforts to support implementation of best practices. This includes encouraging a focus on outcomes other than weight, advocating the prevention of size discrimination, and promoting healthy eating and exercise for a full range of health outcomes. Programs will be less likely to inadvertently promote size discrimination if they are holistic in scope, and do not focus on overweight children. Moreover, universal programs benefit the greatest number of children (see Chapter 41). Professionals should also continue to promote local and federal policies that make it easier for all families and students to make healthy lifestyle choices.

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Section IX
Issues in Treatment

50

The Eating Disorders Treatment Team and Continuum of Care: Saving Lives and Optimizing Treatment

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The Academy of Eating Disorders (AED, 2012) task force, formed in collaboration with the National Eating Disorders Association (NEDA) and the International Association of Eating Disorder Professionals (IAEDP), has identified a multidisciplinary team as a "core treatment component" and concluded that the team should include but not be limited to psychological, medical nursing, nutritional, and psychiatric services. In this regard, the American Psychiatric Association has identified a general trend toward patient-centered care, the centerpiece of which is a "team-based, patient-focused alliance involving all medical professionals: physicians, nurses, social workers, psychologists, and other members of the health care team" (Bernstein, 2011, p. 1023).

The aim of this chapter is to describe the treatment team approach to eating disorders (EDs) across multiple levels of care. Specifically, we will review the overall rationale for a team approach for treatment of EDs, and will then describe in detail the function of each team member, using hospitalized care as the model as a hospital treatment team is likely to be the most extensive and tightly functioning. We will briefly discuss comorbid conditions (see Chapters 15 & 54), and finally will describe how the team changes in members and operation through each level of care starting with outpatient care, the least restrictive treatment environment.

Support for a treatment team approach is indicated by the fact that EDs are complex, multifactorial problems with high rates of psychiatric and medical comorbidity (see Chapters 14, 15, 17, 52, & 54), high rates of service utilization across a range of medical and behavioral health settings, frequent use of high-cost services including medical hospitalization and psychiatric treatment in inpatient and residential treatment settings, significant recidivism, and elevated mortality rates (see Chapter 55; Sullivan, 1995). Not surprisingly, effective treatment requires a large time investment for all diagnostic categories. Studies suggest an average of 5 years for full recovery, although there is substantial variation around the average, and some individuals do exhibit chronic symptoms (Strober, Freeman, & Morrell, 1997) (see Chapters 12 & 55). Furthermore, it is recommended that particularly challenging patients be treated using a multidisciplinary team approach, with the majority of treatment

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occurring in outpatient treatment setting (Wonderlich et al., 2012). For these reasons the team approach represents an essential model for effective treatment of EDs.

The Team Concept

Conceptually, the team serves a number of essential goals including:

1 Accurate diagnosis of EDs and co-occurring psychiatric illnesses, as well as identification of symptoms that do not rise to the level of diagnosis but will likely influence treatment.
2 Medical assessment and stabilization (see Chapters 14 & 52).
3 Treatment education for patients and caregivers, and referral to the next level of care.
4 Identification of nutritional goals that are realistic and support normalization of eating behavior and weight/nutritional status (see Chapter 61).
5 Delivery of evidence-based psychotherapy (individual, group, and family) that takes into account specific patient characteristics.
6 Collaboration with the patient's family and support system.
7 Supporting the patient and family in adapting to changes in clinicians.
8 Providing continuity across multiple levels of care.

The duration of treatment may be a minimum of 6 to 12 months, but more commonly is a number of years or even decades.

An accurate diagnostic formulation using criteria in the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) is an essential part of the initial assessment of psychiatric illness and particularly so with eating disorders (see Chapters 8–16). If the presenting problem is an ED, determining whether the patient has anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED), or another specified feeding or eating disorder (OSFED), when there is considerable overlap between the symptoms of these conditions, is challenging but essential (American Psychiatric Association, 2013). Accurate diagnosis, particularly with atypical eating disorders (Allen, Fursland, Watson, & Byrne, 2011; see also Chapters 4, 11, & 13), will increase the likelihood of successful treatment recommendations and avoid unnecessary costs. The multiple patient contacts that occur within a treatment team are particularly effective in supporting a complete diagnostic assessment, and allow for a greater degree of behavioral observation than is typically available.

Treatment goals for AN must focus on weight gain as an essential element of recovery (Wagner et al., 2006; see also Chapters 61 & 64) and therefore require a member of the treatment team to be monitoring the weight restoration progress. While it is true that normalizing nutrition is not recovery in and of itself, weight gain and normal eating are essential components of recovery. For the male or female with AN, weight needs to be normalized based on an individualized recommendation that is agreed upon by members of the treatment team for each patient. Some individual preferences can be accommodated, but meal plans must be designed to achieve nutritional goals and incorporate adequate amounts of macro- and micronutrients.

Further, nutritionists experienced in EDs recognize that a stated dislike for many foods and food categories and a desire for a lower weight are characteristic of all individuals with EDs (see Chapter 61). In the case of individuals who are not underweight and have problems with binge eating, recommendations for achieving and maintaining a healthy weight range and level

of activity for fitness or athletic competition support the recovery process and overall wellness. Grocery shopping, cooking, and dining with others are also important aspects of nutritional recovery. Co-occurring depression, anxiety disorders, and substance abuse (see Chapters 15 & 54) can undermine patients' ability to be compliant with meal plans. For example, although many patients with EDs have discomfort eating in public settings such as a group dining hall, for individuals with social phobia these fears are amplified and may be present with dieticians and other treatment team members.

Medical assessment and stabilization are required to address emergent medical needs such as cardiac abnormalities, including bradycardia and arrhythmias, and electrolyte disturbances including low potassium and sodium (Mehler, 2011; see also Chapters 14 & 52). Most often screening can be accomplished with a medical history and physical exam, followed by comprehensive laboratory testing including a hemogram, electrolytes, chemistry panel, urinalysis, electrocardiogram, testing of thyroid hormone, as well as a pregnancy test for females and serum testosterone for males. Major factors contributing to medical morbidity and mortality relate to electrolyte and cardiac abnormalities that, ominously, are not typically associated with physical distress. Therefore it is essential that a comprehensive medical examination be a part of the initial assessment of a patient because it helps to determine the appropriate level of care as well as medical follow-up that may be necessary (see Chapters 14 & 52).

The medical team also needs to monitor ED patients who continue to exhibit symptoms such as vomiting or laxative/diuretic abuse while in treatment in order to monitor and treat electrolyte abnormalities. Medical assessment can also identify other medical needs that are less acute, such as ongoing gastrointestinal complaints (e.g., gastroesophageal reflux disorder, or GERD), constipation, and dental concerns (Hermont, Pordeous, Paiva, Abreu, & Auad, 2103). Constipation is a common complaint that may increase the difficulty with normalizing food intake or abstinence from purging. Abnormalities in bone density, including osteopenia and osteoporosis, may influence physical activity during treatment. Finally, menstrual or hormonal irregularities may affect or continue after weight restoration. For medically compromised patients, the team approach is essential (Mehler & Anderson, 2010), as patients who must be admitted to a medical hospital during psychiatric treatment are unlikely to be monitored for restrictive eating, binge eating, purging, and/or excessive exercise.

Evidence-based psychotherapy is an essential part of the recovery process and includes individual and group cognitive-behavioral therapy (CBT; see Chapter 56), and family-based therapy (FBT; see Chapters 53 & 60), particularly for adolescents with AN. Other evidence-based therapies include dialectical behavior therapy (DBT; see Chapter 57) for patients with BN with or without impulsive behaviors (Federici & Wisniewski, 2013), and exposure and ritual prevention (ERP) for patients with co-occurring obsessive-compulsive disorder, social phobia, or food phobias such as fear of choking on foods (Simpson et al., 2013). The effectiveness of therapies is enhanced by the extent to which clinicians possess the skills and training to adhere to the core principles of the treatment (Lowe, Bunnell, Neeren, Chernyak, & Grederman, 2011), as well as the ability to establish a collaborative working relationship with patients.

Ideally, the psychotherapist can help patients to process and accept decisions and recommendations in a supportive way, as well as mediate conflict within the treatment team. The team approach is particularly helpful because difficulties in the therapeutic alliance may arise if therapists take on expanded roles related to nutritional goals, weighing patients, or recommendations for hospitalization or medications. In other words, it is important that psychotherapists are skilled in and enjoy individual, group, and family therapy and not assume that they can or should perform roles of other team members. Consequently, a clear, systematized

approach to team member collaboration with family and/or nonfamily support providers is critical. This is a requirement for treating minors, who must have a guardian to provide informed consent for treatment. For adults, family communication provides a mechanism for explicitly articulating treatment goals, expected progress, and informed decision-making. For example, family communication is helpful when there are expected changes in the intensity of treatment, or life decisions such as moving out of the home, getting a job, or attending school. Such systematized collaboration can also reduce "caregiver burnout" for caregivers who have provided long-term support for an individual with an ED.

Finally, the team needs to be adaptive, because for a variety of reasons, team members will change over the course of treatment for an individual. Effective and integrated communication, generated within a team-based plan, will encourage patients and family members or caregivers to experience the treatment team as continuous, cohesive, and collaborative. Thus, disruptions should be minimized if there are changes in therapists, physicians, or dieticians. Past treatment, both positive and negative, is a form of treatment team change that most patients will have experienced. Consequently, it is important for the current treatment team to take previous treatments into account when planning the current treatment and preparing the patient for a team member change.

Members of the Treatment Team

General Requirements

As in athletics, business, and especially healthcare, ED treatment team members need specialized skills, the ability to work together, and acceptance of a common goal or goals for the patient. EDs are a multisystem illness, and thus all team members must be able to collaborate and effectively communicate across levels of care (see Chapter 53), since it is not realistic to expect that any single team member will have knowledge of the myriad of symptom presentations at different levels of severity. This requires ongoing assessment of varying levels of patient motivation, evolving interventions, and clear documentation at each stage. Still, it is important that the overall treatment approach remains as consistent as possible across different levels of care to avoid patient confusion or having to "restart" during transitions. It is especially helpful to essentially broaden the team for a brief period both before and after transition times to include providers across levels of care. This practice makes successful transitions more likely for providers, patients, and family or support individuals.

Team Leader

Probably the most critical member of the team is the "team leader." This is the member of the team who is taking ownership for identifying the treatment goals, facilitating communication between members of the team, the patient, and family members, and documenting treatment progress. Although the team leader can be from any discipline, important qualities include a passion for ED treatment, excellent communication skills, sufficient experience with the population, and a track record of effective treatment leading to recovery for a number of patients. Typically a licensed masters or doctoral level counselor or psychologist, or medical doctor can function in the role of the team leader. The team leader must be able to lead the team through challenges that arise during the treatment process, working collaboratively with

a team that will include a variety of disciplines and educational backgrounds. He or she also must be able to develop a trusting, supportive, and collaborative relationship with patients, and have effective methods for managing ambivalence toward recovery, which is often characteristic of this illness.

As mentioned previously, treatment is often of a long duration, and team leaders need to be able to work with other providers throughout this process, orient new team members, and provide ongoing consistent communication and resources to family members. The team leader can play a crucial role in helping patients and families make decisions relative to life changes such as attending college, living independently, or returning to work, based on objective indicators of readiness as well as on available resources for treatment or support in new environments. Such life changes typically involve increasing physical distance from family members or nonfamily support providers. The typically chronic course of this illness (see Chapter 55) does require the creation of a clear plan for patient monitoring and development of agreed benchmarks for future step-up or step-down treatment changes. The team leader has a critical role in establishing agreement on the plan with the patient and their support providers, and dealing with challenges such as long-standing poor family communication (see Chapter 53), or frustration with the pace or progress of recovery.

Diagnostician

Diagnostic accuracy for EDs is essential to direct treatment (see Chapters 8–16). The role of a licensed psychiatrist or psychologist is to conduct comprehensive psychiatric assessment to determine psychiatric diagnosis, which typically includes more than one *DSM-5* (American Psychiatric Association, 2013) diagnosis. This assessment also includes a determination of psychiatric stability and safety for the proposed level of care and thus can greatly affect third-party reimbursement for treatment. For example, while ED clinicians are well aware that OSFED does not represent an ED of any less severity, often insurance companies will be less likely to support treatment for OSFED as opposed to AN, BN, and now, with *DSM-5* (2013), BED. An accurate, complete, and consistent diagnosis can avoid potential pitfalls over what may be a long course of treatment. While most licensed psychiatrists and psychologists are fully competent to diagnose ED and co-occurring psychiatric conditions, for complex patients, full diagnosis may indicate referral to other assessment specialists, such as neuropsychologists, for comprehensive testing.

Psychiatrist

In addition to supporting the process of establishing an accurate diagnosis, the psychiatrist has a critical role to play in rationalizing and managing the patient's medication regime (see Chapter 59). While in some cases patients may refuse any psychotropic medications, in most cases patients have been prescribed many different medications from a variety of providers, and may have experienced varying levels of medication management or follow-up. The psychiatrist must review the patient's complete medication history and determine appropriate changes, which may be additions or subtractions. The psychiatrist is also able to determine if there have been past difficulties with compliance, and advise the treatment team on how to manage noncompliance. Good communication with previous psychiatric providers is essential, as in many cases patients may return to their previous providers for follow-up. Finally, as mentioned, patients with EDs perforce have physical conditions

(see Chapters 14 & 52) that may be partially or fully treated with medication, and patients often have other somatic issues that may or may not be related to nutritional status. The psychiatrist must manage indications and contraindications of the patient's entire physical and psychological medication regime (see Chapter 59).

Psychiatric Nurse

In hospital settings, psychiatrists work with psychiatric nurses, who are able to have more frequent contact with patients, and who assist the psychiatrist by providing additional patient observation and assessment. In hospital or residential settings, nurses are able to monitor medication compliance and rapidly notify physicians of side effects that require a rapid response (see Chapter 59). Further, psychiatric nurses are able to guide other staff in recognizing medical issues, and provide communication links with pharmacists and other outside providers.

Dietician

The registered dietician (see Chapter 61) works individually with each patient to (a) conduct a comprehensive nutritional assessment that determines weight recommendations; (b) prescribe an individualized meal plan; and (c) provide ongoing assessment of nutritional progress, including monitoring of weight and normalization of eating. In addition, the dietician provides education about nutrition, guides experience with food preparation, and helps patients gradually reintroduce feared foods into their diet. In residential, inpatient, or partial hospital settings where food is provided, the dietician supervises the work of diet technicians in the kitchen where patients' meals are prepared. In outpatient settings, the dietician can work with families to assess how meals are handled in the home, and in the process educate families on the use of a nutritional exchange system, intuitive eating, ethnic or religious dietary practices, and personal preference. The dietician should be knowledgeable about EDs and be able to collaborate with other treatment team members to ensure a cohesive approach to weight goals and normalizing eating. Finally, dietary recommendations and progress toward nutritional goals must be documented and shared with other team members to allow them to reiterate to the patient and family the importance of nutritional recommendations, as well as to provide support for insurance or third-party payers who are providing financial support for treatment.

Individual and Family Therapists

With proper training, any of the following professionals may serve as individual and/or family therapists: psychiatrists, clinical or counseling psychologists, and clinical social workers, Studies suggest that valued characteristics of the individual therapist include a high level of skill and competence for EDs, the ability to establish an effective and positive therapeutic alliance, and the skills to work well with others (Gulliksen et al., 2012). Although weight gain for patients with AN and reducing abnormalities of food intake and compensatory behaviors provide the foundation for recovery, changes in cognitive symptoms, reducing emotional distress, and developing healthy coping skills are ultimately necessary for full recovery from an ED (Tozzi, Sullivan, Fear, McKenzie, & Bulik, 2003; see also Chapter 64). Given our current knowledge base in the field of EDs, cognitive-behavioral therapy (CBT) has the most evidence as the primary component of effective individual and group therapy across the spectrum of ED diagnoses (see Chapter 56). In addition to the research support for this recommendation, a substantial

behavioral approach makes sense, as EDs are characterized to a large degree by dysfunctional behaviors (see Chapters 1 & 67). CBT aims to give patients (a) a framework with which to understand their symptoms; (b) the ability to identify the many different triggers, thoughts, and feelings associated with ED behaviors; and (c) the ability to identify and discuss obstacles to recovery. Taken together these skills will improve treatment response and ultimately increase the chances of full recovery from an ED. Moreover, CBT can be effectively applied across the treatment spectrum, and this systematic approach can be easily transferred between therapists when patients move from more intensive (inpatient/residential) treatment settings to less intensive locally delivered treatment in partial hospital or outpatient treatment settings. Primary therapists thus must be well trained in multimodality delivery of CBT interventions. This treatment approach also requires that therapists show discipline in adhering to treatment guidelines, as there are often many side issues that can distract from progress.

In summary, the primary therapist has a critical role in the delivery of individual, group, and family therapy. Depending upon experience level and leadership strengths, she or he may provide primary leadership for the treatment team. In addition to basic qualities such as nonjudgment of and empathy for patients and a passion for ED treatment, primary therapists must also be skilled in those therapies that have been demonstrated by empirical studies to be the most effective for EDs, including CBT (see Chapter 56) and FBT (see Chapters 53 & 60). They must be disciplined and remain focused on the most critical aspects of treatment and recovery, while being mindful of the impact of co-occurring conditions such as depression, anxiety disorders, or substance use disorders (see Chapters 15 & 54). Finally, they must be able to support patients' motivation (see Chapter 63) and remain encouraging despite patient ambivalence and what may seem like slow progress.

Experiential Therapists

Experiential therapists work with patients through art, music, dance, psychodrama, or recreation. The experiential therapist assists in exploring personal interests and recreational activities that can support a healthy lifestyle, as well as ways to enhance self-expression, identification of feelings, problem-solving skills, and self-esteem. It is a particularly helpful modality for less verbal patients who struggle with "talk therapy." Art therapy is a useful way to explore body image and self-awareness and different methods of self-expression, and gain new perspectives about what the ED may mean metaphorically to them, and why recovery may be challenging. In recreational therapy, individuals participate in various activities, such as creative games and climbing challenges, which allow them to see their behavior, personality characteristics, and interactions with others in a new light. For example, an individual who is working on reducing his/her strong need to be in control can participate in a game where that control is taken away and given to the other group members. After the game, there is an opportunity to look at what that experience meant relative to personal coping skills and to discuss alternative choices that could have been made. Such guided experiences can provide a powerful treatment experience and can facilitate and enhance the progress in talk therapy, particularly when there is close cooperation between the individual and experiential therapists.

In addition to providing direct experiential therapy, recreation therapists are ideally trained to deal with problems with excessive exercise behaviors, which occur in a substantial number of individuals with EDs (see Chapter 35), and can be particularly dangerous for patients with AN (Davis et al., 1997). For treatment of underweight individuals, the medical doctor on the team must determine when an individual is sufficiently medically stable to participate in

physical activities (see Chapter 52). Once there are positive responses to nutritional and other components of treatment, the team physician and the recreational therapist work together to slowly introduce physical activities and to ensure that patients' responses to activities can be closely observed. Re-engaging in physical activity when it has been suspended for some time is difficult for many individuals with EDs at this level of care, and particularly so for athletes or those who identify closely with athletics (see Chapter 35). The emphasis of recreational therapy treatment for individuals with histories of excessive exercise is on moderation, socialization, and wellness, as opposed to control of weight, shape, or appearance. Recreational therapists experienced in working with individuals with excessive exercise symptoms are skilled at helping patients to understand and challenge their motivation for intense physical activity. These professionals must collaborate closely with the physician and dietician to ensure physical activity is not having a negative effect on weight restoration, and with the individual therapists if the patient maintains beliefs closely associating self-evaluation with exercise intensity or quantity.

Insurance Review Coordinator

Finally, the insurance utilization review coordinator is responsible for working with families and third-party payers to manage financial support for treatment. Insurance companies frequently express frustration with a lack of appropriate treatment goals or of treatment team willingness to move patients to less intense and less costly levels of care when appropriate. On the other hand, treatment providers historically have viewed insurance companies as limiting treatment access without taking into account the seriousness of the patient's illness or understanding the risks if treatment is interrupted. While this natural tension is a reality, the insurance utilization review coordinator can maximize collaboration between insurance companies and the treatment team. The review coordinator acts as a liaison providing insurance companies with treatment plans and progress-to-plan, while communicating to the treatment team additional information required by insurance companies, and informing both the family and the treatment team about financial limitations that may result in changes to the level-of-care.

Family and Caregiver Providers

Family programming or therapy (see Chapters 53 & 60) may be provided by any of the treatment team providers mentioned in this chapter, and is most effective when planned and enacted by all team members. Research indicates that the stress experienced by families and caregivers of ED sufferers is equivalent to that experienced by caregivers of schizophrenic patients, and both exceed stress reported by caregivers of any other mental disorder (Kyriacou, Treasure, & Schmidt, 2008). It is likely that high levels of stress are related to the severe physical consequences and high morbidity of patients with EDs (see Chapters 14, 52, & 55).

Across all levels of care we recommend support groups for individuals suffering with EDs and for family members and other caregivers. Such groups are invaluable in facilitating emotional processing, reducing isolation by connecting with others who are experiencing similar difficulties, and accessing accurate information about the illness and its treatment (Pasold, Boateng, & Portilla, 2010). In a support group setting it is often helpful to have a dietician present, as well as other team members, and this helps to reflect the need for interdisciplinary work in all aspects of treatment. Family members and friends are encouraged to express their frustrations, sadness, and success stories. In this way families and friends relate to each other

and are relieved to hear of others going through the same struggles. They also get ideas and resources from each other, and often form connections that are very valuable for their ongoing support during treatment of their loved ones.

In addition to talk-based support groups, experiential activities provide a positive and engaging connection point to families and caregivers. While residential patients often are exposed to experiential therapy activities several times a week, such activities can also be very helpful in enabling visiting families to join in the treatment experience. Some example activities include ropes course challenges, yoga or relaxation practices, or viewing and hearing artwork done by patients in art therapy and learning from patients the meaning of the work as it relates to their ED. A therapist-guided support group held just for the family and friends can be helpful in recognizing and processing the emotional aspects of having a family member or loved one with an ED.

Comorbidity and Teamwork

We know that psychological comorbidity in EDs is the rule rather than the exception (see Chapters 15 & 54). Untreated comorbidities, including affective disorders, anxiety disorders, and addiction, have been shown to impair treatment effectiveness and reduce the potential for recovery (Fichter, Quadflieg, & Rehm, 2003). Patients with EDs who present with co-occurring anxiety spectrum disorders such as OCD, social phobia, body dysmorphic disorder, panic disorder, or highly ritualized eating behaviors may benefit when the treatment team is extended to include a clinician who specializes in exposure and response prevention (ERP). We have previously presented details of this treatment approach and evidence for positive outcomes in a female population (Bean & Weltzin, 2001). Similarly, we recommend that the treatment team for patients with histories of addiction include a psychiatrist or prescriber of medications who is educated in medications with addiction risks, plus a certified substance abuse counselor (SAC). Support groups such as Alcoholics Anonymous, Narcotics Anonymous, and Overeaters Anonymous are often very receptive to ED patients even when there is not a primary addiction. Al-Anon, a support group for family members of individuals with substance use disorders, can also be helpful for family members struggling with EDs. Support groups are often not available in smaller communities, but there are a growing number of Internet-based support group services that can be helpful (Binford Hopf, Le Grange, Moessner, & Bauer, 2013; see also Chapters 46, 53, & 60).

It is important for the treatment team to identify situations in which outside consultants or specialists should be included either in providing advice to the treatment team or as an adjunct treatment provider. For example, severe depression and associated risks of suicide are not uncommon in ED patients (see Chapters 15 & 54). The low energy and lack of motivation associated with depression can have a significant impact on patients' ability to engage productively in treatment, and even experienced treatment teams may lack targeted assessment and intervention strategies. In addition to evaluation for medication, there are specific psychotherapeutic interventions, such as CBT-behavioral activation, that have been demonstrated to be effective in the treatment of depression (Jacobson, Martell, & Damidjian, 2001). Comorbid post-traumatic stress disorder (PTSD) is another example of a condition that may require that the team obtain expert assistance (see Chapters 15 & 34). PTSD occurs in about 10% of patients with EDs and can create special complexities in treatment. We recommend the use of experts in evidence-based

approaches for trauma, specifically exposure techniques such as prolonged exposure (Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010). Trauma work may increase levels of emotional distress, which may in turn increase ED symptoms, self-harm, or levels of suicidal ideation, so patients must be closely monitored and exposure work must be done in collaboration with other members of the treatment team.

The Treatment Team Across Successive Levels of Care

Outpatient Care

If patients are able to function in a home environment, outpatient treatment is optimal as it is the least restrictive and least expensive, and it usually allows patients the most proximity to family support. Outpatient care continues to benefit from a team approach, at minimum including a therapist and dietician, and usually a psychiatrist for ongoing medication management. In the outpatient setting the treatment team is generally not as tightly integrated, and members are often in different physical locations and may not have experience in working together, creating a potential for poor coordination, including delays and gaps in communication. Such risks can be reduced if providers can make contact at the outset and determine a mutually agreed structure for regular leadership, communication, and coordination. Patients and family members may also have preferences for how care is coordinated across providers. In any case, the approach must be agreed and understood by all stakeholders.

Initially, patients should meet with their therapist and dietician weekly, and if there is symptom improvement session frequency can be reduced. In monitoring treatment goals, psychological providers cannot rely solely on patient self-report, and must include weekly medical care for regular weight monitoring and laboratory testing (see Chapter 52). If patients have a history of treatment resistance or failure (see Chapter 12), weight and laboratory monitoring may be required multiple times a week. If weight and behavioral goals cannot be minimally met, then a higher level of care is indicated, and oftentimes must be initiated quickly and in spite of patients' objections. Strong relationships among providers across different levels of care can be helpful if patients are initially unsuccessful in outpatient care. CBT (see Chapter 56), DBT (see Chapter 57), and FBT (see Chapters 53 & 60) are all typically delivered in an outpatient setting augmented by regular dietician visits (see Chapter 61).

Intensive Outpatient

Intensive outpatient therapy (IOP) typically refers to a group-based treatment that occurs 2–3 days a week for 2–3 hours a day, with at least one meal occurring during the daily sessions (see Chapter 61). In comparison to fully outpatient care, treatment providers in an IOP typically work for a single organization, and thus will have experience of working together as a team and are likely to have a built-in communication structure. IOP allows the treatment team to increase the intensity of treatment in the outpatient setting, so it is convenient and less disruptive because the individual may continue to work for an employer or in the home, or continue in school.

When a patient is recommended to go to IOP this is often a good time to have a team meeting with the patient and family to discuss what options may be necessary if IOP is not successful. IOP is often the first encounter with group therapy with other ED patients as the main treatment modality. Another advantage of IOP is that the team can increase or decrease the intensity of treatment based on the number of days a week the patient participates. This is one way of improving patients' confidence that they can be self-directed in their eating without engaging in ED behaviors.

Partial Hospitalization

The terms partial hospital, day hospital, and day-treatment programs all refer to what we will call partial hospital treatment program (PHP; Zipfel et al., 2002). PHP is designed for individuals needing structured programming for 6 to 8 hours a day, but who do not need 24-hour care. Additionally, patients who have been in an inpatient or residential program can step down to a partial hospitalization program that continues to provide a high amount of structure and support.

Typically these programs include at least two structured meals 5 days a week, and many programs arrange for three such meals a day, 5–7 days a week. PHPs are more similar to residential treatment than to outpatient care or IOP in that PHPs typically follow a daily program schedule with a variety of therapeutic groups focused on education, as well as nutritional and medical monitoring and accountability during the hours of the day identified as most troublesome for patients struggling with an ED. Programs typically have a CBT and DBT emphasis and may also incorporate FBT and ERP techniques to augment treatment.

The team concept is essential to an effective PHP, although different PHP programs may have different team compositions. A typical team would include a psychiatrist, dietician, one or more group therapists, and possibly an individual therapist. Most programs would provide weekly appointments with the psychiatrist and dietician, staff-observed meals, and controlled access to bathroom facilities to encourage avoidance of purging by self-induced vomiting. A patient must be able to avoid purging in most circumstances in order to be appropriate for a PHP, since there is no nighttime support or observation. Admission criteria for PHPs have changed over the last 10 to 15 years; more frequently now patients with AN who are significantly underweight are admitted to and successfully gain weight in the PHP setting. For patients who are severely limiting their intake or engaging in binge and/or purge behaviors on a daily basis, it is often quite helpful to have a brief inpatient admission, ideally on a specialized inpatient ED unit, to improve nutritional intake and to break the pattern of daily ED behavior prior to starting PHP. Alternatively, if a patient is not making noticeable improvement within the first 7 to10 days of PHP, then a brief inpatient stay may facilitate success in the PHP setting.

Residential Treatment

Residential treatment (Bean et al., 2004) can be instrumental in ED recovery for a number of reasons. Residential treatment centers (RTCs) tend to be less intimidating than inpatient psychiatric centers and are ideal when someone probably needs 24-hour care based on the severity of their illness but is not medically unstable, is not actively suicidal, nor is having urges to self-harm. Residential care utilizes medical staff, but not on a 24-hour basis; therefore, residents must be medically stable to be able to be at this level of care. Some individuals

are admitted directly to residential treatment from being an outpatient, while others first go through inpatient treatment and then transfer to a residential program when medically stable (see Chapter 52). Because lengths of stay for residential treatment commonly are 6 to 8 weeks for an uncomplicated treatment and may be a number of months for more complicated cases, individuals typically find the RTC setting much more comfortable than the inpatient setting. Finally, the 24-hour supervision of an RTC can typically provide enough structure to interrupt repetitive and pervasive ED behaviors that are difficult to extinguish in OP, IOP, or PHP levels of care. Thus, residential treatment facilities offer an excellent alternative, providing round-the-clock care in a relaxed, nonhospital setting.

Residential care settings offer an intensive and structured treatment program similar to that for a hospital inpatient, but in a more homelike environment. Individuals in these programs are referred to as *residents* instead of as *patients*. In residential and inpatient treatment settings, in addition to the standard team of psychiatrist, psychologist, dietician, and individual/family/group therapist, team members may include psychiatric technicians, registered nurses, substance abuse counselors, an education specialist for adolescent and college age students, and spiritual counselors. RTCs typically provide daily psychotherapy groups, experiential and art therapies, and nutritional counseling (see Chapter 61). In addition, RTCs offer individual therapy sessions with the social worker to further address each resident's needs. Similar to an inpatient program, families are expected to participate in the treatment process by attending family sessions and group education (see Chapters 52 & 63). Residential care also focuses on developing a thorough discharge plan for when the resident returns home.

An advantage of residential care is that it provides an ideal setting for the entire treatment team to collaborate to develop programming tailored for family and friends of ED sufferers. As family members or supporters may need to travel some distance to attend such a program, a full day of activities is often appreciated. Typical family programs include education on current research, treatment approaches, and treatment outcomes for EDs, as well as hands-on experience in some of the therapy the patients receive in treatment. Guest speakers, particularly individuals who have successfully recovered from an ED and who can effectively share their story, are very valuable to families and can instill a much-needed sense of hope. Families also value practical nutritional education in which a dietician explains how the meal plans are developed and the importance of abstinence from ED behaviors to allow the body to heal from the damage inflicted by restriction, binge eating, or purging. Therapists can conduct a forum with the families to answer questions, further explain the program, and communicate personal experiences in treating many patients with EDs. For patients who are able to manage it, a group lunch including patients and family members can be a positive challenge and can provide tangible evidence of improvement in treatment. This is particularly important, as families have often experienced a great deal of conflict over mealtimes, and may not have enjoyed a conflict-free meal in some time.

Inpatient Care

Inpatient treatment means 24-hour care in a hospital setting, which can be a medical facility, a psychiatric facility, or both. Inpatient treatment at a strictly medical hospital is usually a short-term stay to treat medical conditions or complications that have arisen because of the ED, such as electrolyte imbalances or EKG abnormalities (see Chapters 14 & 52). In fact, many patients with severe EDs may initiate care in a medical-surgical hospital for physical stabilization, after which they typically transition to a specialized ED treatment setting, most

commonly inpatient or residential treatment settings and occasionally partial hospitalization or intensive outpatient treatment.

Treatment at a psychiatric facility may be longer and includes therapeutic interventions. Inpatient psychiatric care (Calderon, Vander Stoep, Collet, Garrison, & Toth, 2007) is necessary for those who need constant nursing care, those who are severely depressed or suicidal, and those who are severely underweight and unable to restore weight in a less restrictive level of care. At psychiatric hospitals in the United States, inpatient treatment typically lasts approximately 2 weeks, long enough to interrupt the cycle of unhealthy eating patterns and stabilize nutritional intake. Patients participate in daily group therapies including cognitive-behavioral education, experiential therapy, and art therapy. The unit dietician meets with patients Monday through Friday to assist in their menu selection and facilitates a nutritional instruction group once a week. Because of the short-term nature of inpatient treatment, a major focus of the stay is to develop a plan for continued treatment of the ED outside the inpatient program. Thus, the case manager role is critically important. This individual is typically an experienced and skilled social worker who works with the individual and the family to first assess what level of care is needed after the inpatient stay, and then to help make arrangements for that care. In other words, the social worker will work with the patient and family to anticipate issues that may arise after the inpatient hospitalization, and then help the individual and family come to a resolution and develop a plan that best meets the patient's needs.

On the inpatient units that regularly treat individuals with severe EDs, the bathrooms are locked to prevent purging episodes, and patients are monitored by a staff member (typically a nurse) for 45 minutes after meals and snacks. All patients begin by eating meals on the unit. The meals planned by a dietician in consultation with the treatment team are prepared by kitchen staff and sent to the unit on individual trays. When patients demonstrate 100% compliance with meals on the unit, they transition to going to the hospital cafeteria to eat. In the cafeteria, patients practice selecting foods from the hot meal line, salad bar, and beverage station and learn to manage portions. If a patient is not able to follow a meal plan and her or his nutritional and/or physical status becomes further compromised, nutritional supplements, such as Ensure, or tube feeding may be necessary.

A high-quality inpatient program that specializes in EDs treatment also provides daily psychotherapy groups, experiential and art therapies, and nutritional counseling. Families of inpatients will likely be asked to participate in one to two family sessions during the treatment stay. These sessions are helpful in providing updates of treatment progress, offering the family an opportunity to voice questions and concerns, and discussing follow-up treatment options. Thus, the treatment team consists of individuals from all of these disciplines, and weekly or biweekly meetings of the entire treatment team are important to rapidly coordinate care and care transitions, given the length of stay is likely to be a maximum of 2–3 weeks.

Conclusions and Future Directions

Eating disorders are complex and disabling psychiatric conditions that typically have significant medical complications and complicating comorbid conditions that often require multiple years of treatment across different levels of care (see Chapters 14, 15, 52, 54, & 55). One of the most important aspects of treatment is a highly collaborative, multidisciplinary treatment team. This team usually encompasses medical doctors, nurses, psychiatrists, psychologists, psychotherapists, dieticians, experiential therapists, and utilization review specialists, but the team is able to expand

and contract as patients transition between different levels of care. Proactive team leadership and frequent, effective cross-disciplinary team communication are essential to management of the myriad and evolving nature of patient symptoms. Leadership and communication, along with flexibility, are also crucial for supporting transitions between levels of care with changing levels of severity, and for ensuring that team members make appropriate care decisions that take into account all aspects of patient behavior in different therapeutic or outpatient milieus. While this treatment process is intensive, we maintain that a collaborative team approach provides the best opportunity for patient recovery from this challenging and multidimensional illness. Future research will help to clarify (a) how best to extend consistency in treatment throughout the course of illness with the aim of optimizing treatment response and recovery; and (b) the importance of targeting factors that negatively affect treatment response (e.g., co-occurring psychiatric problems) using add-on therapies such as behavioral activation, ERP, prolonged exposure, and 12-step programming.

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Addressing Gender and Ethnicity in the Treatment of Eating Disorders

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Eating disorder (ED) researchers have collected limited evidence regarding the optimal treatments for people whose gender or ethnicity differs from a White, female prototype. Beyond even treatment outcome, fine-grained empirical data regarding how to best *address* the issues of gender and ethnicity within evidence-based treatments for EDs are essentially non-existent. Thus, process and outcome treatment research for individuals of diverse gender and ethnicity is still very much needed (Lock & Fitzpatrick, 2009; Strother, Lemberg, Stanford, & Turberville, 2012; Thompson-Brenner et al., 2013). However, researchers and clinicians implementing evidence-based treatments have amassed substantial experience in these areas (see Chapters 16 & 25). The goal of this chapter is to suggest ideas concerning effective treatment content and process that may vary according to gender and ethnicity, and applies across treatment approach, based on treatment research and clinical experience. For both gender and ethnicity, we will consider how therapists may (a) usefully address clients' specific body image and eating issues; (b) thoughtfully address issues of stigma; and (c) fruitfully apply the findings from treatment outcome research. Subsequently, we will discuss considerations that pertain to the *therapist's* gender and ethnicity, including the implications of differences and similarities between therapist and patient, and useful ways to address these issues in therapy.

Addressing Gender in Treatment

Gender has frequently been discussed and studied among investigators in the area of EDs, given that body image and eating habits show strong associations with gender across cultures (see Chapters 19, 23, 27, & 29). In the United States, although more women than men are diagnosed with EDs (Hudson, Hiripi, Pope, & Kessler, 2007; see also Chapter 5), recent research has demonstrated that, for a variety of reasons, the rate of men diagnosed with EDs has risen over the past 20 years (White, Reynolds-Malear, & Cordero, 2011; see also Chapters 36 & 37). In a report from the National Comorbidity Survey Replication study, males were

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characterized as representing 25% of anorexia nervosa (AN) and bulimia nervosa (BN) cases (Hudson et al., 2007). Rates of men and women with binge eating disorder (BED) are even more comparable (Striegel-Moore & Franko, 2003; see Chapters 4 & 11), in part due to improvements in the assessment of ED symptoms more common among men.

Nevertheless, the importance of the explicit discussion of gender within the treatment of EDs depends strongly on the patient, the topic, and the treatment approach. Some approaches—such as feminist approaches to the treatment of body image concerns (see Chapters 19, 43, & 58)—strongly emphasize raising awareness of gender-based body image ideals as important psychoeducational and preventative factors in the treatment of body image concerns and ED symptoms. At the other extreme, treatments that incorporate transdiagnostic principles of behavior or cognitive functioning—such as those focused on cognitive-behavioral therapy (CBT; see Chapter 56), family-based therapy (FBT; see Chapters 53 & 60), or cognitive remediation therapy (CRT; see Chapter 53)—may not raise issues of gender at all, or these issues may arise only within the immediate context of the individual's current cognitive or behavioral patterns targeted in treatment. Nevertheless, gender-specific body image issues (see Chapters 22, 27, 36, & 37) have been identified as particularly productive topics of discussion across treatment approaches. Additionally, it is important to consider gender-specific stigma associated with being overweight, having an ED, or receiving treatment for a mental health issue.

Addressing Gender and Body Image

Research suggests that there are some differences and some commonalities in the body image ideals—and accompanying body image dissatisfaction—among males and females (see Chapters 27 & 37). For example, girls and women vulnerable to EDs have most commonly internalized a thin body image ideal and desire for thinness, whereas boys and men vulnerable to disordered eating and exercise are observed to prefer and desire a body shape that is lean and muscular. Other body image ideals are less gender-influenced and reflect general social concerns or social values (see Chapters 21, 23, & 29). For example, within a cultural context where food is abundant and obesity is epidemic, thinner body image ideals may represent health and social class positioning, as well as sexual attractiveness in both genders (Becker, Burwell, Gilman, Herzog, & Hamburg, 2002).

Research shows that personal but gender-specific pubertal experiences may also contribute to the development of body image concerns among both girls and boys. Aspects of pubertal development that may be risk factors for the development of body dissatisfaction and eating issues among girls include hormonal changes (Klump et al., 2012), early menarche and the development of secondary sex characteristics (Day et al., 2011), or overall weight change associated with puberty (Abraham, Boyd, Lal, Luscombe, & Taylor, 2009). Among boys, slow developers, shorter boys, and both overweight and below-average-weight boys may experience a harder time building a positive body image in adolescence (Andersen, Cohn, & Holbrook, 2000; Paxton, Eisenberg, & Newmark-Sztainer, 2006; Presnell, Bearman, & Stice, 2004; see also Chapter 37). The treatment of body dissatisfaction and EDs may include a review of experiences, cognitions, or behavioral patterns that originated in a gender-specific pubertal developmental context.

Along these lines, gender identity development may be difficult for patients to discuss, but clinicians may sensitively and fruitfully inquire about gender-related experiences of ideals, teasing, and self-criticism in these areas. For example, although both genders may experience teasing, clinicians treating large numbers of men note different themes among males

(Andersen et al., 2000; Bunnell, 2010; Pope, Phillips, & Olivardia, 2000). Because male physical strength and endurance is prized—and because obesity may result in physically "feminine" characteristics such as gynecomastia (breast development) or wide hips—teasing and concomitant self-criticism regarding overweight may have a particular "feminine" theme that is specific to men. Additionally, among boys, thinness may also be associated with weakness; consequently, teasing about thinness may also involve gender-specific attacks on an individual's masculinity. In summary, many treatments that address body dissatisfaction directly—either separately from or in addition to treatment of ED symptoms—include explicit recognition of the gendered aspects of body image ideals, the gendered content of body dissatisfaction, and the relationship between these ideals and eating (see Chapters 22 & 36).

Though data concerning the treatment of transgendered individuals with EDs are scarce, two studies have suggested that EDs may be common in this population, and that it is productive to explore the connections between gender identity issues and ED symptoms (Algars, Alanko, Santtila, & Sandnabba, 2012; Vocks, Stahn, Loenser, & Legenbauer, 2009). Both studies indicated higher levels of ED symptoms than expected in the general population, and the qualitative study suggested that transgendered individuals often use diet and exercise to change the appearance of masculine or feminine features they do not identify with, and/or emphasize the masculine or feminine shape they desire (Algars et al., 2012). Cella, Iannaccone, and Cotrufo (2013) also found that drive for femininity, independent of biological sex, was a risk factor for ED pathology and drive for masculinity a protective factor. Individuals' endorsements of these links between gender identity, body image, and ED symptoms suggest that these issues should be addressed in therapy if relevant.

Addressing Gender and Stigma

The successful treatment of EDs may involve overcoming the effects of stigma, some of which show important gender differences as well. Weight-based stigma is prevalent in the general public (Sikorski et al., 2011) and among medical doctors and other health service providers (Budd, Mariotti, Graff, & Falkenstein, 2011). Stigma attached to individuals with EDs has also been identified (Ebneter & Latner, 2013) and has been linked to the following beliefs: those with EDs are to blame for their illness (Crisafulli, Von Holle, & Bulik, 2008); EDs are trivial illnesses with a basis in vanity (Crisafulli, Thompson-Brenner, Franko, Eddy, & Herzog, 2010); ED symptoms may be transferred from person to person (Crisafulli et al., 2010); and EDs are difficult to treat (Thompson-Brenner, Satir, Franko, & Herzog, 2012). Individuals who internalize stigma—both weight-based stigma and other forms of stigma—are frequently shown to have low self-esteem and poor health (Durso & Latner, 2008). Stigma is also known to negatively affect the accessibility of mental health treatment as well as treatment-seeking behavior (Henderson, Evans-Lacko, & Thornicroft, 2013).

Some stigmatizing attitudes toward individuals with EDs seem to have themes related to biases against women in general, such as the belief that individuals with EDs are simply "vain." Female patients frequently report distress over the process of recognizing or admitting that they have an ED due to their feeling that the illness reflects a negative preoccupation with appearance that is at odds with their other (sometimes feminist, sometimes explicitly anti-female) attitudes. Female patients frequently report concern about admitting their illness to friends, and accessing social support, for similar reasons (Hepworth & Paxton, 2007). Treatment of women who are self-critical for these reasons may be enhanced by a discussion

of the inescapable messages in the media—for example, coexisting criticism of both overweight and underweight women, at times depicted on a single magazine cover—and the clear message that women should be able to control their body weight within a very narrow acceptable range (see Chapters 27, 29, & 36). Corrective psychoeducation regarding the biological contributions to EDs, such as genetics (see Chapter 22) and the biological effects of starvation (Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950; see also Chapter 52), may help to relieve self-criticism and self-stigma.

Among men, however, the stigma associated with having an ED is quite different and therefore gender-specific. Researchers (Andersen et al., 2000) note that for multiple decades EDs have been assumed to primarily affect women. Consequently, though research increasingly recognizes that men in large numbers suffer from poor body image and EDs (see Chapters 5, 36, & 37), there is a stigma associated with men having a "women's disease" and this may lead to a delay in recognizing problematic ED symptoms and seeking treatment (Räisänen & Hunt, 2014). Additionally, research and public perception have stressed the prevalence of EDs among gay men, and therefore heterosexual men with EDs may have been exposed to this bias as well (Pope et al., 2000; see also Chapter 23). Treatment might well address negative experiences associated with stereotyping, such as the invisibility of men's symptoms and distress to others; their exposure toward biased attitudes in the medical or mental health system; and the actual lack of treatment options, such as inpatient treatment facilities that admit men (Anderson et al., 2000; Pope et al., 2000).

The intersection between gender-specific attitudes and the stigma associated with having an emotional illness, or receiving psychotherapy, may also be important to the treatment of particular individuals. Research suggests that men and boys hold more stigmatizing attitudes toward mental illness and treatment for mental illness than do women and girls (Chandra & Minkovitz, 2006), and it appears that the general public holds more stigmatizing attitudes toward males with mental illness than toward females (Stickney, Yanosky, Black, & Stickney, 2012). Among men, specifically, the belief that mental illness is a "weakness of character" has shown associations with stigma against mental illness (Wang, Fick, Adair, & Lai, 2007). Men have been shown to experience levels of negative emotion similar to those of women, but social stereotypes persist that they do not have these emotions, or that expressions of emotion and vulnerability are not masculine (Else-Quest, Higgins, Allison, & Morton, 2012; Fabes & Martin, 1991; Kring & Gordon, 1998). According to experienced clinicians in the ED field, direct discussion of these topics with men may facilitate their engagement in psychotherapy (Bunnell, 2010).

Gender, Treatment Approach, and Treatment Outcome

There is limited research on gender differences in treatment outcome, in part due to the small numbers of males who have participated in treatment outcome studies (Thompson-Brenner et al., 2013). Many research studies in recent decades were in fact limited to women participants, given the prevailing sense that women were far more frequently affected, and that men's EDs may be substantially different in nature or require different treatment. In fact, research and clinical opinion suggests that men with EDs show more similarities in key symptoms than differences, and that, to date, treatments tested primarily with women typically work with men as well.

In treatment outcome studies with enough men to examine gender effects, no differences have been found in dropout rate or in treatment outcome (Munsch et al., 2007; Ricca et al., 2010; Safer, Hagler-Robinson, & Jo, 2010). Although other studies have

shown that ED-associated psychopathology and binge frequency predict treatment outcome (Masheb & Grilo, 2008; Peterson et al., 2000), observed gender differences in these factors have not been linked to gender differences in outcome. Due to the very small number of studies with adequate numbers of male participants, further research is very much needed.

In summary, regardless of therapeutic approach, the treatment of EDs may usefully include gender-specific discussions of body image, cultural standards for masculinity and femininity, and experiences of stigma. Because these are sensitive topical areas for many clients, experts suggest that therapists take the initiative to raise these topics, and attempt to do so without preconceptions regarding the experiences or attitudes of any individual.

Ethnicity in Treatment

Body size ideals, eating norms, and EDs are all understood to be defined or shaped by culture (Becker, 2007; see also Chapters 6, 7, 16, 21, 23, & 25), and clinicians treating EDs should be informed regarding cultural differences in these areas. However, for every generalization regarding an ethnic group—even those founded on strong empirical evidence—there are obvious caveats, because many individuals do not fit the group patterns. It is useful to know the empirical evidence, but no assumptions should be made regarding any individual patient. In the following materials, as in the prior section, we first review clinical recommendations regarding addressing ethnicity, as well as the relationship between ethnicity and stigma that is relevant to the treatment of EDs. Subsequently, we review the small literature concerning differences in clinical outcome and approach according to client ethnicity.

Addressing Ethnicity, Body Image, and Eating

There are many good reasons to include discussion of ethnicity in the treatment of patients with EDs. In addition to research and clinical judgment suggesting that therapists take a more active role in bringing discussions of ethnicity into treatment (Cardemil & Battle, 2003), the recognition that attitudes may have their source in cultural/ethnic group factors may relieve self-blame and assist patients in developing a critical (and/or empathic) perspective on aspects of their experience. However, in bringing discussions of ethnicity into ED treatment, several general recommendations are worth consideration. It is strongly recommended that therapists apply the following guidelines:

- Do not maintain preconceptions about the attitudes held by any particular ethnic group.
- Do not assume that any particular ethnic identity or associated attitudes would be held by a particular patient or their family members.
- Consider how differences between therapist ethnicity and patient ethnicity might affect the interaction between the patient and the therapist.
- Consider how issues of power, privilege, and prejudice may affect the patients' experience, both in the past and in the present (Cardemil & Battle, 2003).

With these excellent recommendations in mind, there are data concerning variations associated with ethnicity in the development and maintenance of EDs (see Chapters 16 & 25), as well as differences in the impact of ethnicity on perceptions of mental illness and patterns of help-seeking, that might usefully inform therapeutic discussions of ethnic and cultural issues in treatment.

Not only is there extensive evidence that body image ideals show cultural differences and are often shared by members of the same ethnic group (Bakhshi, 2011), but also there are cultural norms for appropriate communication regarding body size evaluation. For example, a patient who identifies as second-generation Chinese might note the value placed on thinness by her Chinese female relatives, as well as their habit of commenting critically on other people's weight as part of a reunion. The patient's process of identifying this as a "Chinese" cultural habit, and sorting out her own confusion about whether these comments were meant as personal—and how seriously they were meant—given her own experience as a Chinese American, may be extremely beneficial in the process of addressing her own body dissatisfaction. As noted above, however, it is important not to assume that any patient has any particular body image ideal or identity/acculturation issues as a member of a particular ethnic group. Rather, such issues might be raised as questions regarding aspects of the patient's developmental or ED history. Experienced clinicians suggest that issues of ethnicity and culture should be raised early in the treatment of an ED (Whitehead-Laboo, 2013), for example, via the question "How do you think the culture of your family or your ethnic community might have impacted the development of your eating disorder?"

When therapists have taken the initiative to name ethnicity as an appropriate topic for psychotherapy, they are in a better position to correct themselves when unanticipated ruptures take place around ethnic/cultural misunderstandings. For example, researchers developing a new treatment to prevent weight gain among girls with loss-of-control eating noted that their recommendations regarding regular exercise were met with unexpected resistance. After frank discussion, some African American girls explained their reluctance to exercise as being related to their concerns over the time required to attend to their hair if, as they anticipated, they sweated and thus had to bathe (Osborn et al., 2012). Without open communication regarding these issues, patients may feel resentment regarding the therapists' lack of understanding, and this may contribute to lack of motivation and incomplete therapeutic collaboration.

It is worthwhile remembering that eating habits and food choices can be deeply cultural, emotional issues. Common recommendations regarding healthy food choices may have very different resonances for individuals from different cultural backgrounds. This excerpt from the book *Cultivating Food Justice: Race, Class, and Sustainability* (Alkon & Agyeman, 2011, p. 3) makes this point eloquently:

> For example, Michael Pollan's recently offered list of food rules (2007) is intended to guide consumers toward eating practices aligned with the food movement. However, when Pollan begins his first rule by telling us not to "eat anything your great-grandmother wouldn't recognize as food," he ignores that fact that "our" great-grandmothers come from a wide variety of social and economic contexts that may have informed their perceptions of food quite differently. Some were enslaved, transported across the ocean, and forced to subsist on the overflow from the master's table. Others were forcibly sent to state-mandated boarding schools, in which they were taught to despise, and even forget, any foods they would have previously have recognized. And those who have emigrated from various parts of the global south in the past

few generations may have great-grandmothers who saw the foods they recognized demeaned, or even forbidden, by those who claimed their lands.

Addressing Ethnicity and Stigma

Extensive research suggests that stigma against mental illness and willingness to seek treatment for emotional issues varies as a function of ethnicity, acculturation, and education (Cachelin & Striegel-Moore, 2006; Marques et al., 2011; see also Chapter 16). Research in the field of EDs suggests that such bias may affect access to treatment for certain ethnic groups (Becker, Franko, Speck, & Herzog, 2003). Several studies have demonstrated that ethnic minorities are less likely to seek psychological treatment for an ED compared to White participants with similar symptoms (Cachelin, Veisel, Barzegarnazari, & Striegel-Moore, 2000; Waller et al., 2009). Data from a national screening study indicate that Latinos and Native Americans were less likely than Caucasians with EDs to receive a referral for further ED evaluation, and all minority groups reported being less likely to be asked about symptoms compared to White participants (Becker et al., 2003). Individuals from ethnic minority groups may have relatively greater experiences of being misdiagnosed or underdiagnosed (Cachelin et al., 2000). Research suggests that EDs in ethnic minorities are frequently under-recognized by clinicians (Johnson, Spitzer, & Williams, 2001), and appropriate care is rarely initiated when ED symptoms are disclosed (Crow, Mussell, Peterson, Knopke, & Mitchell, 1999). In addition, individuals of ethnic minority backgrounds with EDs may have difficulty reconciling their diagnosis with their own stereotypes regarding EDs as problems that affect White or privileged individuals. As with other issues of stigma, clinical experts suggest that cultural attitudes toward mental illness should be openly explored and addressed, particularly when they appear to be interfering with treatment (Whitehead-Laboo, 2013; see also Chapter 16).

Ethnicity, Treatment Approach, and Treatment Outcome

The majority of research in the ED field—including the vast majority of treatment outcome research—has been conducted with White individuals and those who have high levels of education (Thompson-Brenner et al., 2013). It is important to have a high degree of caution and humility regarding the conclusions that can be drawn about evidence-based treatments for individuals from minority ethnic groups, and to take into account all our patients' subjective and personal experiences as a topic to address in treatment and a variable to consider in shaping our treatments to be most effective.

A few empirical studies have attempted to identify themes in the treatment of EDs that may be specific to particular ethnic groups. One qualitative study assessed cultural themes in therapy for EDs among a group of primarily second-generation Asian Americans (Smart, Tsong, Mejia, Hayashino, & Braaten, 2011). The therapists reported that therapy focused on the issues of achievement, preference for a thin and petite body size, and culturally specific patterns of communication and emotional expression (Smart et al., 2011; see Smart, 2010, for suggestions regarding cultural competence in the treatment of Asian Americans with EDs). Recently, CBT for BED has been adapted for Mexican Americans with BED (Shea et al., 2012). Changes to the manual suggested by focus groups included additional attention to family issues resulting from generational differences, specific knowledge level and orientation

of Mexican American men, and interpersonal styles in Mexican American families (Shea et al., 2012). Another adaptation suggested specifically for Latino populations is the incorporation of spirituality into treatment (Reyes-Rodriguez, 2013). Many more such qualitative studies are needed to inform culturally competent ED treatment for ethnic minorities.

A major barrier to the discussion of ED treatment among ethnic minorities is that current treatment data concerning minority ethnic/racial groups are extremely limited (Franko et al., 2012). One recent study aggregated data from 12 of the largest clinical trials of treatment for BED in the United States, resulting in a database including more than 2,000 men and women who had participated in a psychosocial treatment trial (Franko et al., 2012; Thompson-Brenner et al., 2013). One of the main findings from that study was that only 102 African American (7.7%) and 64 Hispanic/Latino (4.8%) participants were included in the aggregated database, despite the comparable prevalence rates observed in epidemiological and community studies (Franko et al., 2012).

The few studies that have had adequate power to compare ED treatment responses among different ethnic groups have typically found encouraging results for ethnic/racial minorities in treatment. More specific studies are rare, but there are some signs that certain treatments may be a better fit, in general, for individuals from certain ethnic groups. For example, in the aggregated BED study cited above, despite higher dropout rates, African Americans had good outcomes in the clinical trials, and in fact showed more improvement across psychosocial treatment approaches than other ethnic groups (Thompson-Brenner et al., 2013). In two randomized controlled trials of cognitive dissonance training (see Chapter 44) to reduce body dissatisfaction, Caucasians, African American, Hispanics/Latinas, and Asian Americans all showed similar rates of positive response (Stice, Marti, & Cheng, 2014). One intriguing study from a large multisite trial of CBT and interpersonal psychotherapy (IPT; see Chapter 62) for BN found that Black participants showed more reductions in binge eating when treated with IPT relative to other ethnic/racial groups (Chui, Safer, Bryson, Agras, & Wilson, 2007). This unique finding has yet to be replicated, and again, much more extensive data are needed.

One important adaptation required for treatment to be widely available and comprehensible, by groups of all ethnic backgrounds, is the inclusion of readable language in therapy handouts, patient therapy workbooks, and guided self-help manuals, which are frequently utilized in CBT, CRT, and other structured approaches. Data suggest that ethnic minorities with EDs in the United States tend to have lower levels of education and English literacy than nonminorities with EDs (Franko et al., 2012; Marcus, Bromberger, Wei, Brown, & Kravitz, 2007), and that level of education is associated with treatment outcome in some treatment studies (Thompson-Brenner et al., 2013). However, current assessment measures and treatment manuals are most often written with vocabulary and grammar that exceed the recommended reading levels for clinical materials for individuals with lower levels of education (Peruzzini, Richards, Pratt, Franko, & Thompson-Brenner, 2012; Richards, McHugh, Pratt, & Thompson-Brenner, 2013). Adaptation of treatment manuals and handouts would help to increase access to and retention in empirically supported treatment for EDs among all ethnic groups.

Addressing Therapist Gender and Ethnicity

Across theoretical orientations, most expert clinicians believe that the reactions patients have to their therapist may be important to address in therapy, particularly when those responses have the potential to interfere with the therapeutic alliance or the client's ability to disclose

(see Chapters 16 & 60). Therapists of different theoretical orientations may choose to approach the client's feelings about the therapist differently—for example, interpersonal/psychodynamic clinicians may explore how those feelings pertain to longstanding patterns in interpersonal relationships (see Chapter 20), whereas feminist or multicultural therapists may explicitly support a client's understanding and situate the roots of that discomfort in unequal social experiences (see Chapters 43 & 58). At the beginning of therapy it may be important to bring up the possibility of clients' reactions to the therapist's gender, ethnicity, or other sensitive topics (such as age, body size, class, or other privilege) to help clients with EDs be comfortable in therapy and promote discussion of feelings in general (Whitehead-Laboo, 2013).

Therapist Gender

Because EDs develop in an explicitly gendered context (see Chapters 19, 27, & 43), but the contributions of cultural ideals of gender to body image and other components of EDs (e.g., issues with control) vary by individual (see earlier discussion), the importance of therapist gender may be more immediately salient to some clients than to others, or than other therapist characteristics. Some clients feel that a therapist of the same gender is more likely to empathize with gender-specific experiences, and research suggests this may technically be true—for example, more male therapists report feeling uncomfortable treating clients with EDs (Thompson-Brenner et al., 2012), though there is no evidence that therapist-patient gender match among ED specialists is important to the outcome of ED treatment (Bilker, 1993). It is common practice to inquire respectfully whether a client is comfortable working with a therapist of another gender, and to try to respect client preferences, although the preference may be more difficult to accommodate if a client prefers a male therapist (Lyster-Mensh et al., 2013). Therapists tend to report that gender differences with their clients, like many other differences, are surmountable by careful attention to the therapeutic process and the development of accurate empathy.

Clients with EDs may have many additional subtle and complicated reactions to a therapist's gender than the simple preference for a gender match. For example, women and men with EDs, who may overvalue the importance of shape and weight, may imagine that their same-sex therapist feels competitive or critical because they hold similar values. These feelings are typically more difficult to identify and discuss, and may emerge later in therapy, providing opportunities for rich conversation about body image and interpersonal relationships (see Chapter 20). Clients who have experienced a range of abusive and traumatic experiences with perpetrators of one gender (see Chapter 34) may have strong feelings regarding their safety with a therapist of that gender, which also—if manageable—may lead to productive therapeutic experiences. As with any important and sensitive topic, the therapist's demonstrated willingness to name and explore these issues without defensiveness is key to the patient's openness over time.

Therapist Ethnicity

As noted earlier in this chapter, ethnicity and race, like gender, may be associated with particular cultural and developmental experiences that are important to an individual's therapy for an ED. When the therapist is from a different ethnic/racial background, there may be cultural differences on important issues such as conceptions of mental illness, the relationship of an individual to family or community, and communication style, among many other possibilities

(Cardemil & Battle, 2003). It is impossible to anticipate every possible issue that may come up, and experts again recommend frank, gentle, and open-ended questions regarding the therapist's ethnicity. For example:

> I know that this can sometimes be a difficult topic to discuss, but I was wondering how you feel about working with someone who is from a different racial/ethnic background? I ask because although it is certainly my goal to be as helpful to you as I possibly can, I also know that there may be times when I cannot fully appreciate your experiences. I want you to know that I am always open to talking about these topics whenever they are relevant. (Cardemil & Battle, 2003, p. 281)

In the case of ethnicity/race, important therapist-client differences may also pertain to privilege. Clients may perceive therapists to lack the experience of prejudice or the awareness of privilege associated with ethnicity/race, as well as privilege associated with gender, class, body size, sexual orientation, age, attractiveness, or other attributes (Cardemil & Battle, 2003; Whitehead-Laboo, 2013). Again, experts recommend that therapists who differ from clients on an obvious characteristic that may be associated with prejudice (e.g., ethnicity, body size) demonstrate openness to exploring the feelings that may come up about the therapist in that conversation. For example:

> Today we have been talking about your sense that many of your coworkers are prejudiced. What has this conversation with me been like for you? What has it been like for you to share experiences of discrimination with a White therapist who hasn't had those kinds of experiences? (Cardemil & Battle, 2003, p. 282)

Therapists may at times struggle with personal frustration regarding intractable social forces, or with guilt associated with their ethnic group identification. However, clinicians and patients observe that the opportunity to discuss these issues frankly with a member of a "privileged" group may itself be therapeutic (Cardemil & Battle, 2003).

Therapists from minority ethnic backgrounds may face additional issues, themselves, in the context of therapist-client differences. Clients may hold racist attitudes, deriving from cultural stereotypes and/or from low self-esteem that is temporarily improved by denigrating others. In contemporary society, particularly within the culture of mental health treatment, clients are likely to keep such views to themselves; however, therapists report perceiving racist attitudes that are hard to manage. Such situations pose significant ethical dilemmas across the "helping professions" (Selby, 1999). Ethical guidelines for most disciplines include the necessity of professionalism and the provision of services to all individuals regardless of their attitudes or beliefs. When a therapist perceives that a patient holds racist (or sexist, or size-ist) attitudes, he/she might start with the question, "How do you feel about working with someone who is from a different racial/ethnic background [or sex, or body size] than you?" If that does not seem clinically advisable, or does not resolve the issue satisfactorily, then that therapist may make the judgment that a different therapist is recommended. These issues may have additional complications in the therapeutic communities (see Chapter 50), such as inpatient or residential units (Selby, 1999), or when another therapist is not available.

Given the range of possible sensitive topics and significant differences between therapist and client, it may be overwhelming or unwieldy to query a client about each topic separately. Even more open-ended questions, such as "How does it feel to be talking with a person who looks like me?" may help to start dialogue in areas of personal significance to the client, including differences in gender, ethnicity, age, size, or other relevant issues (Whitehead-Laboo, 2013).

Conclusions and Future Directions

Extensive additional research in the area of gender and ethnicity in the treatment of EDs is needed. The following conclusions for practice and future directions for research are based on our limited knowledge to date.

Experts consistently and clearly state that the practice of psychotherapy suffers when issues of gender and ethnic diversity are not addressed in therapy (see Chapters 16, 25, 43, & 58). Research shows that there are meaningful differences in body image ideals, habits of eating and exercise, and stigma associated with ED and psychotherapy as a function of gender, ethnicity, and strong cultural influences. Although it is impossible to know in advance the cultural influences associated with gender or ethnic/racial group that have affected any individual, it is important to inquire about these issues in the process of psychotherapy.

Researchers who have examined the practice of psychotherapy with different ethnic/racial groups have made specific observations and recommendations regarding common themes that emerge in the practice of psychotherapy with particular ethnic groups, and regarding adjustments to treatment protocols that are suggested by individuals from these groups (Shea et al., 2012; Smart et al., 2011). Therapists treating individuals from ethnic groups different from their own should familiarize themselves with this literature and attempt culturally competent treatment (see Chapter 16). In cases where no prior research has been conducted, therapists should make sure to inquire about the cultural appropriateness of the content and process of psychotherapy.

There are many important future directions for research. Some of the most important future research will concern the efficacy of existing treatment protocols and optimal implementation for minority ethnic groups, males, and other minorities (e.g., transgendered individuals). Another important topic for research and advocacy concerns the stigma associated with EDs among all gender and ethnic groups, as well as among medical professionals. Research is needed concerning the best methods to reduce all types of stigma, and thereby improve the accessibility of treatment as well as individuals' willingness to receive help. Finally, there is a need for fine-grained research that examines the optimal process by which therapists can raise and address these issues in the practice of psychotherapy.

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Medical Complications and Management Strategies in Patients with Severe Restricting and Purging

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In my clinical experience, adults with eating disorders (EDs) often receive suboptimal medical care for a variety of reasons. Front-line medical providers typically lack formal training in ED medicine. This makes many providers reluctant to care for patients with EDs, as does the perception in the medical field that patients with EDs may be difficult to work with, or indeed that anorexia nervosa (AN) and bulimia nervosa (BN) are "just mental illnesses."

Given the increasing prevalence of patients presenting with EDs, ranging from young children to older adults, nearly all of whom require a primary care base for all their medical needs, this unfortunate situation sets up serious issues for providers, patients, families, and the financial well-being of the healthcare system. These patients typically deny the seriousness of their disease, fear recovery, embrace the distortions that trap them in their disordered behaviors, and possess a temperament that tends toward the controlling, anxious, and harm avoidant (see Chapters 2, 3, 18, & 32). Inadequate or harmful medical care causes them to lose faith in the medical system and become less likely to seek and remain in medical care.

For patients with the most severe EDs who have been told they are too medically unstable to admit to an inpatient or residential ED facility, this sets up a "revolving door" of utilization of medical services at critical junctures, followed by a predictable but seemingly uncontrollable return to the same behaviors that created the instability (see Chapters 12 & 50). Enormous amounts of healthcare money are spent on futile, "band-aid" care, patients fail to stabilize sufficiently to enter treatment centers, and a dynamic of mutual suspicion and distaste is created among the patient, the family, and the medical provider.

This chapter reviews in detail the diagnosis and treatment of the many medical complications that arise from severe restriction and purging. In our inpatient facility, with multidisciplinary expertise in the definitive medical stabilization of critically ill men and women with EDs, we admit patients whose body mass index (BMI) ranges from 8 to 14 kg/m^2 (e.g., at 5′4″ [1.63 m], ~ 47–82 lb [21.3–37.2 kg]), so that with proper care they may safely proceed onto inpatient or residential ED treatment (see Chapter 50). Where the literature is insufficient, I will discuss our clinical practices, developed over many years.

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Medical Complications of Severe Restriction

Virtually all organ systems will malfunction in some way in response to sustained caloric restriction and severe underweight. However, in a medical culture that looks to particular parameters such as electrolyte abnormalities for evidence of "instability," pure restrictors (AN, restricting type; American Psychiatric Association, 2013) may persist more easily in their denial of disease because their electrolyte panel may remain normal almost until they collapse. Many pure restrictors, who in their outpatient lives are often thriving and energetic even at astonishingly low weights, note that their primary doctor inadvertently played into their desire to avoid a higher level of care and continue restricting by giving feedback that "labs continue to be normal." While physicians are taught to reassure patients with the message that "everything will be okay," in working with ED patients this strategy may backfire. Denial of disease severity and resistance to treatment are hallmarks of EDs (see Chapter 63), so the physician will serve patients better with direct and clear explanations of all the ways their body is responding poorly to starvation or purging, rather than with reassurances. Complications of pure restricting are adaptive responses of a body that regards itself as starving to death and must conserve energy and preserve vital organs. All body processes slow down, and nonessential processes (such as maintenance of hormonal maturity) regress or cease altogether.

Vital Sign Abnormalities

Patients with severe underweight conserve energy by entering what I call "vital sign hibernation." They cool their inner furnace to burn fewer calories, and thus often present with hypothermia (body temperature less than 35°C, or 95°F). Perfusion (cardiovascular delivery of blood to capillaries) of the extremities is reduced, resulting in cool, often cyanotic (bluish-looking) hands and feet. A sensation of chronic chilliness and cold intolerance accompanies hypothermia, and over time, fine lanugo hair may develop as a heat-trapping mechanism. Only nutritional rehabilitation (see Chapters 60 & 61) will "cure" hypothermia due to AN, with core body temperatures at times remaining low up to a week into treatment before beginning to normalize (Gaudiani, Sabel, Mascolo, & Mehler, 2012). For hospitalized patients we use a forced-air warming system in which warm air flows continuously through a very light blanket, preventing ongoing hypothermic stress and discomfort (see Chapter 14). Patients with severe AN manifest a fever at lower body temperatures, so we regard a body temperature of 37.5°C (99.5°F) as a fever requiring evaluation.

Hypotension, or low blood pressure, is typically defined as systolic blood pressure less than 90 mmHg or diastolic blood pressure less than 60 mmHg. Severely underweight patients likely develop hypotension as a result of heightened vagal tone, reflecting activity of the vagus nerve in suppressing heart rate. This is a key element in the effective functioning of the parasympathetic branch of the autonomic nervous system. Thus, heightened vagal tone mediates bradycardia (heart rate less than 60 beats per min), which is highly prevalent in severely underweight patients and also may be found in patients with a normal BMI who have recently lost a great deal of weight rapidly. Hypotension and bradycardia can cause presyncopal symptoms (lightheadedness and the feeling one might pass out) or syncope itself. We typically find, in the pure restricting anorectic, that blood pressure alters very little with postural change.

It is very important for medical providers to disabuse patients of the notion that their heart rate is 30 "because he or she is an athlete." Bradycardia is an adaptive response to dangerous weight loss, not to past or present athletic pursuits. There is a way to distinguish between

bradycardia arising from starvation versus athletic fitness. People with outstanding cardiovascular fitness and a slow resting heart rate will not markedly change their heart rate when they rise from a chair and walk across the room. By contrast, many patients with AN develop tachycardia (heart rate greater than 100 beats per min) upon minimal exertion, or a doubling or tripling of the resting pulse, even if the pulse does not rise above 100 beats per minute. This is caused by the deconditioning inherent to skeletal muscle loss and underweight, as well as cardiac muscle thinning in some cases, such that the heart must pump faster for the relative "exertion" of movement. This is not, in the restricting anorectic, due to orthostatic changes, in which heart rate accelerates and blood pressure drops when moving from the supine to upright position due to volume depletion or autonomic dysfunction (see Chapter 14). I reserve the term "orthostasis" specifically for these latter situations, because casual use of the term may perpetuate an incorrect understanding of the pathophysiology and management of low and high heart rates in patients with AN. Any significant change in the heart rate from sitting to standing should be explained to the patient as a sign of a body slowed and weakened by an ED, rather than interpreted and dismissed as cardiac fitness. I tell patients, "This is not an athlete's heart. This is a starved person's heart."

Katzman (2005) has provided a full discussion of all cardiac abnormalities in AN, which are important because cardiac causes (e.g., severe arrhythmia) are implicated in a significant fraction of deaths from AN (Papadopoulos, Ekbom, Brandt, & Ekselius, 2009). Recent studies (Krantz et al., 2012) have challenged the previous theory that low potassium levels (hypokalemia) attributable to severe underweight and, perhaps, to many psychiatric medications, were responsible for disruptions in the heart's normal electrical cycle (a prolonged QT interval, corrected for heart rate [QTc]), which increased patients' risk for torsades de pointe and thus for fatal arrhythmia (ventricular fibrillation). Newer hypotheses consider possible precursors to a fatal arrhythmia: heart rate variability; disruption in the electrical depolarization and repolarization of the ventricles (QT dispersion); and bradycardia or tachycardia due to problems at the junction of the heart's atria and ventricles (Krantz, Gaudiani, Johnson, & Mehler, 2011; Melanson, Donahoo, Krant, Poirier, & Mehler, 2004). Much work remains to determine the true precursors of fatal arrhythmias in AN.

Only nutritional rehabilitation and weight restoration (see Chapter 61) will normalize abnormal vital signs in AN. In the patient with pure restricting AN, intravenous fluid should not be utilized purely for bradycardia, as the patient does not have a volume status problem but rather a starvation problem. We additionally recommend against the routine administration of agents used for autonomic dysfunction and orthostatic hypotension with syncope in non-eating-disordered patients, such as fludrocortisone or midodrine. We feel these are "band-aid" treatments and consider them only if bradycardia and hypotension persist after weight restoration, or if another etiology is discovered. Emphasis on documenting abnormal vital signs prominently can help justify needed inpatient medical care. Guidelines from the American Psychiatric Association (2006) recommend medical hospitalization for patients with a pulse below 40.

Refeeding Syndrome

Patients with AN are at risk for refeeding syndrome, a potentially deadly shift of electrolytes that occurs when a starving person begins to take in nutrition (National Institute for Health and Clinical Excellence [NICE], 2006). In refeeding syndrome, low serum phosphorus is the main focus. After a meal is consumed, carbohydrate ingestion appropriately prompts insulin

release, which drives phosphorus from the serum into the cells. Phosphorus is also incorporated into newly synthesized tissues.

Low serum phosphorus can be deadly. Severe hypophosphatemia (serum levels < 3 mg/dL) can cause rhabdomyolysis (muscle breakdown), white and red blood cell dysfunction, respiratory failure, seizure, and cardiac arrest. The high risk of refeeding syndrome argues strongly against severely underweight adult patients trying to refeed on their own in the outpatient setting in order to avoid a higher level of care, unless they have a strong multidisciplinary outpatient team (see Chapter 50) that provides intensive monitoring and laboratory checks multiple times a week. In a hospital setting, we check a chemistry panel with phosphorus daily during the first week of refeeding and begin to replace phosphorus orally when serum levels drop below 3 mg/dL. We use intravenous phosphorus repletion only when the oral supplement causes diarrhea or when serum levels fail to respond even to maximum oral supplementation. Prevention of full-blown refeeding syndrome is key. By monitoring phosphorus levels regularly, and repleting early, clinicians can spare patients the dangerous sequelae of hypophosphatemia.

The rate and mode of early refeeding differs across healthcare systems and varies with the age range of the patient population, the setting, and the severity and intensity of the ED's presentation. The topic is a matter of active research currently (Kohn, Madden, & Clarke, 2011). We increasingly understand that initial "underfeeding"—born of the traditional "start low, go slow" technique to prevent refeeding hypophosphatemia—may be as dangerous as starting calories too quickly (Leclerc, Turrini, Sherwood, & Katzman, 2013). Our adult patients typically have a resting energy expenditure of around 500–700 kcal/day (2,092–2,930 kJ/day), as measured by indirect calorimeter. In light of recent literature, we now start calories at 1400 per day, with the exception of the most gravely ill patients, and increase by 400 calories every 3 days. A carbohydrate content under 50% of total calories is recommended to limit insulin surges, which can exacerbate both hypophosphatemia and edema (Kohn et al., 2011). While our staffing is adequate for 1:1 meal support with almost no utilization of tube feeding, other hospital systems with less expertise may effectively utilize an automatic tube-feeding protocol upon admission for initial calories.

Hepatitis

In an otherwise healthy adult with AN who presents with abnormal liver function tests, the initial presumption should be that this is caused by starvation, with an apoptosis/autophagy mechanism, and will resolve with nutritional rehabilitation (Harris, Sasson, & Mehler, 2013). Further focused workup is appropriate if the patient has concurrent risk factors for hepatitis, such as alcohol abuse, risky behaviors that make viral hepatitis a possibility, or a family history of autoimmune liver disease.

Elevated liver enzymes (aspartate aminotransferase/AST and alanine aminotransferase/ALT) are very common in AN. Fully 75% of our hospitalized patients have hepatitis on admission. However, this finding is not well known by general providers and is often a source of distracting and unhelpful evaluations. Results of liver function tests may continue to worsen in the first week of refeeding, which can be worrisome and puzzling for providers. However, we consistently see that after around 5 days of refeeding, liver function tests will peak and then begin to normalize.

Much more rarely, refeeding itself can cause elevations in liver function tests due to a steatohepatitis mechanism, or "fatty liver." Rising liver function tests during the first week of treatment make this a diagnostic challenge. An elegant solution is to check a right upper quadrant ultrasound every few days if the liver enzymes continue to rise past a week of refeeding. In

starvation hepatitis, the liver will remain the same size, without findings of steatosis on ultrasound, despite rising liver function tests. In this case, we push forward with refeeding. In refeeding hepatitis, serial ultrasounds will reveal that the liver is enlarging as the liver function tests rise, and the liver will have a steatotic appearance. This may lead us to reduce the caloric content of meals and to suspend further caloric increases until liver function tests stabilize, however mild liver function test elevation related to refeeding is well tolerated by the patient, in whom the risk-benefit analysis favors forging ahead with nutritional rehabilitation, as mild refeeding hepatitis should spontaneously subside in most people over time. Thus in mild cases of refeeding hepatitis, we take a watch and wait perspective and continue with calories and weight restoration. Liver function tests greater than three times normal predict hypoglycemia, and the clinician is well advised to use this fact in convincing a resistant patient to enter treatment. Pure starvation hepatitis should resolve completely, and no literature has reported secondary development of cirrhosis.

Levels of serum albumin are almost invariably normal or near-normal, even in severe AN, a fact that perplexes both primary clinicians and medical directors of insurance companies. Medical providers are typically taught that albumin is a marker of "nutrition," and, of course, those with AN are among the least nourished patients imaginable. However, in non-eating-disordered adults who are underweight, serum albumin falls in response to the inflammatory (e.g., rheumatologic diseases), cancerous, or chronic infectious processes that caused the weight loss (Narayanan, Gaudiani, & Mehler, 2009). Indeed, we regard development of low serum albumin in a patient with AN as a "canary in the coal mine" and watch carefully for subsequent manifestations of an infectious process such as a urinary tract infection or pneumonia.

Hypoglycemia

Hypoglycemia, typically defined as a blood glucose less than 60 mg/dL, is a potentially deadly laboratory finding that may bring resistant patients to medical attention, as it can cause a seizure or acute unresponsiveness, with brain damage or death if untreated (see Chapter 14). Hypoglycemia can occur in AN due to starvation, during refeeding, or both. When a patient with severe underweight presents with low blood sugar, or is found to have low blood sugar first thing in the morning or just before meals, starvation physiology is responsible. The liver is too starved to produce glucose or glycogen between meals. However, patients can also develop both (postprandial) hypoglycemia in response to carbohydrate intake during refeeding and low blood sugars after meals. Patients may be asymptomatic during hypoglycemic levels, particularly if they have become accustomed to this state, or they may feel sweaty, confused, dizzy, or sleepy.

In the vast majority of patients with AN, the only way to correct hypoglycemia definitively is to proceed with nutritional rehabilitation to the point where the liver once again possesses the gluconeogenic building blocks to maintain safe blood sugars between meals. In patients with starvation hypoglycemia, we use slow intravenous fluid (e.g., 5% dextrose [D5] normal saline at 50 ml/h) or intermittent oral intake of a liquid nutritional supplement alongside the meal plan until all blood glucose levels exceed 60 mg/dL. Experts outside the United States have found that initial round-the-clock tube feeding prevents postprandial hypoglycemia and may choose not to initiate intravenous glucose (Kohn et al., 2011; Leclerc et al., 2013).

Bone Marrow Dysfunction

Bone marrow dysfunction arises in AN due to starvation-mediated marrow hypoplasia, in which the normal matrix is replaced by an amorphous, non-cell-producing material that can cause reductions in all three cell lines: white blood cells, red blood cells, and platelets. This

process is typically called "gelatinous marrow transformation" or "serous fat atrophy." In discussing this finding with patients and their families, I do not hesitate to point out that the patient is so starved that he or she has bone marrow failure. Such connections of the ED with the objective clinical findings may be very useful in motivating recovery. In young adults with moderately severe AN being treated in inpatient or outpatient settings, the rates of anemia (low red blood cells) and leukopenia (low white blood cells, < 4.5 K/μL) are approximately 30%, while thrombocytopenia (low platelets) runs in the 5–10% range (Hutter, Ganepola, & Hofmann, 2009; Miller et al., 2005). However, in critically ill hospitalized adults with AN, around 80% of patients have anemia and leukopenia, 30% manifest frank neutropenia (absolute neutrophil count < 1 K/μL), and over one third show abnormalities in their platelet count (Sabel, Gaudiani, Statland, & Mehler, 2013).

Gelatinous marrow transformation fully resolves after appropriate nutritional rehabilitation (Abella et al., 2002). Indeed, in very ill hospitalized adults, nearly all cases of frank neutropenia had resolved by the end of medical hospitalization, where the mean length of stay was only 2 weeks (Sabel et al., 2013). In our program, we do not impose "neutropenic precautions" on patients with neutropenia and have never experienced a case of hospital-acquired infection related purely to neutropenia. Additionally, in treating patients with AN-induced leukopenia and neutropenia we strongly recommend against administration of the granulocyte colony-stimulating factors (G-CSF) used in patients after chemotherapy to stimulate recovery of the white blood cell line. G-CSF treatment is expensive, may have side effects, and ultimately dilutes an important message to patients: they do not have a bone marrow problem, but rather a starvation problem.

Anemia (hematocrit < 37%) is very common in patients with AN, but it is almost never attributable to chronic disease or to B12, folate, or iron deficiency (Sabel et al., 2013). This anemia is apparently due to gelatinous marrow transformation that will also resolve with weight restoration. We do not transfuse patients unless their hematocrit drops below 21% over time, or unless they have active bleeding (e.g., from the nose—epistaxis) and they become symptomatically anemic. Once again we recommend not using marrow-stimulating factors, such as erythropoietin, which are unnecessary, expensive, and distracting.

Patients with thrombocytopenia (platelet count < 150 k/μL) may bruise easily. In adults with severe AN who are hospitalized, thrombocytosis (platelet count > 400 k/μL) may develop over the course of refeeding, in the absence of iron deficiency, acute inflammation, or infection. While we do not have bone marrow biopsies to prove this, we hypothesize that this form of thrombocytosis is a sign of early bone marrow restitution. We have not seen any evidence of abnormal clotting in this patient group. We thoroughly assess the thrombocytotic patient for evidence of occult infection and iron deficiency, but we do not otherwise find a high platelet count to be of significant concern.

Gastroparesis

With severe underweight (below 80% of ideal body weight) or rapid weight loss, normal gastric emptying slows, and patients retain food in their stomachs far longer than normal (see Chapter 61). This again reflects a slowed metabolism as the starved body tries to conserve energy for vital functions only. I tell patients, "Your body is so underweight, and wants to preserve calories so carefully, that it doesn't even want to spend calories on emptying your stomach normally." Delayed gastric emptying (gastroparesis) can cause symptoms of early satiety, bloating, mid-epigastric pain, and nausea. Since gastroparesis is nearly universal in patients with severe AN, there is typically no need for a confirmatory nuclear imaging study. Of course,

these symptoms add a physical dimension to the already formidable obstacles to nutritional rehabilitation created by pre-existing mental and emotional barriers. Consumption of large, fiber-rich, or fat-rich meals worsens symptoms of gastroparesis. Smaller, more frequent, calorie-dense, low-fiber meals can help patients maximize nutritional rehabilitation while minimizing gastroparesis symptoms, as can liquid or semisolid supplements such as nutritional shakes, which typically pass through the stomach normally even in the presence of gastroparesis. Our refeeding protocol excludes raw fruits or vegetables during the early weeks of refeeding (Mehler, Winkelman, Andersen, & Gaudiani, 2010).

Two pharmaceutical agents approved for use in the United States may be helpful when a patient's gastroparesis symptoms compromise ongoing nutritional rehabilitation. We generally start by offering a short course (≤2 months) of very low dose metoclopramide, 2.5 mg by mouth, fully 30–45 minutes before meals and at night if needed, which promotes gastric emptying without increasing the frequency of bowel movements. When reviewing this option with patients, we are always careful to note that, in rare cases, long-term use of high-dose metoclopramide can generate potentially irreversible tardive dyskinesia, or twitching of the lips and tongue. Therefore, we advise patients to use the lowest dose for the shortest amount of time possible, and to stop metoclopramide immediately if they develop tardive dyskinesia. Should patients enter treatment with a prior allergy response to, or past failure with, metoclopramide, we may offer erythromycin ethinylsuccinate (EES) at a low dose of 200 mg by mouth, 30–45 minutes before meals. This works very nicely for some patients, but for others it causes nausea and must be discontinued. When initiating EES, particularly if the patient is already taking another medicine which may impact the QTc interval, EKGs are checked on a weekly basis to watch for prolonged QTc intervals. Gastroparesis should ultimately resolve with full weight restoration.

Hypothalamic-Pituitary-Gonadal Axis Dysfunction

Full discussion of the endocrine-fertility dysfunction experienced by men and women with AN is beyond the scope of this chapter, but most women will develop secondary, starvation-related amenorrhea (Lawson & Klibanski, 2008; see also Chapter 8). Many reasons for abnormal vaginal bleeding exist, however (Gaudiani, Heinrichs, Narayanan, & Mehler, 2011). Severe underweight causes a hypothalamic hypogonadism representing a reversion of the hypothalamic-pituitary-gonadal axis to the prepubertal state (Vyver, Steinegger, & Katzman, 2008). A body that is starving knows it does not have the resources to conceive and nurture a child, so it shuts down the fertility pathway. Gonadotropin-releasing hormone secretion is impaired, and women show low levels of luteinizing hormone, follicle-stimulating hormone, and estradiol. Men have low testosterone.

Osteoporosis

Osteoporosis is a condition in which bones becomes more porous and weaker. In AN, osteoporosis is a potentially irreversible complication of protracted underweight. Osteoporosis occurs early and progresses rapidly, and it can be responsible for both traumatic and nontraumatic fractures, vertical height loss, early kyphosis (spinal curvature, causing "humping" of the back), and chronic pain, for instance from vertebral compression fractures. Osteoporosis is particularly severe in AN because bone resorption (i.e., breakdown and dispersal) increases *and* bone formation halts. Bone resorption increases as a result of the hypogonadal hormone state. However, concurrent reduction in bone formation further worsens bone density loss,

due to high cortisol, low testosterone, and low insulin-like growth factor 1 (IGF-1), a nutritionally regulated anabolic hormone (Lawson et al., 2010).

Toward the end of the second decade of life, more than 90% of the peak bone mass has been achieved in healthy teenagers. In men and women who develop AN during this time, this important bone deposition never occurs. As a result, osteopenia (lower bone mineral density, leading to weakening of the bones) is present in 50% of adolescents and 90% of adults with AN, while frank osteoporosis may be found in 25% of adolescents and up to 40% of adults (Bruni, Filicetti, & Potnello, 2006). Indeed, while healthy men typically have 60% fewer osteoporotic hip and vertebral fractures than women, men with AN have greater loss of bone density than women, even though men often have had a shorter duration of their disorder. Furthermore, men may fracture at higher bone density levels than women (Mehler, Sabel, Watson, & Andersen, 2008).

The gold standard of treatment for osteoporosis in AN is full weight restoration. The exact definition of weight restoration is, of course, much disputed between patients and providers and will not be debated in this chapter (see Chapter 61). Studies, mostly of women, consistently show that patients who restore weight may improve their bone density by up to 5% in 2 years, while those who remain underweight may lose a further 4% of their bone density during that time (Olmos et al., 2010). Bone densitometry (DEXA) studies should be performed after 6 months of amenorrhea in a previously menstruating female who presents underweight or is engaged in intensive physical activity, and approximately every 2 years thereafter. While the use of DEXA has been debated as an ideal means of bone density evaluation in this population, it is the one most commonly used and is a reasonable measure. Abnormal DEXA results often motivate patients in their recovery. Clinicians should keep in mind that, since serious athletes tend to have 5–15% higher bone mineral density than age-matched controls, a DEXA result in the low-normal range for an athlete may represent meaningful bone density loss as a result of energy imbalance, and should not be dismissed as "merely normal" (Witkop & Warren, 2010).

In conjunction with full weight restoration, there are a number of best practices to consider. Patients should have their vitamin D-OH level checked, and repletion should be initiated if the level is below 20–30 ng/ml. Virtually all randomized controlled trials argue *against* use of oral or transdermal estrogen for women, if used solely for the purpose of "restarting the menstrual period" or for bone health. These preparations do not promote improved bone density (due to the dual pathophysiology of bone density loss described above), their use obscures the resumption of the natural menstrual cycle that can be a marker of appropriate weight restoration, and they precipitate monthly blood loss in a population already suffering from anemia of bone marrow suppression (Mehler, Cleary, & Gaudiani, 2011).

Many patients persist in exercise regimens inappropriate for their nutritional status, in part because they believe exercise improves bone density. It does in well-nourished people. However, an excellent study has indicated that while patients are underweight, even moderate exercise (such as pacing) can hasten bone density loss (Waugh, Woodside, Beaton, Coté, & Hawker, 2011). On the positive side, once patients fully restore weight, even intense exercise may improve bone density. I share this study with all my patients to help combat their burning desire to "get more active," and to remind them that "serious exercise is a privilege of recovery." Women athletes with a combination of bone density abnormalities, abnormal energy availability with or without a formal eating disorder, and menstrual dysfunction (also called the Female Athlete Triad) can now use an excellent evidence-based clinical scorecard to assess appropriateness of returning to athletic training and competition (De Souza, Nattiv, Joy, Mistra, Williams, Mallinson, Gibbs, Olmsted, Goolsby, & Matheson, 2014).

A good randomized controlled trial by Miller et al. (2011) demonstrated that bisphosphonates, a class of medications that inhibit bone resorption, are effective in improving bone mineral density in young women with early bone density loss due to AN. Further studies are needed to determine benefit in seriously and chronically ill men and women with severe osteoporosis due to AN. In our medical center we recommend that all osteoporotic men and all women past childbearing age, whose vitamin D level is normal, start a weekly bisphosphonate. For younger women, we have a more detailed discussion about bisphosphonate use and decide with the patient whether to start this treatment. A harm-benefit discussion is helpful and necessary, because bisphosphonates remain in the system for a long time, cross the placenta, and have been proven to cause fetal harm in animal studies. Limited human studies suggest that bisphosphonates are reasonably safe in carefully selected populations. In this younger female population we view bisphosphonate use more as a bridge to full weight restoration than as a long-term treatment (Stathopoulos et al., 2011; Vujasinovic-Stupar, Pejnovic, Markovic, & Zlatanovic, 2012). A promising new therapy which has recently shown even more benefit for AN mediated osteoporosis than the bisphosphonate class is Forteo, or teriparatide (Fazeli, Wang, Miller, Herzog, Misra, Lee, Finklestein, Bouxsein, & Klibanski, 2014).

Medical Complications of Severe Purging

At any body weight, severe purging causes medical complications due both to the direct effects of purging and to a separate set of problems when purging stops. Purging usually refers to vomiting, abusing laxatives, or abusing diuretics either concurrent with restricting or after binge eating. Medical consequences of severe purging include electrolyte abnormalities, constipation and potentially permanent colon damage, reflux, and volume depletion, in addition to dental erosion and parotid hypertrophy that may not subside even after sustained recovery (Mehler & Andersen, 2010). The most significant issue with purging cessation is the severe rebound edema (swelling as a result of fluid retention) that can develop, driving the patient back into ED behaviors to combat rapid weight gain and the anxiety-provoking experience of "feeling bloated."

Electrolyte Abnormalities

Purging via vomiting, laxative abuse, or diuretic abuse causes myriad electrolyte abnormalities. Hypokalemia is one of the most common, as potassium is lost in emesis, diarrhea, or urine. Low potassium, especially if less than 2.5 mmol/L, can cause muscle weakness or breakdown, breathing muscle weakness, intestinal dysfunction, and heart arrhythmias. A discussion of potassium repletion will be deferred to the treatment of volume depletion in this population (see below). Volume depletion is the technical term for the depletion of body salt and water, often casually referred to as "dehydration."

Hyponatremia (low serum sodium: < 135 mmol/L) in patients with EDs has numerous etiologies requiring quite varied management strategies. Volume depletion, insufficient free water excretion, and the syndrome of inappropriate antidiuretic hormone secretion (SIADH) can all cause hyponatremia. In volume depletion, total body salt and total body water are low, due to volume losses arising from significant sweating, vomiting, diarrhea, or urinary losses from diuresis. In insufficient free water excretion, the total body sodium is normal, but it is diluted by overly high intravascular water volume because, for a variety of reasons, the kidneys cannot excrete the excess water.

Typically, three conditions cause insufficient free water excretion. One is psychogenic polydipsia, in which a patient cannot resist the drive to drink water and may go to extreme lengths to satisfy that drive, consuming up to 15 or more liters (i.e., ≥ 4 gallons) of fluid a day. Another mechanism may be termed "low solute/poor free water excretion." A person eating a normal amount of food can usually process up to 20 liters of fluid daily, urinating away the excess water and maintaining a normal serum sodium level. However, the kidneys require adequate solute to process water and produce dilute urine. Thus, a person who is severely restricting calories may take in so little solute daily that even two or three liters of water overwhelm the kidneys' ability to excrete water, and the remaining excess water in the bloodstream manifests as hyponatremia (Berl, 2008). This is fairly common in patients who eat very little but persist in significant consumption of tea or water. Finally, SIADH occurs when antidiuretic hormone is present, preventing urinary excretion of water despite too much water in the blood. SIADH can be caused by numerous medications, including narcotics and selective serotonin reuptake inhibitors (SSRIs), and also by pain. Low serum sodium can cause encephalopathy and seizure. Overly rapid correction can also cause a form of irreversible brain damage called central pontine myelinolysis (CPM).

Hypovolemic hyponatremia in patients with AN should be treated with a slow infusion of normal saline (typically no faster than 50 ml/h) and frequent checks of serum sodium. Medical hospitalization should certainly be considered in patients with serum sodium levels below 130 mmol/L, or if any acute confusion or drowsiness develops. Conservatively, serum sodium should not rise faster than 4–6 mmol/L in a 24-hour period. (Sterns, 2015)

With hyponatremia due to low solute/poor free water excretion, a modest fluid restriction (2 liters daily) must be imposed for a matter of days, only until calories have increased sufficiently that the kidneys can once again package up and excrete a more normal fluid intake. In my experience, a patient eating between 1600 and 2000 kcal a day (6,694–8,368 kJ/day) should be able, in the absence of other etiologies of hyponatremia, to drink even relatively large volumes of fluid without risk of dropping the serum sodium. Patients with true SIADH due to psychiatric medicines may need to remain on a 1.5–2 L/day free fluid restriction regardless of calorie intake.

Volume Depletion

As noted previously, patients who purge chronically can develop severe volume depletion. Treatment requires a balanced restoration of salt and water, either by oral or intravenous means, depending on the intensity and seriousness of the illness. Symptoms of volume depletion include lightheadedness (due to true orthostatic hypotension), weakness, fatigue, headache, and muscle cramps. Multiple laboratory abnormalities reflect volume depletion. A high serum bicarbonate (typically > 30 mmol/L) generally reflects a contraction metabolic alkalosis, commonly found in volume depletion due to severe purging. Extremely high bicarbonate levels (>40 mmol/L) increase the risk of seizure, and signal a high likelihood of serious volume overload and edema with purging cessation or intravenous fluid administration.

Pseudo-Bartter Syndrome (Secondary Hyperaldosteronism)

Patients with severe chronic purging live in a state of chronic volume depletion. As a result, their bodies upregulate adrenally produced aldosterone in order to prevent urinary losses of salt and water. This secondary hyperaldosteronism is known as pseudo-Bartter syndrome, and it is this phenomenon that causes the severe, often overnight edema that makes many patients

resist medical care (Bahia, Mascolo, Gaudiani, & Mehler, 2012). Rapid development of edema is particularly problematic for patients who purge chronically, because they often already suffer from distortions and fears around being "hydrated" (even without edema formation or bloating), which may be as strong as their fears of food and fat. I describe this to patients in this way: "Your subconscious brain believes you are dying of dehydration in the desert. It says to itself, 'If we are so lucky as to come across an oasis where there is salt and water, we'd better have upregulated any hormone that will prevent us from urinating away these life-saving nutrients.' And that's exactly what it does with increased production of aldosterone."

Hyperaldosteronism causes urinary wasting of potassium. Both dietary and prescribed potassium will be lost in the urine rather than remaining in the serum. This helps explain why patients who chronically purge may be prescribed enormous doses of potassium without an appropriate serum response. Some fraction of the potassium will be lost in the urine unless the hyperaldosteronism is addressed (Bahia et al., 2012).

Treatment of severe volume depletion and pseudo-Bartter syndrome occurs on three simultaneous fronts. One, the patient must stop purging altogether. Continuation of any purging will continue to drive hyperaldosteronism. Two, the patient must slowly rehydrate. In the outpatient setting this might involve prescriptions of a low-sodium diet and no more than 2–3 liters of total fluid a day. In the inpatient setting, slow intravenous normal saline, usually at 50 ml/h, should be infused until serum bicarbonate is less than 30 mmol/L and serum sodium is above around 130 mmol/L. This may take a few days. Third, concurrently with slow hydration in the outpatient or inpatient setting, the patient's hyperaldosteronism must be blocked pharmacologically until the body naturally downregulates it after 2–3 weeks. We use spironolactone, a direct aldosterone antagonist that is prescribed more commonly as a potassium-sparing diuretic, 12.5–25 mg by mouth daily, for 2–4 weeks. This appears safe even when patients are hypotensive. I remind patients that spironolactone is not a powerful enough diuretic to pose a risk of abuse.

With this clearer understanding of the relationship between purging, volume depletion, aldosterone, and urinary potassium wasting, we now turn to the vexing problem of hypokalemia treatment in patients who purge chronically. Potassium replacement will not work while a patient remains seriously volume depleted, as determined by history, exam, and a bicarbonate greater than 30 mmol/L. Oral potassium supplements are caustic to the stomach and may cause pain and nausea, especially at high doses. Front-line clinicians who find themselves prescribing more and more potassium should instead dedicate their efforts to convincing the patient to present to a more contained level of care (see Chapter 50) as, clearly, purging behaviors cannot be controlled in the present setting. For patients in our unit, we treat the volume depletion as described above, add 20–40 mmol/L of potassium chloride to the slow intravenous drip, give at most 20 mmol oral potassium three times daily with meals, follow EKGs and telemetry, and accept that it may take a few days for the serum potassium to normalize.

Constipation and Atonic Colon from Stimulant Laxative Abuse

Constipation due to energy-conserving slowed gastrointestinal transit is universal in people who are significantly underweight. Patients who abuse stimulant laxatives and enemas prematurely discontinue treatment more often than those who do not abuse laxatives (Brewerton & Costin, 2011). In part, this may be because they can suffer from significant constipation when they enter treatment. For all patients with AN, high-fiber diets actually worsen constipation at low weights, as do dehydration and electrolyte abnormalities such as

hypokalemia. In addition, patients who abuse stimulant laxatives can develop tolerance to their current dose of medication, requiring an escalating dose to obtain the desired results. Although the evidence base is sparse in patients with severe and enduring stimulant laxative abuse, the clinical consensus in our program is that long-term abuse of stimulant laxatives increases risk for atonic ("floppy") colon, in which permanant damage to colonic nerve cells further slows colonic function. Indeed, abuse of stimulant laxatives over time may cause the irreversible cathartic colon syndrome, which can be managed only through colectomy and ileostomy. It is unknown at precisely what dose or duration of abuse this may occur, and it probably differs in each patient (Mehler & Andersen, 2010).

For this reason, I strongly recommend against tapering stimulant laxatives, even under medical supervision. No clinician, no matter how kind or well intentioned, wishes to have directed the ongoing ingestion of a medication that may be having permanent toxic effects. Our program discontinues stimulant laxatives completely on admission, followed immediately by very slow hydration and electrolyte restoration as described above, with a target potassium of around 4.5 mmol/L for optimum bowel function. We also prescribe a moderate dose of an osmotic laxative such as polyethylene glycol (17 g one to three times daily), allow a discrete amount of ambulation on the medical ward, and help set expectations for normal bowel function. A patient accustomed to three bowel movements daily while on stimulant laxatives must be reassured that a reduced frequency is safe and normal. If a patient should fail to have what she or he feels is an adequate bowel movement within 3 days or so, we take an abdominal x-ray and examine the film with the patient at the bedside: if there is little stool, we continue on the prior treatment course, and the patient is reassured. If there is moderate to significant stool, we offer a lubricating suppository such as glycerin, increase polyethylene glycol, and in extreme situations offer a soapsuds enema to relieve discomfort.

Gastroesophageal Reflux Disease (GERD)

Patients with AN frequently experience heartburn, and many take medications to treat GERD. In patients who purge via vomiting, the etiology of GERD makes sense due both to the chronic acid exposure generated by vomiting and to the progressive dysfunction of the lower esophageal sphincter that can result from repeated binge eating followed by induction of emesis. However, pure restrictors frequently have esophageal symptoms as well. The etiology remains unclear. A study of young adults with AN showed that lower esophageal pressures are within normal limits (although lower in purgers than in restrictors) and symptoms did not meaningfully improve with weight restoration (Benini et al., 2010).

The most serious medical consequence of GERD is development of Barrett's esophagus, in which intestinal metaplasia due to chronic acid exposure changes distal esophageal cells into precancerous cells. Up to 5–15% of all patients who present with GERD symptoms may also have Barrett's (Shaheen & Richter, 2009). In addition, esophageal rupture (Boerhaave syndrome) and bleeding from an esophageal tear (Mallory–Weiss syndrome) may occur after forcible vomiting, and both can be life-threatening. The suite of treatments available for GERD is beyond the scope of this chapter, but ED behavior control, frequency and size of meals, reduction of acidic, alcoholic, and caffeine-containing foods, postprandial body position, and pharmacologic options all play a role. In our program, patients with serious purging behaviors for 5 years or more are encouraged to have an upper endoscopy to evaluate for Barrett's, and to repeat this assessment at regular intervals.

Conclusions and Future Directions

Medical clinicians have two main responsibilities to patients with EDs. One is to competently diagnose and initiate treatment for the common medical complications of restricting and of purging (see Chapter 14). It is the clinician's responsibility to refer the relapsing or declining patient—who may fiercely defend his or her right to remain as independent as possible in the outpatient setting—to a higher level of care (see Chapter 50). Indeed, a difficult decision may be required to terminate the medical relationship if the patient will not agree to a contract in which further evidence of physical decline and uncontrolled behaviors will directly result in admission to a higher level of ED care. Evidence of decline and continued loss of control will manifest as one or more of the following: recurrent hypokalemia; failure to restore weight; inability to keep appointments with other members of the treatment team; and refusal of permission that the multidisciplinary team speak freely with each other and with a caring adult in the patient's life (see Chapter 50). No matter how much the patient insists that obligations—be they familial, academic, professional, marital, parental, and/or extracurricular—absolutely prevent admission to a more contained environment, the patient's eating disordered actions say that she or he is in fact unable to resist prioritizing the demands of the ED above all else.

The other responsibility of the medical clinician is to use objective evidence of physiologic suffering from ED behaviors to help the patient break through denial of disease. Indeed, in order to counter the cruel demands of their ED, patients may need regular reassurance that they are in fact physically ill, which may help them resist the temptation to restrict or purge and also may give them a sense of "worthiness" for treatment. While not every patient with an ED has excellent insurance, or even adequate local resources with ED expertise, many do and will benefit from the medical clinician raising the alarm. For those who have access only to emergency services (e.g., because of insurance inadequacy), increased clinician awareness of best care practices can only help. The good news is that the vast majority of medical problems arising from severe restriction and purging will resolve with appropriate nutritional rehabilitation and full weight restoration. As the field of EDs grows, we must hold ourselves to the highest standard of compassionate and competent medical care for these patients, not just mental health care, in order to facilitate the smoothest and swiftest recovery possible.

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Treating Eating Disorders in Children and Adolescents

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An estimated 85% of eating disorders (EDs) have an onset in adolescence (American Dietetic Association, 2001), making early detection and effective treatment of EDs in childhood and adolescence vital. This is especially important given the high mortality and morbidity rates associated with EDs (Arcelus, Mitchell, Wales, & Nielsen, 2011; see also Chapter 55).

The primary difference between the treatment of EDs in childhood and adolescence, compared to treatment of adults, is the greater availability of the family to provide support. Early theories suggested that a specific type of family organization or functioning explained the development of EDs in a particular individual (Minuchin et al., 1975), but there is a lack of convincing evidence to support such a causal explanation (Konstantellou, Eisler, & Campbell, 2012). As a consequence, there has been a shift in focus in the treatment of young people with EDs, away from pathologizing the family toward mobilization of family resources to elicit and support recovery and change (Eisler & Lask, 2008; see also Chapter 60).

In this chapter the evidence base for the most effective treatment approaches for young people with various forms of EDs will be reviewed before looking at recent developments in treatment approaches and levels of care. Possible adjuncts and modifications to core therapy approaches, in the absence of expected treatment progress, will be discussed. We will also review some limitations regarding evidence-based treatments for children and adolescents with EDs and then consider conclusions and future directions.

Family Therapy in the Treatment of Anorexia Nervosa (AN)

To date, the evidence base for the treatment of AN in young people gives precedence to the use of AN focused Family Therapy (FT-AN). FT-AN is sometimes referred to as Family Based Treatment (FBT) or the Maudsley Model of Family Therapy (see Chapter 60). We find both of these terms conceptually misleading. The first suggests that the therapy is defined by who attends rather than by a theoretical conceptualization, while the second implies that it is a distinct model of family therapy (comparable to, e.g., Structural or Strategic FT). What makes FT-AN distinct is its focus on the ED. This requires specific expertise on the part of the therapist about the

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nature of EDs and an understanding of how this affects the family, but conceptually the treatment draws on the full range of systemic family therapy theory and techniques.

FT-AN is recommended in the American Psychiatric Association (2006) guidelines and by the National Institute for Health and Care Excellence (NICE, 2004) in the United Kingdom. There have been eight randomized controlled trials (RCTs) evaluating family therapy for adolescents with AN (Eisler et al., 2000; Geist, Heinmaa, Stephens, Davis, & Katzman, 2000; Godart et al., 2012; Le Grange, Eisler, Dare, & Russell, 1992; Lock, Agras, Bryson, & Kraemer, 2005; Lock et al., 2010; Robin et al., 1999; Russell, Szmuckler, Dare, & Eisler, 1987). These RCTs have provided evidence that FT-AN is an effective intervention for adolescent AN, produces comparatively greater effect sizes than individual therapy, and has good long-term outcome follow-up with low relapse rates of less than 10% (Eisler et al., 1997; Eisler, Simic, Russell, & Dare, 2007; Lock, Couturier, & Agras, 2006).

General Perspective

FT-AN is a treatment *with* the family and not *of* the family, as it emphasizes the family as a resource for managing the eating problems and explores how family functioning has been affected by, and become organized by, the ED (Eisler, 2005; Eisler, Lock, & Le Grange, 2010). A young person's reluctance to eat challenges one of the key priorities of parenthood—to feed and nourish a child. In the face of a child not accepting enough nutrition, parents are likely to feel helpless, disempowered, confused, frustrated, and angry, and the family system can become paralyzed. As a result the ED occupies a key role in the organization of the family, confines family development to the present, and limits its ability to address developing family life-cycle needs, such as adolescent individualization. Available patterns of family interaction become restricted, leading both to a decreasing repertoire of family behaviors and an intensification of certain aspects of previous family functioning organization (Eisler, 2005).

Phases of Treatment

Phase 1 Originally, FT-AN was described as having three phases of treatment (Dare, Eisler, Russell, & Szmukler, 1990), but recently we have differentiated the early stages and suggest that four phases provide a more useful account (Eisler, Simic, & Treatment Team, 2012). The initial phase involves engagement of the family and development of the therapeutic contract, a central part of which is a formal agreement that in order for young people to get better they need their parents to help them by managing their food intake. This requires a shift in the understanding (both on the part of the adolescent and the parents) of the meaning of "parents taking charge of eating" so that it is experienced primarily as an act of caring rather than power and control. The therapist actively looks for opportunities to highlight this while empathizing with the young person's (temporary) loss of independence in this important area of life. Even though the young person may not always agree explicitly with the goals of treatment (which have to include weight gain), or does so only grudgingly, it is important that the therapist works to make an emotional connection and show sympathy with their predicament.

During the first phase the treatment team, which consists of family therapists, clinical psychologists, clinical nurse specialists, and child psychiatrists (see Chapter 50)—all of whom act as the primary therapist in allocated cases—provides psychoeducation about the effects of starvation (Keys, Brozek, Henschel, & Mickelsen, 1950). Similarities in symptoms between starved healthy

people and sufferers of AN are highlighted, both on a psychological level (e.g., fear of losing control, low and/or unstable mood, preoccupation with food) and on a physiological level (e.g., poor circulation, delayed gastric emptying, decreased concentration span; see Chapters 14, 18, & 52). This psychoeducation tends to increase anxiety around the potential physical consequences of AN, which can mobilize the whole family toward constructive action. It also helps families to shift perception of anorexic behaviors away from defiant, willful actions by a young person and toward seeing those behaviors as part of the illness that requires the family to unite against.

This leads to another fundamental concept in the initial phase: externalization of the AN through externalizing conversations (White, 1984), which position the AN as the cause of the destructive behaviors, such as food restriction, and perceptions, such as distorted body image. For example, a conversation might highlight that it is the AN telling the sufferer that she or he is not thin enough, rather than an objective truth. Externalization is another way of framing the parents as a crucial resource in helping the young person to tackle the AN and serves to minimize feelings of guilt and blame. Describing the physical effects of starvation is also a form of externalization as it gives a different meaning to many of the AN symptoms, framing them as aspects of starvation rather than purposeful behaviors or psychological expressions of AN.

Early in phase 1 of treatment, a family meal is arranged at which the therapist helps the parents to build on their own expertise in feeding their child. The aim is to create a safe therapeutic context where the therapist can support the family in tackling the most difficult aspect of their current lives, strengthening the therapeutic alliance, and rebuilding parents' confidence in their abilities to feed their child.

Siblings are encouraged to take part in treatment to support the young person with AN and to explore the impact of the illness on their relationship. The particular role that siblings may take during the course of treatment varies considerably depending on their age and nature of the relationship with the young person with AN; however, they are not encouraged to take any responsibility for feeding their brother or sister or shielding their sibling from "pressures" created by the treatment.

Meal plans, devised by the team dietician, can be used if there are any risks of refeeding syndrome (see Chapters 52 & 61) or if families feel that a meal plan will help them to have more conviction regarding necessary intake for weight gain, or as an aid in minimizing recurring mealtime conflicts around food. Later in therapy it is important that the use of any meal plan is reviewed so that it does not act as a barrier to progress toward return to more flexible family eating.

The final component of the initial phase of FT-AN is to increase the family system's hope and motivation for positive change. This can involve the therapist providing information to families and young people concerning the usual duration of treatment (9–12 months). Use of motivational interviewing techniques (see Chapter 63) may help the family consider the potential losses and gains of moving towards recovery.

Phase 2 The key aim of the second phase is weight restoration and involves the family continuing to challenge anorexic behaviors. Parents are further supported to take charge of the young person's eating, and to develop new strategies and skills to manage the AN and to help the young person to work toward recovery. It is emphasized that this high level of parental control over eating is temporary and that the longer-term goal is for the young person to regain responsibility over eating when his or her physical and cognitive state is improved. Ongoing psychoeducational input about, for example, changes in cognitive functioning with starvation and its restoration with refeeding and weight gain (Hatch et al., 2010) helps reinforce the need for temporary responsibility for weight gain being located with parents during this stage.

Phase 3 The third phase of treatment begins when there is consistent progression toward healthy weight and when anorexic cognitions are not exerting so much power over the young person's behavior. Control over eating is gradually handed back to the young person, which also marks a return to a more age-appropriate position in the family in general. This phase involves exploration of individual and family development, including the young person embarking on developmental tasks that may have been placed on hold due to the AN. The therapist supports the family in differentiating between behaviors driven by AN and everyday adolescent behavior. This process acts in some ways as a partial reversal of the earlier externalization, helping the young person to recapture responsibility for their eating and progression toward individualization. The family as a whole can also start to plan for the future as the journey toward recovery from AN continues.

Phase 4 In this final phase the treatment team and the family negotiate the ending of treatment and advancement toward future plans. Families are reminded that when families are actively involved in treatment, child and adolescent AN has low relapse rates, although some challenges around eating may remain or resurface. Consequently, the young person and family are helped to problem-solve around any possible relapses. The family and young person are supported to continue in their readjustment to family life without AN. Discussions around treatment termination generally elicit a range of related issues, including loss, dependence, and where responsibility lies if problems arise in the future.

Comparison of FT-AN with Other Therapies

Lock et al. (2010) compared adolescent-focused individual therapy (AFT) with FT-AN. AFT helps young people to identify and define their emotions and to tolerate negative affective states rather than numbing themselves through starvation and other weight loss behaviors. In this RCT no statistically significant differences were found between the two groups in terms of full remission at end of treatment, but FT-AN was significantly superior to AFT in the rates of partial remission (89.1% vs. 66.9%) and in the level of weight achieved at end of treatment. There were also differences in rates of full remission favoring FT-AN at 6- (39.9% vs. 18.3%) and 12-month (49.3% vs. 23.2%) follow-up. A recent open (i.e., without a comparison group) follow-up study by Dalle Grave, Calugi, Doll, and Fairburn (2013) provided evidence that cognitive-behavioral therapy (CBT; see Chapter 56) was successful in facilitating weight gain in adolescent females aged 13–17 years alongside a decrease in ED psychopathology and maintenance of positive outcome over a 60-week follow-up period, despite minimal further treatment.

Treatment of Bulimia Nervosa (BN)

To date, there have been only two RCTs evaluating treatment of BN in adolescents. Schmidt et al. (2007) compared FT, modified for use with BN (FT-BN), and a self-help version of CBT. Subjects participating in the study were largely females ages 13–20. Those receiving CBT had significantly lower rates of binge eating post-treatment compared to FT-BN, although the difference was no longer significant at 6-month follow-up, with 41.4% in the FT-BN group and 36% in the self-guided CBT group binge and purge abstinent. Le Grange, Crosby, Rathouz, and Leventhal (2007) compared FT-BN and individual supportive psychotherapy (SPT) in adolescent girls aged 12–19. Remission rates were significantly higher for FT-BN compared to SPT at post-treatment (39% vs. 18%) and 6-month follow-up (29% vs. 10%).

With respect to individual self-help treatments, Pretorius et al. (2009) found that 101 older adolescents (mean age = 18.8 years) who had a brief Internet-based CBT intervention had significant improvements in ED symptoms and reduced service contacts at 3 months and maintained these gains at 6 months. Internet-based interventions and support are becoming an increasingly acceptable intervention of choice for some adolescents who do not have an ED but are at high risk (see Chapter 46), although one should consider the isolative nature of this form of self-help in a population that tends to have difficulties in interpersonal relationships (Arcelus, Haslam, Farrow, & Meyer, 2013). Further research is needed to ascertain the comparative efficacy of FT-BN and individual therapy for adolescents with BN, and to determine which group might benefit from self-help interventions as the first line of treatment. Indeed, there is a clear need for more research on the treatment of adolescent BN in general. This is a group that is poorly identified (House et al., 2012), even though its incidence in late adolescence is comparable to that of AN (Micali, Hagberg, Petersen, & Treasure 2013).

Treatment of Binge Eating Disorder (BED)

Although the prevalence of BED in adolescence appears to be at least as high as that of BN (Le Grange, Swanson, Crow, & Merikangas, 2012), research into the treatment of BED in children and adolescents is in its infancy. To date there have been only three studies: a small pilot RCT of interpersonal psychotherapy (Tanofsky-Kraff et al., 2010; see also Chapter 62), a pilot study of CBT (DeBar et al., 2013), and a CBT- based Internet self-help study (Jones et al., 2008). Results are promising, but a great deal more research is needed.

Treatment of Other Eating Disorders

With the introduction of the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*; American Psychiatric Association, 2013), significant changes have been introduced to the taxonomy of EDs, with important implications for children and adolescents (see Chapter 13). The most significant changes are removal of the Eating Disorder-Not Otherwise Specified (ED-NOS) category and broadening of the classifications of AN and BN (see Chapters 4 & 11). These have implications for research and treatment, because in both adults and adolescents the diagnosis of ED-NOS was more widely applied than the diagnoses of both AN and BN. Research has suggested that children and young people have been less likely than adults to meet the *DSM-IV-TR* (American Psychiatric Association, 2000) criteria for AN and BN (Bryant-Waugh & Nicholls, 2011). The less restrictive criteria for AN and BN in *DSM-5* will incorporate more young people with EDs. This should result in more precise categorization for research purposes and ultimately provide improved guidance for specific, focused treatment.

Other ED classifications in *DSM-5* (2013) that affect children and adolescents (see Chapter 13) are Avoidant/Restrictive Food Intake Disorder (ARFID), which incorporates eating or feeding disturbances in the absence of concerns regarding weight and shape; Pica, a disorder in which people eat "non-nutritive" substances such as paper, sand, and soil; Rumination Disorder (RD), in which food is regurgitated and rechewed and/or reswallowed; Other Specified Feeding or Eating Disorder (OSFED), which encompasses eating or feeding disorders not meeting full criteria for any specific feeding or eating disorder such as AN, BN, or BED (e.g., atypical AN, purging disorder, night eating syndrome; see Chapters 4 & 11);

and Unspecified Feeding or Eating Disorder, which encompasses conditions that are eating/feeding disorders but the reason for not meeting full criteria are not stated.

Treatment of ARFID, Pica, and RD is still being developed but should be guided by comprehensive assessment and formulation of the primary causative and maintaining factors of the presenting difficulties. Behavioral and parental management interventions linked to specific formulations for these difficulties are likely to be the treatment of choice (Fisher et al., 1994). Some evidence indicates that desensitization for specific childhood food phobias, which would be categorized under ARFID, can be effective (Singer, Ambuel, Wade, & Jaffe, 1992). Treatment of OSFED should follow the treatment guidelines of the eating or feeding disorder most closely resembling the presenting difficulty.

Recent Developments in Treatment Approaches

Multi-Family Therapy (MFT)

Multi-family therapy (MFT; Asen & Scholz; 2010; Dare & Eisler, 2000; Simic & Eisler, 2012) involves bringing five to seven families together for an intensive treatment consisting of an introductory afternoon followed by four consecutive days of treatment and then five to eight follow-up days over the course of a year. MFT integrates theoretical concepts of FT-AN with more general concepts of Multi-Family Therapy. These include helping families overcome a sense of isolation and stigmatization; creating new and multiple perspectives through which families can learn from one another; and creating a "hot house" learning environment in which it is safe to practice new behaviors (Asen & Scholz, 2010). This highly integrative approach incorporates systemic, cognitive, and group therapy techniques. Through structured exercises families generate and share different ideas that can lead toward recovery from the ED, while providing each other with mutual support. Recently, a large multicenter RCT (Eisler, 2013) has shown that a combination of MFT and FT-AN improves outcomes in comparison with FT-AN. MFT for BN is currently being piloted at the Maudsley Hospital in London and shows promising initial outcomes (Simic et al., 2011; Stewart, Voulgari, Eisler, Hunt, & Simic, in press).

Dialectical Behavior Therapy (DBT)

Dialectical Behavior Therapy (Linehan, 1993; see also Chapter 57) is a treatment that was originally developed for people with Borderline Personality Disorder (BPD). In our clinical experience, a subset of young people with EDs present with traits associated with BPD, such as difficulties in the areas of emotion regulation, interpersonal effectiveness, and distress tolerance. DBT is designed to increase adaptive skills in these domains and incorporates many strategies from CBT, but balances these with acceptance strategies through approaches such as mindfulness.

A recent systematic review concluded that DBT for EDs significantly reduced ED behaviors, including binge eating, purging, and food restriction, at the end of treatment and at follow-up, with abstinence rates ranging from 29% to 89% post treatment (Bankoff, Karpel, Forbes, & Pantalone, 2012). However, with one exception (Salbach-Andrae, Bohnekamp, Pfeiffer, Lehmkuhl, & Miller, 2008), these were all studies with adults, so research is needed into the use of DBT for EDs in young people, especially for those where clinical assessment indicates underlying dysfunction in emotion regulation, high impulsivity, or concurrent self-harm

through other methods. An uncontrolled trial of an adapted version of DBT (Radically Open-DBT), which specifically targets difficulties in overcontrol, rigidity, and emotional openness, was conducted by Lynch et al. (2013) with 47 adults with AN. Results were encouraging but will need to be replicated with adolescents.

Cognitive Remediation Therapy (CRT)

Cognitive remediation therapy has been shown to improve cognitive flexibility in people with schizophrenia (Wykes et al., 2007). More, recently, CRT for AN has been developed and piloted in adult inpatient populations, based on findings that adults with AN have difficulties in set-shifting, with heightened attention to detail at the expense of holistic processing, and a tendency toward rigid, sometimes obsessive, thinking and behaviors (Lopez, Tchanturia, Stahl, & Treasure, 2008; Roberts, Tchanturia, Stahl, Southgate, & Treasure, 2007; see also Chapters 17 & 18). Although research into the neurocognitive profiles of adolescent AN is sparse, there is some evidence that adolescents with AN have similar set-shifting and global processing difficulties (McAnarney et al., 2011).

CRT utilizes cognitive exercises to help people broaden and strengthen their thinking skills. These include Stroop tasks to practice switching attention between different aspects of a stimulus such as the color or content of a written word, and tasks illustrating the difference between focusing on finer detail versus more global content, such as verbally describing a geometric figure for another person to draw.

CRT has been shown to improve cognitive flexibility in adults with AN (Tchanturia, Davies, & Campbell, 2007) and to enhance efficacy and retention rates of other treatments (Pitt, Lewis, Morgan, & Woodward, 2010). It has been suggested that adolescents, who generally have shorter duration of illness, may be more amenable to increasing their cognitive flexibility than are adults (Tchanturia & Lock, 2011), and initial studies investigating CRT in adolescents with AN (Pretorius et al., 2012) suggest that CRT is a well-received treatment with some promising results.

Levels of Care

Inpatient Treatment

There is no evidence that psychiatric hospitalization for young people with EDs is more effective than outpatient treatment (Crisp et al., 1991; Gowers et al., 2007). There are clear unfavorable implications of admitting young people to hospital, such as separation from family, community, and education. Hospital also provides opportunities to learn a full range of ED behaviors from other young people on the unit, alongside a lack of opportunity to generalize more positive eating and coping strategies to the home environment. Psychiatric inpatient admissions have readmission rates of 25–30% after the first admission, rising to 50–75% for subsequent admissions (Castro, Gila, Puig, Rodriguez, & Toro, 2004; Lay, Jennen-Steinmetz, Reinhard, & Schmidt, 2002). Nevertheless, in some instances hospitalization is warranted. The American Academy of Pediatrics (2003) and the Society of Adolescent Medicine (Golden et al., 2003) have published indications for medical hospitalization of children and adolescents with EDs. These include severe malnutrition and abnormalities in electrolyte balance, heart rate, body temperature, and blood pressure (see Chapters 14 & 50).

There is little agreement across programs and settings on what psychiatric inpatient treatment is and what its aims are. There is also huge variability in the nature, duration, and therapeutic goals of admissions and in links with postadmission treatment. This inconsistency is due in part to theoretical difference, but is also determined by economic and organizational health service factors (e.g., what insurance or health service budgets are willing to pay for). Aside from brief admissions to stabilize physical risk and to commence the initial refeeding process, many inpatient ED programs conceptualize weight restoration to a healthy level as a key aim, when in reality there is little evidence that this needs to occur in an inpatient setting.

Regarding hospitalization with the primary aim of emergency weight restoration, some data (Robb et al., 2002) promote use of nasogastric feeding, but the clinical need for this has not been confirmed (Golden et al., 2003). Long-term benefits of this intervention are also highly questionable, as the young person and family take minimal responsibility for food intake, therefore compromising the opportunity to make a collaborative and progressive plan toward weight gain.

If no alternatives to psychiatric inpatient care are available, or if other forms of treatment have not been successful and admission is unavoidable, then any negative effects can be minimized by keeping the admission short in duration, utilizing opportunities for home leave, involving families in the treatment program, and making use of staff with expertise in EDs. Wallis et al. (2013) have piloted a 2-week hospital-based family residential program wherein the whole family takes part in a structured daily therapeutic intervention in which the locus of control is shifted from hospital staff to the child's parents as either a step down from inpatient care or a step up from outpatient care for nonresponders. This has produced encouraging initial outcomes.

Outpatient Follow-up and Day Treatment

Outpatient psychological treatment of EDs in young people is strongly recommended following inpatient admissions, alongside regular monitoring of both physical and psychological risk. There is evidence to support posthospitalization psychological interventions, with family therapy being more effective than individual therapy (Eisler et al., 1997; Russell et al., 1987). Rhodes and Madden (2005) found that introduction of FT for young people with AN following discharge from hospital resulted in significant improvements in clinical outcomes and reduction in hospital readmission rates.

Day-patient treatment programs for young people with AN have been introduced as an alternative to inpatient care, a step down from inpatient treatment, or when young people are not meeting the expected therapeutic progress in outpatient care. At the Maudsley Hospital in London, the Intensive Day Patient Treatment Program integrates supervised mealtimes with therapeutic groups targeting the maintaining factors of EDs, such as cognitive rigidity, perfectionism, anxiety, and difficulties in interpersonal relationships, including social isolation. Young people take part in mindfulness and therapeutic yoga (see Chapter 33), as well as groups that help increase motivation to overcome the ED, re-engage with school, and broaden the spectrum of self-appraisal beyond weight and shape. Families take an active part in the program through regular family reviews and multifamily meetings that include family meals, learning about the process of the young people's groups, and parent skills training.

In comparison to inpatient treatment, day treatment allows for greater family input as young people can eat with their families in the evenings and on weekends. Any difficulties can be subjected to guided problem-solving in family meetings within the program. Incorporating significant elements of FT and family involvement within day-patient programs is proving promising (Girz, Robinson, Foroughe, Jasper, & Boachie, 2013).

There has been one randomized multicenter trial comparing day-patient treatment after 3 weeks of inpatient care with continued inpatient treatment (Herpertz-Dahlmann et al., 2014). The study, conducted in Germany with adolescents with AN, found that day-patient treatment after short inpatient care was as effective as continued inpatient treatment with regard to weight restoration and maintenance during the first year after admission. Further research is required to establish which elements of intensive day-patient treatment are most effective and to what extent day-patient treatment might accelerate outpatient treatment or decrease the number or duration of inpatient admissions.

Adjuncts and Modifications to Core Therapy

A number of more specific adjuncts to therapy might be indicated when there are comorbid difficulties (see Chapters 15 & 54) or in the absence of expected treatment progress. In young people who present with comorbidities to their ED, such as generalized anxiety, depression, obsessive-compulsive traits, intolerance of uncertainty, dysfunctional perfectionism, or difficulties in regulating emotions or interpersonal relationships—all of which may act as maintaining factors to the ED itself—additional treatment targeting these specific problems may be indicated. However, caution should be used, as some of these difficulties, for example depressive mood, can be resolved with the weight gain. It has not been established whether the delivery of treatment targeting these specific areas should be incorporated into family therapy, added as individual therapy, or delivered in group settings, especially for young people admitted to inpatient and day-patient treatments.

With regard to the treatment of AN utilizing a FT approach, research indicates that certain adaptations are beneficial if there is a lack of progress in treatment or if distinctive family features are present. For example, separated FT (in which the young person and parents are not seen together but the same treatment model is employed) may be beneficial in families where there is high expressed emotion (e.g., parental criticism) directed toward the young person (Eisler et al., 2007). An increased duration of FT may be indicated for young people presenting with more severe eating-related obsessive-compulsive thinking styles and for those from nonintact family systems (Lock et al., 2005).

Limitations of the Current Evidence-Based Treatments

There is shared acknowledgment in the field of EDs in children and adolescents that much remains to be done before we reach clear guidelines for the "holy grail" of personalizing treatment. Continuous shifts in diagnostic criteria, as is evident in the current changes with regard to EDs from *DSM-IV-TR* (2000) to the *DSM-5* (2013), however helpful these shifts may be, result in substantial implications for the research literature. Most studies that previously investigated young people meeting the *DSM-IV-TR* criteria for AN and BN, for example, incorporated EDNOS-AN or EDNOS-BN, which would now be most likely diagnosed as AN or BN according to *DSM-5*. Yet, it is extremely difficult to ascertain whether previous research samples will be fully comparable to the future research samples and therefore whether results from the previous studies will be fully applicable to newly diagnosed cases.

There is a trend in current evidence-based treatment approaches toward operationalizing treatments through manualization. Although this can be very helpful, particularly with less experienced clinicians, and is a prerequisite for conducting certain types of outcome studies, it carries

risks if the manual becomes so prescriptive that the treatment is not adapted to suit the needs and pace of each individual, family, or multifamily group. The skill and experience of trained clinicians to maintain appropriate flexibility, deliver the intricacies of specific therapies, and create a positive and beneficial therapeutic alliance should not be overlooked. There is evidence that general or common therapeutic factors, including client and therapist factors, the strength of the therapeutic alliance, and of hope for, and belief in, change, account for as much as 85% of outcome variance (Assay & Lambert, 1999). It has been strongly argued, therefore, that evidence-based practice should not be reduced to the idea of purely implementing the results of RCTs but should incorporate a broad range of research findings as well as clinical expertise (Kazdin, 2008). Findings from the evaluation of manualized treatments should be interpreted as only one part of the mosaic of empirical evidence. Consequently, training of clinicians needs to promote development of an ability to understand and capitalize on opportune moments for a broad range of advantageous and highly skilled interventions in their treatment approach.

Effective practice is the product of far wider influences than use of a particular treatment. Recent research in the United Kingdom comparing areas of London with direct access to specialist outpatient child and adolescent EDs services to areas where initial treatment was offered in generic Child and Adolescent Mental Health Services (CAMHS) found that the impact of this service level organization was far greater than would be the choice of a specific treatment (House et al., 2012). Thus, in areas with direct access to specialist outpatient services, identification of young people who require treatment was two to three times higher than in areas with no specialist provisions. Specialist outpatient services provided high continuity of care and managed 80–85% of referrals on a purely outpatient basis, in contrast to generic CAMHS, which had to refer as many as 80% of cases seen by them for treatment elsewhere at some point. Moreover, in the nonspecialist care pathway, rates of admission to hospital were 40% or more—more than double the rates of those seen in specialist services.

Conclusions and Future Directions

A solid evidence base is emerging for the effectiveness of family therapy in the treatment of young people with AN. This is a promising advancement, especially considering that within the adult population no specific therapeutic intervention has shown superiority over others in the treatment of AN.

However, it should also be highlighted that treatment approaches bearing the name "family therapy," whether manualized or not, may well vary greatly in their delivery due to the context in which they are applied. For example, treatments delivered within the context of inpatient or day-patient settings are likely to differ significantly from those delivered solely in an outpatient setting because what happens within the therapy session is only a small part of the process of change. For instance, a family intervention designed to support parents in taking an active stance in managing their child's eating can have only a limited impact if the young person is in hospital and staff manage mealtimes. The setting in which a treatment is delivered inevitably modifies the "family-based" treatment or any other evidence-based treatment, resulting in treatment diversity and possibly varied effectiveness of the treatment in question. Similarly, a therapy delivered by a solo therapist in private practice may generate a different impact compared to the same treatment provided in the context of a specialist multidisciplinary team (see Chapter 50), just as treatment provided in a home setting may differ in comparison with the "same treatment" delivered in a clinic.

These contextual aspects of treatment have received very limited attention from researchers and all too often are completely overlooked in discussion of treatment research findings. This results in both an oversimplification of what may and may not enhance therapeutic interventions and a hindrance in progression toward precise treatment guidelines with regard to EDs in children and adolescents.

Potentially important studies are currently investigating whether young people with EDs have premorbid difficulties in cognitive flexibility, set-shifting, risk-taking and emotion regulation, amongst other areas. It remains to be seen if studies on neurobiological markers, attention, and cognitive biases, as well as neuroimaging studies on neural circuits that potentially identify predisposing traits for development and/or maintenance of an ED, will yield data useful for developing novel treatments for EDs in children and adolescents. In addition to important research underway on child and adolescent AN, there is a need for more investigations designed to clarify effective treatments for adolescent BN and for ED diagnoses modified in *DSM-5* (2013).

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54

Issues in Treating Comorbidity in the Eating Disorders

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Clinically, the term *comorbidity* indicates that a disease or other pathological process is occurring simultaneously with another (i.e., that one or more disorders are present in addition to the primary disorder). Statistically, comorbidity refers to an increase in the probability, beyond the population baseline, of one disorder, given the presence of another. Comorbidity in eating disorders (EDs), or the co-occurrence of other psychiatric disorders and personality disorders, is well known (see Chapter 15) and has important implications for assessment and treatment planning, clinician training, and treatment team composition (see Chapter 50).

According to findings from the National Comorbidity Survey Replication study (Hudson, Hiripi, Pope, & Kessler, 2007), anorexia nervosa (AN), bulimia nervosa (BN), and binge eating disorder (BED) are highly comorbid with other major psychiatric disorders. In this study, at least one lifetime comorbid major psychiatric disorder was present in approximately 55% of participants with AN, in 95% with BN, and in 80% with BED. Mood, anxiety, impulse disorders, and substance use disorders (SUD) were most common (see Table 54.1).

Personality disorders (PDs) are also highly prevalent in EDs (Sansone, Levitt, & Sansone, 2006). The most common PD in restricting AN (AN-R) is obsessive-compulsive personality disorder (OCPD), whereas borderline personality disorder (BPD) is the most common PD in the AN binge-eating purging type (AN-BP) and in BN. Individuals with BED have a mixed profile, with both OCPD and BPD being prominent disorders (Table 54.1).

In this chapter we review several common comorbid conditions and briefly address three primary questions:

1 *Prevalence*—how frequently are certain psychological disorders comorbid with ED?
2 *Treatment*—once diagnosed, what psychotherapies and pharmacological interventions (see Chapter 59) are considered "best practice" for this comorbid disorder?
3 *Management guidelines and caveats*—with this particular comorbid combination, what general treatment guidelines and caveats should be noted?

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Table 54.1 Common comorbid lifetime psychiatric disorders among eating disorders.

Anorexia nervosa			
Major depression	39.1%	Any mood disorder	42.1%
Specific phobia	26.5%	Any anxiety disorder	47.9%
BPD (AN-BP)	25.5%	Any impulse control disorder	30.8%
OCPD (AN-R)	22.0%	Any substance use disorder	27.0%
Bulimia nervosa			
Major depression	50.1%	Any mood disorder	70.7%
Specific phobia	50.1%	Any anxiety disorder	80.6%
Post-traumatic stress disorder	45.4%	Any impulse control disorder	63.8%
Social phobia	41.3%	Any substance use disorder	36.8%
ADHD	34.9%		
Alcohol abuse/dependence	33.7%		
BPD†	28.4%		
Illicit drug abuse/dependence	26.0%		
Binge eating disorder			
Specific phobia	37.1%	Any mood disorder	46.4%
Major depression	32.3%	Any anxiety disorder	65.1%
Social phobia	31.9%	Any impulse control disorder	43.3%
Post-traumatic stress disorder	26.3%	Any substance use disorder	23.3%
OCPD†	15.9%		
BPD†	11.7%		

Note. The source is Hudson et al. (2007), *except* for items denoted by †, which are from Sansone et al. (2006). ADHD = attention deficient hyperactivity disorder; AN-BP = anorexia nervosa binge-purge type; AN-R = anorexia nervosa, restricting subtype; BPD = borderline personality disorder; OCPD = obsessive-compulsive personality disorder.

Our management guidelines and caveats are not generally constrained by either age or gender. As a general caution, the following material is largely based upon research using criteria from the *Diagnostic and Statistical Manual of Mental Disorders* (4th edition, text revision; *DSM-IV-TR*; American Psychiatric Association, 2000) and earlier versions of this diagnostic manual. With the introduction of *DSM-5* (American Psychiatric Association, 2013), there may be some need for subtle adjustments.

Mood Disorders

Prevalence

Mood disorders such as major depressive disorder (MDD), dysthymia, and bipolar disorder types I (with mania) and II (with hypomania) are present in approximately 40%, 70%, and 45% of adults with AN, BN, and BED, respectively (Hudson et al., 2007; see also Chapter 15). Prevalence rates for mood disorders in adolescents with EDs appear to be generally lower, particularly for AN (10%) and for BN (50%), with BED at 45% (Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011; see also Chapter 36). Importantly, MDD appears to be the most common comorbid mood disorder across all ED subtypes.

Treatment

Psychotherapy Cognitive-behavioral therapy (CBT; see Chapter 56) and interpersonal psychotherapy (IPT; see Chapter 62) are both evidence-based treatments (EBTs) for depression (Butler, Chapman, Forman, & Beck, 2006; Cuijpers et al., 2011). EBT is the integration of the best available research with clinical expertise in the context of patient characteristics, culture, and preferences (American Psychological Association, 2005). Both CBT and IPT have been modified and adapted to treat EDs and are considered EBTs for BN and BED (Murphy, Straebler, Basdem, Cooper, & Fairburn, 2012; Murphy, Straebler, Cooper, & Fairburn, 2010).

In both the treatment of mood disorders and EDs, core concepts of CBT include direct symptom focus, challenging maladaptive beliefs, modifying cognitive distortions, practicing new skills (i.e., problem-solving, assertiveness, stress management), and relapse prevention. The application of IPT to EDs is very similar to the approach used to treat depression. Theoretical underpinnings suggest that interpersonal functioning is a critical element of psychological adjustment and emotional well-being. Therefore, in IPT, specific ED symptoms are not the primary focus. Instead, patients are encouraged to identify and address current interpersonal difficulties in four core areas: (a) interpersonal deficits; (b) interpersonal role disputes; (c) role transitions; and (d) grief. Throughout treatment, interpersonal functioning is consistently connected to the onset and maintainence of the ED (Tanofsky-Kraft & Wilfley, 2010), with the resolution of interpersonal problems reducing symptoms of both ED and depression.

Pharmacotherapy While fluoxetine (Prozac™) has a Food and Drug Administration (FDA) indication in the treatment of BN (see Chapter 59), its use in the treatment of mood disorders is complicated by its exceptionally long half-life of 16 days—a half-life (i.e., the time it takes to lose half its pharmacological effect) issue that can contaminate follow-up drug trials. The effectiveness of antidepressant medications in mood disorders is generally comparable between and within classes of medications (American Psychiatric Association, 2010). Thus, a number of other factors contribute to selection of a particular antidepressant. These include patient preference, past response history, safety profile, anticipated tolerability, side effects, comorbid psychiatric (see Chapter 15) and medical conditions (see Chapters 14 & 52), pharmacological properties of the antidepressant, and cost.

Nevertheless, for most patients, initial treatment with a selective serotonin reuptake inhibitor (SSRI) or a serotonin norepinephrine reuptake inhibitor (SNRI) is appropriate. We suggest a treatment strategy beginning with an SSRI such as sertraline (Zoloft®), citalopram (Celexa®), or escitalopram (Lexapro®), due to their anti-obsessive effects, minimal weight influences, and exceptional tolerability. If these are ineffective, we suggest changing the pharmacological course to an SNRI, such as venlafaxine extended release (Effexor XR®).

When augmentation strategies are indicated (i.e., the addition of a second drug to enhance the efficacy of the antidepressant), we suggest buspirone (Buspar®), low-dose gabapentin (Neurontin®; with doses of 200–400 mg per day, there is little risk of weight gain), or the relatively weight-neutral atypical antipsychotic aripiprazole (Abilify®). While olanzapine has demonstrated some efficacy in the treatment of low-weight individuals with AN in terms of weight gain (Aigner, Treasure, Kaye, Kasper, & WFSBP Task Force on Eating Disorders, 2011), the potential risks are concerning (e.g., unpredictable weight gain, metabolic disturbances, extrapyramidal side effects). We avoid those medications that cause weight gain such as valproate (Depakote®), lithium, and olanzapine (Zyprexa®), even in AN, due to the inability

to predict a weight outcome. Also, in AN, we believe that weight status must be mastered psychologically, not pharmacologically, when possible.

Management Guidelines and Caveats

Whether mood disorders are present prior to the onset of the ED, concurrent with the ED, or the result of engaging in ED behaviors (i.e., dietary restraint, weight loss, compensatory behaviors), they can persist after the amelioration of ED symptoms. In addition, symptom assignment during diagnosis and classification can be challenging. For example, it is often difficult to determine at intake if insomnia/hypersomnia, fatigue, poor concentration, low self-esteem, isolation and withdrawal, and poor appetite/overeating are symptomatic of an independent mood disorder or the ED, or another disorder such as substance abuse (Keys, Brožek, Henschel, Mickelsen, & Longstreet, 1950; Pollice, Kaye, Greeno, & Weltzin, 1997). If depressive symptoms are the sequelae of malnutrition and ameliorate with ED symptom reduction, they will not likely require separate management.

Fairburn, Cooper, and Waller (2008) outline several clinical features that are suggestive of serious comorbid clinical depression: (a) significant increase in depressive features (e.g., anhedonia) independent of a change in ED behavior or patient circumstances; (b) extreme and pervasive negative thinking independent of weight, shape, or eating concerns; (c) significant decrease in socializing or engaging in previously enjoyable activities; (d) significantly impaired decision-making; and (e) psychomotor retardation (objective slowing of thoughts and behavior). They recommend that severe clinical depression be treated with antidepressant medications prior to engaging in EBT (e.g., CBT) for ED, indicating that effective management of clinical depression will improve mood, concentration, energy level, and the ability to engage in ED treatment (Fairburn et al., 2008).

Note that whether or not a genuine clinical depression is present, treating low-weight individuals with antidepressants usually results in nominal responses. Therefore, when possible, we suggest delaying antidepressant treatment until some weight restoration has taken place, both to confirm that depressive symptoms persist after weight increase as well as to improve antidepressant efficacy.

As for the patient who suffers from bipolar disorder, consultation with a psychiatrist is recommended, as these patients can be very challenging to manage. Bipolar patients typically have significant psychiatric comorbidity (Krishnan, 2005) as well as suicide rates up to 10% (Kahn, Faucett, Morrison, & Brown, 2013); in addition, treatment with medications is often not straightforward. For example, SSRIs have been found to be helpful in some patients with BN; however, use of SSRIs in individuals with an underlying bipolar disorder can precipitate the onset of mania or hypomania (Goodwin et al., 2007). In severe cases (e.g., psychotic symptoms), effective medication management will need to precede psychotherapy treatment for the ED.

Anxiety Disorders

Prevalence

Anxiety disorders affect approximately 50% of individuals with AN, 80% of individuals with BN, and 65% of individuals with BED (Hudson et al., 2007). Social phobia and obsessive-compulsive disorder (OCD) are the most common comorbid disorders in AN, BN, and

BED, while generalized anxiety disorder (GAD) is also common in AN (Godart, Flament, Perdereau, & Jeammet, 2002), and simple phobias and post-traumatic stress disorder (PTSD) are common in BN and BED (Hudson et al., 2007; see also Chapter 34). Anxiety disorders most frequently *precede* the onset of EDs (75% of AN cases; 85–90% of BN cases; Godart, Flament, Lecrubier, & Jeammet, 2000).

Treatment

Psychotherapy Cognitive and behavioral approaches are the primary EBTs for anxiety disorders (Deacon & Abramowitz, 2004). Behavioral approaches view pathological fears as conditioned responses that are reinforced through avoidance of the feared stimuli. Cognitive interventions suggest that fear is an intellectual/cognitive appraisal that takes place when an individual is exposed to physical or psychological stimuli or situations that are considered dangerous or threatening (Beck, Emery, & Greenberg 1985). Therefore, anxiety disorders result from *faulty* beliefs or assumptions that certain situations pose a serious threat and increase personal vulnerability. Subsequently, dysfunctional behaviors organize around these beliefs and promote avoidance of the feared stimuli; avoidance, in turn, reinforces and often intensifies the dysfunctional cognitions and feelings. Systematic desensitization, flooding, prolonged exposure or exposure with response prevention, and in vivo exposure or imaginal exposure therapy are all designed to gradually diminish the anxiety response and provide the patient with corrective information about the avoided situation. Treatments that combine both cognitive and behavioral stategies appear to be most effective in reducing symptoms of anxiety (Beck et al., 1985).

CBT is also an EBT for ED and is considered "first-line" treatment (Agras, Walsh, & Fairburn, 2000; see also Chapters 18 & 56). When the symptoms of anxiety are functionally linked to the ED (eating, food, weight or shape concerns), both disorders can effectively and concurrently be treated using CBT (Steiger & Israel, 2010). If the anxiety symptoms are beyond the scope of the ED (e.g., feeling embarrassed doing common things in front of others, fear of open spaces, or undue fear of harm to self or loved ones, chronic checking, handwashing), then symptoms should be addressed independently using cognitive and behavioral stategies directly tailored for that symptom cluster.

Pharmacotherapy SSRIs are our first choices for treatment because of their broad therapeutic effects with various anxiety disorders, followed by SNRIs. While fluvoxamine (Luvox®) is one of several SSRIs with an FDA indication in the treatment of OCD, this SSRI's relatively high rate of potential negative drug interactions tends to limit its clinical use, especially given favorable SSRI alternatives.

Regardless of the patient's weight status, we avoid medications that cause weight gain, such as paroxetine (Paxil®), because of the inability to effectively strategize an ultimate weight outcome. Buspirone (Buspar®) is an additional option in individuals with GAD and may be dually helpful in patients with alcohol abuse (Malec, Malec, & Dongier, 1996). In the treatment of anxiety disorders we have also been successful augmenting antidepressant therapy with gabapentin (Neurontin®) at low doses (100–300 mg per day), which has minimal effects on body weight. We avoid benzodiazepines because of the risks of addiction and withdrawal, cognitive impairment (particularly in low-weight patients), psychomotor impairment, exacerbation of depression, and behavioral disinhibition (Longo & Johnson, 2000).

Management Guidelines and Caveats

Before conferring a diagnosis of comorbid anxiety disorder, it is important to examine the content and context of the patient's symptoms. In other words, some of the symptoms of EDs resemble features of anxiety disorders (see Chapters 2–4 & 8–13). These include: an intense fear of gaining weight or becoming fat, or engaging in behaviors that prevent weight gain, even at significantly low weights (phobic fears); a preoccupation with and overevaluation of one's body weight or shape; counting calories; a relentless drive for thinness (obsessive thoughts); and lack of control over eating, which leads to engaging in recurrent compensatory behaviors (compulsive behaviors designed to reduce anxiety). If the fears, obsessions, or compulsions are specifically associated with the ED (i.e., food, eating, or weight related) and not evident in other areas of functioning, a formal anxiety diagnosis should be avoided.

In addition, there is significant consensus that both depressive and anxiety symptoms are the sequelae of malnutrition in EDs (American Psychiatric Association, 2006; Keys et al., 1950). This observation reinforces the widely held principle that treatment of anxiety disorders in low-weight patients should begin with nutritional rehabilitation. In many cases, successful treatment of the ED will significantly reduce anxiety symptoms. However, clinicians should be aware that, if independent anxiety disorders are not addressed directly, they will continue to interfere with daily functioning even after the resolution of the ED.

As mentioned previously, anxiety disorders in adolescence increase ED vulnerability, particularly premorbid OCD in relationship to AN (Buckner, Silgado, & Lewinsohn, 2010). Likewise, the adolescent with BN has an increased risk of developing either social anxiety disorder and/or panic disorder in adulthood (Buckner et al., 2010). Unfortunately, both social anxiety and panic disorder tend to be chronic illnesses associated with greater psychiatric and medical comorbidity, higher rates of suicidal ideation and attempts, and SUD (Buckner et al., 2008; Weiller, Bisserbe, Boyer, Lepine, & Lecrubier, 1996). These findings further highlight the need to methodically screen for and treat comorbid anxiety disorders.

Substance Use Disorders

Prevalence

One of the most common comorbid conditions found in EDs is substance use disorder (SUD). Approximately 50% of individuals with EDs are abusing alcohol and/or other drugs (illicit, prescription, or over-the-counter) or Internet supplements (see Chapter 15). These rates are five times greater than those observed in the general population (The National Center on Addiction and Substance Abuse [CASA], 2003). The substances most frequently used and abused by individuals with EDs are alcohol and cannabis (Blinder, Cumella, & Sanathara, 2006; Corcos et al., 2001; Wiederman & Pryor, 1996). However, a significant minority of individuals with EDs also use and abuse diet pills (Reba-Harrelson et al., 2008; Steffen et al., 2010); diuretics, laxatives, and emetics (Steffen, Mitchell, & Roerig, 2007); prescription psychostimulants (Biederman et al., 2007); insulin (Powers et al., 2012); and performance-enhancing substances (Strother, Lemberg, Stanford, & Turberville, 2012).

Alcohol use disorders (AUDs) are most prevalent in individuals with BN (approximately 34%), but also occur in 25% of individuals with AN and 21% of individuals with BED (Hudson et al., 2007). Rates of illicit drug abuse/dependence are approximately 25% in BN, 19% in BED, and 18% in AN (Hudson et al., 2007). Rates of abuse/dependence vary

greatly across AN subtypes, with the AN-BP group reporting a significantly higher rate of AUD (approximately 35%) and drug abuse (32%) than the AN-R group (14% AUD and 6% drug abuse) (Root et al., 2010).

Individuals with both ED and SUD frequently have additional comorbid psychiatric conditions, including MDD, PTSD (Dansky, Brewerton, & Kilpatrick, 2000; see also Chapter 34), and BPD (Bulik, Sullivan, Carter, & Joyce, 1997; see also Chapter 57). Research suggests that individuals with AN-BP or BN and SUD have greater personality-related psychopathology (e.g., BPD) and high levels of novelty seeking and impulsivity (Bulik et al., 1997).

Treatment

Psychotherapy Unfortunately, there are no reported randomized controlled trials (RCTs) or EBT for this comorbid group. Ironically, although researchers suggest that comorbid SUD is *not* a contraindication for ED treatment (Franko et al., 2008), RCTs in the ED field have frequently excluded participants with SUD (Gadalla & Piran, 2007)

CBT is one of the few EBTs to be found effective in the treatment of ED and SUD independently. CBT-Enhanced (CBT-E; Fairburn, 2008; see also Chapter 56) was designed specifically for more complex and treatment-resistant ED patients. This approach incorporates additional treatment components for perfectionism, low self-esteem, mood intolerance, and interpersonal difficulties, and could potentially be effective for ED patients with SUD. In addition, Sysko and Hildebrandt (2009) have outlined a CBT model for BN and co-occuring SUD that addresses the features that are common to both disorders, including motivational issues, interpersonal relationship problems, high levels of reward sensitivity, and impulsivity.

Another promising option in the treatment of ED and SUD is Dialectical Behavior Therapy (DBT; Linehan et al., 1999; see also Chapter 57), which is also an effective intervention for BN and BED in patients with or *without* BPD (Ben-Porath, Wisniewski, & Warren, 2009; Safer, Telch, & Agras, 2001; Telch, Agras, & Linehan, 2001). DBT, which focuses on affect regulation, distress tolerance, mindfulness, and interpersonal relationship strategies, addresses many of the core coping skills deficits found in both ED and SUD patients and is an EBT for BPD.

Motivational enhancement therapy has been used successfully in both the ED field (Geller, Brown, & Srikameswaran, 2011) and the SUD field (Lundahl, Kunz, Brownell, Tollefson, & Burke, 2010). Motivational interviewing (see Chapter 63) is designed to enhance the patient's recognition of the problem, willingness to engage in treatment, commitment to behavioral change, and resolution of ambivalence about recovery. Additionally, some patients may benefit greatly from regular attendance at self-help groups including Alcoholic Anonymous (AA), Narcotics Anonymous (NA), or Cocaine Anonymous (CA).

Pharmacotherapy While a number of substances may result in addiction and withdrawal, there are only three abused substances for which there is EBT in the form of medication: tobacco, alcohol, and opioids (National Institute on Drug Abuse [NIDA], 2009; Pettinati et al., 2010). Medication options for treatment of alcohol and opioid addiction are outlined in Table 54.2. For the treatment of alcohol addiction, there does not appear to be an additive effect between behavioral treatments and pharmacotherapy, although behavioral treatments alone have been effective (NIDA, 2009). For the treatment of opioid addiction, pharmacotherapy treatment combined with behavioral treatment is recommended (NIDA, 2009).

Table 54.2 Evidence-based pharmacotherapy options for alcohol and opioid addiction.

Alcohol addiction
- Naltrexone (marketed, e.g., under the brand name Revia®; blocks opioid receptors involved in the rewarding effects of alcohol as well as the craving for alcohol)
- Acamprosate (marketed under the brand name, Campral®; acts on GABA and the glutamate neurotransmitter system to reduce symptoms of protracted withdrawal)
- Topiramate (marketed, e.g., under the brand name, Topamax®; believed to act on GABA and the glutamate neurotransmitter system)
- Sertraline and naltrexone combined (marketed, e.g., under the brand names, Zoloft® and Revia®; in depressed alcohol-dependent patients, promotes abstinence, and reduces depression and relapse)

Opioid addiction
- Methadone (a form of replacement therapy)
- Buprenorphine (marketed under the brand name, Buprenex®, or, in combination with naloxone, marketed under the brand name, Suboxone®; a form of replacement therapy that reduces or eliminates withdrawal symptoms, but does not produce the euphoria or sedation associated with recreational opioids; has a low risk of overdose)

Note. Sources are National Institute on Drug Abuse (2009) and Pettinati et al. (2010).

Management Guidelines and Caveats

Although previous research has suggested lower rates of AUD and SUD in individuals with AN, clinicians should be aware of several issues associated with screening for SUD in ED patients. First, ED diagnoses are fluid and exist on a continuum (see Chapter 55). For example, up to 50% of AN patients will develop bulimic symptoms during the course of their illness (Fairburn, 1995; see also Chapter 8). Therefore, assuming that an individual who presents with AN-R *does not* or *will not* abuse substances is erroneous. Second, substance abuse can develop before, during, or after the onset of an ED, or even after recovery from an ED. Bulik et al. (1997) found that 28% of participants had AUD prior to the onset of their ED, 38% developed AUD concurrently with their ED symptoms, and 34% developed AUD after the onset of their ED. Additionally, a 10-year prospective follow-up study found that 50% of AN-BP patients and 12% of AN-R patients developed a new comorbid SUD after discharge from inpatient treatment (Strober, Freeman, Bower, & Rigali, 1996). Therefore, we recommend that clinicians screen *all* patients at intake for SUD and continue to monitor patients throughout treatment (Dennis & Helfman, 2010).

Another important consideration in the treatment of EDs and SUD is the role of restriction and starvation. Some individuals with EDs are drawn to substance misuse in order to curb appetite (e.g., misuse of diet pills, cocaine, amphetamines, excessive caffeine, tobacco) or to eliminate excess calories or fluid from their bodies (e.g., misuse of laxatives, diuretics, emetics). Others avoid using drugs that might increase appetite (e.g., cannabis, antidepressants, mood stabilizers), are perceived as "empty calories" (alcohol), or promote loss of control (e.g., alcohol, hallucinogens). However, researchers have found that food deprivation itself leads to increased self-administration of virtually any psychoactive drug, including cocaine, nicotine, amphetamines, alcohol, barbiturates, phencyclidine, and opioids (Specker, Lac, & Carroll, 1994). This suggests that nutritional rehabilitation may simultaneously reduce drug self-adminstration.

Currently, there are no EBTs or established treatment protocols for patients who present with both EDs and SUDs/addictions. Lack of available integrated treatment programs, at all levels of care, has left the dually diagnosed patient vacillating between these two disorders (Dennis, Pryor, & Brewerton, 2014). Patients receiving services in nonintegrated programs have poorer treatment outcomes (Drake et al., 2001). There is evidence in the substance abuse field that when comorbid diagnoses are treated concurrently and integrated on-site, treatment retention and outcome improve (Weisner, Mertens, Tam, & Moore, 2001).

Obsessive-Compulsive Personality Disorder

Prevalence

OCPD is a personality dysfunction characterized by a pervasive preoccupation with orderliness, perfectionism, and mental and interpersonal control. Unlike OCD, which is highlighted by intrusive, threatening, and illogical thoughts and by repetitive behaviors that are believed to magically counter these disturbing thoughts, OCPD is more egosyntonic than OCD and tends to result in less overall dysfunction. It is the most common personality disorder in AN-R (approximately 22%) and BED (15.9%), with somewhat lower prevalence rates in AN-BP (12.%) and BN (11%) (Sansone et al., 2006).

Treatment

Psychotherapy Research on EBT for OCPD is scant. According to the findings of the few available studies, both CBT and psychodynamic psychotherapies (see Chapter 20) are generally comparable in efficacy (Dixon-Gordon, Turner, & Chapman, 2011; McMain & Pos, 2007; Verheul & Herbrink, 2007). Because CBT approaches are strongly advocated in the treatment of EDs, this approach readily adapts itself to the adjunctive treatment of OCPD.

Pharmacotherapy SSRIs appear to exert a nonspecific therapeutic effect on obsessive thinking, rumination, and worry (Andrews, Parker, & Barrett, 1998). While these antidepressants are not indicated by the FDA for OCPD, we typically prescribe them to curb underlying anxiety as well as the symptoms related to "thought overdrive" (agitated, driven, and ruminative thinking). On occasion, we have augmented SSRIs with low-dose (e.g., 20 mg per day) buspirone (Buspar®) with effective clinical results.

Management Guidelines and Caveats

From a clinical and diagnostic perspective, OCPD may be difficult to detect because the symptoms may initially appear in the areas of food, weight and shape concerns, and weight management and therefore may be entirely attributable to the ED. Likewise, starvation may intensify obsessive thoughts and behaviors, resulting in the overdiagnosis of OCPD. In either of these circumstances, we suggest scrutinizing other areas of life functioning, such as academics, sports activities, and environmental orderliness to fully evaluate for this personality disorder. Additionally, it is not uncommon to see both OCD and OCPD in the same ED patient. Because the symptoms of OCPD tend to be egosyntonic and the essence of this personality is insistence on control via maintenance of sameness (and vice versa), psychotherapy treatment of this disorder can be challenging.

Borderline Personality Disorder

Prevalence

BPD is the most frequent comorbid personality disorder in AN-BP and BN (i.e., impulsive eating pathologies), with rates of approximately 25–30% (see Chapter 15). BPD is slightly less frequent in AN-R (11%) and BED (12%) (Sansone et al., 2006).

Treatment

Psychotherapy "The primary treatment for BPD is psychotherapy" (American Psychiatric Association, 2001, p. 4). The specific goals for treating BPD in ED patients include reducing self-harm behavior, improving interpersonal relationships, alleviating affective instability, and promoting global self-regulation. Various types of eclectic psychotherapy treatments have been advocated, with many incorporating contracting, psychoeducation, transference work, psychodynamic psychotherapy, and cognitive-behavioral strategies (Dennis & Sansone, 1989, 1991, 1997; Sansone & Sansone, 2006).

In addition to eclectic approaches, there are two manualized approaches that are specifically designed for EDs and BPD. Standard DBT (see Chapter 57) has been modified to include specific modules that address eating pathology (Safer, Telch, & Chen, 2009; Wisniewski & Kelly, 2003). Likewise, Integrated Cognitive-Affective Therapy for BN (ICAT; Wonderlich et al., 2010) is designed to focus not only on ED symptoms and behavioral change, but also on personality and temperament. ICAT specifically targets self-discrepancy (e.g., discrepancy between perceived self and ideal self), interpersonal insecurity, low self-esteem, negative affectivity, and ineffective interpersonal relationship styles. This approach appears to be well suited for the ED patient with comorbid BPD.

Pharmacotherapy According to the American Psychiatric Association (2001, p. 4), the psychotherapy of BPD should be "complemented by symptom-targeted pharmacotherapy" (e.g., anxiolytics for anxiety, antidepressants for mood disorders, antipsychotics for disorganization). However, this general approach may harbor a number of potential limitations (Sansone & Sansone, 2007), and we therefore advocate an individualized approach.

In addition to an individualized approach, we suggest several modifications in the pharmacotherapy of ED patients with BPD:

1 Avoid medications associated with significant weight gain, as BPD may be associated with a higher body weight in general.
2 Avoid medications that pose a greater risk of death by overdose (e.g., tricyclic antidepressants, lithium, or the SSRI citalopram), as BPD is associated with self-harm behavior.
3 Avoid medications with significant addiction potential (e.g., benzodiazepines, benzodiazepine-receptor-site hypnotics), because BPD is associated with self-regulation difficulties.

Generally, SSRIs are an excellent initial choice for treatment, as their broad clinical effects frequently address the polysymptomatic presentations of patients with BPD.

Management Guidelines and Caveats

Due to the high rates of BPD among individuals with EDs, every patient should be screened for this disorder. Psychotherapy with this population requires a strong therapeutic relationship, management of self-destructive behaviors, the endless realignment of boundaries, limit

setting, and a continuous focus on global self-regulation (Dennis & Sansone, 1991). Individuals with BPD tend to elicit strong emotions and reactions from therapists that can potentially contaminate the therapeutic relationship. Therefore, continual self-monitoring is essential in working with these types of patients. Given the frequency and complexity of this comorbidity, clinicians should be highly trained and skilled in the treatment of severe personality psychopathology; novice clinicians are strongly advised to seek regular supervision with senior clinicians. Because patients with BPD are known for suicidal ideation/attempts, it is important to address and structure this concern early in psychotherapy (Sansone & Sansone, 2006). Finally, because there are few available outcome data to guide clinicians in the pharmacotherapy of BPD, always approach medication trials in a conservative and judicious manner, with a clear understanding that psychotropic medications have modest overall benefits with regard to symptoms in BPD.

Conclusions and Future Directions

Our intention in this chapter was to address the most common psychiatric comorbidities encountered in EDs. We conclude with some of the universal themes that have emerged. In addition to general medical screening, due to the high rates of psychiatric comorbidity in EDs, all patients should be thoroughly screened at intake for the presence of other psychiatric conditions. Effective assessment and treatment planning require attention to all psychiatric conditions, not just the ED. Failure to identify co-occurring conditions can lead to both poor retention and treatment outcome (Grilo, Sinha, & O'Malley, 2002). Thorough assessment will also determine the appropriate level of care, team composition (see Chapter 50), treatment approach (e.g., CBT, DBT, IPT, FBT; see Chapters 52, 56, 57, 60, & 62), and adjunctive treatments (e.g., self-help, and pharmacotherapy; see Chapter 59). Monitoring comorbid conditions should continue throughout treatment, as new conditions can arise during the recovery process and symptom substitution is not uncommon. If anxiety, depression, or substance use is ED-related, often symptom reduction will occur with the normalization of eating patterns and the elimination of compensatory behaviors.

Whether these comorbid conditions affect overall treatment outcome remains empirically murky. Unfortunately, more studies are needed that examine the effects of comorbid psychopathology on ED outcome, as currently there have been relatively few. Davies, Bekker, and Roosen (2011) and Keel and Brown (2010) indicate that comorbid psychopathology generally predicts a poorer outcome among ED patients (see Chapter 55). From a different perspective, there is also a need for more research to investigate Bruce and Steiger's (2006) contention that personality pathology may have more "direct relevance to the course of generalized psychopathology symptoms than to eating-specific pathology" (p. 255), which suggests that ED outcomes may be comparable but that general psychopathology outcomes may be worse. Overall, our reading of the literature and our clinical experiences indicate that these comorbid disorders tend to hamper ED outcomes. However, and keeping in mind the need for further research, we also believe that active assessment and corresponding treatment will result in better overall outcomes with eating pathology, as well as comorbid psychiatric condition(s). Like all treatments, it is important to tailor interventions to the individual needs of the patient.

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The "Natural" Course of Eating Disorders

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This chapter reviews the natural course of eating disorders (EDs; see Chapters 2–4 & 8–11), including anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED), and two *DSM-5* (*Diagnostic and Statistical Manual of Mental Disorders*, 5th edition; American Psychiatric Association, 2013) other specified feeding or eating disorders (OSFED) diagnoses: purging disorder (PD) and night eating syndrome (NES). By "natural" course we refer primarily to the course of EDs in community-based samples. Community samples provide the most representative estimates of the course of EDs in the general population, considering that the majority of individuals who meet criteria for an ED do not seek treatment (Hart, Granillo, Jorm, & Paxton, 2011). Of note, community-based samples are not equivalent to untreated samples, as individuals within the community may also be seeking or receiving treatment. In addition, we have included information from studies with treatment-seeking samples followed over longer durations, as long-term outcomes are likely to reflect the natural course of illness.

When possible, we comment on the effect of age, ethnicity, and gender on ED course and outcome; however, data are often insufficient to draw definitive conclusions and examine moderating effects. There is sometimes limited or conflicting information regarding the influence of age of onset on ED course. A potential explanation for this gap could be the difficulty in accurately pinpointing age of onset in community samples. Regarding ethnicity, many studies fail to report demographic information or are limited to predominantly Caucasian samples. Additionally, treatment-seeking studies are often associated with reduced racial/ethnic diversity, as studies have demonstrated that ethnic minorities are less likely to receive referral information for eating problems and may have limited access to, or be less likely to seek, treatment (Becker, Franko, Speck, & Herzog, 2003; see also Chapter 51). Regarding gender, many studies exclude male participants or have such a limited number of males that gender differences cannot be assessed. This may reflect a lower base rate of EDs in males in community-based studies (see Chapter 5), or potential limitations in understanding how symptoms may present differently in males (see Chapter 37). Further, treatment-seeking samples frequently are limited to female-only samples, which may, in part, be due to EDs being seen as a "female problem," leading males to feel less comfortable seeking help for their eating problems.

The Wiley Handbook of Eating Disorders, First Edition. Edited by Linda Smolak and Michael P. Levine.

Understanding the course and outcome of EDs is essential for several reasons. First, understanding ED course provides important clinical information, as patients are often interested in hearing their prognosis when presenting for treatment. Second, clinicians benefit from information regarding ED course and outcome for the purposes of treatment planning. This is particularly useful for evaluating whether an intervention improves outcome beyond what might be expected from the simple passage of time. Third, information regarding the distinctiveness of the course and outcome of ED diagnostic categories helps evaluate their predictive validity.

The following sections provide information on remission at short-term (up to 1 year), intermediate (1 to 5 years), and long-term (over 5 years) follow-up, along with information on relapse, diagnostic stability, crossover, and mortality for EDs. While data regarding ED course come from studies that evaluated individuals based on earlier diagnostic criteria (*DSM-IV* and *DSM-IV-TR* for some long-term follow-up studies, *DSM-III-R* and *DSM-III* for others; American Psychiatric Association, 1980, 1987, 1994, 2000), evidence of predictive validity was central in developing *DSM-5* (2013) criteria. Thus, the research presented in this chapter is relevant for understanding *DSM-5* disorders, particularly the newly identified OSFEDs (see Chapters 4 & 11).

Anorexia Nervosa

Remission and Relapse

Although a small subset of individuals with AN achieve remission (see Chapter 64) early on in the course of the disorder, the majority of patients continue to struggle with symptoms long-term (*DSM-5*; American Psychiatric Association, 2013). Highlighting the often chronic nature of the disorder, remission rates tend to be low across studies of short-term follow-up. For example, in a mixed clinical and community sample, Milos, Spindler, Schnyder, and Fairburn (2005) found a 9.1% remission rate at 12-month follow-up. The remission rate across intermediate lengths of follow-up in clinical samples is approximately 45%, suggesting that a substantial majority of individuals with AN remain ill, even after treatment (weighted average remission = 44%; Agras, Crow, Mitchell, Halmi, & Bryson, 2009; Clausen, 2008; Fichter & Quadflieg, 2007). At long-term follow-up, approximately 53% of individuals from community-based samples achieve remission (Wentz, Gillberg, Anckarsäter, Gillberg, & Råstam, 2009), while estimates among treatment-seeking samples are higher, with approximately two-thirds of individuals achieving remission (weighted average = 69%; Eisler, Simic, Russell, & Dare, 2007; Fichter, Quadflieg, & Hedlund, 2006; Halvorsen, Andersen, & Heyerdahl, 2004; Nilsson & Hagglof, 2005). Overall, approximately 14–20% of patients remain chronically ill (Steinhausen, 2002; see also Chapter 12). Consistent with an association between length of follow-up and remission, one treatment-seeking sample reported that between 8- and 16-year follow-ups, remission increased from 68% to 84% (Nilsson & Hagglof, 2005). Factors associated with poorer outcome for AN include later age of onset, purging behaviors, obsessive-compulsive personality disorder symptoms (see Chapter 54), and longer period between illness onset and first treatment (Steinhausen, 2002).

Regarding the impact of demographic factors on outcome in AN, age of onset appears to moderate course, with adolescent-only samples having higher remission estimates (57.1%) than mixed samples of adolescents and adults (44.2%) (Steinhausen, 2002). Regarding gender, one study examining male and female inpatients found that, at 1-year postdischarge,

males had lower levels of weight, shape, and eating concerns than females, implying that perhaps the course of AN may be less severe for males than for females (Strober et al., 2006). However, there were no differences in body mass index (BMI) at follow-up or in the number of male versus female patients being rated as much improved (Strober et al., 2006). Little is known about how ethnicity may influence course and outcome in AN, so future studies should consider how this may moderate outcome.

Relapse occurs in a substantial minority of those who have remitted from AN, contributing to the chronic nature of illness. Approximately one third of individuals relapse at some point (Herzog et al., 1999). Predictors of relapse include misperception of body weight or shape and receiving more individual psychotherapy (Keel, Dorer, Franko, Jackson, & Herzog, 2005).

Stability and Crossover

While AN remains one of the most stable ED diagnoses, diagnostic crossover occurs in a considerable proportion of individuals. Fluctuation across subtypes (restricting vs. binge-purge) is common, with up to 49% of participants changing subtypes at some point over a 7-year period (Eddy et al., 2008). The most common pattern of crossover within AN is to a diagnosis of what was known prior to *DSM-5* (2013) as eating disorder not otherwise specified (ED-NOS; see Chapters 4 & 11), occurring in 3–31.5% of individuals (weighted average = 13.6%; Ben-Tovim et al., 2001; Castellini et al., 2011; Fichter & Quadflieg, 2007; Milos et al., 2005; Nilsson & Hagglof, 2005); however, it is notable that such individuals may be viewed as either partially recovered or as having ED-NOS. There is also considerable diagnostic migration to BN, with studies finding between 2 and 34% of individuals changing diagnosis (weighted average = 8.6%; Castellini et al., 2011; Fichter & Quadflieg, 2007; Fichter, Quadflieg, & Hedlund, 2006; Milos et al., 2005; Nilsson & Hagglof, 2005). Notably, crossover from AN to BN is more common for those initially diagnosed with AN binge-purge subtype (Eddy et al., 2008) and is more common across longer durations of follow-up (Agras et al., 2009; Fichter & Quadflieg, 2007). Additional predictors of crossover from AN to BN include unipolar depression, substance abuse, and the absence of a diagnosis of obsessive-compulsive disorder (OCD; Castellini et al., 2011).

AN is 4 to 30 times more likely to remain stable than to crossover to a diagnosis of BN (Agras et al., 2009; Ben-Tovim et al., 2001; Fichter & Quadflieg, 2007; Milos et al., 2005). Of the studies reviewed, no cases of crossover have been observed from AN to BED (Agras et al., 2009; Castellini et al., 2011; Fichter & Quadflieg, 2007). Thus, AN does exhibit some diagnostic crossover to ED-NOS or BN, but not to BED. However, the stability of AN is more common than crossover, highlighting the chronicity of the disorder (see Chapter 12).

Mortality

AN is associated with elevated risk of premature death, with fatal outcomes observed in approximately 1 out of every 20 individuals (Steinhausen, 2002; see also Chapter 14). Across studies, standardized mortality ratios (SMRs) (representing the ratio of deaths observed to those expected in the general population) have ranged from approximately 4.4 to 12.0, reflecting up to a 12-fold increase in the risk of premature death compared to a demographically matched population (Franko et al., 2013; Keel et al., 2003; Löwe et al., 2001; Patton, 1988). Importantly, risk of premature death in AN appears to be highest within the first 10 years of follow-up, and decreases thereafter (Franko et al., 2013). Prospective predictors of

premature death in AN include poor social adjustment, lower BMI, and alcohol abuse (Franko et al., 2013; Keel et al., 2003). In addition to increased risk of death from all causes, AN has been associated with elevated risk of death by suicide, with SMR estimates ranging from 31 to 56.9 (Keel et al., 2003; Preti, Rocchi, Sisti, Camboni, & Miotto, 2011). Examination of methods used by AN patients who completed suicide indicated the use of highly lethal methods that would be fatal regardless of the attempter's physical health (Holm-Denoma et al., 2008). Among those with AN, factors associated with suicide attempts include purging behaviors, depression, substance abuse, and history of physical or sexual abuse (Franko & Keel, 2006).

Summary

In sum, AN often exhibits a chronic course, with fewer than 10% of individuals achieving remission over the short term. These estimates increase to approximately 50% and 67% of individuals over intermediate and long-term follow-up, respectively; however, up to 20% of individuals remain chronically ill over their lifetime (see Chapter 12). Age of onset appears to moderate outcome, with adolescent-only samples having higher remission estimates than samples that include adult onset cases. Males may have a better prognosis than females, at least over short-term follow-up. Relapse is also relatively common, occurring in a substantial minority of individuals. AN is also more likely to stay stable than it is to crossover to another ED. Highlighting the serious nature of the disorder, AN is one of the most lethal psychiatric diagnoses, and is associated with an increased risk of premature death and suicide compared to the general population.

Bulimia Nervosa

Remission and Relapse

BN is associated with a variable course that may be chronic or more intermittent (*DSM-5,* 2013*)*. BN frequently presents episodically, with periods of recurrent symptoms alternating with periods of remission (*DSM-5*, 2013). In studies examining the short-term outcome of BN in clinical samples, approximately 30% of individuals achieve remission (weighted average = 27.5%; Bailer et al., 2004; Schmidt et al., 2008). In a study using a mixed clinical and community sample, 24.1% of individuals were found to achieve remission (Milos et al., 2005).

As with AN, remission rates tend to increase over longer duration of follow-up for BN, with over half (weighted average = 54.5%) of individuals initially diagnosed with BN achieving remission in studies assessing outcome in clinical samples at intermediate length of follow-up (Bogh, Rokkedal, & Valbak, 2005; Clausen, 2008; Fichter & Quadflieg, 2007). Agras et al. (2009) found a similar remission rate of 47% at 4-year follow-up in a mixed community and clinical sample. At long-term follow-up in community samples, approximately 65% achieve remission (weighted average = 65.1%; Grilo et al., 2007; Keel, Gravener, Joiner, & Haedt, 2010; Keski-Rahkonen et al., 2009), while estimates are slightly higher for clinical samples, at around 70% (Fichter & Quadflieg, 2004). Across clinical and community-based samples at long-term follow-up, approximately 20% of individuals with BN experience substantial improvement, and 10% have a chronic course (Keel & Brown, 2010; Steinhausen & Weber, 2009). Factors associated with poorer outcome include comorbid personality disorder pathology, substance use disorders, self-injurious behavior, psychosocial stress, and familial history of obesity or disturbed family relationships (Keel, Mitchell, Miller, Davis, & Crow, 1999; Steinhausen & Weber, 2009).

Information regarding how demographics influence the course and outcome of BN is limited. One study suggests that duration of illness may be shorter for African American and Caribbean Black individuals than other ethnic groups (Taylor, Caldwell, Baser, Faison, & Jackson, 2007). Highlighting the restricted number of studies on males with BN, Steinhausen and Weber's (2009) extensive review of literature on BN course found that males comprised only 1.9% of the entire population studied. Concerning age, the same review highlighted conflicting findings regarding the influence of age on BN outcome, with three studies finding adolescent/young age at onset as a positive prognostic factor, but the majority of studies finding no effects (Steinhausen & Weber, 2009).

Regarding relapse in a majority treatment-seeking sample, approximately one third of remitted individuals experienced a subsequent episode that met full criteria for BN (Herzog et al., 1999). However, estimates of relapse for BN largely depend on how relapse is defined (see Chapter 64), with more strict definitions (i.e., return to full threshold BN) being associated with lower relapse rates (21%) and less restrictive definitions (i.e., some symptoms for 1 month) being associated with higher relapse rates (55%; Olmsted, Kaplan, & Rockert, 2005). Predictors of relapse in BN include poor psychosocial functioning and overconcern with weight or shape (Keel, Dorer, et al., 2005).

Stability and Crossover

Similar to AN, the most common pattern of crossover for BN is migration to ED-NOS, or what would now be considered OSFED subthreshold BN and PD, with crossover rates ranging from 1.2 to 26% (weighted average = 13.4%; Castellini et al., 2011; Fichter & Quadflieg, 2007; Milos et al., 2005). Notably, at some point, up to 83% may crossover to ED-NOS defined by partial remission from BN (Eddy et al., 2008). Migration from BN to BED appears to be relatively less common, ranging from 0.7 to 19% (weighted average = 6.8%; Bogh et al., 2005; Fichter & Quadflieg, 2004, 2007; Keel et al., 2000; Stice, Marti, Shaw, & Jaconis, 2009). Indeed, BN is 49 times more likely to remain BN than to crossover to BED (Fichter & Quadflieg, 2007). Crossover from BN to AN occurs in 1 to 14% of individuals (weighted average = 6.4%; Ben-Tovim et al., 2001; Castellini et al., 2011; Eddy et al., 2008; Keel, Dorer, et al., 2005; Keel, Mitchell, Miller, Davis, & Crow, 2000; Milos et al., 2005). The majority of this migration occurs from BN to the binge-purge subtype of AN, and crossover from BN to the restricting subtype is more rare (Eddy et al., 2008). Importantly, BN is 7 to 10 times more likely to remain stable than to crossover to AN (Agras et al., 2009; Ben-Tovim et al., 2001; Fichter & Quadflieg, 2007). Thus, while BN does exhibit some crossover to ED-NOS (OSFED), diagnostic stability is more common than crossover to either BED or AN.

Mortality

While research has been mixed regarding whether or not mortality is elevated in BN (Crow et al., 2009; Franko et al., 2013; Keel et al., 2003; Nielsen, 2001), a recent meta-analysis found a significantly elevated SMR of 1.93 for individuals with BN, supporting an increased risk of premature death (Arcelus, Mitchell, Wales, & Nielsen, 2011). Risk of premature death in BN is significantly lower than that in AN, as exhibited by the nonoverlapping 95% confidence intervals from the Arcelus et al. (2011) meta-analysis: AN SMR (95% CI) = 5.86 (4.17–8.26); BN SMR (95% CI) = 1.93 (1.44–2.59). BN has also been associated with elevated suicide attempts and increased risk of death by suicide, with results from a recent meta-analysis

providing a SMR of 7.5, with a 95% confidence interval of 1.6–11.6 (Preti et al., 2011). Predictors of suicide attempts in BN include history of substance use disorder, laxative abuse, Cluster B (i.e., dramatic, emotional, and erratic) personality disorder symptoms, childhood sexual abuse, and low self-directedness (Favaro et al., 2008; Franko et al., 2004).

Summary

In sum, research on short-term outcome in BN indicates a remission rate around 30%, while intermediate and long-term remission estimates fall at approximately 50% and 70%, respectively. Highlighting the often episodic nature of the disorder, approximately one third of individuals with BN relapse at some point. Like AN, BN is also more likely to remain stable than it is to crossover to another ED. Recent research supports an elevated risk of premature death and suicide in BN compared to the general population; however, these estimates are significantly lower than those found in AN.

Binge Eating Disorder

Remission and Relapse

Overall, BED has a more favorable course than either AN or BN. Regarding short-term outcomes in community samples, Cachelin et al. (1999) found that, after 6 months, 48% of the 21 individuals were in partial remission. Among treatment studies, remission rates have ranged from 24.7 to 80% (weighted average = 50.9%; Dingemans, Spinhoven, & van Furth, 2007; Grilo, White, Gueorguieva, Wilson, & Masheb, 2012; Schlup, Munsch, Meyer, Margraf, & Wilhelm, 2009). Research on BED course over intermediate length of follow-up demonstrates that, in a clinical population, approximately 65% of individuals achieve remission (Fitcher & Quadflieg, 2007). In a mixed community and clinical sample, the remission rate was slightly higher at around 82% (Agras et al., 2009). Fairburn and colleagues completed a 5-year longitudinal study, tracking illness in 34 community participants every 15 months. The remission rates among the BED sample were 59% at 15-month follow-up, 36% at 30-month follow-up, 55% at 45-month follow-up, and 50% at 5-year follow-up (Fairburn, Cooper, Doll, Norman, & O'Connor, 2000). Supporting the more favorable course of BED, individuals with BED appear to achieve remission in a shorter time than those with either AN or BN (Agras et al., 2009). Regarding the long-term outcomes of BED, Fichter and Quadflieg (2007) conducted a 12-year longitudinal study using a clinical sample ($n = 60$) and found that, at the 6-year mark, 6.7% of the sample still had BED, while 78.5% of the sample did not meet criteria for any ED diagnosis. The percentage of individuals with BED remained constant through the 12-year time point (6.7%), while the percentage of individuals in remission decreased slightly to 66.7%.

Regarding the influence of demographic factors on course and outcome, most studies either did not report demographic statistics or had predominantly Caucasian, female samples. As with BN, research suggests that African American and Caribbean Black individuals may experience a shorter course of illness (Taylor et al., 2007). One community-based study examined how age predicted BED outcome and found that at 3-month, but not 6-month, follow-up older age was a significant predictor of increased objective binge episode frequency (Cachelin et al., 1999).

Concerning relapse, the Fichter and Quadflieg (2007) results suggest that because the number of individuals who met BED criteria at 6- and 12-year follow-up was higher than that

observed at 2-year follow-up, relapse does occur, although at a relatively low rate. In support of this conclusion, Fairburn et al. (2000) found relapse rates among their BED sample to be 10% at 30-month follow-up, 4% at 45-month follow-up, and 7% at 5-year follow-up. Predictors of relapse include being male and reporting a higher frequency of subjective binge eating episodes (Castellini et al., 2011).

Stability and Crossover

As with AN and BN, the most common pattern of crossover within BED is to ED-NOS, with studies finding crossover estimates ranging from 3 to 18.3% (weighted average = 12.8%; Fairburn, et al., 2000; Fichter & Quadflieg, 2007). The rate of migration from BED to BN ranges from 3 to 15% (weighted average = 9.3%; Castellini et al., 2011; Fairburn et al., 2000; Fichter & Quadflieg, 2007). Notably, in the context of the high rate of remission for BED, Fichter and Quadflieg (2007) found that a greater proportion of individuals transitioned to a BN diagnosis than sustained a BED diagnosis. There is no evidence to support a diagnostic shift from BED to AN (Castellini et al., 2011; Fairburn et al., 2000; Fichter & Quadflieg, 2007). Thus, BED does exhibit some diagnostic crossover to either ED-NOS or BN, but not to AN. Considering the research findings regarding remission, a BED diagnosis does not exhibit a high degree of stability over time, as individuals with BED are likely to either achieve remission or meet criteria for a different ED diagnosis.

Mortality

Given the dearth of research regarding BED and mortality, only tentative findings can be discussed. Fichter et al. (2008) reported a crude mortality rate of 2.9% for a clinical BED sample (n = 64) assessed at 12-year follow-up. The SMR for BED was 2.29; however, this value did not differ significantly from a value of 1.0, in which the number of observed deaths is the same as the number expected in a demographically matched population (Fichter et al., 2008). Therefore, no conclusions can be drawn as to whether a diagnosis of BED is associated with an increased risk of premature death.

Summary

In sum, current research on the course and outcome of BED suggests reasonably high remission rates, low relapse rates, and a moderate amount of diagnostic crossover. Short-term outcome research indicates a remission rate of around 50% for BED, while intermediate and long-term remission estimates fall at approximately 75%. Relapse appears to be relatively uncommon, even when considering relapse to any ED diagnosis. BED does exhibit crossover to both ED-NOS and BN, but not to AN. It remains unknown as to whether BED increases mortality risk, as more research is needed on the topic.

Other Specified Feeding or Eating Disorders

In *DSM-IV* (1994, 2000) ED-NOS served as a residual, or "catch-all," category that included all individuals with a clinically significant disorder of eating not meeting full criteria for AN or BN (see Chapters 4 & 11). This led to a myriad of symptom combinations subsumed within the

ED-NOS category, making it difficult to obtain meaningful information on course and outcome (Wilfley, Bishop, Wilson, & Agras, 2007). The *DSM-5* (2013) includes a category of OSFED, within which PD and NES have been named and described, with features that distinguish them from AN, BN, BED, and these conditions in partial remission (see Chapter 11). The following section provides preliminary data on the course of these newly identified syndromes.

Remission and Relapse

Regarding remission in the short term, in a prospective community-based 6-month follow-up of 23 women with PD, Keel, Haedt, and Edler (2005) found that 13% achieved partial or full remission. Two community-based studies have examined course and outcome over intermediate length of follow-up, and found that approximately three fourths of individuals with PD were in remission (weighted average = 74.6%; Allen, Byrne, Oddy, & Crosby, 2013; Stice et al., 2009). This was higher than estimates for BN and BED. Remission rates appear to decrease over long-term follow-up, with approximately half of individuals with PD achieving remission (weighted average = 46%; Allen et al., 2013; Koch, Quadflieg, & Fichter, 2013), which was similar to remission estimates for AN, BN, and BED in these samples. The Koch et al. (2013) study utilized an inpatient sample, which may have contributed to lower remission estimates across diagnostic categories. Little is known about relapse in PD; however, one study indicated a lower likelihood of relapse in PD (5%) compared to BN or BED (41% and 33%, respectively; Stice et al., 2009). Importantly, in this longitudinal study, most cases of PD were identified within the final year of observation, minimizing the opportunity to observe relapse following remission. Thus, the majority of the research on remission in PD suggests that a slightly more favorable course than AN, BN, and BED at intermediate lengths of follow-up, with a similar course at long-term follow-up. However, further research is needed to draw more definitive conclusions.

Studies examining the course of NES (see Chapters 4, 11, & 65) have been rife with methodological issues, including restriction of sampling to obese, weight-loss-seeking samples with limited duration of follow-up. Further, many studies have permitted comorbidity between NES and BED, which limits conclusions that may be drawn about the course of NES independent of BED. With these limitations stated, short-term remission estimates from NES appear to be higher than those for AN, BN, or BED. In a retrospective study of changes in NES in bariatric surgery patients, Latner, Wetzler, Goodman, and Glinski (2004) found that while 55% of the 150 patients exhibited NES prior to surgery, only 2% exhibited symptoms 6-months postsurgery, representing a remission rate of approximately 96% (Latner et al., 2004). Studies examining more intermediate outcomes have found that 1 to 3 years after bariatric surgery, approximately half of patients achieved remission (weighted average = 51%; Adami, Meneghelli, & Scopinaro, 1999; Colles, Dixon, & O'Brien, 2007; Rand, Macgregor, & Stunkard, 1997). Based on the limited information available, it appears that NES may have a more favorable course than either AN or BN over the short-term; however, future studies examining the prospective course of NES in community samples are needed to better elucidate the natural course of the disorder.

Stability and Crossover

Initial estimates of diagnostic crossover appear to support the stability of PD. The most common pattern of crossover appears to be from PD to some other form of ED-NOS, which occurs in 10.8 to 21.8% of individuals (weighted average = 18.1%; Allen et al., 2013; Koch et al., 2013). Crossover from PD to BN is relatively less common, occurring in 4 to 18.9% of individuals

(weighted average = 6.4%; Allen et al., 2013; Keel, Haedt, & Edler, 2005; Koch et al., 2013; Stice et al., 2009). Indeed, the study by Keel, Haedt, and Edler (2005) found that those diagnosed with PD were 20 times more likely to retain a diagnosis of PD than to crossover to BN 6 months later, which was comparable to the stability rate for BN. Similarly, diagnostic migration from PD to AN has occurred in 2.7 to 5.4% of individuals (weighted average = 4.5%; Allen et al., 2013; Koch et al., 2013). Migration from PD to BED is relatively rare, occurring in 0–9% of individuals (weighed average = 1.6%; Allen et al., 2013; Koch et al., 2013; Stice et al., 2009).

Research on the stability of NES has also been limited. Further, in many studies, BED is not an exclusion criterion for a diagnosis of NES, making it difficult to draw conclusions about the stability of "pure" NES or determine accurate estimates of crossover between NES and BED. Indeed, estimates of overlap between NES and BED have ranged from 36 to 40% (Colles et al., 2007; Napolitano, Head, Babyak, & Blumenthal, 2001). Thus, the current state of research renders results about the stability and crossover of NES inconclusive. Future studies would benefit from examining larger samples of NES outside of other EDs to understand the course of the syndrome alone.

Mortality

A recent meta-analytic study demonstrated that ED-NOS has an elevated risk of death compared to the general population, and similar rates of mortality and death by suicide compared to individuals with BN, but lower rates than those with AN (Arcelus et al., 2011). In that meta-analysis, Arcelus and colleagues (2011) reported a SMR of 1.92, with a 95% confidence interval of 1.46–2.52, providing evidence for increased risk of premature death in ED-NOS. Not enough research has been conducted to establish whether individuals with PD or NES are at increased risk for premature death. As PD and NES have often been included in mixed ED-NOS samples, results from the larger studies on mortality in ED-NOS may apply. Further, purging is a medically dangerous behavior associated with adverse medical outcomes (Brown & Mehler, 2013; see also Chapters 14 & 52), thereby increasing risk of death. In regard to empirical research on mortality in PD, Koch and colleagues (2013) found that the crude mortality rate in their inpatient follow-up sample was 5%, which was comparable to the rate in the binge-purge subtype of AN (3.7%) and significantly higher than that in BN (1.1%). This provides preliminary evidence for an elevated risk of death in PD. However, the PD patients were significantly older than those with AN or BN, underscoring the importance of evaluating a standardized mortality ratio rather than a crude mortality rate. In regard to NES, no studies of mortality for this syndrome were identified from our review. Future epidemiological research is needed to ascertain whether risk of death is elevated in community samples of both PD and NES.

Summary

In sum, research on the course and outcome of PD and NES is limited. However, preliminary findings indicate that, over the short term, approximately 13% of individuals with PD achieve remission. Over intermediate lengths of follow-up, PD may exhibit a slightly more favorable course than AN, BN, and BED, while at long-term follow-up the course of PD may be more similar to other EDs. NES appears to exhibit a more favorable course compared to other EDs, with high rates of remission over short and intermediate lengths of follow-up. More research on relapse in PD and NES is needed; however, relapse may be less common in PD than in BN or BED. PD appears more likely to remain stable than to crossover to another ED, while stability

in "pure" NES is unclear, given the high overlap with BED in current research. It is currently unclear whether PD or NES create an elevated risk of premature death.

Conclusions and Future Directions

Because the revisions made from *DSM-IV* to *DSM-5* heavily weighted the importance of predictive validity, diagnostic changes should make little difference on the course and outcome of EDs as defined by *DSM-5*. Supporting this, Castellini et al. (2011) examined the long-term course and outcome according to the *DSM-IV* and the proposed *DSM-5* criteria of 793 patients diagnosed with AN, BN, or BED. As expected, at 6 years following the conclusion of their treatment the recovery rates for patients with AN (*DSM-IV*: 52.1%; *DSM-5*: 52.3%), BN (*DSM-IV*: 49.6%; *DSM-5*: 52.2%), and BED (*DSM-IV*: 59.2%; *DSM-5*: 63.8%), were very similar across *DSM-IV* and *DSM-5*. Regarding ED-NOS, Castellini et al. (2011) found similar recovery rates for anorectic-type ED-NOS presentations between *DSM-IV* (56.5%) and *DSM-5* (57.4%), whereas remission rates for the bulimic-type ED-NOS increased from 63.5% in *DSM-IV* to 80% in *DSM-5*. The larger change for bulimic-type ED-NOS likely represents the removal of BED and migration of subthreshold forms of AN or BN to full-threshold diagnoses. The proposed changes to diagnostic criteria also did not substantially affect crossover rates, which remain similar to those for *DSM-IV* diagnoses.

Questions for future research directions in the field include:

1 How will further investigation of diagnostic changes in *DSM-5* impact our current understanding of course and outcome?
2 How do gender, age, and ethnicity influence the course and outcome of EDs?
3 What are the long-term course and outcome associated with BED?
4 What are the short-term, intermediate, and long-term course and outcome associated with PD and NES (outside of other EDs) in community samples?
5 Does the course and outcome of BED and NES differ between overweight and non-overweight samples?
6 Are sufferers of BED, PD, and NES at increased risk of premature death?
7 Do the course and outcome of PD and NES contribute to the predictive validity of those diagnoses by distinguishing between these disorders and AN, BN, and BED?

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Section X
Therapeutic Approaches

56

Cognitive-Behavioral Therapy for the Treatment of Eating Disorders

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Cognitive theory suggests that in all psychological disturbances, it is distorted or dysfunctional thinking that influences an individual's mood and behavior (see Chapters 18 & 44). Simply put, it is the interpretation of an event, rather than the event per se, that activates negative emotions, certain physiological responses, and dysfunctional behaviors, and these, in turn, reinforce negative cognitions in the form of a vicious cycle. Cognitive therapy was originally developed by Aaron Beck (1967, 1976) for depression and, since then its core principles have been adapted for use with a wide range of disorders, including eating disorders (EDs). Early cognitive theories of EDs posited that the core psychopathology of an ED is extreme concern about weight and shape (Fairburn & Garner, 1986). Cognitive and behavioral theories have been integrated into cognitive-behavioral models of bulimia nervosa (BN; Fairburn, 1981; Fairburn, Marcus, & Wilson, 1993), binge eating disorder (BED; Fairburn et al., 1993), and anorexia nervosa (AN; Garner & Bemis, 1982; Garner, Vitousek & Pike, 1997). More recently, Fairburn and colleagues have developed a "transdiagnostic" model, applicable to all EDs (Fairburn, Cooper & Shafran, 2003).

In this chapter, we will describe the development of the various forms of cognitive-behavioral therapy (CBT) for EDs, and the evidence base for their effectiveness. This will be followed by an overview of the original protocol developed for BN, and the adaptations of this protocol for other EDs. In general, CBT for EDs follows the principles laid down by the early proponents of CBT for depression. Treatment is structured and time-limited, with an agenda set at the start of each session. It focuses on the present and future rather than on the past, and on the factors and processes that are maintaining the disorder. Responsibility for change rests with the patient, with the therapist acting as a guide, providing information, encouragement, and support. For this to occur, a positive therapeutic relationship is vital.

The Wiley Handbook of Eating Disorders, First Edition. Edited by Linda Smolak and Michael P. Levine.

Development of Cognitive-Behavioral Therapy for Bulimia Nervosa (CBT-BN)

CBT-BN, first described in 1981 by Christopher Fairburn, is the most extensively studied and successful of all therapies for BN. The expanded version of the original manual (Fairburn et al., 1993), further elaborated by Wilson, Fairburn, and Agras (1997), has become the "gold standard" for BN treatment. It has been endorsed by the National Institute for Clinical Excellence (NICE; 2004) and the American Psychiatric Association (2006) as a first line treatment for BN.

CBT-BN consists of 19 individual sessions over 20 weeks, moving through three stages. Treatment is based on a model that recognizes the role of both cognitive and behavioral processes in the maintenance of BN (see Chapter 18). Low self-esteem and the overvaluation of weight and shape for determining self-worth lead to the adoption of strict dieting and other extreme weight loss efforts, such as excessive exercise. This rigid dietary restraint is punctuated by episodes of binge eating, which are followed by attempts to compensate for the calories ingested during a binge—most often by self-induced vomiting or other purgative behaviors (e.g., laxative or diuretic misuse). However, these attempts at compensation serve only to reinforce binge eating by reducing anxiety about the potential weight gain resulting from the binge. This binge-purge pattern causes distress and self-condemnation, which further lowers self-esteem and reinforces attempts at weight loss, thus creating a vicious cycle. Treatment aims to break this cycle by replacing strict dieting with regular or "normal" eating and by altering dysfunctional thinking about the importance of shape and weight.

CBT-BN has been found to eliminate binge eating and purging in around 50% of individuals who complete treatment, and to reduce the severity of associated psychopathology (Wilson & Fairburn, 2002). Treatment gains are well maintained in the long term (up to 5 years post-treatment; Fairburn et al., 1995).

Manualized CBT-BN has been compared against alternative treatments in a large number of randomized controlled trials (RCTs). In comparisons between CBT-BN and Interpersonal Psychotherapy (IPT; Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000; see also Chapter 62), CBT led to greater improvement by the end of treatment. However, by 12-month follow-up, differences between treatments were no longer significant. CBT-BN has emerged as superior to behavior therapy alone (Fairburn et al., 1991), exposure plus response prevention (Agras, Schneider, Arnow, Raeburn, & Telch 1989; Carter, McIntosh, Joyce, Sullivan, & Bulik, 2003), self-monitoring only (Agras et al., 1989), nutritional counseling (Hsu et al., 2001), and supportive-expressive psychotherapy (Garner et al., 1993). CBT-BN delivered in a group format is as effective as individual CBT-BN (Chen et al., 2003; Wilfley et al., 1993).

Development of Cognitive-Behavioral Therapy for Binge Eating Disorder

BED (see Chapters 4, 10, & 65), recognized as a formal diagnosis for the first time in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*; American Psychiatric Association, 2013), is the most common ED, with prevalence rates of around 3% (Striegel-Moore & Franko, 2008). The cognitive-behavioral model that underpins CBT-BN remains applicable to BED, although there are several adaptations based on the differing nature of the disordered eating in BED. While BN is characterized by strict dietary restraint accompanied by binge eating and purging, BED patients tend to engage in habitual binge

eating but do not demonstrate the same degree of dietary restraint, nor do they engage in compensatory behaviors (Masheb & Grilo, 2000, 2006). As a result, individuals with BED are commonly overweight or obese (Striegel-Moore & Franko, 2008).

CBT for BED, described in a comprehensive manual by Fairburn et al. (1993), is very similar to CBT-BN. It is the most researched psychotherapy for BED, and the treatment with the most empirical support (National Institute for Clinical Excellence, 2004; Wilson, Grilo, & Vitousek, 2007; Wilson, Wilfley, Agras, & Bryson, 2010).

RCTs of CBT for BED have consistently found CBT to be associated with good outcome in terms of eliminating binge eating in around 50% of patients by end of treatment and improving depression, other psychopathology, and overall functioning (Wilson et al., 2007). Outcomes on these variables for CBT are superior to those for IPT (see Chapter 62), dialectical behavior therapy (DBT; see Chapter 57), psychopharmacotherapy (fluoxetine; see Chapter 59), and behavioral weight loss therapy (Devlin et al., 2005; Grilo, Masheb, & Salant, 2005). However, it has also been repeatedly demonstrated that, although CBT is effective at reducing binge eating, it does not result in clinically significant weight loss in obese patients (Vocks et al., 2009). Several RCTs have also shown that the effectiveness of CBT for BED delivered in self-help format, using Fairburn's (1995) book *Overcoming Binge Eating*, is comparable to individual or group CBT (Grilo et al., 2005; Grilo, Masheb & Crosby, 2012; Striegel-Moore et al., 2010).

Development of Cognitive-Behavioral Therapy for Anorexia Nervosa

A cognitive-behavioral model for understanding and treating AN (Garner & Bemis, 1982, 1985) and a manualized treatment have been available for many years (Garner et al., 1997; see also Chapter 18). In spite of this, there has been limited research on CBT for AN. There are several possible reasons for this. First, AN is relatively rare (see Chapter 5). Second, individuals with AN are often reluctant to seek treatment, and, because of the fear of change that characterizes the disorder, treatment of AN is characterized by high dropout rates. Third, the severity of the illness precludes randomization to waiting list or placebo control groups and necessitates lengthy treatment during which the patient may need hospital admission (see Chapter 50).

Watson and Bulik (2013), in their recent review of 30 years of RCTs of treatments for AN, could include only 48 published RCTs, just four of which involved CBT. Channon, De Silva, Hemsley, and Perkins (1989) compared CBT with behavior therapy and with treatment as usual, and found no difference between the three treatments. Ball and Mitchell (2004) reported improvements amongst those who completed CBT, but also found similar results with family therapy (see Chapter 60). Pike, Walsh, Vitousek, Wilson, and Bauer (2003), studying maintenance interventions, found CBT to be significantly superior to nutritional counseling.

An important study of AN by McIntosh et al. (2005) evaluated two plausible treatment approaches, CBT and IPT, against a control treatment entitled "non-specific supportive clinical management" (NSCM; more recently known as "specialist supportive clinical management," or SSCM; see Chapter 12). NSCM was found to be superior, with 56% of those receiving this treatment having a good outcome. This compared favorably with a good outcome in 32% of those receiving CBT and 10% of those receiving IPT. In the long term, however, the effects of NSCM appeared to wane, such that by the 7-year follow-up there were no differences between the three treatments (Carter et al., 2011). A recent study comparing CBT and SSCM in patients with severe and enduring AN found improvement in both groups, although at follow-up the CBT group showed greater improvement in ED psychopathology

while SSCM led to greater improvement in social adjustment (Touyz et al., 2013; see also Chapter 12). Results from this small number of studies suggest that CBT for AN compares well with existing psychotherapies, but has not been shown to be superior to other treatments apart from nutritional counseling.

Development of Transdiagnostic, Enhanced Cognitive-Behavioral Therapy

Enhanced cognitive-behavioral therapy (CBT-E) derives from the transdiagnostic theory of EDs (Fairburn et al., 2003), which developed out of the original cognitive-behavioral theory of BN (Fairburn, 1981). Because a full and lasting response to CBT-BN was found in only around 50% of patients, the developers augmented the treatment and developed an "enhanced" version suitable for all forms of ED (Fairburn, 2008; Fairburn et al., 2003).

The transdiagnostic model identifies a cycle of ED symptoms: weight/shape overvaluation and severe dieting with rigid dietary rules result in the binge eating and compensatory behaviors typical in BN, and/or the psychological and physiological symptoms of being severely underweight commonly seen in AN (Fairburn et al., 2003). The transdiagnostic model also identifies four additional maintaining mechanisms (mood intolerance, clinical perfectionism, core low self-esteem, and interpersonal problems) that in some patients contribute to the maintenance of ED symptoms. The CBT-E protocol has two versions: a "focused" form (CBT-Ef), which focuses exclusively on the ED symptomatology, and a "broad" form (CBT-Eb), which also addresses, where necessary, the additional maintaining mechanisms described above (Fairburn, 2008).

CBT-E has been evaluated empirically in outpatient populations in four RCTs (Fairburn et al., 2009; Poulsen et al., 2014, Wonderlich et al., 2014; Zipfel et al., 2014) and in three community-based effectiveness trials (Byrne, Fursland, Allen, & Watson, 2011; Dalle Grave, Calugi, Doll, & Fairburn, 2013; Fairburn et al., 2012). Fairburn et al.'s (2009) RCT, involving a transdiagnostic sample with a body mass index (BMI) greater than 17.5, found that, by the end of treatment, 53% of the entire sample and 67% of treatment completers showed clinically significant improvements in ED symptoms and 40% reported no binge-purge behaviors. These improvements were well maintained after 1 year. The Poulsen et al. (2014) RCT, comparing CBT-E with psychoanalytic therapy in people with BN, found cessation of binge eating and purging in 42% of the CBT-E group versus 6% of the psychoanalytic group after 5 months, and 44% versus 15% after 2 years. Wonderlich et al.'s (2014) study found no significant differences in the successful treatment of BN between CBT-E and integrative cognitive affective therapy (ICAT), with 22.5% of the CBT-E sample asymptomatic at both end of treatment and at follow-up. With AN patients, Zipfel et al.'s (2014) RCT found no significant differences between CBT-E, focal psychodynamic treatment, and treatment as usual, although CBT-E showed speedier weight gain.

Of the community trials, the Byrne et al. (2011) study involved a transdiagnostic sample, including AN patients (i.e., BMI < 17.5). After treatment, 43% of the entire sample and 67% of completers showed clinically significant improvements in symptoms, with 46% of binge-purge patients free of those behaviors. In the Fairburn et al. (2012) study, involving adults with AN, 68% of the entire sample and 88% of completers showed improvements in symptoms by the end of treatment, which were maintained after 1 year. In the Dalle Grave et al. (2013) study of adolescents with AN, 79% of the entire sample and 97% of completers reported

improvements in symptoms and, again, these improvements were maintained at 60- week follow-up. Taken together, these studies suggest that CBT-E is effective for patients with all forms of EDs. CBT-E compares well with CBT-BN and CBT for BED, and results in better outcomes than CBT for AN.

The Treatment Protocols: Overview of CBT-BN

In this section, we give an overview of the CBT-BN protocol, then describe each stage of the original protocol, and highlight the ways in which it has been adapted for use with BED, AN, and a transdiagnostic sample. CBT-BN is highly structured, ensuring a systematic approach and providing a framework that encourages adherence to the protocol. Structure is maintained not only throughout treatment, but also within the sessions. The agenda includes addressing dysfunctional behaviors and cognitions, reviewing self-monitoring and other homework, focusing on tasks to rehearse and practice in order to achieve behavioral and cognitive change, summarizing the session, and setting homework tasks. The therapist and patient work together in a process of "collaborative empiricism," for which a strong therapeutic alliance, balancing empathy and firmness, is essential (Beck, 1976).

The structured and focused nature of the treatment is explained to patients at the outset, and a commitment to 5 months of treatment is expected. It is recognized that, although most patients with BN wish to overcome their ED, many are ambivalent regarding change. Interventions to address this ambivalence include taking a motivational approach (Miller & Rollnick, 1991; see also Chapter 63) and identifying the discrepancy between the personal costs of maintaining the status quo and the short-and long-term benefits of change.

Stage 1 of Treatment: The Protocols

CBT-BN

The focus of the first eight sessions (held weekly) is to establish a good therapeutic relationship and to facilitate early behavioral change. Treatment expectations are clarified by explaining that the likely outcome is positive but that "overnight success" is unlikely and progress can continue beyond the end of treatment. Setbacks are identified as opportunities for learning. The cognitive-behavioral rationale for treatment is presented, emphasizing the effect of beliefs and assumptions on behavior and emotions, the active role of the patient, and the need for between-session work to modify behavior.

From the first session, patients are instructed to self-monitor their food and fluid intake and the circumstances under which this occurs, both to increase their awareness of their eating behaviors (thereby helping them regain control over eating) and to provide the therapist with vital information. Patients are instructed to record all food consumption shortly after it occurs, with associated thoughts and feelings, and any binge eating or purging. At the start of treatment, adequate time needs to be given to reviewing these records, to gain a complete picture of the patient's eating habits and to reinforce compliance. Patients are encouraged to weigh themselves (only) once a week, to reduce their preoccupation with weight. In accordance with the cognitive-behavioral model, education is provided concerning the negative effects of rigid dieting, and the physical effects (and relative ineffectiveness) of purging.

By the third session, patients are asked to eat regularly (three meals and two snacks daily) in order to reduce binge eating. Anxiety about normal eating (e.g., that it will lead to binge eating) is addressed by referring to the cognitive-behavioral model of the maintenance of BN. The early focus is on less difficult behavioral change (e.g., developing a regular pattern of eating "safe" foods) in order to facilitate early progress and counter hopelessness.

Education is offered about the subjective experience of fullness (most likely resulting from either fear of weight gain or unfamiliar sensations related to regular eating). Patients are encouraged to wait out aversive experiences of fullness or related anxiety by mindful "urge surfing" until the sensation subsides naturally. Self-control is further increased by introducing planned behaviors incompatible with binge eating (e.g., taking a walk) and by limiting exposure to food (e.g., avoiding shopping) when patients feel that self-control is low.

Modifications for CBT for BED

Because many BED patients are overweight, in many instances their primary goal in treatment is weight loss, rather than merely stopping binge eating. Thus, the issue of weight loss and its relation to binge eating should be addressed from the outset. The therapist needs to acknowledge that weight loss is an important issue for the patient, but that the first step in therapy should be reducing or eliminating binge eating, because control over eating needs to be re-established before any attempt at weight loss can be commenced. The therapist should explore with the patient the benefits of eliminating binge eating, in general, and in relation to weight loss.

For overweight/obese patients with BED, it is important that the therapist provides accurate, up-to-date psychoeducation about obesity, and emphasizes that complex genetic and physiological factors are involved in the development and maintenance of obesity that limit the amount of control an individual has over his or her body weight and shape (see Chapter 65). A discussion about set-point theory should be integral to this stage of treatment, along with information about the ineffectiveness of strict dieting in reducing obesity, and its counterproductive effect on binge eating (cf. Chapter 24). There should also be some discussion about the unrealistic sociocultural norms for thinness and societal prejudice against obese individuals, especially women (see Chapters 19, 21, 27, 29, & 43). Since individuals with BED do not tend to restrict their dietary intake outside episodes of binge eating, introducing a pattern of regular eating should involve addressing *overall* eating behavior, particularly the tendency to overeat, as well as binge eating episodes.

Self-monitoring is extremely important for BED patients, since they may not be fully aware of the amount that they eat, or what constitutes a "normal" (moderate) intake. Patients should be provided with basic nutritional information, if needed, and also be encouraged to increase their day-to-day levels of physical activity.

Modifications for CBT for AN

The first eight sessions are conducted twice weekly and are aimed at establishing the foundations of therapy. It is important to understand the dilemma that patients with AN face: at one level they recognize that AN has deprived them of health and normal development, yet they remain terrified of "letting go" of their symptoms and regaining weight. The pros and cons of the disorder are analyzed, to highlight the perceived functions of the AN. It can be useful to

provide a conceptual framework of the adaptive functions served by the disorder (such as the avoidance of maturation), positive reinforcement experienced as social reinforcement for initial weight loss (admiration from peers or concern from parents), and the sense of mastery that may accompany sustained dietary restriction. Identifying higher order goals such as mastery and autonomy is followed by questioning the current effectiveness of AN in achieving those goals, and then considering the functionality of AN in the future.

Psychoeducation is given to provide information, increase motivation, and challenge cognitions. Learning that many of their symptoms stem from starvation helps patients appreciate the need for improvements to their physical health before the return of psychological health. Armed with information about the body's tendency to defend against weight loss by metabolic adaptation and about the biological pressure to regain weight, patients begin to question their beliefs about the benefits of dieting. Patients learn about the physiological and psychological adaptations that accompany weight loss, how this adaptation often leads to resistance to change, and why maintenance of a submenstrual weight is incompatible with recovery. Thus, they appreciate the need for therapy to address the psychological distress that accompanies weight regain.

Information about the seriousness of AN helps break through denial, particularly the medical and mortality risk (see Chapters 14, 52, & 55), the course of treatment required, the poor outcomes associated with inadequate treatment, and the more positive outcomes associated with appropriate treatment. Also addressed is the possible need for hospital admission, to reduce psychiatric or medical risk (see Chapter 50).

In contrast to CBT-BN, weighing occurs in each session, to provide current weight, but also to elicit cognitive, affective, and behavioral data. A minimum weight is established, below which a hospital admission would be indicated. A target weight range is chosen, corresponding to 90% of expected weight, with a range of 3–5 lb (1.4–2.3 kg) above this threshold, to allow for normal weight fluctuations. Weight gain is introduced at this point as an experiment rather than as a commitment to recovery.

Sessions are structured, as in CBT for BN; however, once patients have been weighed and their physical symptoms reviewed, the agenda items will depend on the patient's current physical state. Initial cognitive interventions develop from efforts at implementing behavioral changes and from the ensuing discussions that reveal underlying beliefs and assumptions, such as fear of bodily changes and of losing control. It is important for the therapist to pursue the questioning with a curious, accepting stance, linking back to the patient's values and goals.

The timing of self-monitoring will depend on the patient's motivation. Early in treatment, meal planning may be more helpful, in the form of a structured guide to the size, type, and timing of each meal. Food is considered "medicine," with an eating plan prescribed to prevent starvation and to "inoculate" against binge eating. Therapists become involved in this intervention rather than referring to a dietician, since the purpose is not merely related to nutrition but to an examination of motivation and beliefs. The quantity of food should be increased to allow for weight regain and the variety of food should be broadened to include previously avoided foods, such as carbohydrates. Classification of foods as "good" or "bad" is challenged and myths are dispelled. The cognitive-behavioral model may need to be introduced later in CBT for AN, once treatment targets have been agreed upon. CBT for AN also includes challenging cultural values regarding weight and shape and encouraging patients to examine the personal implications of adopting the prevailing cultural pressure to pursue thinness (see Chapters 19, 21, 43, & 44).

Modifications for CBT-E

As with CBT for AN, the first eight sessions take place twice weekly. Weighing occurs in the session and weighing at home is discouraged, to reduce both frequent weighing and the equally problematic avoidance of weighing. Education is provided concerning this form of weight checking and normal weight fluctuations. The weight is plotted on a graph weekly, accompanied by a collaborative interpretation of the emerging weight data.

An individualized formulation is created collaboratively by the therapist and the patient, using the patient's particular details and terminology. Whilst the focus of Stage 1 is behavioral change, engagement is the top priority, to ensure the patient is "on board" with treatment and the notion of change. Self-monitoring in CBT-E is established from the outset of therapy with all patients, and is considered as important as attending sessions. Emphasis is placed on "real-time" recording of behaviors, thoughts, and feelings in order to increase awareness and to facilitate the modifying of patients' behaviors and thinking and the changing of their relationship with their ED.

Psychoeducation is given, mainly through "guided reading" about eating problems, purging, excessive exercise, feelings of fullness, and, where relevant, the effects of being underweight. Significant others can become involved to help provide an optimum environment for the patient to change.

Stage 2 of Treatment: The Protocols

CBT-BN

This stage (sessions 9–16, weekly) involves cognitive and behavioral procedures for reducing binge eating and dietary restraint. Binge eating can be triggered by a number of events, including interpersonal stressors and negative emotions. Teaching formal problem-solving gives patients skills so they can cope with situations in ways that are more functional than binge eating.

Patients are reminded that rigid dieting makes them vulnerable to binge eating. Having made changes to their eating patterns (*when* they eat), patients are now encouraged to increase the variety (*what*) and the amount (*how much*) of what they eat. They are asked to create a list (i.e., a hierarchy) of "forbidden foods," which they rank in terms of difficulty. Behavioral experiments help patients tackle these avoided foods, using gradual and repeated exposure to previously feared foods, in order to disconfirm previous beliefs (e.g., "If I eat any chocolate I will binge"). Similarly, patients are encouraged to increase their tolerance of uncertainty by experimenting with eating in previously avoided situations (e.g., restaurants).

Patients will already have had their beliefs challenged by alterations in their behavior, such as eating regularly and consuming feared foods. Formal cognitive restructuring can be used to address dysfunctional cognitions, such as dichotomous thinking and unrealistic attitudes related to shape and weight, in order to develop more reasoned, functional beliefs to guide future behavior. Behavioral techniques are also used to tackle weight/shape concerns. Patients who avoid exposing their bodies by wearing baggy clothing are encouraged to wear more form-fitting garments, and those who scrutinize their bodies are instructed to limit this behavior.

Modifications for CBT for BED

In general, patients with BED share many of the same dysfunctional thoughts and beliefs expressed by BN patients, and these are addressed using the same cognitive strategies as in CBT-BN. However, there are some problematic beliefs that appear to be particularly prevalent

amongst individuals with BED. Foremost is the belief that he or she is "addicted" to food or certain types of food (see Chapters 54 & 65). Holding this belief means that individuals are often reluctant to relinquish their strict dietary rules, which, in turn, serves as a barrier to eliminating binge eating. The therapist should acknowledge that while there may seem to be some commonalities between the processes involved in binge eating and addiction problems (e.g., substance abuse), there are important differences and there is no evidence that specific foods are "addictive" (Wilson, 1993). In addition, total abstinence from food is impossible, so it is important that individuals learn to develop healthy yet flexible eating habits. Guided exposure and response prevention can be conducted either in vivo or as behavioral experiments, for example, exposing the patient to a small portion of an "avoided food" and then helping them to resist further consumption.

Overweight BED patients also tend to have comparatively high levels of concern about weight and shape, poor body image, and a self-critical mindset with regard to their weight and shape (see Chapter 22). An emphasis is placed on *body acceptance* as opposed to *body satisfaction* (see Chapter 33). Patients are encouraged to accept that, although they may be larger than they would prefer, self-criticism and the resulting negative emotions serve only to perpetuate a negative cycle of continuing low self-esteem, low mood, and poor control over eating.

Avoidance is another important behavioral feature to address. Many overweight BED patients will have been avoiding setting goals for engaging in activities such as social events, leisure activities, job challenges, or relationships–putting off starting these until "after losing weight." Avoidance behaviors need to be identified, and appropriate behavioral experiments are collaboratively designed to reduce the avoidance. For example, an individual who has been avoiding joining a social club is encouraged to commit to attending an orientation evening.

Finally, therapists should be aware that even when binge eating has been eliminated, some BED patients continue to overeat. The therapist should carefully approach the issue of reducing overall energy intake, making sure that the patient does not adopt rigid dietary rules but, rather, follows flexible, healthy dietary guidelines.

Modifications for CBT for AN

The second phase of CBT for AN (from session 9, weekly, and constituting the majority of treatment) continues to address motivation, psychoeducation, and progress, and sustains the emphasis on normal eating and weight regain sufficient to achieve normal biological functioning. Individuals with AN typically hold rigid schemas that tend to lead to dysfunctional interpretations of experiences (see Chapter 18). These dysfunctional thoughts (such as weight/shape being the major frame of reference for self-evaluation) are identified, explored, and challenged.

Patients are helped to understand that their lack of behavioral change stems from beliefs and fears that can be examined and modified, rather than from defiance or self-sabotage. Automatic thoughts and cognitive schemas are addressed and, following increased commitment to change, more formal cognitive restructuring is introduced. Beliefs are challenged via behavioral experiments, by highlighting the dissonance between competing beliefs, and by "decentering" (i.e., evaluating beliefs from different perspectives). Self-concept deficits are also addressed, by working to improve self-esteem and self-awareness.

Because of the marked interpersonal deficits in AN patients, interpersonal themes need to be integrated into treatment and are addressed with standard CBT approaches, highlighting the limits that AN places on social and intimate relationships. Family involvement is included

whenever the patient is living at home or financially dependent, and in older patients, when there is severe conflict. Family sessions can be conducted from within a cognitive perspective, assisting the patient and the family to identify dysfunctional beliefs and prescribing behavioral change.

Modifications for CBT-E

CBT-E incorporates an extra stage, a review, as Stage 2 (sessions 8 and 9), before progressing to address the more cognitive elements of the ED in the remainder of treatment (CBT-E's Stage 3). This review comprises a joint analysis of progress, taking into account the patient's behavior change and attitude toward the treatment approach, while barriers to change are identified (e.g., fear of change, rigidity, core low self-esteem, clinical perfectionism, interpersonal difficulties, and substance misuse). The formulation is reviewed and revised if necessary.

Stage 3 of CBT-E (weekly; sessions 10–17 in the short version and sessions 10–37 in the long version) is determined by the formulation. It is designed with consideration given to whether to use the broad version of CBT-E, and the relevance of each key maintaining mechanism for the particular individual. As noted previously, these key factors include overvaluation of weight and shape or control over eating; dietary restriction and dietary restraint; being underweight; and changes in eating triggered by moods or events. Only the focused version will be described here.

Stage 3 often starts with addressing weight/shape overvaluation and its consequences. Creation of a "pie chart" helps patients identify the typically large contribution of weight/shape to their self-worth and leads to a discussion concerning the negative effects of having such a dominant "slice." These include the riskiness of having "all one's self-evaluation eggs in one basket," the narrowing of interests, and the reality that weight and shape are not fully under one's control. This leads to identifying and enhancing previously marginalized domains for self-evaluation.

CBT-E includes a protocol to assess and treat obsessive shape checking, via questioning and educating about this phenomenon. Patients' beliefs about checking are challenged, and their behaviors (such as comparisons with others and overuse of mirrors) are systematically reduced, with the use of behavioral experiments. Avoidance, equally problematic, is also addressed, with behavioral experiments involving exposure. Another new focus found in CBT-E is the examination of "feeling fat." This subjective experience tends to be unrelated to weight but triggered by mislabeling certain emotions or body sensations. Patients are educated about this and, through monitoring of episodes, learn to identify triggers and masked feelings.

A further CBT-E strategy is teaching patients ways to control their "ED mindset," which is switched on by certain contexts. The core psychopathology of EDs is seen as a "frame of mind" that leads patients to filter their experience (e.g., by noticing only thin people), mislabel physical and emotional experiences (as "feeling fat"), and engage in behaviors associated with EDs (e.g., purging, rigid dieting, excessive exercising). Therapeutic strategies, designed to challenge key ED features and to dismantle the processes maintaining the ED, will have, by this point in treatment, begun to erode the ED mindset. It is now, when patients are experiencing moments or days free from the hold of their ED mindset, that they are helped to address it head on. The mind is compared to a DVD player, with an "ED DVD" that was once stuck but that is now no longer locked in place. Patients learn to recognize triggers in real time, to identify warning signs (the "first track" of the ED DVD) before the ED mindset gets locked back into place, and to displace ("eject") the mindset by engaging in positive behaviors learned in treatment (e.g., returning to a pattern of regular eating).

In this stage of CBT-E, dietary rules are revisited. Patients need to be reminded about the role of dieting (which they highly value) in the maintenance of the ED (i.e., as a cause of eating binges rather than a solution) and in impairment (i.e., the preoccupation with thoughts about food and eating, limiting eating socially, believing they have failed). Patients' remaining dietary rules are identified and behavioral experiments are designed to break a rule, including the consumption of avoided foods and the replacement of "rules" with "guidelines."

Event- and mood-related eating are addressed in CBT-E if they are playing a role in the maintenance of the ED. Patients identify examples of such episodes from real-time self-monitoring. Binge analysis is a specific strategy for understanding and learning from residual binge eating. Mood-related eating episodes, especially common in patients with mood intolerance, can be addressed with problem-solving. When this does not sufficiently prevent an escalation of mood, cognitive restructuring and mood acceptance (e.g., education about the ability to "ride out" moods) are introduced. Patients are encouraged to practice functional mood modulatory behaviors, such as playing soothing music, and to place barriers in the way of noneating dysfunctional mood modulatory behaviors (e.g., removing drugs or tools of self-injury, such as knives).

Stage 3 of Treatment: The Protocols

CBT-BN

In the final stage (sessions 17–19, at 2-week intervals) the therapist takes more of a backseat role. With a focus on maintaining progress following treatment, patients identify the key aspects of treatment they found particularly helpful. Preventing relapse and reducing the chance of a small lapse becoming a full-blown relapse are aided by identifying high-risk circumstances. Coping strategies are rehearsed and incorporated into a written maintenance plan. Self-monitoring ceases.

Modifications for CBT for BED

Treatment parallels that of CBT-BN, except that Stage 3 may need to be extended in duration, so that all of the changes in eating attitudes and behaviors have time to become well practiced. Treatment may be spread out with 2–4 weeks between sessions. There is also a distinctive focus on overall lifestyle modification and on the value of developing a broader range of life activities (other than eating) from which to derive satisfaction, fulfillment, and pleasure.

Modifications for CBT for AN

The future is discussed in terms of possible vulnerability and the potential for returning for further treatment, if warranted. Those who are still symptomatic should agree to continued medical management, and some may require hospital admission.

Modifications for CBT-E

In CBT-E the final stage is Stage 4 (sessions 18–20 in the short version, fortnightly, and 37–40 in the long version, 2–3 weeks apart). Patients start to weigh themselves at home weekly, at a predetermined time and day. The sessions become more future-oriented, with a

focus on holding realistic expectations, identifying strategies to be continued, and developing a long-term maintenance plan.

Conclusions and Future Directions

The cognitive-behavioral theory of BN and the treatment that stems from it (CBT-BN), first described by Fairburn (1981), have been extensively studied in a large number of experimental studies and RCTs. There is widespread agreement that CBT is the treatment of choice for BN, and the publication of detailed CBT-BN treatment manuals has allowed for the treatment to be widely disseminated. Various modifications of the original CBT-BN have evolved over the past two decades, allowing CBT to be applied to BED and AN. The evidence for the efficacy of manualized CBT is robust for BED and emerging for AN, and these treatments have been shown to be amongst the most effective currently available for these disorders.

More recently, the original CBT-BN has been refined and adapted to make it suitable for all forms of EDs, making it "transdiagnostic" in its scope. This new version is termed Enhanced CBT (CBT-E). The transdiagnostic model of EDs encompasses processes that are hypothesized to maintain all EDs: a dysfunctional scheme for self-evaluation; strict dieting; low weight and the associated "starvation syndrome"; and binge-eating and compensatory behaviors. It is also broader in focus than the original CBT-BN theory, in that it includes four additional maintaining mechanisms—clinical perfectionism, core low self-esteem, difficulty coping with intense mood states, and interpersonal difficulties—which are external to the ED psychopathology but in certain cases serve to maintain this psychopathology and prevent change.

Data are emerging to suggest that CBT-E may be even more efficacious than the original CBT for BN, resulting in significant improvements in both ED and general psychopathology. Looking ahead, it is important that further well-designed studies of the efficacy of CBT-E for all forms of EDs are conducted, in order to add to these promising data. In particular, in addition to RCTs, it is important that the findings from efficacy studies of CBT-E can be replicated outside controlled treatment trials, so that they can be generalized to patients treated in routine outpatient clinic settings. The results of recent "effectiveness" trials of CBT-E (Byrne et al., 2011) compare favorably in many respects to those reported in the only RCT of CBT-E (Fairburn et al., 2009). These findings provide strong evidence that manualized CBT-E is generalizable to treatment conducted in "real-world" clinical settings by therapists with a range of clinical experience.

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Dialectical Behavioral Therapy

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Clinicians continue to have concerns about the number of patients failing to respond to or relapsing after evidenced-based treatments for eating disorders (EDs) such as cognitive-behavior therapy (CBT; see Chapter 56) or interpersonal psychotherapy (IPT: see Chapter 62). Analysis by the U.K.'s National Institute for Clinical Excellence (NICE, 2004) asserts that CBT is the leading evidenced-based treatment for bulimia nervosa (BN) while IPT can achieve comparable results over a longer period of time. However, a recent study of CBT-enhanced (see Chapter 56) revealed that at 60-week follow-up a substantial portion (48.7%) of treated individuals still had a level of ED symptoms one standard deviation above the community mean on the Eating Disorder Examination (EDE; see Chapter 38) (Fairburn et al., 2009). As Christopher Fairburn (2008), a strong advocate of CBT for EDs, has noted in commenting on future directions for CBT, "First, the treatment needs to be made still more effective" (p. 261). Some have sought to accomplish this with various versions of dialectical behavior therapy (DBT).

The Widespread Use of DBT

With or without empirical research to support its use, DBT has been used in the treatment of EDs since the mid-1990s. A recent survey of treatment centers for EDs in the United States advertising in an EDs publication revealed that 39 (73.6%) of 53 programs employed DBT as a treatment modality across the spectrum of ED diagnoses (Bishop, 2013). Thus, many treatment centers are employing DBT tracks for their difficult patients. This raises the question, what might be the basis of the obvious popularity of this therapy in treatment centers?

DBT was originally developed to treat the parasuicidal or self-harming individual commonly diagnosed with Borderline Personality Disorder (BPD; Linehan, 1993; see also Chapters 14, 32, & 54). While ED behavior can be construed as self-harming and often occurs comorbidly with other self-injurious behaviors encountered in BPD (Zimmerman & Mattia, 1999; see also Chapters 14 & 54), the shift to DBT and other newer therapies seems to be due to a change in treatment emphasis from more cognitive approaches to affect regulation models (Bankoff, Karpel, Forbes, & Pantalone, 2012; Chen & Safer, 2010; see also Chapter 22).

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However, there are a number of additional reasons that make DBT potentially effective with ED patients. DBT has strategies for dealing with ambivalence about treatment (see Chapter 63), maintaining commitment to treatment, reducing affect intolerance, and minimizing the affect-driven or mood-dependent behavior generally seen in "difficult patients." In this chapter, I will briefly describe this treatment approach and its allure, as well as the empirical basis for its use with EDs.

Theoretical Foundations

Standard DBT is primarily an outpatient cognitive-behavioral treatment strategy. DBT was developed by Marsha Linehan (1993) at the University of Washington in the 1970s to improve the treatment of patients with marked emotional dysregulation and self-destructive behavior, usually in the context of BPD. The hallmark features of this approach were the application of behavioral science, mindfulness, and dialectical philosophy instead of the prevailing psychodynamic theories and treatments for these patients. Linehan called DBT a CBT approach because it employed behavioral principles of assessment and treatment using techniques such as self-monitoring, behavioral analysis, contingency management, cognitive restructuring, skills training, and exposure procedures (Koerner & Dimeff, 2007).

Linehan (1993) elaborated a biosocial theory of BPD in which emotion dysregulation is the product of emotional vulnerability and difficulty modulating emotional reactions. Emotional vulnerability results from increased sensitivity to emotional stimuli, intense emotional responses, and a slow return to one's emotional baseline. Problems with emotion modulation reflect difficulties inhibiting mood-dependent behaviors, organizing behavior in service of personal goals and values, regulating physiological arousal, diverting attention from emotionally evocative stimuli, or experiencing emotion without avoidance or extreme secondary emotion, such as feeling distressed or anxious because one is so upset.

The "social" aspect of Linehan's biosocial theory asserts that BPD develops in the context of an invalidating environment in which interpersonal transactions negate, punish, or question the internal experience of the emotionally vulnerable person. Invalidation also occurs when, in other circumstances, caretakers react erratically or inappropriately to understandable responses of the individual (Chen & Safer, 2010). Invalidating environments tend to oversimplify life's problems so that the child does not learn distress tolerance or form realistic goals. The child often evinces extreme emotional displays to elicit helpful responses from the environment. Often this reaction to the invalidating environment only reinforces the invalidating behavior in a vicious circle. With this pattern of interaction, amplification of emotional responses or avoidance of emotions may occur, giving rise to some of the characteristic features of BPD (Linehan, 1993).

Overview of Treatment and Central Strategies

Several randomized controlled trials (RCTs) have found DBT to be effective in reducing suicidal and self-harming behavior, psychiatric hospitalizations, substance abuse, and interpersonal problems in patients with BPD (Chen & Safer, 2010). Standard DBT is a complex treatment package consisting of four modules of psychoeducational skills training groups offered over a period of approximately 26 weeks. The modules are core mindfulness, emotion regulation, distress

tolerance, and interpersonal effectiveness. In addition to the group skills training, patients have weekly individual psychotherapy aimed at increasing more skillful management of emotions and decreasing ineffective behavior. As with CBT, self-monitoring with diary cards and other homework assignments are employed to encourage and reinforce the practice of skills outside of therapy sessions. Patients are also provided with telephone consultations to deal with issues and crises that occur between treatment sessions. Efforts are made to not reinforce self-injurious behavior by imposing a 24-hour delay of telephone contact after such behavior. Instead, patients are encouraged to call the therapist *before* urges are converted into behaviors. Beyond the direct involvement with the patient in treatment, therapists attend weekly supervision and support to ensure treatment fidelity and to help prevent burnout due to, for example, dealing with constant crises with suicidal or self-harm behavior (see Chapter 54). With standard DBT, patients are asked to commit to a year of treatment, encompassing two skills training cycles.

In her early work with emotionally dysregulated individuals, Linehan (1993) found that the usual relentless CBT focus on identifying dysfunctional thoughts and changing them to functional ones was too invalidating for her BPD patients and resulted in high rates of treatment dropout. She introduced the concept of "dialectics" to the behavioral treatment model (hence, the name DBT). Dialectic resolution means systematically juxtaposing opposites or apparent contradictions and finding the "middle path." The primary dialectic or tension to be addressed in DBT is acceptance versus change. In addressing EDs this would entail, as Wisniewski and Kelly (2003) have suggested, that one achieve a dialectical synthesis of overcontrolled eating versus mindless eating.

Linehan also observed that therapy with BPD patients was almost always ineffective due to behavioral contingencies operating outside of the therapist's awareness. The sheer volume and intensity of problems presented by the patient monopolized the time available in therapy at the expense of learning more effective behavior. By focusing on self-management skills and reduction of therapy-interfering behaviors, and by de-emphasizing emotional catharsis, DBT potentially offers a more direct route to acquiring more effective strategies for living. Thus, DBT directly targets the mood-dependent behaviors, to use Linehan's term, that are self-defeating in BPD patients. Metacognitive functioning (e.g., knowledge about one's own thinking, as well as when and how to apply certain problem-solving strategies) is enhanced through mindfulness techniques that help the person identify the state of mind in which she or he is functioning, that is, emotional mind, reasonable mind, or wise mind (Linehan, 1993).

As will be discussed below, it is not a huge leap to conceptualize ED sufferers, who tend to have extremely high harm-avoidant temperaments (Fassino, Amianto, Gramaglia, Facchini, & Abbate Daga, 2004; see also Chapter 32), as being exceptionally emotionally vulnerable and to conceptualize their ED behavior as mood-dependent. In other words, the heightened sensitivity of highly harm-avoidant ED sufferers inclines them to control and avoid negative affect in part by using ED behaviors to facilitate experiential avoidance. Targeting emotional vulnerability and mood-dependent eating behavior with DBT could possibly benefit those in general who are non-responders to CBT for EDs, and not just those with an ED and comorbid BPD (see Chapter 54).

The Central Strategies of Dialectical Behavior Therapy

According to Chen and Safer (2010), the central strategies of DBT, each of which balances change and acceptance, are dialectical, core, stylistic, case management, and integrated. I will highlight only the first four strategies. As the name indicates, and as noted briefly above, dialectics

are central to the theory and practice of DBT. The central dialectic of DBT is the tension between acceptance and change. Therefore, DBT emphasizes finding a balance between life's polarities, including the frequent need to look beyond what is immediately apparent in pursuit of solutions. As noted above, Wisniewski and Kelly (2003) have proposed that the primary dialectic in ED patients is "over-controlled eating versus absence of an eating plan" (p. 134). Linehan (1993) described other dialectical tensions in BPD patients that are not uncommon in ED patients without that diagnosis: emotion dysregulation versus self-invalidation, inhibited grieving versus unrelenting crisis, and apparent competence versus active passivity. Common dialectical tactics applied by the therapist are use of metaphor, stories, experiential exercises, and playing the Devil's advocate, all in service of alternating between ambiguity and certainty, while acknowledging and exploring paradox. One significant, recurrent, and dialectical challenge in therapy is gauging the balance between accepting the patient's vulnerabilities and changing what is possible in the current moment.

One of the principal core strategies aimed at a healthy balance between promoting acceptance and facilitating behavioral change is behavioral chain analysis. This descendant of Skinner's functional analysis entails a structured examination of problematic behavior, including its topography, intensity, frequency, duration, situation, antecedents, and consequences (Chen & Safer, 2010). Thus, chain analysis helps the patient understand the cues, maintaining factors, and function of the problematic behavior. It helps the clinician to understand the obstacles to effective behavior and thereby enable the patient to acquire appropriate metacognitive and behavioral skills. Another important core strategy is validation of the person's intense feelings and concerns, while later engaging with the person to question the appraisal processes that generated those strong, often maladaptive reactions. Validation strategies help the patient be mindful of and accept his or her experience, while facilitating development of the strong therapeutic alliance necessary for exploration and modification of maladaptive cognitions and behaviors.

Stylistic strategies encompass reciprocal communication and irreverent communication (Chen & Safer, 2010). Reciprocal communication conveys empathy and involves interpersonal warmth, responsiveness to the patient's concerns, and strategic self-disclosure. In contrast, irreverent communication can use a confrontational tone, paradoxical expression, or an outrageous, humorous, or blunt style; all of these are intended to shock or grab the patient's attention, especially when he or she is engaging in therapy-interfering behavior. For example, if the patient says that she did not have time to complete her diary (self-monitoring) card, the therapist might irreverently remark, "yet you found time for bingeing and purging." Keep in mind that these kinds of remarks should be context sensitive.

Case management strategies subsume three broad tactics: consultations with the patient, for the patient, and about the patient. The consultation-with-the-patient strategy helps the patient develop skills for interacting with the environment. The therapist acts on the patient's behalf, that is, consults for her or him, only when absolutely necessary, such as when the patient is in a powerless situation. When consulting with other professionals who are working with the patient, as is frequently the case with ED patients (see Chapters 50, 54, 59, & 61), this is done in the presence of the patient whenever possible. Often, patients have to be educated about how to deal with other medical personnel who are less familiar with EDs. Finally, consultation by the therapist about the patient is a special feature of DBT work in which the treatment team supports the therapist in the challenging clinical work and helps to insure that DBT-informed treatment is delivered.

Application and Evaluation of DBT in the Treatment of Eating Disorders

DBT for ED patients has been applied in its standard form especially with comorbid BPD or, for research purposes, in a form adapted for patients with binge eating disorder (BED) and BN (see Table 57.1). The most extensively researched is DBT adapted for BN (Chen & Safer, 2010). I will now review the current research findings for these approaches and their variations.

Standard DBT

There are no RCTs, but several uncontrolled studies of the effects of standard DBT with minimal modifications in ED patients with BPD have been published (Ben-Porath, Wisniewski, &Warren, 2009; Chen, Matthews, Allen, Kuo, & Linehan, 2008; Federici & Wisniewski, 2013; Kröger et al., 2010; Palmer et al., 2003). With the exception of one male participating in the Ben-Porath et al. (2009) study, all participants in these studies were female. There were no participants with AN in either the Chen et al. (2008) or Palmer et al. (2003) studies. In the Chen et al. (2008) study of eight patients, standard DBT was employed with the usual targets of reducing imminent high-risk suicidal and life-threatening behaviors, therapy-interfering behaviors, and quality-of-life interfering behaviors (e.g., economic or health issues), while replacing maladaptive responses with skillful behaviors useful for pursuing healthy individual goals. In contrast to standard DBT for BPD, more emphasis was given to ED-specific problems, in particular binge eating. The active treatment was only 6 months rather than the recommended 12 months. The investigators conducted a systematic assessment with instruments evaluating suicidal and self-injurious behavior, ED pathology, *DSM-IV* (*Diagnostic and Statistical Manual of Mental Disorders*, 4th edition; American Psychiatric Association, 1994) Axis I and II (personality) disorders, and social adjustment.

Table 57.1 Dialectical Behavioral Therapy (DBT) modifications at a glance.

DBT Variations with ED	*Modifications of Standard DBT*	*Target ED Diagnoses*
DBT for comorbid BPD and ED (Chen et al., 2008; Federici & Wisniewski, 2013)	Standard DBT with minimal modifications such as the addition of an ED module, e.g., Weight and Eating	AN, BN, BED, EDNOS
Appetite-focused DBT for ED (Hill et al., 2011)	Abbreviated Stanford adaptation of DBT of 12 weeks with the addition of Appetite Awareness Training	BN, ED-NOS (subthreshold BN)
DBT for BN and BED (Safer et al., 2001; Telch et al., 2001)	Stanford adaptation of DBT: 20 weeks of group or individual therapy with elimination of interpersonal effectiveness component	BN, BED
DBT for adolescents with ED (Salbach-Andrae et al., 2008)	Standard DBT adapted for adolescence with the addition of Food and Body Image and Emerging Autonomy modules	AN, BN

Note. ED = eating disorder; BPD = borderline personality disorder; AN = anorexia nervosa; BN = bulimia nervosa; BED = binge eating disorder; ED-NOS = eating disorder not otherwise specified.

At 6-month follow-up post-treatment, there were large effect sizes on all outcome measures in the desired direction, suggesting efficacy in spite of the modifications to standard DBT.

Palmer et al. (2003) applied the standard DBT using all four basic elements but with the addition of a special skills training module designed for ED patients. Seven patients were treated, with no dropouts from the program and a substantial reduction of hospital confinement and self-harm. At 18-month follow-up, no patients met full criteria for an ED, although four had a partial syndrome diagnosable as eating disorder not otherwise specified (ED-NOS; see Chapter 4).

The other three uncontrolled studies of DBT in ED patients were done at higher levels of care and included AN patients. Ben-Porath et al. (2009) investigated whether ED patients with BPD differed from those without BPD on measures of ED symptoms and general distress over the course of DBT-informed treatment at the partial hospitalization level, and whether the two groups differed on post-treatment expectancies concerning individual ability to regulate negative affect. The treatment significantly reduced ED symptoms but there were no significant post-treatment differences between those patients with or without BPD on the key dependent variables. As expected, at pretreatment those with ED and BPD were significantly less able to regulate affect than those without BPD, but at post-treatment there were no statistically significant differences. Ben-Porath et al. (2009) noted that their findings were concordant with at least one previous study (Zeeck et al., 2007) showing that comorbid BPD has minimal impact on ED treatment outcomes. Other studies have implicated BPD as a significant predictor of outcome (Johnson, Tobin, & Dennis, 1990; Wonderlich, Fullerton, Swift, & Klein, 1994).

Kröger et al. (2010) reported the outcome of adapted inpatient DBT of 3 months duration with an added CBT module in 24 women with BPD (9 with comorbid AN and 15 with comorbid BN). All of the patients had failed to benefit significantly from previous ED-related inpatient treatments. ED symptomatology was significantly reduced at post-treatment and follow-up. At 15-month follow-up the remission rates for AN and BN were 33% and 54%, respectively, although no patients received DBT-informed treatment between post-treatment and follow-up. For AN patients the mean weight was not significantly increased at post-treatment but did increase somewhat at follow-up.

A third uncontrolled study (Federici & Wisniewski, 2013) consisted of a case series of seven participants who had previously been unresponsive to CBT-based treatment with DBT components. Four patients were diagnosed with AN and three with ED-NOS as well as other psychological disorders. Most had comorbid BPD or traits. The program was rigorously standard DBT delivered at the intensive outpatient (IOP; sometimes called "Day Treatment") level 3–5 days per week with a 6-month commitment. One patient was advised to discontinue treatment due to frequent hospitalizations during the first month of the program. For the others, however, there were large reductions in ED behaviors and in therapy-interfering behaviors, as well as increased medical stability at post-treatment. Unfortunately, no follow-up data were collected.

In another uncontrolled outpatient study (Salbach-Andrae, Bohnekamp, Pfeiffer, Lehmkuhl, & Miller, 2008), 12 adolescents with AN and BN participated in a DBT program expanded to 25 weeks. Only one patient had comorbid BPD. In addition to the standard four DBT modules, Salbach-Andrae et al. (2008) developed and implemented two modules called "Dealing with Food and Body Image" and "Walking the Middle Path." In the latter, the parents were included during the whole module as opposed to once in each of the other modules. At post-treatment, of the six patients diagnosed with AN only one continued to have that diagnosis. Of the six patients diagnosed with BN, three continued to meet the criteria for BN but two

shifted to ED-NOS. One of the BN patients prematurely discontinued treatment. In contrast to the findings of Ben-Porath et al. (2009), Salbach-Andrae et al. (2008) proposed that the lack of remission among the BN patients was related to the more severe comorbidity in that group. However, across all 11 adolescents who completed treatment, there were significant reductions in ED behaviors (as measured by subscales of the Eating Disorder Inventory-2) and in a measure of general psychopathology (the Global Severity Index of the SCL-90).

These uncontrolled studies show that ED symptomatology can be reduced by standard DBT with some modification to focus on ED behavior with or without comorbid BPD. However, they do not indicate that DBT is better than other accepted treatments or inform us about what moderates or mediates treatment effects.

Highly Adapted DBT for Disorders Involving Binge Eating

The other trend in the literature is the use of highly adapted DBT for EDs. The ED professional group at Stanford University, led by Safer, Telch, and Agras, has produced the majority of these studies, starting with case reports, case series, and RCTs with waitlist control groups, and culminating with a study utilizing an active comparison group control. The modifications ("adaptations") of standard DBT in these studies were variable and extensive. Modifications included constraining the treatment to 20 sessions so as to be comparable to studies of CBT (see Chapter 56), eliminating individual sessions in some instances (mainly with BED), or having only individual sessions while excluding group treatments in others (mainly with BN). The Stanford series of studies also eliminated the "Interpersonal Effectiveness Module" of standard DBT in order to isolate better the effect of affect regulation and distinguish the treatment from IPT (see Chapter 62).

The Stanford team, along with research in Colorado done in collaboration with Hill and Craighead, has conducted four RCTs, two with follow-up data and two without (Hill, Craighead, & Safer, 2011; Safer, Robinson, & Jo, 2010; Safer, Telch, & Agras, 2001; Telch, Agras, & Linehan, 2001). Two of these studies were conducted with BN or subclinical BN patients (at least one binge/purge episode per week) and the other two studies were conducted with BED patients. The two BN studies involved individual therapy sessions and had no follow-up assessments.

Hill et al.'s (2011) study of BN or subclinical BN patients added appetite awareness training to a reduced version of the manualized DBT approach described by Safer, Telch, and Chen (2009). In this study 32 patients were randomly assigned to DBT-appetite-focused (DBT-AF) therapy or to a 6-week delayed treatment (i.e., waitlist) control. The components of DBT-AF were appetite awareness training and emotional regulation skills. Together, these were used to teach individuals to have greater awareness of emotions and appetite signals, to replace binge eating and purging with adaptive emotion regulation skills, and to use internal signals of hunger and fullness to direct eating behavior. The time frame of 15 hours of intervention delivered in 12 sessions over 12 weeks was chosen to fit the treatment into the typical college semester. Compared to the waitlist condition at 6 weeks, DBT-AF produced significant reductions (with moderate to large effect sizes) in objective binge and purge episodes and in scores on several measures of ED and general psychopathology. At the completion of the study, of the 26 subjects who actually entered treatment, 7 (26.9%) were abstinent from binge/purge episodes over the previous month and 16 (61.5%) no longer met full or subthreshold criteria for BN. From the standpoint of dropouts, two subjects in the initial treatment condition (11.1%) and two from the waitlist failed to participate long enough to complete the 6-week assessment.

Safer et al. (2001) randomly assigned 31 women with BN or subclinical BN to receive DBT or to a waitlist. Treatment was delivered in 20 individual 50-minute sessions. At post-treatment there were significant reductions (with moderate to large effect sizes) in episodes of binge eating and purging; approximately 29% of participants in the DBT group were abstinent from binge/purge behaviors compared to none in the waitlist group. Five DBT participants remained symptomatic and met *DSM-IV* (1994) criteria for BN. There were no significant differences between groups on secondary measures of, for example, mood regulation and depression, after correction for multiple comparisons.

The two RCTs with follow-up were done at Stanford with BED patients in a group therapy format. Telch et al. (2001) randomly assigned 44 women to a waitlist control or to a 20-session manualized DBT treatment condition previously shown to be effective in an uncontrolled study (Telch, Agras, & Linehan, 2000). Four participants in the treatment group and six in the waitlist group dropped out of the study. Analysis was done on completers only—although Safer et al. (2010) later did an intent-to-treat analysis of the data. The group DBT intervention produced significant reductions at post-treatment in binge eating days as well as total number of binge eating episodes. Abstinence from binge eating was found in 89% of the DBT group versus 12.5% of the controls post-treatment. Those receiving group DBT also reported significantly lower scores on measures of eating in response to anger, as well as concerns with weight, shape, and eating. At 3- and 6-month follow-up those who completed treatment were 67% and 56% abstinent, respectively. Interestingly, when waitlist participants were offered treatment, 14 accepted, 4 dropped out and, of those completing, 90%, 80%, and 67% were abstinent at post-treatment, 3-month, and 6-month follow-up, respectively.

Although the results were promising, the Telch et al. (2001) study does not shed light on the mediators of effect or whether this adaptation of DBT per se accomplished the effect. As the researchers pointed out, the data offered no support for the hypothesis that the treatment worked by reducing negative affect or improving expectancies for regulation of negative affect. However, the lower scores on the measure of eating in response to anger may reflect a reduction in the urge or impulse to eat in the face of negative emotions, rather than a diminution of negative affect.

In an effort to address these questions, Safer et al. (2010) randomly assigned 101 men and women to receive either a DBT group treatment or an active comparison group therapy (ACGT). Using an "active placebo" controlled for the placebo and/or other nonspecific effects of DBT as a form of psychotherapy. Additionally, the authors added a measure to test more directly the purported effect of DBT-BED on the important mediating variable of difficulties in emotional regulation. Safer et al. (2010) found that the DBT-BED group had a significantly lower dropout rate (4%) than the ACGT group (33.3%). At post-treatment, 64% of the DBT-BED participants were no longer binge eating, as compared to 36% in the ACGT condition. However, there were no significant differences at 12-month follow-up, indicating that there was little or no longer-term advantage of group DBT over the ACGT.

Interestingly, unlike at post-treatment, at the 12-month follow-up small effect sizes in favor of ACGT were found on the self-report measures of negative affect and difficulties in emotional regulation; this certainly fails to support the hypothesis that DBT-BED works by reducing negative affect. Safer et al. (2010) concluded that both DBT-BED and ACGT were beneficial in reducing binge eating. Of the two treatments, DBT-BED was probably more acceptable to patients, as evidenced by the lower dropout rate and higher follow-up assessment completion rate. DBT-BED produced more rapid changes on the primary measures at post-treatment, but this advantage was largely lost at 12 months follow-up. Further analysis of data from this cohort of patients found that avoidant personality or earlier onset of overweight and

dieting was associated with worse outcome in the ACGT compared to the DBT-BED group (Robinson & Safer, 2012).

The RCTs reviewed above show that the Stanford form of modified group DBT reduces ED symptomatology in comparison with waitlist controls but does not significantly improve outcome for BED at follow-up compared to active group therapy, although DBT-BED retained patients in treatment significantly better than the comparison form of group therapy. Further analysis indicated that subgroups of BED patients may benefit more from DBT than group therapy in general.

The Place of Dialectical Behavior Therapy in Eating Disorders Treatment

Aside from the finding that strictly defined EDs (28% were ED-NOS) are comorbid in about 34% of BPD patients over a 6-year period (Zanarini, Frankenburg, Hennen, Reich, & Silk, 2004; see also Chapters 15 & 54), there are a number of reasons for using DBT with ED patients. A frequently cited rationale for applying DBT to EDs with and without BPD is the association of negative affect with binge eating, indicating that the latter is a mood-dependent behavior (see Chapter 32). Heatherton and Baumeister (1991) are often referenced for their theory of binge eating as a means to escape a spiraling cycle of negative affect and self-awareness. There is support for this in ecological momentary assessment data showing reduction of negative affect after engagement in ED behaviors (Berg et al., 2013). In other words, binge eating and purging can be seen as linked forms of experiential avoidance in which the increasingly aversive awareness of increasingly aversive affect is diminished. Experiential avoidance specifically refers to the attempt to escape awareness of the contents of one's mind (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996). Consequently, the various adaptations of DBT seek to minimize use of binge eating and purging to avoid painful emotional experiences while building a skill set that allows the individual to direct behavior toward more effective living.

Several theoretical explanations have been given for the less than optimal outcomes of various currently accepted treatments for ED patients (Vanderlinden, 2008). Current treatments, including CBT, have difficulty motivating patients to give up ED behaviors (see Chapters 12, 18, & 63). When there is a direct agenda of changing so-called dysfunctional thoughts about shape, weight, and their control, some ED patients find this too threatening and thus resist because they feel the ED is the best or their only "solution" in terms of traditional problem-solving. The ED removes looming and often extremely aversive experience from awareness, thereby providing reliable, temporary, and fairly immediate relief. Thus, symptom-focused strategies may fail. DBT, on the other hand, employs broader containment and acceptance strategies for negative emotions or thoughts without necessarily focusing on the ED symptoms per se. DBT also uses strategies to help patients commit to behavior that effectively and skillfully advances them toward desired goals and purposes that could have more intrinsically reinforcing effects.

The Hierarchy of Treatment Goals

With the substantial prevalence of comorbid BPD in ED patients treated at the higher levels of care, DBT in some form is likely to continue to be a substantial component of treatment. Given this situation, it is important to consider the stages of intervention in DBT, because this helps to organize thinking about treatment with ED patients.

Linehan (1993) described a hierarchy of primary treatment targets in DBT that consisted of four stages, including pretreatment (she later added another stage, which will not be discussed here). According to Linehan, in the initial stage of pretreatment (Stage 1) the targets should be orientation to treatment and agreement on goals. In the first stage of treatment (Stage 2), which she called "attaining basic capacities," the targets are decreasing suicidal behaviors, therapy-interfering behaviors, and quality-of-life-interfering behaviors, and increasing behavioral skills in mindfulness, interpersonal effectiveness, emotion regulation, and distress tolerance. Thus, in Stage 2, DBT aims to increase behavioral capabilities and motivation for treatment (Chen & Safer, 2010). In Stage 3, the target is decreasing post-traumatic stress (see Chapter 34), which Linehan thought best delayed until Stage 2 targets were under control. In the final segment (Stage 4) of Linehan's model, the goals are increasing self-respect and achieving personal goals.

Most of the adaptations of DBT for ED described above have worked within the context of Linehan's Stages 2 and 4. However, DBT for severe and complex EDs, such as that used by the Cleveland Center for Eating Disorders (Wisniewski, Safer, & Chen, 2007), addresses multiple stages of treatment and is employed at all levels of care. These patients have multiple comorbidities and thus are often highly dysregulated, so treatment targets must be prioritized toward controlling life-threatening behavior. In extremely acute cases of AN and BN, the ED behavior itself often falls into this category. Cases with comorbid PTSD may also be so dysregulated as to require the addressing of Stage 3 targets.

Conclusions and Future Directions

My review of the literature and my extensive clinical experience support the conclusions that Bankoff et al. (2012) drew from their critical review of the literature addressing the use of DBT in treating EDs. This form of therapy appears to be a promising alternative to CBT and IPT for EDs, especially when there is comorbid BPD. DBT reduced dropout rates, while targeting and reducing ED behaviors and other comorbid symptoms.

Recalling that DBT was originally developed for the treatment of highly dysregulated patients whose behavior was often life-threatening, the studies done thus far may not have fully tested the strengths of this treatment approach for addressing refractory patients (see Chapter 12). Unfortunately, the literature yields no conclusive instruction about what level of care is served better by DBT or what elements of DBT must be included to achieve the best effects. As Bankoff et al. (2012) noted, as yet there have been no RCTs that directly compare DBT in any form (standard or otherwise) with evidence-based treatments such as CBT or IPT. Nor have moderation or mediation studies been done to identify the factors and processes that facilitate change. Similarly, there are no data teasing apart the relative beneficial effects of each component of standard DBT, although a mediation analysis done in a study of the treatment of BPD showed that use of DBT skills fully mediated decreased suicide attempts and depression, as well as increased control of anger over time (Neacsiu, Rizvi, & Linehan, 2010). Without knowledge of the factors that mediate the treatment effects of standard DBT, the modifications of DBT in ED treatment trials to date have been dictated mainly by delivery restraints and research questions, which may have diluted the potential effects of standard DBT.

There are many questions that need to be answered before DBT-ED, as an exclusive treatment approach, can be accepted as an alternative to current empirically supported

treatment such as CBT and IPT. While standard DBT and CBT have demonstrated success in reducing problematic behavior, they continue to lack a coherent theoretical base that accounts for their efficacy (Hayes, Luoma, Bond, Masuda, & Willis, 2006). There continue to be questions regarding which components of these treatment packages contribute most to outcomes (Neacsiu et al., 2010). Does emotional regulation skills use actually improve emotion regulation? And does use of distress tolerance skills actually contribute to the improved behavioral control often produced by standard DBT (Neacsiu et al., 2010)? Without this information, it is difficult to design DBT treatments best suited for patients with EDs.

It was probably too soon to test the affect regulation model of binge eating, as enticing as it might be, without these questions being answered. Nevertheless, the results obtained in the uncontrolled studies strongly suggest that adapted DBT applied in a fashion more closely aligned to standard DBT produces clinically significant positive effects. Further study should be undertaken to ascertain whether the full package of DBT is better suited for certain patients. Robinson and Safer (2012) have suggested a stratefied approach based on patient characteristics. At least with the Stanford adaptation of DBT-ED, BED patients with avoidant personality and earlier onset of overweight and dieting did better with DBT than with the general group therapy. Likewise, other adaptations of DBT-ED, such as appetite-focused DBT, need to be tested against standard approaches and evaluated for what actually mediates their effects.

Although Fairburn et al. (2009) have advocated a transdiagnostic application of CBT-ED, DBT has not been rigorously evaluated in the treatment of AN. Several uncontrolled studies (Ben-Porath et al., 2009; Federici & Wisniewski, 2013; Salbach-Andrae et al., 2008) have suggested that DBT may be useful in this population, even if the affect regulation link may not be so apparent as with BN or BED patients. AN patients tend to be extremely intolerant of affect and therefore could be considered affect-phobic or experientially avoidant (Schmidt & Treasure, 2006). Adaptations of emotion regulation and distress tolerance skills for AN may be possible, promoting containment and acceptance so that strong negative emotions do not continue to be obstacles for effective action.

The behavior of most ED patients, and especially those with AN, seems to be dominated by excessive avoidance and control of potentially aversive experiences. This has been exploited in some treatment settings to motivate patients to eat by negative reinforcement or punishment. Although this may motivate (i.e., "get") patients to eat and restore weight in the short run, this type of contingency management frequently backfires once they are out of the controlled treatment environment. This raises the question of the place for increased emphasis on positive emotions and intrinsic values, particularly in the context of interpersonal effectiveness. DBT has always focused on effectiveness in pursuit of the patient's idenitified goals and purposes (Linehan, 1993), so perhaps this could be sharpened in DBT-ED. Combining DBT-ED with the processes of valuing and committed action emphasized in Acceptance and Commitment Therapy (ACT; Hayes et al., 2006) may improve motivation for recovery from ED. DBT can readily incorporate acceptance stategies within the acceptance versus change dialectic. A study using ACT groups in a residential ED treatment facility found that the strongest mediator of change was willingness to have distressful thoughts and feelings (Juarascio, 2011). This suggests that DBT's traditional focus on willingness versus willfulness may be productive.

In conclusion, there is much to be learned about the potential uses of DBT with ED patients. It is too soon to either endorse DBT wholeheartedly or write it off completely as an alternative treatment of EDs. Hopefully, researchers will take on the challenges of this work and expand our knowledge in this promising area.

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58

Feminist Therapy

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> Feminism is a movement to end sexism, sexist exploitation, and oppression. (Hooks, 2000, p. 4)

Susie Orbach's *Fat is a Feminist Issue* (1978) began a wave of writing that characterized women's troubled eating patterns and weight-related issues as responses to complex social, political, and economic forces. The wave continued through the 1980s as themes such as women's contradictory gender roles, changing body ideals, and women's ownership of their sexuality were explored. By the 1990s feminist approaches to therapy for eating disorders (EDs) gained considerable momentum as three books were published in the United States (Bloom, Gitter, Gutwill, Kogel, & Zaphiropoulos, 1994; Fallon, Katzman, & Wooley, 1994) and Canada (Brown & Jasper, 1993). These works drew parallels between the violence women experienced in their day-to-day lives and the "medical model" of treatment for EDs. They critiqued the use of force or coercion in refeeding and proposed, instead, collaborative therapy models that made connections between the contexts of women's lives and their eating and body image difficulties.

More recent works (McGilley, 2009; Piran, Jasper, & Pinhas; 2004; Tantillo, 2006) propose key elements or concepts in feminist practice with EDs and recognize that these elements can be integrated with diverse approaches to therapy, including cognitive-behavioral (see Chapter 56), interpersonal (see Chapter 62), psychoanalytic (see Chapter 19), narrative, or psychoeducation. Malson and Burns (2009) use a critical feminist approach, applying post-structuralist and postmodern perspectives to theorizing and therapy for eating "dis/orders." Brown (2007) integrates a similar feminist perspective with a specifically narrative therapy approach.

As there is no definitive feminist approach to treating EDs, each of the broad categories of feminisms—liberal, cultural, radical, socialist, and finally, critical social and postmodern feminisms—will be briefly described here with accompanying examples of applications to therapy for EDs. These feminisms have influenced one another, but have also made distinct contributions. Each sees sexism as a source of oppression (see Chapters 19 & 27), and some see it as one of an *intersecting* set of systemic oppressions (see Chapter 43). Feminist therapy works toward empowering women to step out of roles, relationships, and practices that subjugate

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them and encourages them to challenge the system that produces and perpetuates these. Ultimately feminism seeks to end oppression. What is unique to each type of feminism is what it identifies as the focal point for change: laws, institutions, theories, societal structures (e.g., patriarchy), economic systems (e.g., capitalism), and/or dominant stories. Examples are given to show how each perspective can be integrated into ED treatment. Understanding the principles of each type of feminism may enable therapists and other readers (e.g., as activists) to generate many more ideas of how to apply them in practice.

Liberal Feminism

Liberal feminism (Friedan, 1963; Stanton, 1898/2006; Wollstonecraft, 1792/2012) holds that women should have rights and access to opportunities that are equal to those of men. It encourages women to challenge inequality as it is encoded in laws, social rules, and institutions. Liberal feminists spearheaded the actions that won women the right to vote, and they championed affirmative action to promote equal access for women, lobbied for paid maternity leave for women, and challenged divorce laws. Liberal feminism has been criticized for considering the plight of privileged White women only and not recognizing the need to change socioeconomic systems that maintain the intersectionality of oppressions (Crenshaw, 1991, as cited in Davis, 2008; Hooks, 2000) upon which White privilege depends.

Three examples of ways that liberal feminist concerns might be brought into therapy with girls and women with EDs are described below.

1 Facilitate a group that fosters consciousness-raising about socially rooted restrictions and demands on girls and women, then encourage and empower them to challenge these (see Chapter 43). For example, the group could explore the social imperative for girls and women to be "nice and kind" (Prentice & Carranza, 2002), as well as what this imperative may cost them in terms recognizing their own needs and wishes. Participants can learn how to bring themselves more fully into relationships using assertiveness skills that are taught in the group.
2 At ED assessments for teens, parents are frightened for their child's future. Often, they want to know how it happened that their child has an ED. A short version of some risk factors, including gender-related factors (see Chapters 19 & 27), can be given: that she is female; she lives in a country where body ideals are unrealistic; and she has a temperament that can make a girl more vulnerable to anorexia nervosa (AN)—for example, being highly sensitive, detail-oriented, perfectionist, very structured, and with exceptional focus (Boachie & Jasper, 2011). The therapist can say something to the effect of: "If I needed brain surgery or a bridge to be designed, I would want someone like your daughter to do it, because she will take her time and do the right thing" (Boachie & Jasper, 2011, p. 38). Reframing individual risk factors in such terms gives the message to parents and their daughter that, although she is seriously ill now, she has a future that is full of possibility, including not being constrained by gender role stereotypes. The adolescent hears that her characteristics are not pathological and are valuable in social terms; thus, although she fears her dream of perpetual thinness is dying, her potential to fulfill more substantial dreams is affirmed.
3 Resist affirming gender-based prescriptions and proscriptions. Theresa Bernardez (1996) comments about the ways in which therapists tend to guide girls and women away from,

for example, the expression of anger, because there is a cultural proscription against it. Young women who do express anger may be denigrated with the label "bitch." It is not unusual for teens in ED treatment to become angry (expressed through yelling, being insulting, using the "f-word") with staff and with their parents when, for example, the adults insist on an inpatient admission or partial hospitalization (see Chapter 50), on adequate food intake and weight gain (see Chapters 52 & 61), or for purging to stop. It is important not to target the anger itself as unacceptable or to shame girls/women for being angry, but at the same time to stay firm about ED-related expectations. Limits about not breaking things or using abusive language should be set and kept, without communicating that it is bad to be angry. Later in the process of recovery, anger may surface related to underlying issues the patient has previously been silent about (Lask & Bryant-Waugh, 2007). We can first validate that she is telling us about something important and then, if she is being abusive or demeaning, ask her to use more respectful language.

To summarize, feminist therapy from a liberal perspective is concerned with equality for girls and women, which means challenging gender-related inequities, and working to empower girls and women to take an equal place in the world.

Cultural Feminism

Cultural feminism revalues "the feminine," which has typically been seen as less valuable than "the masculine." Relational cultural feminists like those of the Stone Center (Jordan, 1991), as well as Gilligan (1982) and Chodorow (1978), have taken issue with dominant male-centric models of human development that imply, if not proclaim, that women are essentially inferior to men (Erikson, 1950; Freud, 1905/1962). Jean Baker Miller (1991), for instance, proposed a relational model of development derived from observations of girls, families, and women, and how girls grow and mature into women. Its main premises are that throughout the lifespan, human beings grow through and toward connection, rather than via separations (Erikson, 1950); that all human beings need connections to flourish; and that disconnections are the source of psychological problems, especially for women (see Chapters 33 & 43). Using this model in therapy "the perspective shifts from control and self-sufficiency to one of relatedness and mutuality" (Jordan, 1997, pp. 140-141), which leads the therapist "to subtly and sometimes directly encourage turning to others for support and assistance rather than emphasizing an ultimate state of self-reliance and independence" (Jordan, 1991, p. 288).

Many young people with EDs valiantly try to be self-sufficient, when in fact they need relationships (including family ones) in which they can be emotionally vulnerable and feel supported. As Jordan (1997) observes, when this primary need for emotional connection is not met, the need to feel special will dominate. An ED can provide a way of feeling special through control of appetite and weight, especially in comparison to the common "failed dieter," or through the spotlight that medical crises and treatment bring. Recovering means letting go of these ways of feeling special, a task that is eased by developing emotional connections and mutuality in relationships. Tantillo and colleagues describe the way multifamily groups for adults with AN can foster intra- and inter-personal connections that contribute to full recovery (Tantillo, 2006; Tantillo, Sanftner, & Hauenstein, 2013). McGilley (2009) describes a group for adolescents with EDs, and Piran et al. (2004) describe a case of binge eating disorder (BED), both of which are also informed by relational-cultural theory.

Consider the relational-cultural model in the context of a partial hospitalization program (PHP). In the example that follows, adolescent participants attend 5 days a week and on those days eat all their meals and snacks during program hours, with staff supervision. Parents take over meal support on evenings and weekends. Average length of stay is about 6 months, and the adolescents enter and leave the program at different times. This type of program provides intensive and helpful behavioral support for renourishment, weight gain or stabilization, and prevention of compensatory behaviors like vomiting and overexercise. Because of significant time spent there, the milieu also has the potential for supporting recovery through fostering *growth in connection*, thereby helping teens shift away from competitive pursuit of specialness based on body size and illness severity, and toward mutually supportive relationships.

Two staff supervise each meal and snack and eat the same foods as the patients, modeling the possibility of eating without fear and with acceptance of their varying body shapes and sizes. Staff also support everyone to eat all of their food within the time limit.

Case Example

Susan

Susan is a 15-year-old, and the second daughter of a white, upper middle class, intact family from South Africa. She has been struggling with AN for several years. Her body is showing the effects of this struggle: she has been hospitalized several times due to very low heart rate and she has osteoporosis (see Chapters 14 & 52). She has been attending a body image group as part of her PHP. One day at the start of the group, when members were invited to share and decode experiences of "feeling fat" (Friedman, 1993), Susan said with great distress that she felt "as big as a house." She did not know why she felt this way, and she could not tell the group anything that might help them understand her experience. She had not felt so big yesterday, or even earlier that day. Other girls in the group thought they knew something about what might be troubling her, so they offered observations. Susan accepted their help and together they were able to piece together the story. Susan had successfully taken what she saw as a big risk the day before by eating dinner at a fast-food restaurant, and she had planned with the dietician to take another big risk at dinner by including a "fattening" dessert on her menu.

Both of these challenges were acceptable to her, even though she was preoccupied with them. She and other members of the group agreed that at times they need a "push" to try new foods and to challenge their ideas about what is "safe" to eat. However, just prior to the group Susan learned she was expected to eat something at dinner that she felt took her beyond what she could tolerate. She believed she could not handle another challenge right then, would struggle at the meal, and was afraid her peers would be angry with her at dinner for making them stay late because they would have to "process" (Faith, Pinhas, Schmelefske, & Bryden, 2003). "Processing" occurs at the end of a meal where one or more patients do not complete the meal on time. Each of them writes answers to a short set of questions about what contributed to their being able/unable to complete the meal on time, what it felt like to complete their meal on time when someone else did not, and what they could do next time to help themselves and/or others. They then discuss their answers to these questions in the group.

As Susan's story unfolded, other young women in the group sympathized with her and expressed their dismay at what they saw as the unfairness of staff expectations. At the

suggestion of a group facilitator, they discussed what action they might take. They decided to bring the issue to the community meeting to raise the question of unfairness. They talked over who would speak to the issue at the meeting and who would take a more supportive role. Susan felt less distressed. She was surprised that her body felt smaller at the end of the group. The discussion had allowed her to identify her emotions in relation to her real situation, to express her fear of disconnection from her peers, to experience the empathy and support of her peers, and to protest what she felt was an unfair and oppressive expectation. She knew the protest would be taken seriously.

As it happened, the program dietician was requiring that Susan change her dinner option because she had chosen the lowest calorie dinner as a way of allowing herself to have the "fattening" dessert—bargaining with her ED, so to speak. When the principle behind the dietician's decision was discussed at community meeting, a girl who was close to graduating from the program volunteered to eat the same dessert to support Susan to eat her full meal at dinner, which she did.

At first, Susan's body was doing the talking for her: she was disconnected from her own emotions and said she felt as big as a house. Group members helped her make connections between her big-body experience and her situation while empathizing with her, which allowed her to reconnect with her emotions and with them. Susan was able to take one more step toward recovery. Such "growth in connection" is a key aspect of relational cultural feminism.

Socialist Feminism

Socialist feminism (Kitzinger & Perkins, 1993) sees women's oppression as part of systemic and intersecting oppressions related to gender, race, and class that benefit capitalist economies. Women are amongst those whose labor is exploited (not paid or not paid adequately) for the benefit of a White, male-dominated, profit-making class. Capitalist systems also benefit by encouraging insecurity about appearance in women, who can then be sold products to "correct" their appearances (see Chapter 29). Social conditions or problems are individualized, and often medicalized, such that people see themselves as having a problem or even an illness rather than seeing the source of their trouble in the culture and political economy.

This feminist perspective insists that so-called "psychological" issues must be socially situated, and therefore the individual should not be pathologized. This means that individual therapy must be used with great caution (i.e., not to help the individual adapt, but to be more conscious), and social action and social change are needed. Activating grassroots community-based support and groups, especially self-help groups, is preferred to individual therapy. In groups, a subculture can be created in which alternate values can be fostered, as in, for example, a community-based group where women support one another to eat well rather than diet, or to take care of other aspects of their health even when they are unwilling to eat. A contemporary example might be establishing an online pro-recovery or body size acceptance community. While socialist feminism is not interested in improving the practice of individual therapy, it influenced other feminisms to recognize the intersecting oppressions of race, class, and gender. It encourages women, including professional women, to be activists for social justice (see Chapters 43, 48, & 66).

Radical Feminism

This form of feminism also recognizes the intersecting oppressions of race, class, and gender, but radical feminism identifies patriarchy as the source of women's oppression, while highlighting women's bodies as the site of patriarchal control. Under traditional forms of patriarchy, women have literally been the property of men through marriage. Physical violence (or threat) is the ultimate form of patriarchal control. Sexual assault, abuse, and body-based harassment are expressions and extensions of the idea that males have, and are entitled to, rights over women's bodies (see Chapters 19 & 27). Radical feminists have been instrumental in extending sexual assault laws to protect women from rape within marriage, insisting on women's right to control their own bodies and therefore to make any and all decisions related to them, including whether or not to have children (Burstow 1992; Chesler 1972; Firestone 1970). Some radical feminists have advocated that women create separate lesbian communities (Rich, 1980) as the only way to live safely and fully independently of patriarchy.

In feminist therapy, as influenced by radical feminism, the concern is to not replicate practices that assert *power over* women and women's bodies. Fundamentally, women have a right to make decisions about their own bodies and to value their bodies apart from any appeal or use they have to men. For instance, a woman who has been sexually abused might find that not eating or that controlling her body size helped her re-establish some control over her life by numbing traumatic memories or by making the abuser lose interest (see Chapter 34). It is problematic on many levels if a doctor or therapist then takes *control over* her eating and body weight to "help" (force) her to "recover," as this is likely to be experienced as a revictimization (Burstow, 1992). Such concerns have fueled feminist criticism of medically-based ED treatments for their replication of an obsessive focus on food and weights (Gremillion, 2003), for taking control away from the woman (Brown, 1993, 2007), and for paying too little attention to the role of sexual abuse, sexual assault, and body-based harassment in the etiology of and recovery from EDs (Larkin, Rice & Russell, 1996; Wooley, 1994).

Case Example

Wendy

Wendy is a 16-year-old girl, the eldest of four children in a middle-class intact French family. She has had several inpatient admissions for a restrictive ED. After discharge, she rapidly lost weight and became medically unstable. Her frightened and discouraged parents brought her back to hospital each time. She was offered a place in a PHP, to support her not to lose weight immediately after discharge again.

During a group in which some other young women talked about bullying and abuse experiences, Wendy talked about an intensely humiliating experience she had in middle school when, during a menstrual period, blood had leaked through her clothing and became visible on her skirt. Some boys in her class noticed and loudly spread this "news" to her entire class. Wendy was mortified. Relentlessly, these boys referred to her as "Leaker"—a body-based harassment they used with impunity.

Wendy felt it was her body rather than the harassment that was the problem. She learned in health class that significant weight loss could stop menstruation, so she had dieted with the intention of making this happen. Understandably, as she gained weight

in treatment, she experienced and expressed increasing anxiety about her periods returning. She started having suicidal thoughts and self-harming urges, because, as she said, she would rather die than be humiliated again.

Some staff members' anxiety mirrored Wendy's and reflected patriarchal attitudes toward menstruation as disgusting. Conversation in the team rounds tended toward finding a way to stop her periods from coming so she could continue her progress with weight gain without self-harm or suicide, and possibly also to end the discussions of this "disgusting" subject. Some were attracted to the idea of using Depo Provera, because it would ensure that Wendy would never have to have periods.

An alternative formulation, reflecting a radical feminist perspective, was suggested. Wendy's experience could be recognized as a real trauma that was the result of body-based harassment by boys at her school. Such harassment is common (see Chapter 27). As Maine (2000) reports, the vast majority of girls have encountered harassment in school. This harassment is permitted and because it is permitted boys are reinforced in the idea that they have rights over women's bodies.

In addition, menstruation could be reframed as something to be valued, as part of respecting and valuing the female body—revaluing the female is also an aspect of cultural feminism. The team could thus reframe Wendy's experience as a trauma, for which she was not to blame, and from which she could recover fully. In support of this reframe, it was observed that in spite of her increasing and powerful anxiety, Wendy had not missed a single day of the program, had not refused any meals, and was not significantly restricting when at home on the weekends. Her weight was continuing to increase, bringing her closer to what she dreaded, but also to what she wanted, which was to get on with her life.

Thinking of Wendy's situation this way, the team became calmer. It was decided to offer her oral contraceptives, so that she would be able to predict when her first period would arrive. She was also offered an inpatient bed for those days so that she would have support not to self-harm or attempt suicide while she allowed her first period to arrive. Wendy accepted both of these offers, but did not use the inpatient bed. After her first period, her anxiety decreased markedly and she completed treatment.

Wendy started treatment thinking that her body was the cause of her humiliation. She was offered a perspective that identified her trauma as the real effect of patriarchal body-based harassment, and this perspective became the foundation of support to recover from this trauma. She was also offered a woman-positive perspective from which to relate to her body and menstruation, all key aspects of radical feminism.

Critical Feminist Theory and Postmodern Feminism

Postmodern feminism can be traced to Simone de Beauvoir's (1953/1989) *The Second Sex:* "One is not born a woman, but rather, becomes a woman" (p. 267). Beauvoir was saying that the position of women, the characteristics ascribed to them, the devaluing of the labor they are assigned, and the way they learn to see themselves as "less than," has nothing to do with what is "natural" to females or with any "essence" of femininity.

Postmodernism challenges key notions of modernism, including the ideas that there is a single, discoverable, and irrefutable reality; that inherent natural or essential characteristics

underlie and justify our binary categories, for example "one is either male or female" or "it is *natural* to be heterosexual and unnatural (perverted) to be homosexual"; and that the self is stable and enduring. Postmodernism asserts that there are many "truths," each of which is related to the standpoint of a narrator. That is, reality is socially constructed (these constructions serve the interests of some over those of others), even though we speak as if nature made things the way we construct them, for example, "it is natural for women to take care of children." In addition, there is no transcending stability of the self; "personality" is a construct and our identities are not fixed, implying that gender identity and sexuality are fluid.

Postmodern understandings of power emphasize that we are constrained not only by the concentrated "power over" us wielded by legislators and institutions, but through diffuse power created by the measurement and assessment of behavior that defines and redefines what is normal, acceptable, and deviant (Foucault, 1977/1995). We then become vigilant about the ways we or others might not be "normal," and in so doing effectively "police" ourselves and others. Critical feminist theory is another perspective that sees gender as one among several intersecting systems of oppression (race, gender, class, sexuality, ethnicity, size, and disability/ability; Davis, 2008) that maintain an economic system that privileges the few over the many. For any particular woman, gender may not be the most salient system of oppression in her life at a given time.

Piran and Teall (2012; see also Chapters 27 & 43) use a critical feminist and postmodern perspective in the development of the construct of embodiment, which they suggest better captures the complexity of the lived body experience of girls and women over time and social location than does the notion of "body image." Embodiment is seen on a continuum from embodied agency, care, and joy to disrupted embodiment and is comprised of three domains:

- **Physical freedom versus constraint/corseting.** This is a continuum that at one end represents freedom of movement, safety and care, comfort with appetites, desires, and age-related changes in the body (see Chapter 33). At the negative, unhealthy end is limited freedom of movement, becoming a target of violations of body ownership, restriction of appetites and desires, and disruption of comfort with age-related body changes.
- **Mental freedom versus constraint/corseting.** At one end of this continuum is "the freedom to explore and determine one's own sense of identity" (Piran & Teall, 2012, p. 186), whereas the other end represents being pressured by oppressive social discourses either to fit molds of femininity that may disrupt connection with one's body, or to be ostracized; for example, "tomboys" become "losers," "butches," or "manly girls."
- **Social power versus social disempowerment.** On this continuum one end represents experiences that reflect equity, freedom from marginalization, and the experience of respect and power within a community. The other end represents experiences that reflect inequity, marginalization, and lack of control and power within a community. One socially approved and strongly encouraged method girls use to change their status and acquire more social power is to change the appearance of their bodies.

The developmental theory of embodiment may explain, at least in part, why some but not all girls and women who live in the "same" culture develop disordered eating. They do not, in fact, live in the same culture. There are, rather, very different journeys of embodiment, some of which hold a greater burden of socially adverse experiences (Piran & Thompson, 2008) and consequently a more disrupted embodiment than do others.

In the following case example of "Tina," therapy with a young woman with an ED is discussed using critical feminist, postmodern, and developmental embodiment theories and a narrative therapy model. Briefly, narrative therapy from a feminist perspective works with dominant narratives such as those that characterize women as weak, emotional, and valuable primarily in relation to how they can support men; and whose bodies are valued to the extent they are attractive to men (Brown, 2007). In a highly collaborative way, narrative therapy uses the clients' own strengths, values, and unique outcomes to co-construct nonoppressive or alternative narratives that support their preferred identities (White, 2007).

Case Example

Tina

Tina is the 15-year-old daughter of a single mother and is of Aboriginal heritage. She is an outstanding hockey player and has been playing since she was 8 years old. When she talks about hockey, she lights up. It is clear she enjoys her power and strength, as well as the aggression and skill involved in skating fast and whipping the puck away from another player. Tina recently made it onto a team in a more competitive league. She has been bothered about "whether I'm good enough" ever since she overheard the White father of another player say that his daughter should get more ice time and could replace Tina, who he said was "a lazy player."

Tina found herself with less ice time. Wanting to improve her performance, she found nutrition advice on an Internet site for athletes. After several weeks of eating the prescribed foods, she found herself really hungry at the end of the day and binged on "junk food." Feeling "gross" and afraid of the consequences of eating this food, she made herself vomit. Within a few weeks she was binge eating and purging once or twice a week. She felt she'd discovered the perfect solution, but then her mom found her vomiting in the bathroom late one evening.

Tina's mother and grandmother brought her to an ED assessment, which resulted in a referral for outpatient family-based therapy (Le Grange 2011; Lock & Le Grange, 2012; see also Chapters 53 & 60). At the assessment and initial appointment, they were given information about the relationship between dieting and binge eating. Tina was relieved to hear it was not her fault, but rather that her body needed more food. If she would eat more and eat regularly, she would very likely stop having urges to binge. Tina didn't want to gain weight, but she did want to stop bingeing and purging, so she accepted her mother and grandmother's support and encouragement to complete her meals and snacks and not to purge. She did not talk about the conversation she had overheard between her teammate's father and her coach. Within 5 weeks, she was able to take charge of her own meals and snacks and needed successively less family support.

A few months later Tina lost some weight. She told the therapist that she'd been careful not to go long stretches without food, but she ate less overall. Her family was then enlisted to support Tina to have adequate food and, not long after, she was able to take control for food intake back again. At this point Tina described feeling "too masculine"; she wanted to be smaller and more feminine. Her mother said that Tina had talked about having a crush on a boy at school. In individual sessions that followed, Tina was invited

by the therapist to tell her more about her understandings of, and her feelings toward, "masculine" and "feminine." A few times at school, some boys standing at the top of the stairwell had called her a "butch." The therapist asked Tina how she felt as she was coming up the stairs. Tina said she was really annoyed by the way the boys stood there looking down and judging girls as they walked up the stairs. Usually, the boys said nothing to her, but this time they did. Taken by surprise and feeling shamed, Tina could think of nothing to say. She liked being with girls—playing hockey and hanging out, and did not feel a need for a boyfriend—did that make her lesbian? The thought panicked her and she found herself wondering if maybe something was "wrong with me." The therapist asked Tina if she noticed these boys calling other girls out similarly. She did, especially girls who didn't wear makeup or who dressed in loose or sporty clothes.

Maybe it would be better, Tina thought, just to act normal. But what was normal? Most girls in her grade tended to wear tight, revealing clothes and make-up. She had been feeling some pressure to change the way she dressed, to "look more like a girl" (Legge, 2011), but she did not want to be stared at and the clothes would be "binding, really uncomfortable." The therapist asked Tina to say more about the pressure she felt to "look like a girl." Tina replied that she had just noticed a lot of girls who used to dress in a more sporty way were now dressing in a "girly way." This resulted in more attention from boys, and that attention made them important. Some boys she had enjoyed playing hockey with in earlier grades did not even greet her in the hallway anymore. According to Tina, after the boys on the stairs called her a "butch," she "realized I was very masculine looking," and she thought she would look more feminine if she were smaller.

Although Tina did not identify as either heterosexual or not heterosexual, she seriously and painfully considered changing her body size so she would look more feminine and perhaps avoid being defined and condemned by this dichotomy. The therapist asked Tina to talk about the part of her that loves to play hockey. In answering, Tina reaffirmed that she was proud of her muscular strength and her physical power and did not want to lose these key aspects of her current identity (Nagasawa, 2013). Yet, another part of her was excited about the good things looking more feminine could bring, even to skillful athletes (Poplack, 2013; see also Chapter 35). She would feel less marginal at school. But Tina's "hockey-loving self" then felt angry and betrayed. For years hockey had provided her a sense of belonging, respect, and excitement, on her team if not at school. Without that Tina wanted to be accepted by her peers at school and longed to be part of the center of social life there. Tina's voice broke and she started crying. She told the therapist about what she'd heard her teammate's father say to the coach. She realized how much she had lost, leaving her old teammates and then playing so much less of each game with the new team. She didn't know whether it was worth it.

Over the course of several conversations, Tina was encouraged to talk about the racism inherent in the father's comment and to question the either/or framing of what she could be and do. She looked more closely at the lives of athletes she admired, including a member of her extended family, and saw there were women who were remarkable in sports that required strength and power and who also had families. She looked around her at school and in the broader community and noticed that not all boys and men had partners who were smaller than them. She was also aware of female athletes who had female partners. Breaking down stereotypes and not accepting the dominant narratives gave Tina a different picture of what could be possible for her. She talked to her mother and grandmother

about whether they wanted her to "get a boyfriend," and about her experience of racism with the new hockey team. She considered going back to her old hockey team, but with family support decided to stay on and persist in demonstrating her skills to the new coach, enacting a value she appreciated and learned from her grandmother. Tina decided to change the way she dressed for school to attract less "negative" attention, but without sexualizing her appearance. She thought she might also try wearing makeup. On the weekends she liked to wear the clothes in which she felt most able to move with ease. With these changes, Tina found a way to limit the constraints of the dominant narratives upon her. She was able to eat well again without direct family supervision.

Tina moved from more embodied agency to disrupted embodiment and then back to a somewhat more embodied agency (Piran & Teall, 2012). Molds of femininity, specifically the "girly girl" mold, influenced her sense of herself and her sexuality, pressuring her to make herself smaller and to consider wearing clothes she would not feel comfortable in, while body-based sexual harassment made her feel unsafe and sexually marginalized (Legge, 2011). The limited social power she felt as the daughter of a single parent with no White father to advocate for her made her look to changing her body to increase her social and physical power. Tina was supported in therapy to negotiate her dilemmas, challenging dominant and unrealistic dichotomies and oppressive narratives along the way.

Controversies

Feminist therapists working in the area of EDs have tended to leave responsibility for weight or other symptom change in the hands of the client. For example, Brown (2007) writes, "I began by telling Sarah [a client] that I wasn't going to engage in power struggles about her weight and eating—that she was in charge of this. I explained that we would not focus too much on weight and eating, but more on exploring the meaning they had for her" (p. 291). In this view, once the client is able to give voice to the meanings of her eating behaviors and weight control, she will be equipped to make choices and most likely she will choose not to restrict or binge and purge, because she will have found a more satisfying way of dealing with her situation; either way, it will be her choice. Burns (2004, quoted in Gremillion, 2009) says further: "the cessation of purging activities as a measure of wellness for women with bulimia is based on notions of normativity and functionality, that potentially have little to do with a woman's experience of well-being or her (possibly ongoing) negotiation with cultural messages concerning women's bodies" (p. 249). In other words, a woman might continue to restrict, binge eat, and/or purge, as part of her "ongoing negotiation with cultural messages" and thus we should see this as part of her own definition of well-being, rather than part of an illness.

It is understandable that as feminist therapists we have not wished to engage in, let alone replicate and reinforce, power struggles with women over their bodies. Nevertheless, EDs present difficulties for this hallmark feminist position. One is the sad and potentially fatal irony that they can render a person increasingly less able to exercise choice and control because of their real effects on body and brain. Children and teens in particular stand to lose out developmentally if their ED symptoms are allowed to persist. Growth in height, bone density,

and brain development can all be compromised (see Chapter 13). There is a limited window of time during which such growth occurs and once that time has passed, it will simply not happen. Therefore, while it is most appropriate to support developing autonomy in adolescents, it is a mistake to interpret the "voice" of the ED as the voice of autonomy; a protest against eating a meal or gaining weight might look like an expression of personal choice when it is actually an aspect of the ED. Not addressing it as such can unintentionally empower the ED rather than the person. Recent developments in feminist theory may support this view as nature is reintegrated with the social and cultural. As Gunnarsson (2013) writes: "a crucial aspect of our freedom, which is an essential goal of feminist politics, is to have our needs met. And to the extent that our needs are not particularly indeterminate but part of enduring natural structures, freedom involves submitting to forces that are both structured and beyond our control" (p. 10).

The urgency that arises from developmental and biological pressures could lead to the use of oppressive practices rather than collaborative ones, even when collaboration is safe. Feminist approaches can thus comprise a safeguard against this kind of "power over" mode and contribute much to respectful and ethical therapy practice. Some of the case examples above show how feminist approaches can be integrated with treatments that directly address weight and eating behaviors.

Conclusions and Future Directions

Feminism has a role to play in therapy for EDs, imparting skills that are empowering, revaluing what has been devalued, facilitating reparative connections in relationships, encouraging the voicing of silenced aspects of girls' experiences including harassment and trauma, and collaboratively understanding the impact of harmful and dominant narratives to make room for alternative stories and preferred identities. We don't know how to ensure a full and lasting recovery for everyone with an ED, but we do have evidence that adverse social experiences contribute to the development of disordered eating (Piran & Thompson, 2008; see also Chapters 19, 21, 27, 34, & 43). Bringing an awareness of how oppression affects girls and women over the lifespan (see Chapter 36) may improve our outcomes.

While feminist therapy does not easily lend itself to manualization and randomized control trials, there are at least two approaches described here that should be considered for research and/or treatment programs. Tantillo et al. (2013) argue that "disconnection" is a mediating process in the development and perpetuation of AN (see Chapter 33) and suggest a research program that could explore this. Piran and Teall's (2012) developmental theory of embodiment could provide the basis for establishing group work in treatment programs, replacing the "body image" work that is currently standard, with working toward "embodied agency."

Finally, it is a basic commitment of feminism to work toward social change. The toxic social, political, and economic structures that make women vulnerable to EDs appear to be expanding their influence across continents, genders, and age such that "all of our bodies are being mined for profit" (Orbach, 2013), and therefore the change we want is not "*a larger slice of the poisoned cake*" (Devaki Jain, cited in Krondorfer, 2008; emphasis added). It is, rather, a more fundamental transformation that has yet to be fully imagined and strategized.

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59

Psychopharmacology in the Treatment of Eating Disorders

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Notwithstanding the primacy of psychological treatments, pharmaceutical agents have an important role in the management of eating disorders (EDs). Those addressed in this chapter include psychotropic medications and weight management therapies (for information on replacement nutrients see Chapter 61). Empirical literature was identified using previously conducted broad-based literature searches of MEDLINE and Scopus databases by the authors up to September 2012 (Hay, 2013; Hay & Claudino, 2012). Psychotropic medications whose use in the treatment of an ED is supported by evidence for efficacy are summarized in Table 59.1.

Psychotropic Medications

Antidepressants

In addition to a role in treating comorbid depression, antidepressants appear to have a specific effect in appetite regulation in EDs characterized by recurrent binge eating, namely bulimia nervosa (BN) and binge eating disorder (BED; Goldstein, Wilson, Ascroft, & al-Banna, 1999). Systematic reviews and meta-analyses (Bacaltchuk & Hay, 2003; National Institute for Clinical Excellence [NICE], 2004; Reas & Grilo, 2008; Stefano, Bacaltchuk, Blay, & Appolinário, 2008; Vocks et al., 2010) have consistently found improved binge eating remission rates (by up to 70% in the short term) and reduced binge frequency when antidepressants have been compared with placebo in double-blind randomized controlled trials (RCTs) of BN and BED. However, when antidepressants are used without any concurrent psychosocial intervention the pooled abstinence rates for people with BN have been found to be better than rates for placebo (10%) but still less than 20% (Bacaltchuk & Hay, 2003).

Trials testing antidepressants in the treatment of BN have included tricyclics (TCAs), monoamine oxidase inhibitors (MAOIs), and selective serotonin (5-HT) reuptake inhibitors (SSRIs), in particular fluoxetine. Other SSRI antidepressants have relatively weaker evidence of efficacy in treating BN. Longer-term maintenance of change is also unclear. A frequently cited study by Agras et al. (1992) found that one third of the 25% of patients who were abstinent

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Table 59.1 Role and evidence base for psychotropic medications in treating eating disorders.

Class of Drugs	*Eating Disorder*	*Indication*	*Evidence Base*
Antidepressants	Bulimia nervosa	Reduction in binge eating, especially when combined with psychotherapy	Level I: tricyclics, monoamine oxidase inhibitors, SSRIs Level III: other antidepressant classes
		Relapse prevention	Level II: conflicting evidence, high attrition
	Binge eating disorder	Reduction in binge eating, especially when combined with psychotherapy	Level I: SSRIs Level II: atomoxetine, duloxetine Level III: other antidepressant classes
	Anorexia nervosa	Improved weight gain or relapse prevention	No robust and/or conflicting evidence
Antipsychotics	Bulimia nervosa	No indication	Not applicable
	Binge eating disorder	No indication	Not applicable
	Anorexia nervosa	Improved weight gain	Level I: inconsistent support Level II: olanzapine, amisulpride only
		Reduction in ED symptoms	Level I: not supportive
		Improved mood	Level I: mixed support
		Reduction in anxiety or ruminations	Level I: inconsistent support
Mood-stabilizing medication	Bulimia nervosa	Reduction in binge eating and purging	Level I: topiramate
		Improved health-related quality of life	Level II: topiramate
	Binge eating disorder	Reduction in binge eating	Level I: topiramate
		Improved weight loss in obese people	Level I: topiramate Level II: zonisamide but problematic adverse effects
	Anorexia nervosa	No indication	Not applicable

Note. Levels: I = systematic review of level II studies; II = one or more randomized controlled trials; III/IV = controlled (nonrandomized) study, uncontrolled trial or case-series (NHMRC, 2009).

at the end of treatment relapsed over time. Two relapse prevention studies (Fichter, Kruger, Rief, Holland, & Dohne 1996; Romano, Halmi, Sarkar, Koke, & Lee, 2002) have reported a positive effect of continuing pharmacotherapy in the treatment of BN. Romano et al. (2002), however, reported very high (around 90%) attrition at 12 months follow-up, limiting the interpretation of the impact of fluoxetine in preventing relapses. Dropout rates are also notably higher (up to nearly 40%) when a single drug is used compared to the 30% or less when the treatment is psychotherapy alone or when patients receive a combination of antidepressant and psychotherapy. Attrition is lower with SSRIs, due to their more benign side-effect profile.

In the treatment of BED investigators have tested SSRIs and TCAs against placebo. Two meta-analyses (which pooled seven SSRI trials) found antidepressants to produce remission of binge eating in about 40% of cases. Effects on mood were smaller and effects on weight reduction mixed (Reas & Grilo, 2008; Stefano et al., 2008).

Trials of newer antidepressants, including those that act to inhibit noradrenergic reuptake, have had positive results, although the studies have had small sample sizes. Bupropion (a noradrenergic and dopamine reuptake inhibitor) appreciably reduced binge eating and purging in one study of BN, but was associated with an unacceptable number of indiscriminate tonic-clonic seizures (Horne et al., 1988). Since the literature search date of September 2012 another study (double-blind RCT) testing bupropion in overweight women with BED has been reported. The drug was well tolerated and produced greater short-term weight loss (though very modest), but there was no evidence of positive effects on ED symptoms or depression (White & Grilo, 2013). An RCT of atomoxetine (a selective noradrenergic reuptake inhibitor) reported significantly reduced binge eating and weight in 40 binge-eating obese patients (McElroy, Guerdjikova, et al., 2007). Sibutramine (a noradrenergic and serotonergic reuptake inhibitor) showed promise in clinical trials for BED, but it has been withdrawn in most jurisdictions because of unacceptable adverse cardiovascular effects in obese persons.

Other open-label (i.e., not double-blind and placebo-controlled) studies or case reports (Bernardi & Pallanti, 2010; Leombruni, Lavagnino, Gastaldi, Vasile, & Fassino, 2009; Malhotra, King, Welge, Brusman-Lovins, & McElroy, 2002) have reported that newer dual noradrenergic and serotonergic reuptake inhibitors such as venlafaxine or duloxetine have positive effects on BED. One double-blind RCT with several follow-ups investigated the effects of duloxetine in obese individuals with BED and depression (Guerdjikova et al., 2012). Reduction of binge frequency and weight, as well as improved global clinical impression with regard to binge eating and depression, were confirmed in the primary, longitudinal analysis, but retention was under 70% (27/40). However, these positive findings decayed by the secondary endpoint and thus need replication in larger trials.

Currently, the recommended dose of fluoxetine in the treatment of BN is 60 mg/day (American Psychiatric Association, 2006), as lower doses have been found to be significantly less effective (Fluoxetine Bulimia Nervosa Collaborative Study Group, 1992; Goldstein, Wilson, Thomson, Potvin, & Rampey, 1995). A further analysis of these two studies by Sysko, Sha, Wang, Duan, and Walsh (2010) indicated that early therapeutic response (more than 60% reduction in binge eating or vomiting frequency in the first 3 weeks of treatment) was strongly predictive of later overall positive response to treatment.

Compared to psychotherapies such as cognitive-behavioral therapy (CBT; see Chapter 56), TCAs (Agras et al., 1992; Leitenberg et al., 1994; Mitchell et al., 1990) and SSRIs (Goldbloom et al., 1997; Jacobi, Dahme, & Dittman, 2002; Mitchell et al., 2001; Walsh et al., 1997) have in most studies of BN performed less well. Similar results have been found in RCTs of interventions for BED (Devlin et al., 2005; Grilo, Masheb, & Wilson, 2005; Molinari, Baruffi, Croci, Marchi, & Petroni, 2005). The body of research evaluating antidepressants in BN or BED thus supports their role as adjunctive to, rather than as an alternative to, psychological therapies, unless psychotherapy is not available.

In contrast to BN and BED, there have been several RCTs of antidepressants in anorexia nervosa (AN; Claudino et al., 2006), but these yield little support for their use. Four RCTs have addressed the effects of antidepressants during refeeding, while two have examined post-weight restoration. In only one RCT investigating refeeding was there a significant effect. Halmi, Eckert, LaDu, and Cohen (1986) found that the TCA amitryptyline or the antihistamine cyproheptadine reduced the time to achieve weight regain in the acute phase of the illness

(low weight). Kaye (2008) argued that the lack of efficacy for antidepressants in AN is due to alterations in 5-HT1A receptor activity due to starvation. The two maintenance trials reported conflicting outcomes. The second and larger trial of 93 patients randomized to the SSRI fluoxetine or placebo added to CBT did not find an advantage for fluoxetine in preventing relapse for 1 year (Walsh et al., 2006). Evidence is thus mixed and generally does not support a role for antidepressant use in AN, but more trials are needed.

Antipsychotics

The evidence base and use of antipsychotics in EDs are quite different from that of antidepressants. The only ED for which there is evidence pointing to utility is AN, in which the postulated mode of therapeutic action for the antipsychotic medication is in modulating serotonin and/or dopamine dysfunction (Kaye, 2008). Early trials of typical antipsychotics, such as chlorpromazine, pimozide, and sulpiride, reported inconsistent findings and problematic adverse effects. More recent trials of atypical antipsychotics, particularly olanzapine, have, however, yielded somewhat more promising outcomes.

There have been five RCTs to date of olanzapine, a 5-HT2/D2 receptor antagonist (i.e., although its effects on neurotransmission are complex and incompletely understood, olanzapine blocks serotonin [5-HT] and dopamine [D] neurotransmission at certain postsynaptic receptor sites). Mondraty et al. (2005) compared olanzapine to the typical antipsychotic chlorpromazine in a small ($n = 15$) nonblinded RCT. Their main finding was of reduced ED ruminations in those receiving olanzapine. A further trial of adjunctive treatment compared olanzapine to a placebo in the treatment of 30 outpatients, all of whom were receiving CBT (Brambrilla et al., 2007). Subgroup analysis revealed that only patients with the binge-purging type of AN had improved weight gain and decreased mood symptoms when receiving CBT plus olanzapine. In a placebo-controlled RCT of olanzapine added to a 10-week day hospital treatment (see Chapter 50), Bissada, Tasca, Barber, and Bradwejn (2008) also found those receiving olanzapine achieved weight regain more quickly and were more likely to reach a body mass index (BMI) greater than or equal to 18 (87.5% vs. 55.6%). However, over half of eligible participants declined to be randomized, indicating a low acceptance rate. A small preliminary outpatient RCT of olanzapine by Attia et al. (2011) also has reported improved rate of weight regain. However, another small RCT conducted by Kafantaris et al. (2011) not only did not support the adjunctive use of olanzapine in adolescents with AN engaged in an ED program, but also found increased fasting glucose and insulin levels at the end of the study in the drug group.

In addition to these RCTs of olanzapine, there is one RCT of the atypical antipsychotic amisulpride, which differs in action, having D2 and D3 (dopamine; see Chapter 30) presynaptic receptor antagonism at low dose and direct postsynaptic antagonism at high dose. Ruggiero et al. (2001) compared low-dose amisulpride to fluoxetine and clomipramine in 35 inpatients with AN. The investigators reported few differences but there was higher weight regain with amisulpride. Two RCTs of the atypical antipsychotic quetiapine, which is thought to work like olanzapine by blocking D2 and 5-HT2 receptors, have compared it to treatment as usual and to placebo, respectively (Court et al., 2010; Powers, Klabunde, & Kaye, 2012). Few advantages for adding quetiapine were found and it produced no improvements in weight regain. Recruitment in the second study was also problematic, adding to the perception that people with AN are disinclined to take medication with a known effect of weight gain. Risperidone, a medication that antagonizes the actions of many neurotransmitters, was also tested in a RCT (Hagman et al., 2011) that included adolescents (mean age 16 years). There was no consistent impact on weight gain or ED psychopathology.

Adding to this mixed evidence for the effectiveness of antipsychotics in the treatment of AN, two meta-analyses (Kishi, Kafantaris, Sunday, Sheridan, & Correll, 2012; Lebow, Sim, Erwin, & Murad, 2013) found no significant effects for atypical antipsychotics on weight regain or ED symptoms—and one meta-analysis published since our search date of September 2012 reported a possible worsening of anxiety along with improvement of mood (Lebow et al., 2013). Kishi et al. (2012) reported increased drowsiness and sedation with antipsychotics, but attrition rates did not differ between any one of the antipsychotic (olanzapine, pimozide, quetiapine or sulpiride) treated groups versus a placebo group. Moore, Watson, Harper, McCormack, and Nguyen (2013) also reported increased binge eating associated with atypical antipsychotic use. Finally, a third systematic review (Balestrieri, Oriani, Simoncini, & Bellantuono, 2013) also published since our search date, which focused on effects of psychotropic drugs in adolescent samples, similarly reported a lack of evidence for any positive effects. Despite these mixed effects and the caveats that accompany them, research reporting on clinical practice indicates that their use is common, in adolescent as well as adult samples, particularly as an adjunct to refeeding regimes in order to improve mood, reduce anxiety, and thus enhance weight gain (Powers et al., 2012; Moore et al., 2013).

Mood-Stabilizing Medications and Anticonvulsants

Apart from a largely forgotten (albeit positive) small RCT of lithium in the treatment of AN (Gross et al., 1981), the use of such mood-stabilizing agents (Goodwin & Malhi, 2007) or older anticonvulsants has been little studied in the treatment of people with EDs. However, there has been renewed interest in this group of drugs with the introduction of novel anticonvulsants, such as topiramate and zonisamide, that are postulated to reduce impulsivity and enhance weight management in overweight patients with binge eating.

An RCT conducted by Hoopes et al. (2003) found significant reductions in binge eating and purging in people with BN treated with topiramate. Furthermore, there was a small but significantly greater weight loss (1.8 kg [~4 lb]) in those in the topiramate arm versus those in the placebo arm who had an average increase of 0.2 kg (~0.4 lb). A second placebo-controlled RCT by Nickel et al. (2005) also found that topiramate significantly reduced binge eating and purging, as well as improved health-related quality of life, in participants with BN. Mean weight loss with topiramate was greater (3.8 kg [~8.4 lb]) compared with placebo in this trial, suggesting that risk of weight loss should be considered if topiramate is to be used in patients who suffer from BN and are in the lower range of normal weight. In addition, topiramate has notable adverse effects, particularly cognitive impairment and neurological symptoms (e.g., unusual sensations—paresthesia), and should not be used in the treatment of pregnant women with BN because it is potentially teratogenic.

There have been two placebo-controlled RCTs of topiramate in the treatment of obese patients with BED (McElroy et al., 2003; McElroy, Hudson, et al., 2007). Mean doses used were 213 mg/day and 300 mg/day, respectively, which are similar to the dose of 250 mg/day in the Nickel et al. (2005) BN trial. As in the BN studies, topiramate was associated with significantly reduced binge eating and weight reduction and improved general psychopathology. Similar problems with adverse effects and high attrition were found, however. It is also worth noting that there has been one RCT of the anticonvulsant zonisamide in 60 obese women with BED (McElroy et al., 2006). Despite positive results, attrition was high (50%) and side effects also problematic. The only double-blind RCT that tested the anticonvulsant lamotrigine in the treatment of obese individuals with BED found that, although this drug was well

tolerated, there was no evidence of efficacy in reducing ED symptoms or improving metabolic indices, and a small weight loss was observed (Guerdjikova et al., 2009).

Based on a meta-analysis ($n = 290$) of the three trials of the anticonvulsants topiramate and zonisamide that were reviewed above, Reas and Grilo (2008) concluded that the use of these medications caused positive and large significant reductions in binge eating and weight (relative risk [RR] for binge eating abstinence, given use of these anticonvulsants vs. placebo = 0.63; 95% CI [0.51–0.78]), and that the medications did not increase dropout rates. It is unclear what the future holds for anticonvulsants in the management of binge eating disorders such as BN and BED. Their use may be confined to those with more intractable symptoms where the benefits outweigh unwanted side effects. As is the case for antidepressants in the treatment of BN, use in combination with psychological therapy may be both efficacious and more acceptable to patients (Grilo, Masheb, & Wilson, 2005). In line with this, Claudino et al. (2007) have reported a positive outcome in a placebo-controlled RCT of topiramate added to CBT in the treatment of obese participants with BED.

Anxiolytic and Other Agents

There is no substantive evidence base for using anxiety-reducing (anxiolytic) medications such as benzodiazepines in the treatment of EDs. In a very small study ($n = 14$, with 11 completers) participants with AN were prescribed D-cycloserine, a glutamate partial agonist (i.e., it binds to and has some activating effect on glutamate receptors), as an adjunct to exposure therapy (Steinglass et al., 2007). The drug produced little positive benefit in increasing meal intake.

On the basis of the observation that EDs resemble addictive disorders and the knowledge that the endogenous opioid system is involved in eating behavior, there have been some studies of the effects of the opiate antagonist naltrexone in the treatment of AN and BN. Results have, however, not supported further use or investigation (Marrazzi, Bacon, & Kinzie, 1995; Mitchell et al., 1989). In line with this, a placebo-controlled pilot trial published since our search date, which tested ALKS-33, a compound that acts primarily as an antagonist of the μ opiod receptor, failed to produce positive effects in 62 obese individuals with BED during 6 weeks (McElroy et al., 2013).

Baclofen (a centrally acting GABA-B receptor agonist) and memantine (a noncompetitive NMDA receptor antagonist) have received some research interest but no RCTs have been done. Also, acamprosate, a drug with glutamatergic effects (antagonist of NMDA and other glutamatergic mGLUR5 receptors) has been tested in one double-blind RCT in individuals with BED, but findings from longitudinal analysis did not support its use, although some positive effects on binge days, craving, quality of life, and small weight loss were found in the secondary analysis (McElroy et al., 2011). In contrast, ondansetron, another agent used in treatment for addiction disorders because it acts as a serotonin 5-HT3 receptor antagonist, reduced binge eating and vomiting and improved eating patterns in one RCT of people with BN (Faris et al., 2000). In sum, so far there is insufficient evidence for the value of anxiolytic and anti-addiction agents in treating eating disorders.

Anti-Obesity Medications

Lipase is an enzyme involved in the processing of dietary lipids (e.g., fats, triglycerides) during digestion. Orlistat is used as an anti-obesity medication because it inhibits lipase. In patients with obesity and an ED, most commonly BED, orlistat has been found to significantly aid

weight diminution in two placebo-controlled trials, one combined with a reduced-calorie diet and one with guided self-help CBT (Golay et al., 2005; Grilo, Masheb, & Salant, 2005). In addition, both trials reported improved ED symptoms and significantly reduced binge eating in the orlistat groups compared to placebo. In contrast, another more recent double-blind RCT of the effects of orlistat combined with behavioral weight loss therapy (BWL) on economically and disadvantaged obese Latinos with ($n = 40$) or without BED ($n = 39$) and with psychiatric comorbidities did not find that this treatment was more effective than a placebo in reducing either weight or ED symptoms (Grilo & White, 2013). Thus, although there is support from initial trials that orlistat can be helpful and safely used in ED patients with comorbid obesity, further studies in populations commonly affected but usually underrepresented in trials of BED are needed to clarify generalization of these findings.

Finally, it should be noted that the high placebo response rate (around 40%) found in short-term pharmacological trials in BED have raised concerns about its significance and about the use of drugs to treat this disorder. A study that pooled data from 10 double-blind placebo-controlled pharmacological trials in BED (234 participants) identified 38% as responders (75% reduction in binge eating episodes) and 26% attaining binge cessation, with lower baseline binge frequency predicting placebo response in general (Blom et al., 2014). It is advisable that future trials include participants with more severe pathology in order to improve the quality and usefulness of the evidence for use of pharmacological agents in BED.

Conclusions and Future Directions

Research on pharmacological interventions for EDs provides evidence supporting the efficacy of some drugs or classes of drugs in reducing ED behaviors in both BN (antidepressants—mainly fluoxetine) and BED (antidepressants, topiramate, sibutramine, and orlistat). Whilst psychological interventions constitute the first line in ED, pharmacological agents have a useful role as adjuncts in treating EDs and in reducing other psychological symptoms such as anxiety. The only medication that could be recommended as a sole treatment is high-dose fluoxetine for BN, although patient preference and tolerability for it are less than for psychotherapy, and long-term maintenance of change is unclear. The evidence for use of medication is also based largely on trials in adults, though some adolescents were included in trials testing antidepressants for BN. As women are much more likely than men to have AN or BN (see Chapter 5), trials have also included much greater numbers of women. Although gender distribution is more similar in BED, drug trials have systematically included more females also (above 85% in most trials).

However, a clear understanding of the clinical impact of these findings and their generalizability is limited at the moment by many questions that remain unexplored. This is mainly due to the fact that trials investigating long-term clinical effects of interventions, both following short-term use and discontinuation, or when drugs are offered for longer periods of time, are seldom done. In addition, studies have often excluded participants who best represent the routine treatment seekers by limiting the participation of those suffering with comorbid physical (see Chapters 14 & 52) or psychiatric conditions (see Chapters 15 & 54) or by including volunteers responding to research advertisements, who may differ from many people with an ED in the degree of severity of their psychopathology.

Information on the effects of pharmacological treatments in males, adolescents, and different ethnic groups or cultural contexts is also restricted due to the small numbers of these participants in studies of drug treatments for EDs. Also, predictors of treatment response—both

patient and treatment factors—need to be more systematically investigated in trials that might gather such data as symptom profile, duration of illness, previous interventions, motivation to change, and other features.

There is also a need for more trials that examine the efficacy of SSRIs and other common antidepressants in the treatment of BN and BED. Early trials, such as that indicating the value of using a high dose of fluoxetine (60 mg/day) in treating BN (Fluoxetine Bulimia Nervosa Collaborative Study Group, 1992), require replication because at this dose adverse effects are more likely to be clinically problematic. Similarly, there is a need for larger trials evaluating the use of the atypical antipsychotics olanzapine and quetiapine in the treatment of AN, rather than continually rushing to conduct more small trials of the next "new" antipsychotic. As in other areas of research (Laws, 2013), perhaps more attention needs to be paid to the merits and importance of replication studies to build the evidence base in the psychopharmacology of EDs.

Although many medication trials have tested the same drugs/classes of drugs for BN and BED, it is still uncertain whether these disorders respond similarly to the same type of pharmacotherapy. A growing interest in understanding the complex neurobiology of eating/feeding behavior and weight regulation through neuroimaging (see Chapter 17) and preclinical research (e.g., animal models of binge eating) might help in clarifying differences and therefore in developing new specific pharmacological targets. So far, the research has supported a potential role of drugs that affect neurotransmitter systems (serotonin, norepinephrine, dopamine, opioids, and glutamate) and hormones or peptides (e.g., neuropeptide Y) involved in eating and weight control mechanisms, but it is expected that the ED field might further benefit from collaborative research with the obesity and the addictions field (McElroy, Guerdjikova, Mori, & O'Melia, 2012). With regard to the latter, though initial evidence from pharmacological studies does not support a clear role for anticraving drugs in treating EDs, growing biological research suggests that a common pathophysiology (involving the same brain reward circuitry) might be involved in drug addictions and food craving (and binge eating disorders) (Gearhardt et al., 2011; Van den Eynde et al., 2010), supporting further research in this area. In addition, as in the obesity field and other psychiatric areas (e.g., bipolar disorders), the testing of drug combination strategies that might enhance effects by acting on multiple targets deserves exploration in the field of EDs (Crow, Mitchell, Roerig, & Steffen, 2009; McElroy et al., 2012).

In regard to AN, besides some of the unanswered questions raised above for other EDs (e.g., effects of longer-term treatments or the impact of treatments on ED patients with different demographic or clinical characteristics), pharmacological research is even more challenging. Not only are there difficulties in recruiting adequate numbers and in managing the high rates of attrition in clinical trials, recent findings suggest that even if power is increased with larger studies, it might not be possible to find a substantial clinical effect of drugs such as antipsychotics (Kishi et al., 2012). Whether this will be confirmed or not in future trials, the question of what makes AN so resistant to so many pharmacological interventions is intriguing and deserves exploration. As Crow et al. (2009) discuss, is this resistance explained by both the acute impact of the starvation state and the long-term effects of the disorder on metabolism and neurotransmission, as well as the potential to affect brain development (synaptogenesis) during adolescence? And, what are the anatomical and physiological bases of the behavioral and psychological features (control of eating behavior, personality variables such as obsessionality and/or cognitive disturbances) that shape the development and/or maintenance of the disorder and thus need to be addressed (see Chapters 17 & 32)? We anticipate that further investigation of the neurobiology behind these basic alterations might lead to the identification of more adequate targets for biological interventions as pharmacotherapy.

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Family Approaches to Treatment

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This chapter provides an overview of the 40 years of research on family approaches to the treatment of eating disorders (EDs) in adolescent boys and girls. While structural family approaches and other family treatments are discussed in the context of the review of published studies, this chapter focuses on the most extensively studied family-based approach, Maudsley family therapy (see Chapter 53). This treatment is often also called the Maudsley Approach or Family-Based Treatment (FBT). We include a brief description of the FBT approach, a discussion of known mediators and moderators of treatment, and a discussion of future and ongoing research directions.

Historical Perspectives on the Role of the Family in EDs

Perspectives on the role of the family in eating disorders (EDs) have shifted considerably over the past two centuries. Beginning in the 1870s families were perceived to have a negative impact on ED treatment. In fact, in the 1940s the slang term "parentectomy" emerged in the ED treatment field, a term based on the widely held belief that it was essential to remove the negative influence of parents during the treatment of anorexia nervosa (AN) in order to achieve recovery. These attitudes persisted until the 1960s, when Minuchin and colleagues (Minuchin et al., 1975) at the Child Guidance Center in Philadelphia created a permanent shift in the conceptualization and treatment of AN. Their successful treatment program emphasized the modification of family dynamics in the "psychosomatic family," whose overprotective, rigid, conflict-avoidant, and enmeshed characteristics were understood as the unintentional context for the development and maintenance of AN.

In the 1980s and 1990s, researchers at the Institute of Psychiatry and the Maudsley Hospital in London (Dare & Eisler, 1997) shifted the focus toward the family disturbances that emerge when an ED is present *because of* the disorder. This approach used parents as a resource in treating their ill child while emphasizing the unhelpful and often paralyzing nature of blaming

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the family as a cause of the disorder. The subsequent research based on Maudsley family therapy has drastically changed both the landscape of ED treatment in adolescents and the way we conceptualize the role of the family in adolescent EDs.

Description of Maudsley Family Therapy for Adolescent EDs

Manuals for clinicians treating adolescent AN (FBT-AN; 1st ed.: Lock, Le Grange, Agras, & Dare, 2001; 2nd ed.: Lock & Le Grange, 2013) and adolescent BN (FBT-BN; Le Grange & Lock, 2007) provide a detailed description of Maudsley family-based therapy (FBT). Each manual includes a list of goals and suggested interventions for each treatment session, annotated session transcripts, and sections dedicated to troubleshooting for commonly encountered challenges. FBT typically consists of 15–20 outpatient sessions spaced over a 6–12-month period. Each session begins with a 5–10-minute check-in and weigh-in conducted with the patient alone. Then the therapist, typically a psychologist or clinical social worker, but in some cases a medical physician or psychiatrist, conducts a 45–50-minute session with the entire family. All family members with significant interaction with the adolescent are encouraged to participate in treatment.

Briefly, FBT consists of three phases of treatment. Phase 1 is focused on uniting parents to take temporary control of all food-related decisions in order to directly interrupt disordered eating behaviors, such as dietary restriction and weight loss (AN), or problematic eating patterns such as bingeing and purging (bulimia nervosa; BN). Phase 2 consists of transitioning control back to the adolescent as he or she is learning to make independent, developmentally appropriate choices about eating. This phase is initiated when parents feel confident their child can make healthy food-related decisions. This typically occurs when the AN patient reaches 90–95% of their ideal body weight (IBW) or when the BN patient's eating and binge-purge patterns have been normalized. Phase 3 consists of several sessions at the end of treatment that target areas of adolescent development that may have been derailed because of the ED, for example, peer and dating relationships. The need for adolescent individuation and reorganizing parental roles in the family are also commonly covered in Phase 3.

The therapist works with the family to reinforce the idea that neither the parents nor the adolescent are to blame for causing the disorder, so treatment does not focus on etiology. In general, the therapist works with the family as an "expert consultant," taking a nonauthoritarian stance in order to encourage and empower parents to take the lead in whatever ways are most effective in helping their adolescent overcome this disease. The initial focus is on behavior and symptom targeting. Other psychopathology (e.g., negative body image) is not addressed until eating is returned to near normal, weight is restored (in the case of AN), and binge-purge patterns have been disrupted (in the case of BN). Consequently, FBT emphasizes early parental control. Treatment is immediately focused on empowering parents to become united in taking complete control of food-related choices on a day-to-day basis for their child for a temporary period. Siblings are instructed to provide support for their ill brother or sister and to refrain from involvement in parental refeeding efforts. The purpose of these instructions is to reorganize family structure into a natural hierarchy with parents in charge and siblings as a subsystem.

Throughout this work the illness is externalized; metaphors and academic illustrations are used to demonstrate the ways the ED is separate from the adolescent and therefore, out of his or her control. For example, AN is often described in a similar manner to a cancerous tumor, such that food is analogous to the chemotherapy that is often required for a patient with cancer.

Thus, early intervention is very important, as is completing a full course of treatment. Externalization reduces blame and criticism (expressed emotion) that may be directed toward the adolescent, while creating a sense of urgency in the family's need to accept the belief that without parental intervention the disorder will not subside.

Some of these "essential" components of FBT-AN have also been studied empirically. Ellison and colleagues (2012) asked therapists providing FBT-AN to rate weekly the extent to which they adhered to five components of FBT that share significant overlap with the fundamental elements (e.g., increasing parental control, externalizing the illness) described above. Weight gain during treatment was significantly correlated with higher total adherence across all five FBT elements as well as each individual FBT component, with the exception of sibling support. Increasing parental control over refeeding was the strongest predictor of outcome and was also significantly associated with lower treatment dropout rates.

Empirical Support for Family Therapy in Adolescent EDs

The evidence base for family-based treatment of adolescent EDs includes uncontrolled case-series studies as well as randomized controlled trials (RCTs). It should be noted that there is no consensus regarding how best to define remission or recovery in adolescent EDs (see Chapter 64). A comprehensive review and commentary on this topic can be found in Couturier and Lock (2006).

Early Uncontrolled Studies of Family Therapy for AN

Early reports on the treatment of children and adolescents with AN were far from encouraging (Blitzer, Rollins, & Blackwell, 1961). This state of affairs changed when Minuchin et al. (1978) reported that their structural family therapy approach yielded recovery rates above 80% in a case-series trial of 53 adolescents with AN. Two similar case-series studies reported comparable long-term recovery rates when treating adolescents with acute AN using family systems therapy in combination with individual and/or inpatient treatment (Martin, 1985) or using family systems therapy alongside treatment from a pediatrician (Herscovici & Bay, 1996). Family systems therapy included exploration of developmental issues and facilitating conflict resolution.

A number of additional small case-series studies, such as that by Mayer (1994), reported on the success of Maudsley family therapy as a stand-alone treatment for adolescent AN. A larger case-series study (Stierlin & Weber, 1989) of AN reported conservative recovery rates at follow-up (~50%) but failed to report on differences between adolescent and adult patient responses. Several additional case-series studies of family therapy based on the Maudsley approach reported positive outcomes, with the majority of patients achieving recovery from AN or improvements in weight and/or ED symptoms (Lock & Le Grange, 2001; Wallin & Kronvall, 2002). These early uncontrolled trials demonstrated the feasibility of various family therapies and established the need for larger, RCTs to assess the specific advantages of family therapy.

Early Controlled Studies of Family Therapy for AN

To date, eight controlled trials of Maudsley family therapy or a variant of this approach for AN have been reported. To our knowledge, there have been no RCTs examining family systems therapy or structural family therapy for adolescent AN. The initial RCT was conducted at

Maudsley Hospital (Russell, Szmukler, Dare, & Eisler, 1987). Following inpatient weight restoration, adolescents and adults with AN or BN ($n = 84$) were randomized to receive 1 year of Maudsley family therapy or individual treatment that was supportive, educational, and problem-centered. At end of treatment, participants were divided into four subgroups based on diagnosis type, age, and length of illness. Participants in the AN subgroup with shorter length of illness (<3 years), and younger age (<18) had significantly better outcomes when treated with family therapy. Specifically, six of 10 patients in this subgroup who received family therapy achieved "good" post-treatment outcome according to Morgan–Russell criteria (Morgan & Hayward, 1988; see also Chapter 64), three achieved "intermediate" outcome, and one achieved "poor" outcome. In contrast, one of 11 patients in this subgroup who received individual therapy achieved "good" outcome, one achieved "intermediate" outcome, and nine achieved "poor" outcome. In the AN subgroup with longer length of illness (>3 years), and younger age (<18), participants did equally well in both therapies, as did participants with BN. Participants with AN who were older (>19) fared slightly better in individual therapy. A 5-year follow-up of this trial (Eisler et al., 1997) showed a similar pattern of results. These findings suggested that family therapy was best suited for adolescents with a shorter length of illness, so subsequent studies of family therapy have focused on this population.

Further studies at the Maudsley Hospital examined family therapy delivered on an outpatient basis with the whole family (conjoined format) or in separate sessions for parents and the adolescent (separated format). In a pilot RCT, adolescents ($n = 18$) with a short duration of AN (<3 years) received 6 months of either conjoined or separated family therapy. All participants in both conditions improved significantly on multiple criteria (Le Grange, Eisler, Dare, & Russell, 1992). In a subsequent RCT (Eisler et al., 2000), adolescents ($n = 40$) with a short duration of AN were stratified by levels of baseline family critical comments. Results supported the efficacy of both treatment formats, but separated family therapy was superior for families with high levels of maternal criticism. A 5-year follow-up (Eisler, Simic, Russell, & Dare, 2007) revealed no long-term difference in outcomes between conjoined and separated family therapy, although families high in pretreatment maternal criticism continued to demonstrate poorer outcomes in conjoined treatment.

The first RCT of family therapy outside of the United Kingdom was conducted in the United States. Robin et al. (1999) compared approximately 1 year of behavioral family systems therapy (BFST) with ego-oriented individual therapy (EOIT) among adolescents ($n = 37$) with a short duration (<12 months) of AN. Weekly EOIT sought to promote adolescent individuation and improve coping skills and included occasional collateral parent sessions. BFST was based on the Maudsley approach, included the whole family, and consisted of weekly treatment containing behavioral, cognitive, and family systems components. At post-treatment and 1-year follow-up, participants in BFST had superior body mass index (BMI) outcomes. The majority (80%) of participants in BFST had reached their target weight by 1-year follow-up, as compared to 68.8% in EOIT. Post-treatment, BFST (94%) was superior to EOIT (64.4%) in the proportion of participants who resumed or started menstruation, but the treatments were equivalent on this outcome by 1-year follow-up (92.9% in BFST vs. 80% in EOIT). Eating attitudes, depressive symptoms, ego functioning, and family conflict around eating were equivalently improved in both groups at post-treatment and follow-up.

In a Canadian study (Geist, Heinmaa, Stephens, Davis, & Katzman, 2000), 25 adolescents entering an inpatient ED treatment program for AN were randomized to receive 4 months of either family group psychoeducation (FGP) or family therapy (FT) closely resembling the Maudsley approach (eight 45-minute sessions of biweekly therapy attended by all family

members). FGP consisted of eight 90-minute biweekly sessions attended by multiple families. All participants received medical management of their ED and additional psychosocial treatments such as group and milieu therapy. Once participants were medically stable, they were discharged to an outpatient program in which FT or FGP sessions were continued. Post-treatment, participants in FT and FGP had increased percent body weight and reported reduced eating pathology, with no differences between groups.

Recent Controlled Studies of Manual-Based Family Therapy for AN

The first study using the FBT-AN manual was conducted at Stanford University (Lock, Agras, Bryson, & Kraemer, 2005). Adolescents with AN (n = 86) were randomized to either 10 sessions of outpatient FBT conducted over 6 months, or 20 sessions of FBT conducted over 12 months. There were no significant differences post-treatment in BMI outcome for short- versus long-term FBT, but participants in long-term FBT were significantly improved on the Eating Concerns subscale of the Eating Disorder Examination (EDE; see Chapter 38) and the Internalizing subscale (e.g., anxious, depressed, withdrawn) of the Child Behavior Checklist. However, analysis of a subset of these participants an average of 4 years post-treatment (Lock, Couturier, & Agras, 2006) revealed no differences between short- and long-term FBT in terms of BMI, EDE changes, need for additional treatment, and hospital admissions for medical problems related to AN.

A second RCT of FBT-AN was conducted at Stanford University and the University of Chicago (Lock et al., 2010). Adolescents (n = 121) with acute AN received 1 year of either FBT-AN or adolescent-focused individual therapy (AFT). FBT consisted of 24 one-hour sessions with the whole family. AFT was based on Robin et al.'s (1999) EOIT and consisted of thirty-two 45-minute individual sessions plus up to eight sessions with parents alone. AFT treatment included behavioral weight gain goals and focused initially on behavior change as well as on identifying, defining, and tolerating emotions rather than using starvation as a coping tool (see Chapter 57). Parent-only sessions assessed parental functioning and updated parents on patient progress. Results revealed no differences between FBT and AFT post-treatment in the number of participants who reached full remission (≥95% expected body weight and mean EDE global score within 1 SD of published means). However, a greater number of participants receiving FBT achieved full remission by 6- and 12-month follow-up. FBT was also superior to AFT in number of participants achieving partial remission, total BMI percentile increase, and reductions in eating-related psychopathology post-treatment; these differences were no longer present at follow-up.

A recently completed, large six-site study across the United States and Canada compared FBT and systemic family therapy (SyFT) for adolescents with AN (Agras et al., 2014). This study was well suited to address whether general involvement of parents (as in SyFT) is the key to success of treatment or whether the specific involvement of parents in refeeding (as in FBT) is what affects outcome. Results indicated that the two treatments promoted similar rates of remission from AN but FBT incurred lower treatment costs. The authors concluded that FBT remains the preferential treatment for adolescent AN.

The results from these eight RCTs highlight the success in treating adolescent AN with family therapy based on the Maudsley approach. For adolescents with a short length of illness, family therapy is more successful than supportive or ego-oriented individual treatments in restoring weight status during treatment and facilitating remission over follow-ups as long as 5 years. The specific effect of family therapy on changes in eating pathology and other psychosocial

outcomes is less clear, although it appears that family therapy is at least as effective as individual therapy in improving these outcomes. With some exceptions (e.g., in families with high levels of criticism), family therapy can be successfully implemented with parents alone or with the whole family together, and treatment can also be delivered in doses as short as 10 sessions. Despite these encouraging findings, much is still unknown about the effectiveness of this intervention for older adolescents, for individuals with a longer course of illness, and within inpatient settings.

Uncontrolled and Controlled Studies of Family Therapy for BN

As described above, the early RCT of family therapy conducted at the Maudsley Hospital indicated that participants with BN (n = 23) did equally well in family therapy or individual therapy both post-treatment and at 5-year follow-up (Eisler et al., 1997; Russell et al., 1987). Several case-series studies continued exploring family approaches with BN, driven in part by evidence that the binge eating/purging subtype of AN (AN-BP) responded favorably to family therapy, suggesting that parents are able to help their child both gain weight and normalize problematic eating patterns. An uncontrolled study conducted on eight adolescents with BN at the Maudsley Hospital demonstrated that a family therapy approach based on the treatment protocol for AN was promising (Dodge, Hodes, Eisler, & Dare, 1995). The majority of participants experienced significant improvement in bulimic behaviors post-treatment. A descriptive case report outlining development of a manual for FBT-BN (Le Grange, Lock, & Dymek, 2003) further highlighted the promise of FBT with adolescent BN.

Two RCTs of family therapy for BN have been reported. Le Grange, Crosby, Rathouz, and Leventhal (2007) randomized adolescents (n = 80) with full- or sub-threshold BN to 20 sessions of family therapy based on the FBT-BN manual, or 20 sessions of individual supportive psychotherapy (SPT) over a 6-month period. At post-treatment, a significantly greater percentage of participants in FBT-BN (39%) reported abstinence from bulimic behaviors in the past month, as compared to SPT (18%). At 6-month follow-up, a significantly greater percentage of participants in FBT-BN (29%) remained abstinent, as compared to SPT (10%). FBT-BN also elicited faster reductions in eating pathology by mid-treatment.

A second RCT of family therapy for adolescent BN (Schmidt et al., 2007) randomized adolescents (n = 85) with full- or sub-threshold BN to receive 6 months of family therapy based on the Maudsley treatment for AN, or cognitive-behavioral therapy guided self-care (CBT-GSC) adapted from a manual for adults. At post-treatment, patients who received CBT-GSC reported abstinence from binge eating in the past month to a greater degree than those who received FBT, although this difference was no longer significant at 6-month follow-up. No treatment differences were found with respect to vomiting or ED pathology at any time point, or when binge eating and purging were combined into one outcome. In a cost comparison, however, CBT-GSC was found to be lower in direct treatment costs compared to FBT.

These limited studies suggest that family therapy based in the Maudsley approach is likely beneficial in the treatment of adolescent BN. Family therapy is more successful than nonspecific individual therapy in promoting sustained abstinence from bulimic behaviors, and more immediate changes are observed in patients treated with family therapy. Family therapy appears to be at least equivalent to individual guided CBT in reducing bulimic psychopathology and other eating pathology over time. Although more research is needed, it is encouraging that parents are able to help their children manage bulimic behaviors, suggesting that Maudsley-based interventions are useful across the ED spectrum.

Mediators, Moderators, and Predictors of Outcome in Family Therapy

A few consistent findings across studies have increased our understanding of how family-based treatment works and elucidated for whom family therapy works best and why. Several studies suggest mechanisms of change that may be related to treatment outcome. The most robust findings point to the importance of early response to treatment. Specifically, three studies found that early weight gain in FBT-AN was related to positive outcomes (Doyle, Le Grange, Loeb, Doyle, & Crosby, 2010; Le Grange, Accurso, Lock, Agras, & Bryson, 2014; Lock et al., 2005). Early bulimic symptom reduction in FBT-BN may also predict treatment success (Le Grange, Doyle, Crosby, & Chen, 2008). Other potential mechanisms of change include improvement in behavioral problems and family relations during FBT-AN (Lock, Couturier, Bryson, & Agras, 2006) and improvement in ED pathology within FBT-BN (Lock, Le Grange, & Crosby, 2008). Yet some studies have failed to identify mediators of treatment outcome (Lock et al., 2010). Since research on mediators of treatment is in its early stages, more research is needed to better understand exactly how family therapy works.

Several studies have identified pretreatment moderators of specific treatment effects in family-based treatment. For example, AN patients with greater ED pathology, including individuals with AN-BP, may benefit more from FBT compared to individual treatment. Within the multisite study comparing FBT-AN to AFT (Lock et al., 2010), participants with greater eating-related obsessionality and ED pathology had higher remission rates post-treatment when treated with FBT as compared to individual therapy, and participants with AN-BP were more likely to be remitted during follow-up when treated with FBT (Le Grange, Lock, et al., 2012). Similarly, in Lock et al.'s (2005) comparison of short- and long-term FBT-AN, participants with greater eating-related obsessionality did better in long-term FBT. In contrast, BN patients with *lower* ED pathology may benefit more from FBT than individual treatment. In the RCT comparing FBT-BN to SPT, participants with less severe baseline ED pathology were more likely to be at least partially remitted at follow-up when treated with FBT (Le Grange, Crosby, & Lock, 2008).

Two studies assessed whether family structure (i.e., intact versus nonintact families) is related to treatment outcome. In the Stanford study comparing short- and long-term FBT-AN, nonintact families in longer treatment did better in terms of global EDE changes (Lock et al., 2005; Lock, Couturier, & Agras, 2006). In contrast, the study comparing FBT-BN to SPT found no differences in outcome at post-treatment or follow-up for intact versus single-parent families (Doyle, McLean, Washington, Hoste, & Le Grange, 2009). It appears that family structure may be most important to consider in the treatment of AN, where longer family therapy is more beneficial for divorced or single-parent families.

Research has also identified pretreatment variables that are related to outcome independent of the type of treatment received. The most consistently identified nonspecific predictors of remission following family-based treatment for adolescent AN are related to length and severity of illness. Participants who are older (Le Grange, Lock, et al., 2012; Lock, Couturier, Bryson, & Agras, 2006), have had prior treatment including hospitalizations (Eisler et al., 2000, 2007; Lock et al., 2010), and have a longer duration of illness (Eisler et al., 2000; Le Grange, Lock, et al., 2012) experience worse outcomes across studies. Greater baseline psychopathology is also related to poorer outcome for both AN and BN: ED pathology in BN (Le Grange, Crosby, & Lock, 2008); eating-related obsessionality in AN (Lock, Couturier, Bryson, & Agras, 2006); and comorbid psychiatric symptoms in both AN and BN (Eisler et al., 2000; Le Grange, Crosby, & Lock, 2008; Lock, Couturier, Bryson, & Agras, 2006). It is less clear how

baseline weight status in AN relates to outcome. Some research shows that lower pretreatment weight is linked to poorer outcomes (Eisler et al., 2000), whereas other studies have found that higher pretreatment weight is associated with a lower probability of achieving remission status through follow-up (Le Grange, Lock, et al., 2012). It is noteworthy that some studies (Le Grange, Crosby, & Lock, 2008) have not identified factors like age, duration of illness, and diagnosis type as predictors of outcome.

The Role of Expressed Emotion in Family Therapy

First discussed within the fields of schizophrenia and depression, expressed emotion (EE) refers to family member attitudes and behaviors toward an ill family member. Specifically, families high in EE display greater levels of critical comments, hostility, and emotional overinvolvement, as well as lower levels of positive remarks and warmth (Brown, Birley, & Wing, 1972). Although the relationship between EE and outcomes in family-based treatment for EDs has been extensively explored, findings are inconsistent and the role of EE in family therapy remains poorly understood. Some research has found that high levels of EE, particularly critical comments, are associated with treatment dropout and poorer outcome in family therapy for AN (Le Grange, Eisler, Dare, & Hodes, 1992; Lock, Couturier, Bryson, & Agras, 2006; Szmukler, Eisler, Russell, & Dare, 1985). One study pointed to the particular importance of critical comments that come from mothers (Eisler et al., 2000, 2007). Yet, other studies have failed to find associations between EE and outcome. The 5-year follow-up of the first Maudsley Hospital trial (Eisler et al., 1997) found no relationships between EE and type or outcome of treatment; this was also the case for the multisite study of FBT-AN versus AFT (Lock et al., 2010). Recent research suggests that families with AN have low overall levels of expressed emotion (EE) and that baseline warmth may be a better predictor of outcome (Le Grange, Hoste, Lock, & Bryson, 2011).

Not surprisingly, it is unclear what effect treatment itself has on EE within families. The initial Maudsley Hospital study comparing conjoined to separated family therapy found that critical comments from fathers and mothers increased in conjoined family therapy but decreased in separated family therapy (Le Grange, Eisler, Dare, & Russell, 1992). In contrast, the subsequent study of conjoined versus separated family therapy found that critical comments decreased in both forms of treatment, while warmth increased (Eisler et al., 2000, 2007). Robin, Siegel, and Moye (1995) reported similar results in a study of BFST compared to EOIT. Family conflicts and negative communication were reduced throughout both family and individual therapy. Further, compared to mothers in individual therapy, mothers in family therapy reported greater reductions in negative communication around eating and improvement in positive communication around eating (Robin et al., 1995).

Dissemination and Implementation of Family Therapy

Despite the growing evidence base supporting its use with both AN and BN, anecdotal evidence suggests that family-based treatment has not been widely adopted by clinicians. The relatively recent manualization of FBT-AN and FBT-BN facilitates implementation in clinical settings, and several dissemination studies based on these manuals are reviewed below. Several adaptations of family therapy for use in different clinical contexts are explored, and potential barriers to implementation of family therapy are discussed.

Dissemination of Family Therapy

Although most research on FBT for AN has been conducted with adolescents, two studies suggest that FBT is a viable treatment for children as young as 9 years old. An open case-series study examining FBT-AN provided in an outpatient university-based ED clinic (Le Grange, Binford, & Loeb, 2005) reported no differences in outcome between patients who were younger (ages 9–14) versus older (ages 15–18), those who received a greater dose of treatment (>20 sessions versus ≤ 20 sessions), or those who were treated by a senior therapist versus a trainee therapist. Another case-series study examined the use of manualized FBT-AN in younger children ages 9–13 (Lock, Le Grange, Forsberg, & Hewell, 2006). Using a comparison cohort of adolescents ages 13–18, results indicated that while EDE scores were lower in children both pretreatment and post-treatment compared to adolescents, the results of treatment were comparable. FBT appears to be an effective approach in a younger age range.

FBT-AN has also been implemented successfully in open trials with therapists outside of the Maudsley group or the developers of the FBT manuals. Studies have demonstrated the effectiveness of FBT for AN with adolescents in Brazil (Turkiewicz, Pinzon, Lock, & Fleitlich-Bilyk, 2010), Canada (Couturier, Isserlin, & Lock, 2010), Sweden (Paulson-Karlsson, Engström, & Nevonen, 2008), and the United States (Loeb et al., 2007). Couturier et al. (2010) also assessed treatment fidelity by coding clinician adherence to elements of treatment based on videotapes of sessions. Treatment fidelity was rated as "considerable" (rated at least a 5 out of 7) in 72% of Phase 1, 47% of Phase 2, and 54% of Phase 3. This same study also found that treatment was generally acceptable to adolescents and their parents. It appears that FBT can be successfully implemented with a variety of patients across multiple settings, although it should be noted that in each of these studies the therapists providing FBT were trained at the Maudsley Institute and supervised by either one of the manual developers or someone who trained with these individuals.

Adaptations to Family Therapy

Several research groups have started assessing the efficacy of family-based treatment delivered in modified formats, for example, in multifamily groups or when extra parent consultation is added to standard treatment. Researchers at the University of California at San Diego (Rockwell, Boutelle, Trunko, Jacobs, & Kaye, 2011) created a 5-day intensive family-based treatment program for adolescents with EDs. Program content was based on Maudsley family therapy but also included a range of other treatment strategies, including systemic family therapy, parent coaching, behavioral contracting, and training in coping and distress tolerance. There was also psychoeducation regarding the effects of malnutrition, as well as the short-term and long-term medical and psychological complications associated with EDs. Rockwell et al. (2011) report that, of 19 AN patients ages 10–18, all but one had achieved a sustained weight gain (as assessed by percent of IBW) post-treatment.

Two studies have assessed an enhanced parental component of family therapy. Rhodes, Brown, and Madden (2009) randomly assigned families of a child with AN to receive standard FBT-AN or FBT-AN with an added consultation session with parents who had recently completed FBT. The majority of parents receiving this consultation indicated it had distinct benefits (e.g., feeling less alone) above and beyond the positive effects of FBT-AN. A pilot study of an Internet-based parent support group tested the feasibility and acceptability of a weekly, structured, psychologist-facilitated online group to supplement FBT-AN (Binford Hopf,

Le Grange, Moessner, & Bauer, 2013). Parents who participated in the group reported satisfaction with the support program and indicated that it helped them to implement FBT and cope with their child's ED.

Barriers to Implementation of Family Therapy

A therapeutic alliance exists when the therapist and patient have common goals, trust each other, and feel comfortable working together. Studies using outside raters have found high ratings of therapeutic alliance in FBT. These studies also consistently find that higher ratings of therapeutic alliance are associated with better outcomes and lower treatment dropout (Forsberg et al., 2013; Isserlin & Couturier, 2012; Pereira, Lock, & Oggins, 2006). Research assessing therapeutic alliance via self-report by patients and parents also has generally found that family therapy generates a strong therapeutic alliance. In the study of essential components in FBT (Ellison et al., 2012) therapeutic alliance was rated by parents on a weekly basis. Mothers gave higher alliance ratings than fathers overall. Somewhat paradoxically, higher maternal alliance predicted greater weight gain and lower dropout, whereas higher paternal alliance predicted lesser weight gains. A study assessing adolescent-rated therapeutic alliance in one of the RCTs of family therapy for BN (Le Grange et al., 2007) found that therapeutic alliance and ratings of treatment expectations and suitability were similar in family and individual therapy (Zaitsoff, Doyle, Hoste, & Le Grange, 2008). There was no relationship between therapeutic alliance or treatment acceptability and outcome.

Despite these findings, therapists in training may perceive that therapeutic alliance is in jeopardy during FBT, particularly due to the challenge of interacting with multiple family members concurrently. To help understand potential barriers to implementation, a recent qualitative study assessed clinicians' perceptions of the strengths and weakness of FBT (Couturier et al., 2013). Therapists (n = 40) working with children and adolescents with AN had generally favorable views of FBT, but also reported significant barriers to implementing this intervention. Perceived advantages of family therapy included its clear structure, empirical support, long-term benefits, and outpatient nature, which helps avoid hospitalization. Specific advantageous intervention components were also identified, including reducing blame, increasing parental control, and focusing on refeeding. Reported barriers to treatment implementation included the amount of time required of families, the lack of attention to comorbid symptoms, the need for parental consistency, the lack of family meals in the real world, and the need for sibling involvement in therapy, which is typically recommended. Many therapists also noted the barriers of working without a dietician, conducting weekly weigh-ins, determining how best to utilize the family meal session with patients, and uncertainty about the level of support from their organization for incorporating FBT in their practice. Couturier et al.'s (2013) study is a first step toward addressing the challenges in disseminating and implementing family therapy on a wider scale.

Conclusions and Future Directions

Despite historically negative views regarding the role of the family in EDs, research on family therapy over the past 40 years has created a permanent shift to include families as an essential component of treatment for adolescent EDs. Family therapy has emerged as the most efficacious treatment to date for adolescent males and females diagnosed with AN. Studies

have shown that family therapy outperforms individual therapy for adolescents under age 18 with a short duration of illness (Eisler et al., 1997; Lock et al., 2010; Robin et al., 1999; Russell et al., 1987). Treatment is effective when delivered with children as young as age 9 (Le Grange et al., 2005; Lock, Le Grange, et al., 2006), in doses as short as 10 sessions (Lock et al., 2005; Lock, Couturier, & Agras, 2006), and is equally effective when provided in conjoined or separated format (Eisler et al., 2000, 2007; Le Grange, Eisler, Dare, & Russell, 1992). Although research on family therapy for BN is in its early stages, results from two randomized trials for BN suggest that families can successfully help to interrupt disordered eating behaviors and reduce bulimic symptoms during family therapy (Le Grange et al., 2007; Schmidt et al., 2007).

Several ongoing research studies will likely improve our understanding of FBT for EDs over the next 5 years. First, a recently completed multisite study at the University of Chicago and Stanford University compared FBT with CBT (see Chapter 56) and supportive psychotherapy in the treatment of adolescents with BN. Participants (n = 130) received 18 sessions of treatment over 6 months. One-year follow-up is underway and data analysis is pending. Second, investigators at Mount Sinai School of Medicine and University of Chicago recently completed a treatment development study of FBT for overweight adolescents and their families. Seventy-seven families participated in this study comparing FBT with a nutritional education curriculum; data analysis is pending. Third, an ongoing study in Melbourne, Australia, is reviving the question of the best format for the delivery of family therapy. This investigation is comparing regular FBT and parent-focused therapy (PFT) in a sample of adolescents with AN. A therapist applying PFT pursues the specific goals of traditional FBT but works only with the parents, not the whole family. In PFT the pediatric team medically monitors the adolescent (see Chapter 52), and appropriate referrals for psychiatric assessment are made when this is indicated (see Chapter 54). The main goal is to test whether a more streamlined approach to involving families in treatment is efficacious.

Several additional ongoing studies are attempting to refine the family therapy model for treatment of nonresponders. Another multisite study at the University of Chicago and Stanford University is comparing FBT for adolescents with AN to an enhanced version of FBT called "Adaptive FBT." This is a study of modifications to the FBT treatment for individuals who do not meet weight goals in the early stages of treatment. A second treatment development study involving the University of Chicago and a community ED program focuses on adolescents with AN who will receive a modified version of FBT that includes skills training (for parents and the patient) borrowed from the DBT literature (see Chapter 57). Based on the hypothesis that many families struggle in FBT because of poor distress tolerance in the patient, parents, or both, this study seeks to develop and evaluate the feasibility of an alternate version of FBT for this patient population. In addition to these studies, several ongoing studies at the University of Chicago and Stanford University are evaluating the feasibility of disseminating FBT to a wider therapist base by training therapists through in-person and online trainings. Additionally, a study at the University of Chicago is investigating the use of telemedicine (videoconferencing) to implement family-based treatment.

Although research on family therapy has flourished in the past few decades, ongoing research is needed to clarify key components of this treatment and treatment mechanisms. Future studies should also continue to explore issues related to dissemination, barriers to implementation, and adaptations for use of family therapy in other populations.

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Medical Nutrition Therapy for Eating Disorders

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Medical Nutrition Therapy (MNT) is widely recognized as an essential component in the treatment of eating disorders (EDs). Multiple professional groups have advocated MNT in the treatment of disordered eating and EDs, including but not limited to the American Psychiatric Association (2006), the Academy of Nutrition and Dietetics (American Dietetic Association, 2011), the Academy for Eating Disorders (n.d.), the National Eating Disorders Association (n.d.), and the United Kingdom's National Institute for Clinical Excellence (NICE, 2004). Guidelines for nutritional care are general in nature and do not define specific techniques or strategies, but specifically endorse MNT within the context of a multidisciplinary treatment team (see Chapter 50).

This chapter provides an overview of theoretical, scientific, and applied information related to nutrition therapy in the treatment of EDs. Readers will gain knowledge regarding the treatment environment, qualifications and therapeutic qualities of providers, and best practice strategies in the provision of nutrition therapy as part of a multidisciplinary approach.

Basic Elements of Medical Nutrition Therapy

Having a poor relationship with one's body and food is an integral component of EDs (see Chapters 2–4, 8–13, & 22). The registered dietician (RD) trained in working with serious mental illness and psychopathology provides technical, collaborative, and therapeutic expertise within the ED treatment team (Tholking et al., 2011). MNT generally applies to nutrition counseling provided by a RD, although by law Medicare (n.d.) also recognizes MNT provided by nutrition professionals who meet specific criteria.

MNT consists of a comprehensive nutritional assessment (see Table 61.1) and individualized care that addresses a host of factors including, but not limited to, individual preferences, food intake, physical activity (see Chapter 35), supplements, medications (see Chapter 59), medical treatment (see Chapters 14 & 52), and psychological therapies (see Chapters 51, 53, 54, 56–58, 60, 62, 63). MNT has been integrated into treatment guidelines for a number of diseases.

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Table 61.1 ABCDEs of nutrition assessment.

Anthropometric	Vital signs
	Body height
	Body weight
	Bone density
Behavioral	ED signs and symptoms
	Adaptive function of symptoms
	Executive function, traits—personality and temperament
	Anxiety and phobias
	Compulsions
	Obsessions
	Rituals
	Picky eating
	Mindfulness and interoceptive awareness
Clinical	Medical history
	Psychological history
	Dietary and weight history
	Physical exam
	Medications and supplement use
	Blood
	Urine
	Saliva
Diet	Quantity
	Quality
	Timing
	Tolerances and allergies
	Stimulants (e.g., caffeine)
	Laxatives (e.g., fiber and supplements)
	Hydration
Environmental	Culture (ethnic and sport)
	Family, friends, coaches
	Stressors

Note. Source of the category entries is Tholking et al. (2011).

The key components of MNT are nutrition diagnoses, intervention based upon a nutrition care plan, and ongoing monitoring and evaluation. The Academy of Nutrition and Dietetics standards of practice paper for RDs working with disordered eating and EDs (Tholking et al., 2011) serves as a resource for organizations and individuals seeking direction in maximizing the probability of safe and competent MNT by providers who meet professional practice and regulatory body standards. The Nutrition Care Process and Model (Writing Group of the Nutrition Care Process/Standardized Language Committee, 2008) defines the specific and consistent steps a dietetics professional uses when delivering MNT based upon the highest quality of information available for making practice decisions. These are guidelines, as the steps are not linear and approaches may vary depending upon the patient's dynamic needs, level of care, and care setting.

MNT for EDs occurs along a continuum of settings, with practitioners working in specialized medical settings, hospitals, partial hospitalization, and residential health settings, although the majority of this work is in outpatient settings. Provision of MNT ideally follows a progression from greater structure, support, and supervision toward more flexibility. RDs recognize

and address a host of factors that influence eating behaviors, including hunger and satiety. When and where food, nutrition, body weight, and/or shape or size are emphasized, the risk for disordered eating and EDs increases. For example, athletes (see Chapter 35), vegetarians, people with diabetes, models, actors, and health and fitness professionals are all considered to be at greater risk than the general population.

Patients with eating pathology experience nutritional deficits and other nutritional risks that progress if not resolved. The behaviors comprising disordered eating and EDs cause disruption in multiple facets of the digestive process from ingestion to absorption of nutrients. Inadequate intake places the body on alert to face potential "famine." Both over- and under-consumption patterns may affect availability of vitamins and minerals. Altered nutritional status affects cognition (Bourre, 2006; Markus, 2008; Sathyanarayana, Asha, Ramesh, & Jagannatha Rao, 2008), impacting patients' ability to engage in psychological treatment for the ED. Nutrition assessment is essential for identifying nutrient deficits and risks, and addresses them through empathic nutrition counseling. The goal is to improve both nutritional status and the patient's relationship with food and with his or her body, as well as supporting other treatment interventions for the ED (Tholking et al., 2011). The goal of MNT is ultimately to interrupt the dysfunctional relationship the patient has with food and to support a healthy state, both physically and emotionally, related to food intake and the body.

Nutritional Rehabilitation

How to advise another person on managing her or his eating has implications regarding the connection between you and that other person. According to Satter (2007, p. S145), it is "about trusting or controlling" and "about accepting or rejecting." Thus, the initial phase of MNT begins with establishing a trusting therapeutic relationship between the RD and patient or parent/caregiver.

Nutrition assessment by a RD begins with evaluation of the following: severity of nutritional compromise; body image, as understood within a sociocultural paradigm (see Chapter 21); sociocultural aspects of knowledge about food, as well as food acceptability, food availability and choice; and the level of resources, readiness, and ability to consume and tolerate improvements in nutritional intake. During the first phase of care, nutrient deficiencies are identified through diet history and biochemical assessment, and then corrected; consistent eating patterns are established as a foundation toward improvement of overall eating behavior and nutritional status. Challenges in nutritional rehabilitation differ, depending on the length of illness, dysfunctional behaviors, physical compromise, co-occurring medical issues, and individual patient circumstances.

Patients often fit into one of four categories at the onset of treatment:

1 Severe protein-calorie malnutrition with a need for weight restoration, whether or not the patient is engaging in compensatory behavior.
2 Protein-calorie malnutrition with a need for minimal weight restoration or with no weight restoration indicated—with/without compensatory behavior.
3 Reasonable macronutrient status and reasonable weight status but engaging in compensatory behaviors that interrupt access to nutrition.
4 Weight that has been driven above normal via excessive intake of energy relative to expenditure in the absence of compensatory behaviors.

The focus and priority of nutrition intervention will vary among these groups and will need to be adapted by the RD and treatment team as each individual case progresses.

Energy Requirements

Energy needs are discussed using a variety of terms. Total Energy Expenditure (TEE), also referred to as Estimated Energy Requirement (EER), is the predicted average dietary energy intake needed to maintain energy balance in healthy adults and children. Equations for predicting EER take into account age, gender, weight, height, and level of physical activity (Woodruff, Hanning, & Barr, 2009). However, EER equations estimating caloric need may be drastically different than the actual caloric intake that supports healthy weight maintenance among patients presenting for assessment. This difference can be explained by either *adaptation,* which is defined by the Institute of Medicine (IOM, 2005) as a change in energy balance over an extended period of time that allows "maintenance of essentially unchanged functional capacity" in spite of some "altered steady-state conditions" (p. 149), or *accommodation*, which applies to "maintenance of adequate functional capacity under altered steady-state conditions" (p. 149), typically a short-term adjustment. Human survival in diverse environments is supported by physiological changes in response to complex feedback mechanisms. Shifts in energy intake or energy expenditure trigger a metabolic switch in which hormonal and nervous system responses maintain body weight, although in a compromised state (IOM, 2005).

Resting Energy Expenditure (REE), also referred to as Resting Metabolic Rate (RMR), is often measured or calculated using predictive equations. Resting energy needs reflect the largest component of TEE/EER, and represent the amount of energy needed to maintain bodily functions when the body is at rest. Activity factors are used to arrive at TEE/EER from REE/RMR. Once a patient has been assessed, recommendations for feeding are established. Currently, no formula exists for calculating resting metabolic rate (RMR) in ED patients. Standard equations used to calculate RMR in hospitalized patients are poor predictors of energy requirements for the ED population (Cuerda et al., 2007).

Indirect calorimetry is the most accurate way to measure RMR among ED individuals (Haugen, Chan, & Li, 2007; Schebendach et al., 1995), however, it is not commonly used in practice. Indirect calorimeters are costly and not very portable. Handheld calorimeters have several limitations. Research using handheld equipment is limited to healthy ambulatory participants (Van Loan, 2007), and no studies have used ED participants (Reiter & Graves, 2010). Furthermore, handheld equipment uses a constant for the respiratory quotient (RQ), a comparison of oxygen inspired and carbon dioxide exhaled, and ED behavior, such as purging, affects both components of RQ. This means that the expected respiratory compensation for ED behaviors would not be detected with a fixed RQ, potentially compromising accuracy. In addition, the measurement of metabolic requirement may reinforce patients' overconcern with energy balance and thereby unwittingly reinforce patients' obsessive measurement and monitoring of their body. As a result, many clinicians adapt standard equations to estimate energy expenditure. This estimate is then combined with diet history, clinical skill, and experience to determine initial energy need and to begin the process of developing and monitoring the feeding/eating plan to achieve treatment goals.

Multiple factors must be considered when determining energy recommendations for ED patients. For example, among patients who have restricted nutritional intake, the length of illness, degree of energy deprivation, and malnutrition are significant factors. Aggressive feeding in these patients, who are likely to have marked metabolic downregulation in response

to sustained negative energy balance, increases the risk of refeeding syndrome (RS). It is important to remember that RS may occur among individuals who appear to be of reasonable weight status. RS, which affects multiple body processes, is characterized by hypophosphatemia, hypokalemia (low potassium), hypomagnesaemia, hypocalcemia, fluid retention, and thiamine deficiency (Korbonitis, Blaine, Elia, & Powel-Tuck, 2007; O'Connor & Goldin, 2011; Solomon & Kirby, 1990).

Although RS occurs commonly in the early stages of nutritional rehabilitation and individual risk cannot be predicted in advance, intense monitoring and early intervention allow safe management (Gaudiani, Sabel, Mascolo, & Mehler, 2012; see also Chapter 52). Traditionally, severely malnourished patients are started on 30–40 kcal/kg (126–167 kJ/kg; American Psychiatric Association, 2006); for a 100-pound (45-kg) patient this is approximately 1350–1800 kcal. Establishment of consistent intake while limiting risk has been the first priority, with weight recovery second. Once the window of risk (1 to 3 weeks in most cases) has passed, the pace of feeding becomes more aggressive, with weight recovery taking priority (Cockfield & Philpot, 2009). Balancing energy intake, nutrient balance, and supplementation with needed micronutrients is necessary to safely alter the metabolic state from catabolic (tissue breakdown to release energy) to anabolic (growth and development based on adequate reserves).

Cautious feeding is often standard, but some recent research suggests that a more rapid pace of correcting nutritional intake is appropriate for adolescents (Garber, Michihata, Hetnal, Shafer, & Moscicki, 2012; Whitelaw, Gilbertson, Lam, & Sawyer, 2010). Ideally, patients at higher risk for RS would be treated using multidisciplinary medical care, including careful monitoring, by clinicians experienced in ED treatment (Gaudiani et al., 2012). However, many high-risk patients are currently managed in an outpatient setting for a variety of reasons, including patient refusal to obtain a higher level of care, or lack of access, including financial hardship.

Once metabolic safety, confirmed by biochemical assessment, is established, feeding can be advanced to achieve a desired pace of nutritional recovery. Rate of weight restoration is generally 2–3 lb (0.91–1.36 kg) per week for patients in higher levels of care, and 0.5–1 lb (0.23–0.45 kg) per week in the outpatient setting (American Psychiatric Association, 2006; NICE, 2004). In anorexia nervosa (AN), energy requirements may be higher than expected, with some patients requiring up to 70–100 kcal (293–418 kJ)/kg/day (American Psychiatric Association, 2006); for a 100-pound (45.4-kg) patient, this translates to 3150–4500 kcal (13,180–18,828 kJ)/day. Possible explanations for the increased need for energy include the increased thermic effect of food; challenges with gastrointestinal absorption; and recovery-sabotaging behaviors by patients (e.g., compensatory exercise, discarding food, or dishonesty in reporting food consumption) due to loss of the ED as a means of life management and to fear of weight gain (Rigaud, Brondel, Poupard, Talonneau, & Brun, 2007).

With normal-weight or overweight patients, the initial focus of meal planning is healthful eating behavior, weight stabilization, and balanced nutrition. Research has not yielded consistent findings regarding energy requirements for patients with bulimia nervosa (BN) or binge eating disorder (BED; de Zwaan, Aslam, & Mitchell, 2002), whereas weight-restored patients with AN have higher energy needs than control participants (Weltzin, Fernstrom, Hansen, McConaha, & Kaye, 1991), increasing the challenge of achieving weight maintenance. RDs bring scientific knowledge of food, nutrition, physiology, growth, and development, as well as counseling skills, to the process of counseling and supporting ED patients as they work to (a) establish appropriate energy intake to meet nutrient needs, and (b) set healthy boundaries around eating and nutrition-related behaviors. In addition, RDs provide psychoeducation to

assist patients in developing a deeper understanding of energy balance and of their relationship with food and physical activity.

Route of Feeding

Nutritional intake may occur via several routes. Oral feeding with whole foods involves chewing, swallowing, and digesting. Enteral nutrition, also called tube feeding, provides a liquid formula via a tube to the stomach or intestine, bypassing the need to chew and swallow. Parenteral nutrition (also called total parenteral nutrition, or TPN) involves feeding a person intravenously, bypassing digestion and gastrointestinal absorption. Oral feeding using whole foods is considered the first choice for nutritional rehabilitation (American Psychiatric Association, 2006; NICE, 2004). The oral route has a lower risk of RS and allows a more straightforward challenge to ED thoughts and behaviors. Often, however, tube feeding is used as a secondary choice or as a supplemental feeding route. Some research suggests that supplemental use of tube feeding in both AN and BN may lead to superior improvements in abstinence from ED behavior, and in lean body mass, mood, and quality of life (Rigaud, Brayer, Roblot, Brindisi, & Verges, 2011; Rigaud, Brondel, et al., 2007). Parenteral nutrition support is considered a last resort due to the risk of infection and RS complication. TPN may also inadvertently allow avoidance of the challenges of eating and delay psychological healing from the ED; it is generally reserved as a last resort in the treatment of severe AN.

Macronutrient Needs

At this time there is no clear scientific protocol for manipulation of either macronutrients (carbohydrates, proteins, or fats) or micronutrients (vitamins and minerals) specific to the treatment of EDs. For aggressive treatment of young patients with AN, feeding less than 40% of energy from carbohydrates, prophylactic use of an oral phosphorus supplement, and utilization of nocturnal tube feeding may be beneficial and minimize RS risk (Kohn, Madden, & Clarke, 2011). Manipulation of macronutrients often presents practical challenges, due to patients' fear of dietary fat. Patients' distortions regarding the quality of food tend to follow fad-dieting trends and often involve specific energy nutrients, carbohydrate, protein, or fat. Meal planning establishes a baseline of adequate intake and a template for attaining overall nutrient balance among food groups. Nutritional manipulation occurs with input from the interdisciplinary team regarding physiological (e.g., RS risk) and psychosocial readiness (e.g., emotional tolerance of challenge, cultural acceptability, and economic or environmental barriers to food access). Because carbohydrate provides for energy needs over the course of the day, nutrition education emphasizes carbohydrate-rich foods as "fuel for the brain," "power for muscles," and a vehicle for fiber and phytochemicals (e.g., antioxidants). Over time, the goal is to lessen dietary rigidity and increase flexibility in overall eating.

Although most patients are aware of the importance of protein intake for the provision of amino acids for lean tissue synthesis and repair, many are less aware of the importance of protein in other metabolic processes. The concept of maintaining overall dietary adequacy and appropriate carbohydrate and fat intake in order to spare amino acids for such functions is useful in helping patients achieve a balanced distribution of energy-producing nutrients in early recovery. For example, inclusion of adequate dietary protein helps establish sustained energy as well as satiation (Lakhan & Vieira, 2008). Consistent energy availability and satiety after eating can be especially helpful to patients struggling with binge eating or those fearful of a loss of control when eating.

Discussion of the need for adequate energy coupled with protein intake to allow bioavailability of precursor amino acids and micronutrients for neurotransmitter production can also be useful in challenging distortions regarding nutritional needs, especially among those with a complicating mood disorder. Evidence for the value of specific amino acid supplementation in the treatment of mood disorder is inconclusive at this point (Lakhan & Vieira, 2008). Nevertheless, the role of nutrition in neurotransmitter production is well established, and evidence clearly supports the proposition that depression is better managed in nourished individuals (Layman, 2009; Markus, 2008). In nutrition education and counseling, the roles of protein may be presented as that of a "bridge" supporting satiety signals, as well as a "connector" for building neurotransmitters to achieve harmony in energizing and calming messengers.

Dietary fat is essential to a balanced eating plan. In addition to providing essential fatty acids and vitamins, fat provides a protein-sparing energy source, limits the respiratory load for severely malnourished patients, and contributes to food palatability and satiety. In contrast to characteristic patient convictions about dietary fat as a trigger for loss of control over both eating and body weight, dieticians often refer to dietary fat as an "antibinge" nutrient when educating patients about the role of fat in satiation and quality of diet. Furthermore, fat also plays a role as an "antidepression" nutrient, with lower rates of depression associated with higher intakes of omega-3 fatty acids (Freeman et al., 2006). Many ED patients severely restrict dietary fat either by overt food avoidance or use of modified foods (fat-free and reduced-fat foods). While improvement in fat intake is a priority in nutritional rehabilitation, pace of change is balanced with the potential for early satiety and readiness for change.

Micronutrients

Frank micronutrient deficiency occurs less than expected in the ED population, but ED behaviors do limit availability of vitamins and minerals (Hadigan, Anderson, & Miller, 2000). Consequently, although adequate oral intake of vitamins and minerals via food is ultimately preferred, it is routine practice for patients to be supplemented with a complete vitamin/mineral preparation during treatment. Specifically, laboratory evaluation is typically followed by therapeutic supplementation of thiamine, folic acid, vitamin B12, zinc, vitamin D, magnesium, phosphorus, iron, and calcium. The role of B vitamin insufficiency in mood disorder (Ross, 2007) further supports supplementation with vitamin B12, niacin, and folic acid. Deficiencies in zinc and folic acid may persist past the rehabilitation phase of treatment (Castro, Deulofeu, Gila, Puig, & Toro, 2004), warranting supplementation over a longer period of time. Some research suggests that 5-methyltetrahydrofolate may be preferable to other forms of folates, due to lower risk of adverse effects and enhanced bioavailability among patients with a genetic polymorphism that reduces the ability to methylate folate to this centrally active metabolite (Papakostas, Cassiello, & Iovieno, 2012).

Bone health is a significant treatment issue for the ED population (Mehler & MacKenzie, 2009). As a result, many patients are routinely evaluated for bone density and vitamin D status, with subsequent dietary recommendation to protect bone mass. While calcium should be supplemented if dietary intake is not sufficient, supplementation may need to be delayed for severely malnourished patients early in nutritional rehabilitation. Calcium binding to phosphorus reduces phosphorus availability necessary for adenosine triphosphate (ATP) production, thus increasing the risk for RS. Vitamin D supplementation should be administered if indicated by a serum 25-hydroxyvitamin D assessment; definitive thresholds for serum vitamin D are uncertain, but optimal concentration thresholds are proposed to fall in the range of 40–70 ng/ml (Larson-Meyer & Willis, 2010). Vitamin D increases bone health,

immune function, inflammatory modulation, and muscle function, while reducing risk for chronic and autoimmune disorders and cancer (Larson-Meyer & Willis, 2010).

Meal Planning

As noted above, evidence-based guidelines for feeding patients with EDs are lacking. In general, healthful eating behavior must be developed, reinforced, and practiced throughout the course of treatment, regardless of level of care. MNT addresses nutritional adequacy (energy and nutrient intake), variety within and between food groups, desensitization to "fear" of "bad" foods that have been avoided or used to binge and purge, and normalization of eating behavior. Planning is a natural part of healthful eating, so meal plans assist patients in establishing consistent intake, while providing for essential nutrition and satiety. These plans also provide a guideline for developing a consistent pattern of eating that allows the patient to progress in ways that foster health, reduce fear, and build confidence about eating. Plans vary from simple to highly structured and may include a variety of self-monitoring strategies to increase awareness and accountability. Food journals or dietary recall may be used as a means of assessing intake relative to environmental triggers and to ED-related thoughts, urges, or behaviors.

Two common foundations for establishing meal plans are *MyPlate* and the *Exchange Lists for Meal Planning*. *MyPlate* was established by the U.S. Department of Agriculture (n.d.) as a web-based set of guidelines for improving eating habits within the general public. Eating is guided utilizing a plate concept to include foods from the major categories of fruit, vegetables, grains, protein, and dairy. The *Exchange Lists* were developed as a joint effort between the American Diabetes Association and the Academy of Nutrition and Dietetics (Krebs-Smith & Kris-Etherton, 2007). Foods are categorized (e.g., fruit, vegetable, starches, meat, milk, and fat), such that one can "exchange," or select, foods from within each group to develop variety and flexibility, while maintaining basic macronutrient balance. Patients are provided a guideline for what food category/exchanges to include at each meal/snack. *MyPlate* and the *Exchange Lists* both have limitations, as neither was intended for ED treatment. One potentially problematic limitation is that when used literally, as originally written, both systems can increase dietary restraint and rigidity, especially for foods containing fat and sugars. RDs must be aware of the limitations of meal planning and be prepared to update plans throughout the treatment process, altering MNT according to coordination of care by the interdisciplinary treatment team.

RDs working as part of a treatment team along the spectrum of care employ differing strategies to guide the patient through the nutritional rehabilitation process. Desire to eat is influenced by biological/homeostatic need and hedonic (relating to preference, liking, or reward) drives. Monotonous diets, typical of ED individuals, especially those with AN, result in a reduced hedonic value. As hedonic value of food decreases, intake of that food generally decreases (Hetherington, Foster, Newman, Anderson, & Norton, 2006). Dietary variety (altered flavor, color, texture, temperature, and shape) enhances food intake at a meal by as much as 40% when successive courses are involved (Hetherington et al., 2006). Thus, improvement in dietary variety is essential in decreasing dietary restraint and improving flexibility in eating patterns.

Patients often begin with a more structured approach to eating and then are encouraged to move toward more variety and flexibility throughout the course of treatment. In this process, the RD provides guidance regarding timing and portioning of foods for meals and snacks, and dosages for supplements. While meal plans are useful to patients who will benefit from a sense of safety in the therapeutic relationship with a dietetics professional, meal plans (external regulation) may also be problematic in terms of discouraging recognition and response to internal

signals/regulation. The experienced RD will assess the patient for the type of meal plan (e.g., highly structured to more flexible) that best serves the individual over the course of treatment.

For caregivers engaged in family-based therapy (FBT) for EDs (see Chapters 53 & 60), MNT is not provided directly to the patient. Instead, RDs may provide consultation to support parents/caregivers, assisting them to develop increased confidence in their ability to effect change in the nutritional status and eating behavior of the ED child or adolescent. The goals are to provide (in an empathic, confident, and authoritative fashion) psychoeducation about nutrition and to empower the family in supporting their child's food intake regarding what, when, and how much. When appropriate developmentally and medically, the RD will assist in the transition of control of eating so that parents steer their child toward a more mindful eating approach without inadvertently giving permission to return to ED-related behaviors and thought processes. Mindfulness supports greater flexibility in food choice and the ability to recognize and utilize internal cues of hunger and satiety to regulate eating (see Chapter 33). Currently there is no empirical guide for determining when, or to what extent, a patient is ready to proceed toward exploration of a mindful eating approach guided by internal cues. This means that RDs must use utilize clinical experience and trial and error in their guidance.

Nutritional rehabilitation frequently involves the introduction of foods and beverages of higher caloric density to meet energy needs. Not only is this true for those suffering with AN, but also it is often necessary for treatment of BN, BED, and eating disorder not otherwise specified (ED-NOS) in order to restore metabolic rate following metabolic adaptation or accommodation. Some patients with BN and ED-NOS (now either Other Specified Feeding or Eating Disorder, or Unspecified Feeding and Eating Disorder; see Chapters 4 & 11) who are within their "healthy" body weight range as predicted from BMI, may be under their optimal weight based upon genetic endowment and muscle mass (e.g., if they are athletes), negatively impacting cognitive and physical function. Evidence suggests that dietary energy density score (DEDS) predicts the success of treatment among recently weight-restored patients (Schebendach et al., 2008; Schebendach, Mayer, Devlin, Attia, & Walsh, 2012); patients who continue to avoid calorie-dense dietary fat and calorie-containing beverages are more likely to have a poor long-term outcome. Providing less selection to patients early in treatment for AN may improve rates of weight restoration without enhancing overconcern with eating, weight, and shape (Leacy & Cane, 2012).

Supervised and therapeutic meals are an opportunity to fully assess a patient's relationship with food and to work with the patient to develop normalized eating behavior. Other benefits of supervised, therapeutic meals include the opportunity to challenge limited variety, verify the patient's report of intake, observe eating, explore recognition of hunger and satiety, and desensitize food- and eating-related anxieties using experiential eating activities and graduated exposure to distressing contact with food and eating environments. Effective meal therapy requires considerable preparation, including development of a clear plan for each meal event, identification of specific goals, anticipation of challenges, and processing of the event after the meal. The decision to talk about food, or not, during the meal should be part of the plan.

Weight Goals

Establishing a weight restoration goal is one of the most challenging areas of nutritional rehabilitation for EDs. Although outcome research has yet to provide an empirically supported guide for target weight, recent research suggests that, for patients who enter intensive treatment, two problems predict the need to return to an intensive level of care: failure to

address weight recovery early in the treatment process (Lund et al., 2009), and failure to complete nutritional rehabilitation, including weight restoration to a functional weight with time to stabilize weight (Kaplan et al., 2009; Meguerditchian et al., 2009).

The RD is often the treatment team member responsible for assessment and recommendation of weight range goals. Goals should be based upon family history, the patient's growth and development, personal weight history, and functional status, both biologically and psychologically. Biologically, patients need to achieve a weight that supports normal function of all body processes (e.g., reproductive function, endocrine function, and normal laboratory values) and that limits bone loss and other longer-term medical complications of the ED (see Chapters 14 & 52). Pelvic ultrasound may be used to evaluate reproductive function in female patients (Mason, Key, Allan, & Lask, 2006).

Psychologically, patients need to be capable of adequate cognitive processing to make use of psychotherapeutic interventions. As previously mentioned, some patients may appear normal by societal standards but be underweight for their biological heritage, leading to significant loss of function. Healthy weight is highly variable, and weight alone is a very limited predictor of overall health. Consequently, weight goals should be reassessed throughout treatment to ensure that all domains of function are addressed. The treatment team needs to develop consensus supporting a team recommendation and thereby establishing a unity and consistency in the weight restoration process (Purtillo & Doherty, 2011). Failure to reach and maintain this consensus may contribute to distrust in treatment, conscious or unconscious "splitting" of team members by the patient seeking a lower goal weight, and increase in the risk of the ED interfering with treatment progress, leading to an unproductive lengthening of care.

Therapeutic Role of the RD

While the technical and biochemical aspects of nutritional care are recognized as essential to ED treatment, the therapeutic role of the RD within the treatment team is also very important. J. Lichota (personal communication, April 21, 2013) aptly summarized the RD's role:

> Dietitians must provide much more than just nutritional rehabilitation and nutrition education. As a member of the treatment team, they work to both dispel the ED and to support patients in their quest to find what they are truly searching for, a sense of security and a trusting, connected relationship. Resistance should not be interpreted as a lack of desire to get well, but rather as a sign of the strength of the relationship the individual has developed with their ED, providing comfort, companionship, safety, trust, predictability, control and even love. The RD is in the role that most directly challenges this relationship; the interactions in the RD-patient relationship are at times the most important.

Patients value being treated by providers who are considered experts (Gulliksen et al., 2012). Because the RD is considered the expert in food, body weight, energy balance, and metabolism, patients rely on the RD to understand their concerns regarding body dissatisfaction, fear of fat, and so forth. RDs who are trained and experienced in ED treatment are judicious in discussing information on caloric value, metabolic rates, body weights, body composition, and so forth; they have learned to skillfully move the patient's focus in directions that are consistent with the patient's personal values as deemed constructive to recovery (Gulliksen et al., 2012; Nordbø, Espeset, Gulliksen, Skårderud, & Holte, 2006). The RD provides authority regarding

food, nutrition, and readiness/appropriateness of physical activity. MNT enhances feelings of confidence and safety for patients who feel insecure about change in their beliefs and behaviors related to food and physical activity.

RDs at various levels of treatment may assist patients in meal planning and preparation skills. RDs trained in EDs also have counseling skills to discuss ED behaviors and urges to engage in destructive behaviors, and to direct patients to get support around feelings rather than using the behaviors to numb/self-soothe. RDs support the work of psychotherapists who address the internal and relational pieces, reinforcing behavioral changes to be more sustainable. MNT for EDs mixes directives with empathic, supportive statements that both challenge and encourage through consistent confirmation of the patient's ability to develop and use skills. The RD trained in ED treatment is familiar with various therapeutic modalities and thus reinforces efforts by other members of the treatment team through close collaboration. Essentially, the RD in a coordinated treatment team supports recovery from the inside out, recreating and reinforcing the relationship with emotions, body, and food.

It is a widely held belief that people "can and should control their body weight and shape" (see Chapter 21) and that an ED simply represents either a strong commitment to such endeavors or a helpless abandonment of them. Other mental health disorders are not characterized by such attitudes toward illness (Roehrig & McLean, 2010). The RD is in a position to address these societal attitudes, but in the process will be personally challenged by patients' direct and indirect comments regarding the RD's appearance. The RD working with ED patients needs a strong personal foundation to navigate the increased awareness and concern about food, eating, and compliance with cultural ideals (Warren, Crowley, Olivardia, & Schoen, 2009).

Moving Toward "Normal" Eating

Once a patient is physically healthy and has established a baseline pattern of eating without ED behaviors, the focus of the MNT shifts to stabilization and normalization of weight and eating patterns. This is accomplished in a number of steps to support adequacy of intake and prevent weight loss or gain that is unhealthy for that individual. MNT toward normalized eating assists the patient in moving away from the meal plan (externally managed eating) and toward healthy attitudes and behaviors that utilize internal signals to regulate eating. Specifically, MNT for AN and BN emphasizes developing increased flexibility and comfort within eating patterns and expanding the repertoire of selected foods so that the patient can eat in a variety of settings. Development of consistent and adequate eating patterns that include flexible but reasonable limitations, and establishment of strategies to build skills in managing trigger foods are the focus of MNT for BED.

"Normal eating" is often confused with what is "normative" in a society or culture. Satter's (2007) definitions of normal eating and eating competence have raised awareness about the disruption and threat to health caused by restricted feeding, restrained and/or disinhibited eating, unreliable availability of food, overconcern with body image, and lack of attunement with hunger, satiety, and appetite (see Chapters 18 & 33). One tenet of normal eating is to apply mindfulness to structured and planned eating episodes. MNT includes assessment and development of mindfulness skills relative to eating. When mindfulness is deficient, greater external structure (e.g., meal planning and mealtime support) is needed to maintain progress. Patients are encouraged and helped to continue to explore social eating and family support systems needed for recovery.

Thus, MNT for EDs generally progresses through three phases. The shift from a directed, highly structured meal plan (phase 1) to greater flexibility (phase 2), and then optimal eating (phase 3) is akin to going from the strong external structure of a "cast" to "rehabilitation," and finally to "synergy." Nutritional recovery is similar to progressing from use of crutches to support recovery from a physical injury, to rehabilitation (including painful change), and then to full potential. The role of the RD shifts from directive to more collaborative, with the patient increasing her or his flexibility in food acceptance, experimentation with food environments, and greater decision-making. Finally, if and when the patient reaches the third phase, we see a dramatic paradigm shift to a patient-inspired drive toward personal growth and optimal nutrition, with the RD as ally. Full nutritional recovery takes time. As in physical therapy once an injury is healed, continued reliance on crutches (the meal plan) would become an obstacle to normal life. Patients who achieve full recovery will have healthy attitudes and behaviors around food, moving beyond reliance on external guidance, and achieving "vitality" for continuation of a meaningful and purposeful existence, where the time and attention placed on food and eating are only one piece of a healthy, balanced life.

Providing MNT Along a Continuum of Care

Nutrition therapists participate in behavioral interventions, and apply exposure therapy, response prevention, and cognitive restructuring around food, eating, and body image, all along the continuum of care. Most experts agree that MNT should be delivered at the least restrictive level of care possible based upon a patient's severity of illness and medical stability at presentation for treatment (American Psychiatric Association, 2006). Lack of progress warrants progression toward a higher level of care. Preparation of the patient for matriculation from one level of care to another has not been discussed in the nutrition literature; however, our experience indicates that this subject deserves great attention, as ED sufferers often struggle with set-shifting and are challenged to trust care providers. Nutrition therapists must attend to the transition process to provide high-quality care.

Conclusions and Future Directions

Eating disorders are recognized as biologically based (Klump, Bulik, Kaye, Treasure, & Tyson, 2009). It stands to reason, then, that nutrition as a biochemical factor plays a major role in the onset, severity, and duration of mental states that commonly accompany EDs, such as anxiety and depression. It also stands to reason that RDs play a major role in the assessment, planning, psychoeducation, and behavioral interventions that are part of multidimensional treatment for EDs.

While knowledge of nutrition in athletic performance, health, and physical illness is quite well developed, scientific consensus is lacking regarding the role of nutrition in mental illness. Nutritional neuroscience is a growing field that is generating new understanding of factors that affect the signaling molecules involved with cognition, emotion, behavior, and stress-induced psychological disorders (Gautam et al., 2012; Sathyanarayana et al., 2008). Further research is needed regarding the roles that food, nutrition, nutritional supplements, eating, and disordered eating play in human biology, and particularly in psychological disorders and metabolic disease. This information will allow improvements in the assessment of individual needs and in making individualized dietary recommendations based upon nutritional factors

relevant in brain signaling pathways. Further investigations of homeostatic and hedonic mechanisms that regulate eating are also needed to help in crafting of more effective nutritional therapies for EDs.

In the meantime, nutrition professionals will continue to play an important role in the continuum of care for people of all ages who suffer from EDs. At this time, resources for treatment, including insurance reimbursement for MNT, may be limited. Therefore, nutrition professionals are needed to educate the public and professionals, and to influence policy toward enhanced coverage of MNT for ED.

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Interpersonal Psychotherapy in the Treatment of Eating Disorders

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Interpersonal psychotherapy (IPT) is an evidence-based, time-limited therapy that focuses on improving an individual's interpersonal functioning by relating psychiatric symptoms to interpersonal problem areas and then developing strategies for dealing with those domains (Freeman & Gil, 2004; Klerman, Weissman, Rounsaville, & Chevron, 1984). The aim of IPT is to improve interpersonal functioning within four social domains: grief, role transitions, interpersonal role disputes, and interpersonal deficits. As these domains are the focus of IPT, they are sometimes referred to as focal areas. A basic principle of IPT is that development and maintenance of some psychiatric illnesses occurs in a social and interpersonal context such that onset, response to treatment, and outcomes are influenced by significant interpersonal relations between patients and others. The overall goal of IPT is thus to identify and change the maladaptive interpersonal context in which the psychiatric problem is developed and maintained and to reduce or eliminate psychiatric symptoms by improving the quality of the patients' current interpersonal relationships and social functioning.

IPT originated as a standardized form of interpersonally oriented psychotherapy for use in treatment outcome research on depression (Klerman et al., 1984). IPT was modified in the late 1980s for patients with bulimia nervosa (BN; Fairburn et al., 1991; Fairburn, Jones, Peveler, Hope, & O'Connor, 1993) and then adapted into a group format for individuals with binge eating disorder (BED; Wilfley et al., 1993, 1998, 2000, 2002). Randomized controlled trials (RCTs) with sufficient follow-up periods have demonstrated that IPT is an effective treatment for both BN and BED.

This chapter will review interpersonal theory and how it provided the foundation for IPT. The central role that interpersonal functioning plays in the development and manifestation of eating disorders (EDs) will also be discussed. This chapter will then review empirical evidence supporting the efficacy of IPT for the treatment of EDs, as well as explain the delivery of IPT for EDs with a description of the major tenets of the treatment. We will also outline a novel

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adaptation of IPT for the prevention of excess weight gain and, finally, propose future directions for this treatment.

History and Theoretical Issues

Interpersonal psychotherapy is grounded in theories developed by Adolf Meyer, Harry Stack Sullivan, and John Bowlby, each of which recognizes interpersonal function as a critical component of psychological adjustment and overall well-being. In the late 1950s, Meyer postulated that psychopathology was rooted in maladjustment to one's social environment (Frank & Spanier, 1995; Klerman et al., 1984; Meyer, 1957). Sullivan similarly theorized that individuals could not be understood in isolation from their interpersonal relationships; enduring patterns in these relationships could either encourage self-esteem or result in psychopathology. IPT is also associated with Bowlby's (1982) work emphasizing the importance of early attachment for later development of relationships and emotional well-being. IPT incorporates aspects of these theories and acknowledges a two-way relationship between social functioning and psychopathology: disturbances in social roles can serve as antecedents for psychopathology and, conversely, mental illness can produce impairments in an individual's ability to perform social roles (Bowlby, 1982).

Interpersonal Functioning and Eating Disorders

The interpersonal model of IPT asserts that social problems create an environment in which ED symptoms are developed and maintained. As a result, patients with EDs experience unfulfilling social relationships and/or become socially isolated, thereby increasing negative affect, a potent antecedent to binge eating (Wolfe, Baker, Smith, & Kelly-Weeder, 2009) and to interpersonal problems (Rieger et al., 2010). IPT addresses these social deficits by helping patients to identify patterns of binge eating as a coping strategy for dealing with negative affect and interpersonal stressors and to improve interpersonal skills in ways that promote self-esteem and improve negative affect.

IPT has been highly efficacious for patients with BN and BED (Rieger et al., 2010). Patients with BN and BED commonly report adverse social and family experiences and interpersonal stressors. These patients often experience loneliness, low perceived social support, poor self-esteem, and difficulties in social situations (Fairburn et al., 1998; Fairburn, Welch, Doll, Davies, & O'Connor, 1997). IPT is particularly well suited to address these problem areas. IPT has been evaluated for patients with anorexia nervosa (AN) in comparison to cognitive-behavioral therapy (CBT; see Chapter 56) and specialist supportive clinical management (SSCM; Carter et al., 2011; McIntosh et al., 2005), though no specialty treatment has demonstrated superiority for adult patients with AN (Watson & Bulik, 2013).

Empirical Evidence for the Efficacy of Interpersonal Psychotherapy

Bulimia Nervosa

As noted, IPT is an efficacious treatment for individuals with BN (see Chapters 3 & 9). In fact, IPT is the only psychological intervention that has demonstrated long-term results equivalent to CBT (Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000; Wilson & Shafran, 2005), which is

currently the most well-documented efficacious treatment for BN (Wilson, Grilo, & Vitousek, 2007). IPT for EDs was originally adapted from IPT for depression for use as an alternative psychological treatment in comparison to CBT (Fairburn et al., 1993). IPT was selected as an active comparator to CBT because IPT matched CBT in terms of "nonspecific" therapeutic factors associated with psychological interventions but contrasted with CBT in that IPT focused on patients' current interpersonal functioning and did not include behavioral and cognitive procedures to address specific ED symptoms (Fairburn et al., 1993). Initial trials comparing CBT and IPT for BN demonstrated similar short- and long-term treatment effects (Fairburn et al., 1993, 1995). Specifically, in a trial comparing focal IPT to CBT and behavior therapy among 75 female adults, patients receiving focal IPT and CBT had significantly greater abstinence rates (i.e., no binge eating or purge episodes) at 6-year follow-up (52% and 50%, respectively) compared to those who received behavior therapy (18%; Fairburn et al., 1993). A subsequent multisite study was conducted with 220 adult patients with BN (Agras et al., 2000). Results demonstrated differential time courses for CBT and IPT in reducing binge eating among patients with BN. Specifically, CBT outperformed IPT in the short term (at end of treatment, 29% of patients who received CBT were recovered compared to 6% of patients who received IPT), but at 12-month follow-up, the two treatments did not significantly differ in their outcomes (28% of the CBT condition were recovered and 17% of the IPT condition were recovered). These results suggest that both treatments are efficacious for improving binge eating among patients with BN.

It has been noted, however, that delivery of IPT for BN in this type of comparative outcome research has lacked core features associated with treatment delivery, which may have attenuated treatment effects. For example, ED symptoms were not discussed and therefore not routinely linked to the interpersonal context. In addition, use of an extended assessment reduced the amount of time over treatment to work on change (Rieger et al., 2010). IPT for BN has since been modified for delivery in clinical practice (IPT-BNm; Arcelus et al., 2009) and subsequently condensed into a brief, 10-session format (IPT-BN10), given the demand for time-limited therapies (Arcelus, Whight, Brewin, & McGrain, 2012). Results of pilot testing suggest these treatment adaptations may be feasible and warrant additional study.

Moderators of IPT treatment effects have been identified. African American participants experience greater reductions in binge eating from IPT compared to CBT (Chui, Safer, Bryson, Agras, & Wilson, 2007). It may be that the personalized nature of IPT is acceptable to various cultural groups and backgrounds (Chui et al., 2007), but further research is needed to evaluate the effects of IPT among different racial and ethnic groups (see Chapters 16, 23, & 25). Individuals with high expectations for improvement also show positive outcomes in CBT and IPT (Constantino, Arnow, Blasey, & Agras, 2005). Finally, lower initial levels of psychosocial functioning have been associated with greater relapse among women with BN (Keel, Dorer, Franko, Jackson, & Herzog, 2005), which may explain the long-term benefits of IPT for BN, given the treatment's focus on interpersonal functioning.

Binge Eating Disorder

Given the initial efficacy of IPT for BN (Fairburn et al., 1991), Wilfley and colleagues adapted IPT for BED (see Chapters 4 & 10) using a group format (Wilfley et al., 1993, 2000). Patients with BED tend to self-stigmatize and experience shame from their ED behaviors, which patients often keep hidden from others. The group format can help patients to create a "live" social network, thus reducing isolation while encouraging the development of new relationships and serving as a model for relationships outside of therapy (Waldron, Tanofsky-Kraff, & Wilfley, 2013; Wilfley, Frank, Welch, Spurrell, & Rounsaville, 1998). To test the efficacy of

IPT for BED, group IPT was compared with group CBT and with a waitlist control condition in a RCT with 56 female adult women with BED without comorbid depression or substance abuse. CBT was used as the active comparison condition, given the extensive evidence base supporting the efficacy of CBT for BED (see Chapter 56). Thirty-three percent of participants dropped out of CBT and 11% of participants dropped out of IPT; however, this difference was not significant. Results showed no differential treatment effect between IPT and CBT, with 55% and 50% reductions in number of days binge eating among patients in the CBT and IPT conditions, respectively, from baseline to 1-year follow-up. This study clearly demonstrated support for the efficacy of IPT in reducing binge eating (Wilfley et al., 1993).

A second RCT comparing group IPT with group CBT was conducted to evaluate IPT's efficacy using a larger sample of adult men and women (n = 162). Rates of treatment compliance (83% completion of CBT and 88.5% completion of IPT) and dropout (9.9% from CBT and 8.6% from IPT) were not significantly different between conditions (Wilfley et al., 2002). Patients in both conditions showed equivalent rates of abstinence from binge eating (72% for CBT and 70% for IPT) and global ED psychopathology below that of a nonbingeing obese sample (82% for CBT and 79% for IPT) at 1-year follow-up (Wilfley et al., 2002). Results also supported specificity of treatment effects; although the two treatments showed a similar time course for all other measures of psychopathology, CBT led to quicker improvements in dietary restraint, although there were no differences between IPT and CBT in this measure at 1-year follow-up (Wilfley et al., 2002). At 4-year follow-up, 52% of patients in CBT and 76.7% of patients in IPT were abstinent from binge eating. There was a significant treatment × time effect, but there were no significant differences between conditions at any time point (Hilbert et al., 2012).

Given the efficacy of group IPT for BED, a third multisite trial was conducted to evaluate whether patients with BED require specialty treatment. Using the largest sample to date of male and female patients with BED (n = 205), IPT (a specialty treatment) was compared with behavioral weight loss treatment (BWL, designated a nonspecialty treatment because it does not specifically target the core ED psychopathology of BED) and with CBT delivered in a guided self-help format (CBTgsh, a less costly and easier to disseminate form of specialty CBT treatment). Over 2-year follow-up, IPT and CBTgsh outperformed BWL (odds ratios: CBTgsh vs. BWL = 2.3; IPT vs. BWL = 2.6; CBTgsh vs. IPT = 1.2), and individuals with greater psychopathology showed the greatest improvements with IPT (Wilson, Wilfley, Agras, & Bryson, 2010). IPT also demonstrated significantly lower dropout (7% for IPT compared to 28% and 30% for BWL and CBTgsh, respectively), and patient ratings of treatment suitability for addressing their problems were higher for IPT than CBTgsh (Wilson et al., 2010). Results suggest that CBTgsh and IPT are efficacious interventions for individuals with BED, and IPT is recommended for individuals with greater psychopathology (particularly those with low self-esteem and high psychopathology; Rieger et al., 2010; Wilson et al., 2010). In sum, results across these trials suggest that IPT is an efficacious treatment for individuals with BED.

Anorexia Nervosa

Interpersonal psychotherapy has been evaluated in comparison to CBT and SSCM (an educational intervention focused on weight restoration; see Chapters 12 & 56) in a RCT among 56 women with AN (see Chapters 2 & 8). At post-treatment, patients receiving SSCM were more likely to have a good outcome (defined as minimal to no AN symptoms) compared to IPT (56% vs. 10%, respectively) but were not significantly different from CBT (32%). IPT

and CBT were not significantly different from each other in terms of percent of patients with a good outcome (McIntosh et al., 2005). Long-term follow-up results (mean 6.7 ± 1.2 years) showed 49% of patients evidenced a good outcome, with 41% of patients in CBT, 64% of patients in IPT, and 42% of patients in SSCM evidencing good outcome (Carter et al., 2011). However, treatment differences were not significant at long-term follow-up. Novel approaches to improve rates of remission and increase the rate of treatment response are warranted for the subset of patients who remain symptomatic (see Chapter 12).

Considerations When Choosing a Treatment Modality

Interpersonal psychotherapy is similar to many psychotherapies in its focus on helping patients to gain a sense of mastery and to reduce isolation. However, the strategies used in IPT, the conceptualization of the cause of patients' problems, the typical length of treatment, and its treatment foci make IPT distinct from other psychotherapies (Tanofsky-Kraff et al., 2010; Wilfley, 2008). IPT is time-limited, focused on specific problem areas, examines current rather than past relationships, and recognizes rather than centers on intrapsychic conflict. IPT also differs from cognitive and behavioral approaches in that maladaptive thoughts and behaviors are addressed only as they apply to problematic interpersonal relationships or functioning; the goal of IPT is to change the relationship pattern rather than associated cognitions. Functional analyses of ED symptoms (e.g., episodes of binge eating) are conducted in relation to the interpersonal context in which the behavior occurred. The treatment focuses on improving interpersonal functioning rather than on providing specific behavioral strategies for the alleviation of ED symptoms. Also, in contradistinction to other psychotherapies, IPT recognizes but does not directly focus on the patient's personality characteristics.

When selecting a treatment modality, patients and clinicians are encouraged to evaluate the advantages and disadvantages of all treatment approaches. It is beneficial for clinicians to consider their comfort level and expertise in providing certain treatment approaches. Given that IPT is a specialty treatment, it should be delivered by therapists who have received proper training. However, evidence suggests that IPT is easily learned and implemented with high fidelity (Birchall, 1999).

IPT is well suited for individuals with difficulties in social functioning who have a broad range of disordered eating and general psychopathology (Markowitz, Skodol, & Bleiberg, 2006; Wilson et al., 2010). IPT may also be particularly beneficial for certain minority groups (e.g., African Americans; Chui et al., 2007) or specific age cohorts (e.g., adolescents; Tanofsky-Kraff et al., 2010). Finally, for individuals who express discomfort or experience difficulties with elements of CBT such as homework and self-monitoring, IPT may be more acceptable, as it does not utilize at-home monitoring assignments.

Delivering Interpersonal Psychotherapy for Eating Disorders

Interpersonal psychotherapy aims to help patients improve current interpersonal problems associated with the onset or maintenance of ED behaviors. IPT focuses on four interpersonal problem areas common for patients with EDs (Wilfley, Stein, & Welch, 2005). The problem area of *role transitions* is common for individuals experiencing changes in their life status, such as moving, starting or ending a job, divorce, or the onset of a medical illness. Treatment goals

are to help patients to mourn the loss of the old role, identify positive aspects of the new role, and build mastery for the new role. The problem area of *grief* arises when patients experience complicated bereavement following the loss of someone of personal importance. Treatment goals are to help patients to mourn the loss of the relationship and develop new, fulfilling relationships and activities to replace the loss. *Interpersonal role disputes* are a treatment focus when patients experience problems or conflicts with significant others, such as family members, friends, or coworkers, as a result of differing expectations of the relationship. Treatment goals focus on helping patients to identify interpersonal conflicts and to develop more effective communication strategies to resolve disputes. Finally, the problem area of *interpersonal deficits* is common for individuals with a history of unsatisfactory or unfulfilling relationships, social isolation and loneliness, or difficulty in establishing or maintaining relationships. The goal of therapy is to help build patients' social networks by developing new relationships or enhancing the quality of existing relationships. Across each of these problem areas, therapists help patients to consistently connect current interpersonal functioning to the maintenance of ED behaviors throughout treatment.

Structure of IPT

Interpersonal psychotherapy is delivered in three phases, usually conducted in 15–20 sessions over 4 to 5 months. The *initial phase* of IPT is typically delivered over five sessions. The focus of the initial phase is to assess the patient's ED symptoms and obtain a history of relevant life events and ED symptoms. The therapist provides a rationale for IPT and explains how addressing interpersonal problems will help to alleviate ED behaviors. During this phase, the therapist conducts an *interpersonal inventory* to assess the patient's current relationships, interpersonal functioning, and relationship expectations and patterns. The interpersonal inventory is used to develop an interpersonal formulation, identify problem area(s), and determine the focus of treatment.

The *intermediate phase* typically occurs over 8–10 sessions and is considered the "work" phase. Each week, the therapist and patient review problematic interpersonal situations that occurred and link them to disordered eating behaviors. Patients learn to identify the ways in which interpersonal problems trigger ED symptoms and to develop strategies to alter the interpersonal context in which disordered eating behaviors occur. This allows the patient to interrupt the cycle of the ED. Therapists are also encouraged to reinforce situations in which patients describe positive connections between interpersonal functioning and eating behaviors. For example, praising patients when they do not engage in binge eating following an argument with a significant other can help patients to build confidence and reinforce mastery over behavior changes.

Therapists also help patients explore and express affect, which may help to improve patients' interpersonal relationships (Wilfley et al., 2000). Given that negative affect is a common antecedent to binge eating (Wolfe et al., 2009; see also Chapter 32), it is important for the therapist to assist patients in acknowledging painful emotions, experiencing affect that might otherwise be commonly suppressed, and using affect to help drive interpersonal changes (Wilfley, 2008; Wilfley et al., 2000). For example, helping a patient to express feelings of hurt or frustration with a significant other, rather than suppressing the emotions, can be a constructive therapeutic target. During the intermediate phase, therapists may also employ communication analysis to help patients improve communication skills. For this technique, patients describe a recent interaction with a significant other. Therapists then use this detailed

analysis to identify patients' ineffective communication patterns and work with patients to improve their communication strategies.

Over the course of treatment, therapists must remain attuned to helping patients make connections between interpersonal problems and ED symptoms. Though patients may be eager to discuss distressing ED symptoms in session with the therapist, therapists must reinforce treatment goals by gently but firmly directing patients back to focusing on the interpersonal context in which the ED symptoms occurred.

The final phase of treatment is the *termination phase*, in which plans for the end of treatment are explicitly discussed. This final phase typically lasts four to five sessions. Therapists and patients work together to evaluate and consolidate gains made in treatment and plan for goals following the end of treatment. Patients are encouraged to identify early warning signs for relapse (e.g., binge eating, dietary restriction) and discuss strategies for effective coping. Patients are reminded to remain aware of times that may trigger problematic ED behaviors (e.g., high stress, low mood), as well as how their coping strategies can facilitate relapse prevention.

IPT Delivered in a Group Format

Interpersonal psychotherapy delivered in a group format is similar in structure to individually delivered IPT and proceeds through three phases. However, prior to initiating group treatment, patients complete an individual session with a therapist to conduct an interpersonal inventory, identify problem areas, and establish treatment goals. Often, IPT groups are facilitated by trained co-therapists. The group format allows for a safe environment in which to practice new behaviors in session with peers. This interpersonal "laboratory" affords the co-therapists an opportunity to provide in-the-moment coaching and problem-solving for more effective interpersonal relationships.

Conclusions and Future Directions

Interpersonal psychotherapy is efficacious for the treatment of BN and BED, and it can be delivered effectively in individual and group formats. However, several important areas require further study. In an ongoing effort to improve IPT and broaden its utility, we initially review a newly emerging adaptation for IPT to prevent excess weight gain in adolescents and then propose other research directions, including other applications for IPT with diverse populations. Finally, we discuss the important next step of translating IPT for EDs from specialty care centers to use in the primary care setting and in other nonresearch clinical settings where clinicians could be trained to deliver IPT in a novel and effective manner.

Adapting IPT for the Prevention of Excess Weight Gain

A novel adaptation of IPT is being tested for the prevention of excessive weight gain in adolescents who report loss of control (LOC) eating patterns (Tanofsky-Kraff et al., 2007). LOC eating refers to situations in which one cannot control what or how much is being eaten, regardless of the actual amount of food consumed (Tanofsky-Kraff, 2008). LOC eating is common among youth, is associated with distress and overweight (Tanofsky-Kraff, 2008), and predicts excessive weight gain over time (Tanofsky-Kraff et al., 2009) as well as the development of partial or full-syndrome BED (Tanofsky-Kraff et al., 2011).

IPT for the prevention of excess weight gain (IPT-WG) was adapted from IPT for the prevention of depression in adolescents (IPT Adolescent Skills Training, or IPT-AST; Young, Mufson, & Davies, 2006) and from group IPT for BED (Wilfley et al., 2000). The intervention evolved from the finding that individuals with BED who cease binge eating tend to maintain their body weight during and after treatment (Agras et al., 1995; Agras, Telch, Arnow, Eldredge, & Marnell, 1997; Devlin et al., 2005; Wilfley et al., 1993, 2002). Therefore, it was hypothesized that treatment of LOC eating among youth may reduce excess weight gain and prevent development of full-syndrome EDs (Tanofsky-Kraff et al., 2007). Preliminary data suggest IPT-WG may be a promising intervention for the prevention of excess weight gain and BED (Tanofsky-Kraff et al., 2010).

IPT-WG is delivered in a group format over 12 weeks. Group size is typically five, enabling counselors to keep adolescents engaged (Tanofsky-Kraff, 2012). Overweight adolescent girls recruited from the community for treatment are assessed for LOC eating via the Eating Disorder Examination (Fairburn & Cooper, 1993; see also Chapter 38). To address developmental differences in participants, girls are assigned to groups based on younger (12–14 years) and older (15–17 years) age.

Several factors indicate that IPT is particularly appropriate for the prevention of obesity in high-risk adolescents with LOC eating. IPT-WG addresses an individual's overconcern with shape/weight and with negative self-perceptions of body image, and focuses on negative affect linked to LOC eating. Peer relationships are a crucial standard of self-evaluation for youth (Mufson, Dorta, Moreau, & Weissman, 2004; Tanofsky-Kraff, 2012; see also Chapter 31), and weight gain over time is highly influenced by perceived social interactions and social standing (Lemeshow et al., 2008). Adolescents who are overweight are also more likely to regard themselves negatively based on their shape and weight compared to adolescents who are normal weight (Fallon et al., 2005; Schwimmer, Burwinkle, & Varni, 2003; Striegel-Moore, Silberstein, & Rodin, 1986). This may result from increased appearance-related teasing, rejection, and social isolation (Strauss & Pollack, 2003; see also Chapter 21), which could be directly targeted and alleviated by IPT. By decreasing depressive symptoms and negative affect (Mufson, Dorta, Wickramaratne, et al., 2004), IPT also reduces LOC eating that results in excessive weight gain. Additionally, IPT is posited to increase social support, which is associated with improvements in weight loss and weight maintenance among adults who are overweight (Wing & Jeffery, 1999) and children who are overweight (Wilfley et al., 2007).

Adapting IPT-WG for Diverse Populations

Loss-of-control eating patterns show comparable prevalence rates among African American, Hispanic, and Caucasian adolescent girls and are endorsed with substantial prevalence in low-income urban and rural communities (Croll, Neumark-Sztainer, Story, & Ireland, 2002; Field, Colditz, & Peterson, 1997; Smith, Marcus, Lewis, Fitzgibbon, & Schreiner, 1998; Snow & Harris, 1989; Story, French, Resnick, & Blum, 1995; Striegel-Moore, Wilfley, Pike, Dohm, & Fairburn, 2000; see also Chapter 25). Given the strong indication that African American patients show greater reductions in binge eating frequency when treated with IPT (Chui et al., 2007), further study of IPT with ethnically diverse populations who are at heightened risk for adult obesity or adult onset EDs is necessary. Additionally, developing training strategies to reach therapists in resource-poor communities is needed (e.g., using Internet-based tools), given the prevalence of LOC eating in low-income urban and rural communities.

Adapting IPT into Child and Family-based Formats

Additional considerations for improving IPT should include adaptations for utilizing IPT with children in a family-based format, given the importance of parental involvement with children in behavioral interventions (Wilfley, Kass, & Kolko, 2011). A pilot study of family-based IPT for treating depressive symptoms in children ages 9 through 12 demonstrated that it was feasible and acceptable to families (Dietz, Mufson, Irvine, & Brent, 2008). Given that LOC eating in youth is associated with excess weight gain and social problems (Tanofsky-Kraff et al., 2009, 2011, 2012), targeting LOC eating in children may reduce risk for obesity/excess weight gain and improve social functioning.

Enhancing IPT for BN and BED

Interpersonal psychotherapy emphasizes the importance of connecting ED symptoms and interpersonal problems (Hilbert et al., 2007; Wilfley et al., 2002). Other treatment modalities are beginning to incorporate intervention components that link interpersonal functioning to ED symptoms. For example, Fairburn and colleagues demonstrated the efficacy of including an interpersonal module into a recently modified version of CBT for EDs (enhanced CBT for EDs; Fairburn, 2008; see also Chapter 56). IPT, in its current form, already seamlessly incorporates aspects of other treatment modalities with such techniques as the collaborative, behavioral formulation during the interpersonal inventory; however, it is possible that incorporating still more outside techniques into IPT delivery may enhance treatment potency. IPT therapists may, for example, consider using such techniques as self-monitoring as a method for patients to become more aware of negative affect that surrounds ED symptoms.

Testing the Efficacy of IPT for AN

There is a relative lack of research examining the utility of IPT for AN. It is possible that the IPT model could be applicable to patients with AN, since they tend to have deficits in social-cognitive skills that impede their capacity to create and experience validating social interactions (Rieger et al., 2010). Couples-based interventions for adults with AN have demonstrated preliminary efficacy. These interventions incorporate a focus on interpersonal and relationship issues (Bulik, Baucom, & Kirby, 2012; Bulik, Baucom, Kirby, & Pisetsky, 2011; Goddard et al., 2011, 2013), which suggests that interventions such as IPT that focus on interpersonal functioning may be efficacious for patients with AN.

Increasing the Dissemination of IPT

Despite the pressing need for effective interventions, there is substantial evidence that patients in routine clinical care are not receiving evidence-based interventions such as IPT (Beidas & Kendall, 2010; National Collaborating Centre for Mental Health, 2004; Proctor et al., 2009; Resnick, 2005; Wilfley et al., 2002; Wilson et al., 2010) or that these interventions are often not competently delivered (Shafran et al., 2009). Consequently, an important next step is to translate IPT from delivery in specialty care centers to primary care and other nonresearch clinical settings. Increasing the number of therapists trained to deliver IPT will improve dissemination and implementation of IPT. Practical, low-cost training strategies such as train-the-trainer (in which an internal champion in an organization is trained to deliver an evidence-based

intervention, who then trains and supervises other trainees in that setting) may help to achieve this aim (Fairburn & Cooper, 2011).

Use of an online program for clinical training also overcomes translational barriers and has several key advantages (Fairburn & Cooper, 2011). First, online training can be offered to geographically dispersed trainees using minimal person-based resources. The website can be accessed anytime and anywhere to effectively accommodate therapists' busy schedules. Second, online training enables trainees to review material continually (e.g., at work or at home), thereby reinforcing session content across multiple contexts. Third, the process of training to fidelity can be customized to the trainee through the use of tailored quizzes, feedback, and refresher courses. Fourth, incorporating an asynchronous online discussion board allows trainees to interact with other trainees and a training guide; this collaborative forum can encourage trainee dialogue (e.g., ask questions, share common problems) and reinforce the continued practice of IPT with patients. Finally, the website can be updated regularly, facilitating efficient dissemination of new information. Thus, online training may overcome barriers that limit current training and supervision approaches and has strong potential to increase the number of therapists trained to deliver evidence-based interventions, resulting in substantial clinical impact for individuals with EDs.

In conclusion, IPT is a specialty treatment, efficacious for the treatment of adults with BN and BED, and shows promise for the prevention of excess weight gain among adolescent girls who are overweight. Continued research is needed to examine subpopulations for whom IPT is most efficacious, to further investigate IPT for use with AN, and to adapt IPT for use with children or for delivery in a family-based format. Finally, widespread dissemination of IPT, such as through the use of scalable training strategies, will increase the number of clinicians trained to deliver IPT, resulting in greater reach and sustainability of IPT for individuals with EDs.

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63

Motivational Interviewing and Readiness for Change

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In this chapter we consider the use of motivational interviewing (MI) in the field of eating disorders (EDs) in relation to helping both sufferers and carers. The definition of "carer" used is that of the United Kingdom's Princess Royal Trust for Carers (Carers Trust, 2012): "A carer is someone of any age who provides unpaid support to family or friends who could not manage without this help due to illness, disability, mental ill-health or a substance misuse problem." MI is a form of interaction used to negotiate behavior change in a style that is both patient-centered and gently directive. The technique has been adjusted over time in the light of a large body of research and practice (Miller & Rollnick, 2013). It is now established as one of the key behavior change strategies (Abraham & Michie, 2008).

Research has shown that MI as a style of interviewing has the most benefit for people who are ambivalent and hostile about change. People with anorexia nervosa (AN) are typically less ready to change than people with other EDs, such as bulimia nervosa (BN) or binge eating disorder (BED; Blake, Turnbull, & Treasure, 1997; see also Chapter 12). Although distressed by their symptoms, people with BN or BED typically seek help of their own accord, making it easier to form a treatment alliance and effect change. A key feature of AN, however, is that it is highly valued by the person herself or himself, even when faced with the prospect of death (Schmidt & Treasure, 2006; Treasure & Schmidt, 2013; see also Chapters 2, 8, & 18), and it has a social impact as the illness is highly visible to others (Treasure & Schmidt, 2010). With AN, presentation to services often involves a degree of coercion from family members, teachers, or occupational health physicians (Treasure, Lopez, & MacDonald, 2011). MI is a useful tool in engagement and addressing ambivalence in such cases. MI can also help with key behaviors such as meal support, which have been described as a battleground within inpatient settings (Long, Wallis, Leung, & Meyer, 2012).

In the United Kingdom, the National Institute for Clinical Excellence (NICE, 2004) guidelines recommend that most people with AN be managed on an outpatient basis. Given that people with EDs are frequently dependent on their families, either because of their age or because of the severity of their illness, the NICE policy places primary responsibility on family members, and in this regard NICE recommends including families in the treatment of younger patients in particular (see Chapter 60). Nevertheless, caregivers frequently report that they

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lack the skills and resources required to care for their loved ones (Haigh & Treasure, 2003; Treasure et al., 2001). Furthermore, the manner in which the family attempts to reduce the symptoms often inadvertently plays a role in the maintenance of the problems (Treasure, Sepulveda et al., 2008). Consequently, MI can also be a useful tool in helping families decide to change those behaviors and communication patterns that may contribute to these cycles of maintenance. In turn, carers can use some of these skills to address the ED symptoms (Treasure et al., 2011).

This chapter outlines the principles of MI and its use in the field of EDs. Its relationship to the transtheoretical model and the concept of "readiness to change" will also be discussed. Practical examples of MI being used with both sufferers and carers will be illustrated. The closing sections of the chapter examine the evidence base with regards to effectiveness of MI interventions in the treatment of EDs, as well as areas for improvement.

Motivational Interviewing, Readiness for Change, and Eating Disorders

Miller and Rollnick (2013, p. 29) have provided two helpful definitions of MI: (a) "a person-centered counseling style for addressing the common problem of ambivalence and change"; and (b) "a collaborative, goal-oriented style of communication with particular attention to the language of change. It is designed to strengthen personal motivation for commitment to a specific goal by eliciting and exploring the person's own reasons for change within an atmosphere of acceptance and compassion."

There are several reasons for the appeal of the use of MI in providing treatment and support for health-related problems. First of all, it was originally developed to address resistance to change. Second, it can be used in the form of motivational enhancement therapy as a stand-alone intervention or in conjunction with other therapies. Third, research evidence supports both the efficacy and effectiveness of MI with health-related problems, with relatively few sessions (Arkowitz, Westra, Miller, & Rollnick, 2008).

At the heart of MI lie four key interrelated elements. *Partnership* involves active collaboration between experts. *Acceptance* of what the client brings to the session consists of recognition of the client's absolute worth, accurate empathy for the client's experience, acknowledgement of the client's autonomy, and affirmation of his or her strengths and efforts. *Compassion* refers to active promotion of the client's welfare and prioritization of her or his needs, while *evocation* emphasizes bringing forth and developing what is already present, not installing what is missing.

The Transtheoretical Model of Change

There is often confusion between MI and the transtheoretical model of change (TTM; Prochaska & DiClemente, 1984), mainly because they were developed around the same time during the early 1980s. A fundamental insight of the TTM is that because people have different stages (i.e., levels) of readiness to change health-related behaviors, at each stage certain behaviors to facilitate change can be particularly constructive or destructive and the therapist will have to use a range of different approaches to help patients move forward. During the *precontemplation* stage, for example, the individual has no intention to change behavior in

the near future. Therefore, the therapeutic process involves helping the patient to step back and take a more global perspective on her or his values and beliefs about their life. In the *contemplation* stage, there is recognition of the problem, albeit with a degree of ambivalence, so the therapist will work to increase any dissonance between where the patient is now and where she or he would like to be. *Preparation* involves introspection and reaffirmation of the need and desire to change behavior. This may be the point at which the patient suddenly recognizes the inherent contradictions in his or her current pattern of behavior and commits to making the change. Once an individual is committed to change, she or he is in the *action* stage. Here standard behavior change principles are useful, such as setting goals, designing behavioral experiments, planning their implementation, and predicting and preventing obstacles derailing the process (Treasure, 2010).

Motivational Interviewing and Stages of Change

Miller suggests that people who are at or beyond the stage of readiness (*preparation*) for change do not need MI, and there is some evidence that it may even retard their progress relative to action-oriented treatment (Miller & Rollnick, 2009; Project MATCH Research Group, 1997; Rohsenow et al., 2004; Stotts, Schmitz, Rhoades, & Grabowski, 2001). On the other hand, people who do not see a need for change (precontemplation stage) or who are ambivalent about changing (contemplation stage) benefit most from MI. Thus, there is an art in using MI, especially in complex problems such as EDs, where various levels of symptoms are targeted for change. A wise therapist will flexibly step between MI and more action-oriented approaches such as cognitive-behavioral therapy (CBT; see Chapter 56) as and when needed.

Rationale for Using MI with Sufferers and Families

A key aspect of AN is that the individual typically does not recognize that she or he has a problem (see Chapter 18), and therefore presentation to services often involves a degree of coercion or limit-setting from others (Treasure et al., 2011). The person's disregard for danger and apparent inability to see the risks and adverse consequences of her or his maladaptive, or even self-destructive, behavior leads to high levels of expressed emotion and distress in family and friends (Zabala, Macdonald, & Treasure, 2009). Eventually, there will be an understandable desire for confrontation from those around the person with AN in an effort to make him or her "see reason" and "get help."

MI theory posits that confrontation, no matter how well meaning, frequently leads to increased resistance and less change (Miller & Sanchez, 1994). Indeed, this type of intense, hostile reaction, be it controlling or rejecting, alienates the individual, who then retreats further into ED behaviors and social isolation. This negative cycle makes meal support, often provided by carers in outpatient settings and nurses in inpatient settings, a difficult task. A recent study reported that a motivation-based script delivered on mobile technology reduced anxiety and increased the amount of a nutritional test meal drunk by outpatients (Cardi, Krug, et al., 2012; Cardi, Lounes, Kan, & Treasure, 2012). This suggests that the skills of MI may be helpful in supporting restorative eating.

Patients with AN also often want to please and will tend to avoid expressing irritation, anger, or rebellion directly, especially during outpatient therapy. Instead, they will listen and agree whilst continuing to lose weight. This form of passive aggression can easily lead to therapist, as

well as family, burn-out (Treasure & Ward, 1997). MI provides a useful approach designed specifically for use when people are not ready or are ambivalent about change; MI explicitly offers a framework to work *with* ED sufferers rather than *against* them (Treasure & Schmidt, 2010). MI may also be an effective approach for reducing maladaptive interpersonal responses to the symptoms within the family, as well as in the professional arena.

In terms of the relational aspects of EDs and their potential effects on outcome, Schmidt and Treasure (2006) developed a cognitive interpersonal model to explain the maintenance of ED symptoms. The model proposes that ED symptoms induce caregiver anxiety and also confrontational interactions (high expressed emotions; see Chapters 53 & 60) that can promote resistance. Caregivers' anxieties, frustration, and associated behaviors lead to patient anxiety that, in turn, accentuates ED symptoms. Interventions targeting the elements emphasized by this model have been developed. Self-management tools for carers include some of the basic principles of MI (Treasure, Smith, & Crane, 2007), and various settings and forms of interventions (workshops, telephone coaching, individual and group sessions) have been used to teach carers these skills (Goddard et al., 2011; Goddard, Macdonald, & Treasure, 2010; Macdonald, Murray, Goddard, & Treasure, 2011; Sepulveda, Lopez, Macdonald, & Treasure, 2008; Treasure, Sepulveda et al., 2007).

The Use of MI in Treating and Managing Eating Disorders: Working with Sufferers

In their latest edition of *Motivational Interviewing: Helping People Change,* Miller and Rollnick (2013) describe MI as consisting of four overlapping processes: engaging, focusing, evoking, and planning. The following sections use excerpts based on our clinical experience to explain MI's component processes and illustrate how they can be used with a sufferer.

Engaging

The first process is the establishing of a mutually trusting and respectful helping relationship, a vital component of all therapeutic approaches. According to Miller and Rollnick (2013, p. 46), five client factors influence engagement or disengagement at all points in therapy:

1 Desires or goals—"What is it that you're looking for?"
2 Importance—"How much of a priority is it?"
3 Positivity—"Did you feel good about the experience"? "Did you feel welcomed, valued and respected"?
4 Expectations—"What did you think would happen?" "How did the experience fit with what you expected?"
5 Hope—"Do you think that this situation helps people like you to get what you're seeking?" "Do you believe it will help you?"

The key behavioral components involved in engaging clients include open questions, affirmations, reflective listening, and summarizing (OARS; Miller & Rollnick, 2013) These are described further in Table 63.1. In addition, there is a compassionate, respectful style of relationship in which a high level of listening, accurate empathy, and behavior change skills are used to gently

Table 63.1 The OARS process for engaging clients during motivational interviewing (MI).

Open questions	Open questions are self-explanatory. Closed questions that are likely to elicit monosyllabic answers should be avoided in favor of open questions that open the opportunity for the patient to speak (e.g., how, what, tell me more, etc.). Overall questions should be limited (never more than three in a row). Reflections should be used in preference to questions) with the therapist aiming for a ratio of two reflections to every one question
Affirmations	The therapist has a compassionate, accepting stance and reflects upon strengths, and reinforces positive moves toward more helpful behaviors
Reflective listening	The therapist uses reflections as an implicit mark of listening and understanding the client's perspective. A "simple reflection" is nothing much more than an acknowledgement, via repetition to the person, of what he or she has said. A "complex reflection" feeds back what the person is saying with the aim of eliciting reflections about or plans for change, or increasing the client's self-confidence for change. Complex reflections are one of the directive strategies in MI
Summaries	A summary is a short précis that encapsulates the gist, particularly of the pro-change ideas, intentions, or behaviors. Summaries are used at intervals to enable the patient to hear what she or he is thinking and saying

Note. Source is Miller and Rollnick (2013).

move the interaction toward change. In the following excerpt, the key components of OARS are used to engage Eve (a pseudonym), who has been asked to report to the school counselor by teachers concerned about what they consider clear and distressing signs of an ED. The counselor, in true MI spirit, uses empathy and acknowledges autonomy to make Eve feel valued and respected. He also explores her expectations and avoids any expert-driven directing.

COUNSELOR: *Good morning, Eve. Thanks for coming.*

EVE: *Yeah, well, I wasn't really given much choice. It was more of a case of "turn up with Mr. Ross on Tuesday morning at 10:20."*

COUNSELOR: *Ah, OK, so you're feeling as if you've been coerced into coming here this morning.* [MI: complex reflection] *I appreciate your honesty.* [MI: affirming]

EVE: *Yeah, well, it would have been better if somebody somewhere explained what's going on.*

COUNSELOR: *Sure, I understand where you're coming from.* [MI: empathy] *So you've no idea why some of your teachers have aired some concerns?* [MI: closed question]

EVE: *No* (sighs)*…Well, I guess a couple of them have mentioned weight loss. My Mum's also been on my case, saying that I'm not eating properly and that I'm too thin.*

COUNSELOR: *So there's pressure not only at school but at home, too.* [MI: complex reflection]

EVE: *I know I've lost some weight, but I don't really think there's anything wrong with that. I'm perfectly fine.*

COUNSELOR: *People around you are vocalizing their concerns yet you don't understand why.* [MI: complex reflection]

EVE: *Well,* (sighs) *kinda…sometimes…Well, it gets to you, doesn't it, when people around you are making a fuss…*

COUNSELOR: *It confuses you.* [MI: complex reflection] *So if you did think that coming here for a chat over the next few weeks would help, how would that make you feel?* [MI: open question]

EVE: *Well, I suppose you're not a teacher…you're not my mother. I may find it useful just to speak to somebody else…*

Focusing

In MI, focusing is a collaborative process of finding a mutually agreeable direction. When there is a reasonably clear set of possibilities, the task of agenda mapping is to choose and prioritize. However, when the goals are less clear, then "orienting" may be required. In this process the practitioner helps the client put the pieces together in a way that generates possible ideas about where to begin.

Motivational therapists suppress any propensity to show a "righting reflex" (Miller & Rollnick, 2013, p. 5), that is, to help solve problems and set things right by giving advice. Information is offered indirectly, elaborating a reflection, or with permission. A sequence of *elicit-provide-elicit* is also used. The therapist first checks in to find out what the patient knows, then gives information, and finally checks in to see what has been understood. Again, this emphasizes the patient's active involvement in her or his own healthcare and is intended to enhance motivation for behavior change. In the following dialogue the therapist uses complex reflections to help the patient focus on the bigger picture with respect to the meaning and context of the current difficulties.

COUNSELOR: *Last time you spoke about your excitement of going to university in September, and at the same time the fear and anticipation that this move will bring. You also told me a little bit about the situation at home and the pressure to do well.* [MI: summary of reflections]

EVE: *Yeah, I feel incredibly swamped. I've got to make my choice by the end of the week…I'm not sure if I want to do law, or I want to do business and economics…but I can't face telling Dad…It's like it's expected of me. So there's that, and the other night Mum caught me vomiting after dinner…And all hell was let loose but, hey, that's the only way I can cope right now…It's all way too much for me to handle.*

COUNSELOR: *So right now you're feeling overwhelmed with choices you need to make. The anxiety builds, and your way of responding to the pressure is either to restrict or to vomit, and then this brings on more anxiety when you see the effects that this has on the rest of the family. You're not quite sure how to break out of the vicious circle.* [MI: complex reflection summary]

EVE: *Yup…you've hit the nail on the head.*

COUNSELOR: *The uncertainty you are facing with your career choices is tough for you to manage and it takes a toll on your health, which, in turn, makes your family anxious.* [MI: complex reflection]

EVE: *Yeah, this course and university choice is the first thing I really need to sort out.*

COUNSELOR: *Dealing with uncertainty and anxiety and how it impacts on your health would be an important area to focus on.* [MI: complex reflection, agreement with a twist]

Evoking

During the evoking stage, the counselor reflects on what "change talk" he is hearing from Eve, whether he is steering her too far or too fast in any one direction, and whether his own righting reflex may be pushing or pulling him to argue for change. In the helping professions there is obviously a tendency to want to do specific things that help people. With the best of intentions, professionals (and carers) will have a strong desire to fix what seems to be wrong with people and to set them on a better course (the righting reflex; Miller & Rollnick, 2013).

Yet, we have already discussed the challenge of ambivalence in treatment of EDs. According to Miller and Rollnick (2013), an ambivalent individual will already have the two arguments

inside her or his own head. Therefore, a helper who follows the righting reflex and argues for change is siding with one voice in the sufferer's head. This has a predictable negative outcome: "Argue for one side and the ambivalent person is likely to take up and defend the opposite" (Miller & Rollnick, 2013, p. 7). Denial or resistance is a common reaction to the righting reflex or being steered or directed too quickly. Consequently, in the following dialogue, the counselor evokes and makes a note of Eve's *own* reasons for change.

EVE: *So I had a talk with my Mum and Dad and discussed how I felt and how I was a bit confused as to career directions.*

COUNSELOR: *That must have taken some courage. Dealing with uncertainty and anxiety by enlisting the support of others is a great life skill.* [MI: affirming and highlighting the patient's use of robust emotional regulation strategies]

EVE: *Yeah, they were actually cool with it. They said they just wanted me to start eating. That might be a little more difficult.…I really don't see what the big deal is. I'm fine with eating…really.*

COUNSELOR: *So, on the one hand, you're not quite so sure why your parents are fussing, but you respect their opinion and so you come here.* [MI: complex reflection] *What do you think they might have noticed?* [MI: open question]

EVE: *Well…I get tired more often these days and can't seem to concentrate like I used to…but I do have a lot on my plate…literally speaking!*

COUNSELOR: *So you've noticed some changes since you lost the weight.* [MI: complex reflection]

EVE: *Well, sometimes I have trouble in concentrating and stuff…*

COUNSELOR: *That must be tough at this point in your studies when concentration is important to your goals.* [MI: empathy] *Any other changes you may have noticed since your weight has gone down?* [MI: open question]

Planning

Planning is evident when people start to think about change and how they might make those changes. It involves both developing commitment and formulating a specific plan of action.

COUNSELOR: *Last session you spoke about how your health may have suffered in the face of anxiety and uncertainty. How have things been going since we last spoke?* [MI: open question]

EVE: *Yep, life has been such a grind these last few months, especially around this food thing. I mean breakfast is OK, and lunch I can generally handle.…However, by dinnertime I can be a bit of a basket case. I just get so wound up.*

COUNSELOR: *You're pretty adept at pinpointing areas that are difficult for you.* [MI: affirming] *By the end of the day your anxiety is at its highest, and you're not sure where to take this.* [MI: complex reflection]

EVE: *Actually I have been doing a lot of thinking about possibilities…like when I have such an urge to go throw up after dinner. I guess I could perhaps ask my parents for some support with this—maybe a walk or some other distraction would be helpful. They seem different these days; they've had a bit of support and guidance, although they don't talk very much about it. My eating disorder is still very much the white elephant in the room.*

COUNSELOR: *I am impressed at your ability to reflect on how others can help you. People who move towards recovery show this skill.* [MI: affirmation] *You also recognize that more open communication between you and your parents may be helpful, and this may be an ideal opportunity to discuss some of the challenges.* [MI: complex reflection]

Planning is also illustrated in the next excerpt, in which the counselor sits with Eve in the school cafeteria during lunch in order to offer some "meal support." The counselor accepts and rolls with Eve's resistance, acknowledging her autonomy as well as her difficulty with trust, attachment to others, and the emotions these connections might produce. Later he affirms her previous plans and wise emotion regulation strategies, and then he elicits from her other possible options that would help with current anxiety levels. He also uses distraction to remind her of her future plans and engages in non-ED talk with her.

COUNSELOR: *Ah, I see you went for the fish option today. I thought I'd try the chicken.*

EVE: *I'm not overly hungry, really; in fact I'm not hungry at all. Had a big breakfast.…I'm really only here because I'd have felt bad about breaking the appointment when we'd arranged it.*

COUNSELOR: *I appreciate your honesty. It's really brave of you to turn up at all—I know how difficult it must be for you.* [MI: affirmation and empathy]

EVE: *Yeah, well, I think I can manage after this one on my own. I have my meal plan and I know what I need to do.*

COUNSELOR: *Part of you seems more confident this week about being able to keep up with your meal plan. Last time we met you were talking about the anxiety around mealtimes and how you found it useful having somebody around to keep your mind away from the anorexic thoughts.* [MI: simple reflection] *Anything in particular that's changed, new strategies that we can discuss?* [MI: open question]

EVE: (sighs) *No…not really. I guess if I just feel a bit "watched" when I eat with you.…You know, like I'm "under observation."*

COUNSELOR: *Ah, right, on the one hand it's like sitting with the "food police," and on the other hand having someone to help with the anxiety perhaps produces mixed feelings such as safety, but perhaps with a twinge of shame that you need compassion and support.* [MI: complex reflection]

EVE: (smiling) *Something like that.…Sometimes I just think all this effort that everybody goes to is just so…so futile.*

COUNSELOR: *It is as if you do not deserve help or support. I mentioned the last time how impressed I was by your ability to look out for those options and any necessary support that may ease the struggle, particularly during the more difficult times.* [MI: complex reflection] *We are social animals and getting help and connection from others is a wise, "wired-in" strategy.* [MI: giving information and affirmation]

EVE: *Mmm…*

COUNSELOR: *I was also really interested last time in hearing about your travel plans for this summer. You seem to really have the eye on the bigger picture.* [MI: complex reflection] *How do you feel if we continued this conversation through lunch today?* [MI: open question] *I've got that information you asked for on youth hostelling through France.* [MI: giving information]

EVE: *I guess…*

COUNSELOR: *Jenny up in the library has also put together some pamphlets, so after lunch we could pop upstairs to collect them.…*

The Use of MI in Treating and Managing Eating Disorders: Working with Carers

As discussed above, carer programs based on the cognitive interpersonal model (Schmidt & Treasure, 2006; Treasure & Schmidt, 2013) not only equip and empower carers with psychoeducational information and skills training, but also provide training in MI skills. The goal of

using MI to work with carers is to support carers in an empathic, accepting, and compassionate fashion in order to boost their self-reflection, self-efficacy, and action planning. When working with families, light-hearted animal analogies are used to address challenging interpersonal interactions (Treasure, Sepulveda, et al., 2007). These include situations whereby carers fall into extreme patterns of emotional responding: either too intense an emotional reaction with anguish or anger (jellyfish), or denying that they have any emotions (ostrich). In terms of directedness, carers can be overly protective (kangaroo) or overly directive (rhinoceros). The goal is to become like a dolphin and use warmth, gentle nudging, and negotiation. If families can be helped to develop more effective forms of social communication, this may help them facilitate a restoration of nutritional balance (Treasure & Schmidt, 2013).

In the dialogues that follow, we have used the word "coach" to reflect our current work with "experienced" carers, that is, carers who have lived with a person suffering from an ED and have had brief training from professional therapists in how to work to support other carers. The dialogues illustrate how the coach uses an MI approach to help the carer think about change and put effective behavior change strategies into place.

Scenario 1: Empathy

CARER: *I just get so frustrated. Last night, for example, I sat with her and she refused point blank to eat. She just sat there screaming "Shut up, shut up, shut up, shut up…" I'm at my wits' end; I just don't know what to do when she's like this. We end up screaming at each other for what seems like hours and in the end there's nothing achieved, nothing gained.…*

COACH: *It's very tough on you having to cope with such resistance—with rhinoceros battles—on a daily basis. The frustration and fighting must be very difficult on your stress levels, and effective communication is lost.* [MI: complex reflection]

Scenario 2: Boosting Self-Efficacy

CARER: *I mean, every day it's apparent that there's something that I'm not doing right, or have done wrong, or could have done better.…So I go away and try to analyze it.…I guess I do get drawn into being a rhino.*

COACH: *You reflect on how the eating disorder pulls you to react.* [MI: affirming] *You are open to coming up with new problem-solving strategies.* [MI: complex reflection]

CARER: *Yeah, I guess…*(pause)*…If I talk to somebody else who has a problem, I can say "Well, if you try that" and I think "Well, yeah, I have done that so I must be doing something right"…But sometimes you need somebody from the outside to say "Yeah, you've done that well."*

Scenario 3: Encouraging Self-Reflection and Goal Setting

COACH: *You said you tend to make decisions in all areas of her life, as if the eating disorder keeps you in kangaroo mode.* [MI: complex reflection] *What ways can you think that you could do the dolphin nudge?* [MI: open question]

CARER: *Well, because she's not really doing very much, it's quite hard to sort of see how I can… but I suppose, really, it's just a way of encouraging her to try and get more involved in things outside of home and the illness and to try and make arrangements to actually go out and see people and do things.…I mean the exercising thing is a big problem for us because she goes mad with it, and that's all she really wants to do, so I need to try and encourage her to do other things.*

COACH: *So you seem to have a good grasp of what's needed.* [MI: affirming] *I sense that you also find it difficult to put these into place.* [MI: complex reflection]

CARER: *Yeah, but I can see that, if I want her to change her attitude, then I probably need to change mine.*

Fidelity to MI Model and Quality Control

Using MI flexibly and effectively takes practice, extended learning, and appropriate supervision. Based on research findings and on Miller and Rollnick's own experiences in teaching MI, there is little relationship between self-reported competence in MI and objective measures of skills based on observed practice (Miller & Mount, 2001; Moyers, Martin, Manuel, Hendrickson, & Miller, 2005). The fidelity and quality of MI can be quantified for both supervision and research using the Motivational Interviewing Treatment Integrity (MITI) Code 3.1.1. This is a validated and reliable behavioral coding system used to measure treatment integrity in using MI (Moyers, Martin, Manuel, Miller, & Ernst, 2010). It consists of two proficiency components: global scores and behavior counts. This can be used as part of the supervision process, by coding transcripts of sessions, ideally by the clinician who is being supervised. A recent study that measured treatment integrity to MI in both professional coaches and lay carer coaches, for example, showed that whilst prior clinical training resulted in professional coaches reaching MITI recommended competency, the "lay carer coaches" were able to deliver MI to a level of basic competency (Macdonald et al., 2014).

Conclusions and Future Directions

Motivational interviewing is a style of interaction that is of particular benefit for managing ambivalence about change rather than as a stand-alone therapy. In the treatment and management of complex problems such as EDs, which are marked by low confidence about change and by emotional difficulties, MI is often combined with many other forms of behavior change and stress management. Therefore, it is difficult to dissect and define the particular benefits of using this therapeutic style in ED treatment. For example, this style is embedded within therapy applying the Maudsley Model for Treatment of Adults with AN (MANTRA); Schmidt et al., 2013; Wade, Treasure, & Schmidt, 2011) and in cognitive remediation (Tchanturia et al., 2008).

Nevertheless, the evidence base for the effectiveness of MI and of MI principles applied to brief motivational enhancement therapy (MET, e.g., for substance abuse) is relatively strong, as seen in the results of several very large, multisite randomized controlled trials (Ball et al., 2007; Carroll et al., 2006). Evidence for the effectiveness of MI for other measures of behavior change (e.g., body mass index [BMI], cigarette smoking, HIV risk, systolic blood pressure) has also been synthesized in several systematic reviews (Burke, Arkowitz, & Menchola, 2003; Dunn, Deroo, & Rivara, 2001; Lundahl, Kunz, Brownell, Tollefson, & Burke, 2010; Rubak, Sandbaek, Lauritzen, & Christensen, 2005).

Two recent literature reviews have concluded that MI can increase "readiness to change" in people with EDs (Dray & Wade, 2012; Macdonald, Hibbs, Corfield, & Treasure, 2012). There is some evidence also that the transtheoretical model of change is relevant to the treatment of EDs, as the more advanced a person's initial stage of readiness for change, the better the

treatment outcome for his or her ED (Dray & Wade, 2012). No study to date, however, has used a design that can establish whether the process of using an MI style during therapy impacts outcome. Only 5 of the 13 studies in the review by Macdonald et al. (2012) used some form of monitoring to ensure fidelity to the MI model. In order to draw confident conclusions about the place of MI in the field of EDs, future research needs to focus on discrete aspects of behavior (e.g., during test meals) while using process measures of treatment fidelity.

In conclusion, MI and related approaches may be highly relevant and applicable to the field of EDs, particularly for the treatment of AN. Yet it is important to keep in mind that MI is a technique that is useful at certain points of therapy and is not an intervention in its own right. For example, it may be contraindicated when individuals are in the *action* stage such that standard behavior change principles can be applied. On the other hand, it may be of particular relevance when, as is often the case for people with AN, there is ambivalence or hostility to change, for example, when meal support is needed or for engagement into the treatment process. Studies in progress are examining whether teaching carers to use a motivational style alongside other skills is of benefit for change in the patient with an ED. As mentioned above, preliminary evidence shows some promising results in both carer and patient data (Goddard et al., 2011; Hibbs et al., 2014).

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Section XI

Controversies and Future Directions

64

Perspectives on the Measurement of Outcome

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Roughly a century ago, the publication of Emil Kraepelin's (1921) *Manic Depressive Insanity and Paranoia* transformed the study of psychiatric illness. Kraepelin's brilliance was the understanding that by linking a meticulous study of clinical history to a comparably detailed investigation of how illness behavior evolves over time, valid differences between disorders would emerge. The analysis, from which the separation of schizophrenia from affective disorders was deemed valid, was the foundation for modern psychiatric nosology.

Today, Kraepelin's synthesis is being recast. As evidence grows that psychiatric disorders with different symptom profiles can share both genes and abnormalities in neurocircuitry, what the conventional diagnostic boundaries mean and how they should now be drawn are new, intriguing questions (Morris & Cuthbert, 2012). Why, given these commonalities, do outcomes differ between persons with similar afflictions, receiving similar treatments? Why do short-term outcomes often correlate poorly with outcomes measured years later? What influences come into play that might explain a shift in illness trajectories? The implications are straightforward: without precision in our definitions of outcome, consistency in their application, and a common conceptual foundation for understanding their rationale, rigorous study of the mechanisms that give rise to different illness trajectories is not possible. Systematically defined clinical outcomes are integral to clinical research (Frank et al., 1991; Kraemer, Frank, & Kupfer, 2011; Kraemer & Kupfer, 2006).

The significance of terms like *remission* and *recovery*, which only recently has received attention in our field (Bardone-Cone et al., 2010; Couturier & Lock, 2006a, 2006b; Kordy et al., 2002), has been debated in other areas of psychiatric research for some time, perhaps best illustrated by studies of the affective disorders (Frank et al., 1991). In this chapter we review why these distinctions are important and the questions they beg for future study of the eating disorders (EDs). The chapter is not a review of outcome studies in EDs; for this summary, interested readers are directed elsewhere (Berkman, Lohr, & Bulik, 2007; Steinhausen, 2002; Steinhausen & Weber, 2009; see also Chapters 55–63).

The Wiley Handbook of Eating Disorders, First Edition. Edited by Linda Smolak and Michael P. Levine.

As the pioneering discussion by Frank et al. (1991) shows, terminology applied to research on illness course and outcomes rests on more than semantics. Important theoretical and clinical considerations are involved (Frank et al., 1991). A simple point suffices: the complex nature of psychiatric illness is well accepted, so we recognize that factors conferring risk are not necessarily identical to the mechanistic alterations that induce symptoms, and both sets of processes may be different from the adaptations that cause illness to endure. At the heart of these differences is, of course, the question of how genotype, environment, and phenotype link together, one that only now is being considered in EDs research (Klump, Burt, McGue, & Iacono, 2007). But a wealth of research on the molecular and genomic phenotypes in addiction supports the premise (Nestler, 2012). It is from this amalgam that psychiatric phenotypes emerge.

The processes determining how illness progresses and resolves are not static. At the heart of clinical research are the fundamentally important questions of how to interrupt the mechanisms that give rise to and sustain dysfunction, and how to identify the critical periods during which interventions are optimally beneficial. In short, if we are to gain insight into the factors promoting illness behavior in the hope of optimizing our treatments, definitional clarity and consistency in the measurement of outcomes are imperatives.

Defining Recovery in Eating Disorders

Anorexia Nervosa (AN)

The historical precedent for defining "recovery" in AN—the Morgan–Russell criteria (G. Morgan & Russell, 1975; H. Morgan & Hayward, 1988)—measured physical, psychopathological, and psychosocial functioning. A "good" outcome required attainment of 85% of average expected body weight for age and height, and in postpubertal females, a return of cyclical menstruation. An average outcome score is derived from these ratings. The index has been used widely, but revisions using physical health parameters plus a reduction in eating attitudes within one standard deviation of a normal comparison group have been proposed (Pike, Walsh, Vitousek, Wilson, & Bauer, 2004).

A simpler, categorical index of improvement is the transition from full-syndrome illness to symptoms of subthreshold intensity; however, any assumption that such change implies the underlying psychopathological process is arresting, or trending down, is premature (see below). But a quick point: we lack unequivocal knowledge of exactly what level of weight restoration, once attained, predicts continuing, uninterrupted reduction of ED psychopathology and offers robust protection against later relapse. We routinely invoke the term "ideal body weight" (IBW) when arguing for target goals in AN, as if one particular weight threshold has wide appeal and reliable empirical support, but what the term "ideal" connotes is unspecified. In what sense is a weight "ideal"? How does the attainment of "ideal" weight relate to other parameters of risk? Moreover, the use of body mass index (BMI) thresholds can be difficult, since for younger patients continued growth makes standardized BMIs unreliable. BMI percentiles are considered a better index (Le Grange et al., 2012), but the obvious notwithstanding—children and adolescents below the 10th percentile for age, height, and gender have increased likelihood of meeting criteria for AN (Hebebrand, Casper, Treasure, & Schweiger, 2004; Hebebrand, Himmelmann, Hesecker, Schaefer, & Remschmidt, 1996; Hebebrand, Wehmeier, & Remschmidt, 2000)—there is no consensus on a specific BMI percentile that defines "optimal weight" and this is likely to vary from person to person. Of course weight restoration is critical; it hardly needs mention. So the caveats argue only for more

rigorous study of what these different thresholds portend for short- and long-term clinical morbidity (psychological and physical) and functional impairment (Hall & Crisp, 1987; Lock et al., 2010; Pike et al., 2004).

The second component of the Morgan–Russell criteria is the return, or initiation, of cyclical menstruation. It was originally deemed important as an objective signal of a normalized endocrine process. Yet we know now that the absence of cyclical menstruation doesn't imply active psychopathology, risk of symptom re-emergence, or future relapse (Roberto, Steinglass, & Walsh, 2008). Further, the weight at which menstruation begins or resumes in young people with AN is highly variable (Swenne, 2004); in fact, there is no fixed expectation for menstrual regularity in younger adolescents (American Academy of Pediatrics and American College of Obstetricians and Gynecologists, 2006; Swenne, 2004). Furthermore, an outcome that incorporates a menstrual criterion obviously excludes males, and it also excludes females on certain oral contraceptives.

Bulimia Nervosa (BN)

As is the case with AN, recovery from BN has lacked a universally accepted definition, accounting in part for inconsistencies in definitions of treatment "response." Percent change in frequency of binge eating/purging has been the custom (Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000), but the long-term meaningfulness of the criterion remains unknown, just as it remains unknown if complete abstinence from binge eating and purging in the short term is predictive of the length of abstinence over extended periods (Halmi et al., 2002). Unsurprisingly, definitions of remission in BN in terms of duration also vary but are typically 4 to 8 weeks (Agras et al., 2000; Fairburn, Peveler, Jones, Hope, & Doll, 1993; Halmi et al., 2002; Mitchell et al., 2011). Thus far, how relapse probability varies as a function of this temporal criterion for remission (Grilo et al., 2007) is undetermined. It is also unknown whether inclusion of eating-related psychopathology in the definitions of remission and recovery has long-term predictive value (Cogley & Keel, 2003), and what definitions reflect valid outcome indicators in BN (Le Grange, Crosby, Rathouz, & Leventhal, 2007; Schmidt et al., 2007).

Binge Eating Disorder (BED)

The unanswered questions about defining outcome are the same for BED as with the other ED diagnoses. Although BED appears in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*; American Psychiatric Association, 2013), predictors of sustained remission or long-term outcome patterns are unexplored. Differences in the proposed minimum duration of time that qualifies as abstinence exist, ranging from 1 week (Claudino et al., 2007) to 2 months (Wilfley et al., 2008). But questions remain about whether abstinence should include both objective and subjective binge eating; the merits of defining remission dimensionally (Fichter & Quadflieg, 2007; Grilo et al., 2007); and the validity of defining remission and recovery by using discrete binge eating episodes versus the number of days that binge eating occurs in a given period of time.

Transdiagnosticism

Eating disorder phenotypes can transform, most often from AN to BN (Eddy et al., 2008; see also Chapter 55). Taking account of this, and considering further evidence of common symptoms across AN and BN (Fairburn & Bohn, 2005; Fairburn et al., 2009; Fairburn,

Cooper, & Shafran, 2002; Loeb, Lock, Greif, & Le Grange, 2012) and of shared familial transmission of the syndromes (Strober, Freeman, Lampert, Diamond, & Kaye, 2000), it is reasonable to consider recovery in transdiagnostic terms (Bardone-Cone et al., 2010). But how to do so is far from straightforward. Specifically, what period of prospective observation is needed before the likelihood of syndrome crossover has expired and recovery can be assumed? In the case of AN, data exist (Eddy et al., 2008; Tozzi et al., 2005), but their incorporation in a transdiagnostic schema would need to be tested prospectively.

The Translational Relevance of Outcome Research in Affective Disorders

How, then, might our field benefit from research in other areas of psychopathology where at least some of these questions have been considered? Indeed, treatments of unipolar and bipolar affective illness have been studied empirically for decades, and many long-term naturalistic studies of outcome have been published (Goodwin & Jamison, 2007). The implications to be drawn from this body of research are strong: like the modern narrative of AN and BN, the affective disorders are often unsparing in their long-term negative consequences (Judd et al., 2003; Paykel et al., 1995). We also know that affective symptoms can appear early in life, episodes of illness vary appreciably in their cycle lengths, and acute illness often leaves prolonged states of subsyndromal intensity in their wake, sometimes lasting for decades.

It was for these reasons, terms like remission (either partial or full), recovery, and relapse were invoked by Frank et al. (1991), who argued that different process and state factors may be driving illness trajectories over time. In this vernacular, **partial remission** refers to a brief period (typically defined as upwards of several months) in which there is a substantial (but not complete) reduction in symptom intensity (i.e., syndromal diagnostic criteria are no longer fulfilled), whereas in **full remission** *all* clinical symptoms—every single one—have abated, so that there is no evidence of *any* of the diagnostic psychopathological behavior—of any form or character. By contrast, **recovery** is invoked when symptom abatement (as with remission, partial or complete) has been maintained for a longer period (generally 3 to 6 months minimum). Thus, the duration of "significant improvement" or "wellness"—the difference between remission and recovery—has important theoretical and clinical implications. It is that the acutely applied treatment can be ended because an underlying disease, or pathogenic process, has resolved, *or* that the treatment (or treatments) applied to the acute symptoms can be continued with the aim of preventing their return.

According to Frank et al. (1991), full or partial **relapse** into a state or condition that once again warrants the diagnosis denotes a return of symptoms—meeting either full or subthreshold diagnostic criteria, respectively—within the temporally defined period of remission, whereas, in line with the definition of recovery, **recurrence** (partial or full) is the return of subsyndromal or syndromal levels of illness, respectively, *following* recovery. The practical and conceptual issue of importance here—distinguishing remission and recovery—is that as the duration of *complete* symptom abatement lengthens, the likelihood of a subsequent relapse/recurrence declines, at least hypothetically, though longitudinal studies supporting this are not available. There is a related point, which is that recovery, as defined above, does not mean that illness has resolved permanently. It may have, but as recovery is a dynamic concept, once recovery is attained, the risk of a subsequent relapse is not predictable given current knowledge. It is often assumed that you never "recover" from psychiatric illness; that the illness is always hibernating so to speak. In truth, people often enter a period of recovery that endures for the remainder of their life.

Research support for these distinctions is considerable. As Fava, Ruini, and Belaise (2007) note in a recent review of depression research, achieving full symptom remission during acute therapy for depression is a challenge that clinicians must heed, given the abundance of research showing that mild symptoms, social impairment, and negative attitudinal and cognitive biases often linger after initial clinical improvement. The point is crucial: when these features persist—characterizing a remission deemed to be partial—the risk of future relapse is elevated. Even one or two persisting symptoms of mild intensity after a period of acute therapy, whether pharmacological or psychological, have been shown to double the risk of early relapse into full depressive illness. This implies that a patient who is "very much improved" is not a patient whose short-term risk is negligible. It is not clear whether mild, persisting clinical symptoms drive the postacute social problems and dysfunctional attitudinal biases that many patients exhibit; if these social/cognitive anomalies are "scars" of an episode; or if these features are predisposing vulnerabilities that stand on their own as risk factors of long-term importance (see Chapter 67). Regardless, partial remission confers early risk of worsening symptoms, whereas a transition from acute illness to full remission, then from remission to recovery, decreases the future odds of syndromal relapse.

Still, there are caveats. In the case of bipolar illness, there is much evidence (Baldessarini & Tondo, 1998) that patients who have been maintained well on lithium prophylaxis can experience a recurrence of their disorder within months of discontinuing their medication, presumably because an active pathophysiology has remained present, albeit suppressed by the drug. Long-term *clinical* recovery, regardless of treatment approach, does not imply the absence of a persisting, "hidden" biological abnormality.

Empirical Studies in Eating Disorders

Is there value in generalizing these same concepts to the prospective study of remission and recovery in AN and BN? Reports by Strober, Freeman, and Morrell (1997) and by Kordy et al. (2002) are illustrative.

Full Recovery

In a 10–15-year prospective study of young patients with AN, Strober et al. (1997) defined recovery as a complete absence of behavioral and psychological features of illness, requiring a minimum of 8 weeks of symptom-free functioning. The results showed that, while the median time to full recovery in the cohort was lengthy, patients who achieved this status had zero risk of a subsequent recurrence of illness up until the end of the follow-up. Kordy et al. (2002) showed much the same thing using a definition of recovery as a minimum BMI of 19 (e.g., at 65 in. [1.65 m], 114 lb [51.7 kg]), a lack of extreme fear of weight gain, and no binge eating or purging for 12 months. Although the follow-up duration in their study was far shorter—2.5 years—the same trajectory was found for AN. If full recovery is achieved, risk of recurrence is very low. Importantly, the trajectory in BN was found to be quite different; specifically, the risk of relapse and recurrence following remission or recovery was substantial. Other data concur (Field et al., 1997; Halmi et al., 2002; Olmsted, Kaplan, & Rockert, 2005). Clearly, then, AN and BN reflect important differences in the processes impacting the short- and long-term course of illness.

Analogously, do empirical studies of AN and BN support the validity of applying stringent versus less stringent criteria to definitions of remission/recovery? The answer parallels what has been found in depression research. Specifically, the presence of lingering ED attitudes—for example, elevated anorexic attitudes and residual concerns about weight and shape—has been

shown to predict post-weight-restoration relapse in AN (Carter, Blackmore, Sutandar-Pinnock, & Woodside, 2004; see also Chapter 55). Cogley and Keel (2003) showed that excluding undue influence of shape and weight from recovery criteria renders differences between persons who have recovered from BN and non-ED controls negligible on measures of negative affect, body dissatisfaction, and social adjustment. Along the same lines, Bachner-Melman, Zohar, and Ebstein (2006) reported that only individuals with AN in whom cognitive and behavioral symptoms had abated were comparable to controls on measures of body dissatisfaction, disordered eating, and drive for thinness. Bardone-Cone et al. (2010) reported similar findings of negligible functional impairment and psychiatric comorbidity when recovery was defined as the absence of ED psychopathology and the presence of normal weight.

Where to place the cut-point for outcome criteria remains uncertain. Lock et al. (2010) found that relapse from full remission (defined as 95% of expected mean body weight plus scores on the Eating Disorder Examination within one standard deviation of the community mean; see Chapter 38) at the end of family-based treatment for adolescent AN (see Chapters 53 & 60) was only 10% at 1 year follow-up. In contrast, lowering the standard for remission to only a weight at 85% of expected mean body weight at end of treatment shifted the relapse rate at follow-up to 17%, suggesting that the highest possible threshold for denoting symptom abatement appears to have clinical merit. On balance, the observation dovetails with the "protective" effect of the stringent criteria for recovery shown in the studies conducted by Strober et al. (1997) and Kordy et al. (2002).

Still, the optimal duration of symptom-free observation for denoting recovery remains insufficiently studied. Up to now, varying durations have been employed, ranging from eight consecutive weeks to 1 year. Strober et al. (1997) and Kordy et al. (2002) provide good support for a criterion of 2 months of a complete absence of behavioral and cognitive symptoms in the case of AN, but a duration of 4 months for BN may be needed. Bear in mind that these studies used risk of subsequent relapse/recurrence as the validating criterion, rather than diagnostic crossover or the development of a new ED. Other arguments have been promoted. For example, 1 year of complete absence of behavioral and cognitive symptoms has been advocated, based on evidence that risk of relapse/recurrence tends to concentrate in the year following treatment (Herzog, Schellberg, & Deter, 1997).

So it is intuitive that as the stringency of outcome definitions increases, the proportion of participants in treatment and follow-up studies who meet the threshold lowers. For example, in Couturier and Lock's (2006a) study of remission in a trial of treatment for adolescent AN (Lock, Agras, Bryson, & Kraemer, 2005), depending on the criteria set employed, the proportions of sustained recovery ranged from a high of 96% to a low of 3%. As regards the validity of these differences, the question looms: does the satisfaction of a particular definition bear on risk of future relapse, or the subsequent likelihood of recovery? Concerning this question, Couturier and Lock (2006b) conducted a predictor analysis on follow-up data with a mean follow-up assessment point of 3.95 years (range 2.3–6 years) post-treatment. Defining recovery as weight greater than 95% IBW, higher end-of-treatment BMI and lower age were predictors of full recovery.

The Challenges Ahead

Eating disorders rarely emerge without prior developmental indicators of risk. As revealed in numerous studies (Bulik et al., 2006; Fairburn, Cooper, Doll, & Welch, 1999; Fairburn et al., 1998; Fairburn, Welch, Doll, Davies, & O'Connor, 1997; Field, 2004; Jacobi, Hayward,

de Zwaan, Kraemer, & Agras, 2004; The McKnight Investigators, 2003), both AN and BN are commonly foreshadowed by a number of anxious, obsessional, and compulsive traits (principally in persons with AN), and by a variety of behavioral and contextual factors in persons with BN, including body image and eating attitudes. The unfolding of AN and BN is deservedly viewed as developmental in character.

Two interconnected points follow then. First, as in other psychiatric disorders, residual symptoms (not the least of which is persisting low body weight or sporadic binge eating) frequently track a patient's course over time. Second, beyond the fact that long-term outcome trajectories are not easily predicted, correspondences between premorbid risk factors, acute morbidity indictors, persisting symptoms, nonsymptom features of functional impairment, contextual factors, and long-term clinical outcome have yet to be properly studied. How long these features remain after weight restoration or abstinence from binge eating, and in which subsets of patients their persistence is more likely, remain unknown. On the other hand, clarifying these temporal trajectories is a tall order. For example: Do individual factors correspond to specific state and process factors? Are certain contextual influences more relevant than others? Do factors such as early life stress, affectional bonds, or parenting styles bear a relationship to predisposing biological abnormalities? How are these processes expressed in gene, brain, and environmental transactions? These are but a few of the meaningful questions to resolve given the unique complexities that shape EDs.

Unfortunately, some of the questions meet, at times, with no small measure of irritation in clinical discussions: "We need to stop debating the cause of our patients' suffering and get moving on the business of getting them well," has been argued in conference forums. Granted, psychiatric research has a long list of failures to deliver on the promise that knowledge of cause will lead to plausible therapies. But this is not always true. A case in point is research on maintaining factors in mood and anxiety disorders, which has led to effective adjunctive therapies (Miklowitz et al., 2013; Peris et al., 2008).

There are myriad ways in which treatment approaches can be sequenced over time (see Chapter 50). There are also complex and dynamic interactions that must underlie the timing and patterning of their effects, and the possibilities for enriching knowledge of treatment optimization—how to change illness trajectories—can't be divorced from considering how to characterize remission and recovery in future research studies. From the vantage points of remission and recovery, crucially important therapeutic questions remain for which outcome research is central: What illness processes are best targeted early? Do multiple, acutely delivered interventions accelerate time to remission? Do particular symptoms forecast early relapse in cases of partial remission? Do persisting temperamental or social impairments after full remission increase risk of recurrence? What individualized interventions might sustain a longer maintenance of remission and recovery, and for which subsets of sufferers? Exactly what periods of observational study are needed to determine that a given patient's long-term outcome status can be reliably assumed?

Conclusions and Future Directions

Importantly, given the heterogeneity within AN and BN (see Chapters 2, 3, 8, & 9), whether we are examining treatment effects or outcomes observed during the course of naturalistic, prospective study, what holds for one subset of patients may be only half true for another. This is why identifying prognostic indicators will be crucial for anticipating contrasting outcomes

across a range of different interventional modalities. More problematic, or at least challenging in different ways, is the question of how to define outcomes—naturalistic or in treatment trials—in chronic, unremitting presentations (see Chapter 12). In such cases, recovery is sometimes not a reasonable expectation for a proposed course of therapy, but optimization of function and improving the patient's sense of well-being are desirable and reasonable outcomes (Hay, Touyz, & Sud, 2012; Strober, 2010). The point is that outcome in persons with longstanding illness doesn't lend itself to determinations of remission and recovery as conventionally defined. This caveat underscores the importance of thinking broadly about outcomes.

We have provided an overview of the importance of coming to terms with the need to define outcomes, particularly remission and recovery in EDs. Without clear definitions of these outcomes the meaning of our treatments for our patients and their families is frustratingly vague, and the ability to compare effectiveness of treatment strategies is not possible. Elements to address in defining remission and recovery include change in symptoms, duration of change, and markers for relapse, but there is little agreement about how to do this. At the same time, our review of the progress to date on this topic indicates that, although we are far from being able to agree on a common definition of recovery in EDs, we have made some progress in understanding the main dilemmas in facing this issue, including the role of diagnosis, use of particular outcomes, duration of abatement of symptoms, and the problem of setting realistic expectations given current therapeutic tools. More challenging is the relationship of remission and recovery from EDs to health and well-being more generally.

In closing, the landscape of illness behavior has changed dramatically in the last decade. How this knowledge extends to EDs is unclear, but the opportunities are many. Today, we marvel at technologies that shed light on the organization of complex behaviors. For example, we are able to view the brain's properties with subcellular resolution. We understand better the separate facets of emotional experience and the neural architecture that supports them. We can see how gestational and postnatal events could influence future response to challenging environments and how these biological processes mediate these contrasting adaptations, as well as how polygenes affect response to psychiatric treatments. The findings that emerge remind us not only that the dialogue between genome, biology, experience, and disease has profound consequences for behavioral change throughout development, but also that outcome research is important to a further understanding of this dialogue. In short, determining in a particular sample the proportion of patients within different treatment protocols who attain full or partial remission or recovery is no more or less an important question than what set of factors influence differences in outcomes throughout the life cycle. The opportunities for future progress in shedding new light on the underpinnings of vulnerability and resilience in our patients is great.

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65

Is Obesity an Eating Disorder?

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Obesity is a term used to describe the condition of having an elevated body weight, and is most commonly defined by the construct of body mass index (BMI), a ratio of weight divided by height squared (kg/m^2), with cut-points determining classes for "normal weight" (18.5–24.9 kg/m^2), "overweight" (25–29.9 kg/m^2), and "obese" (≥30 kg/m^2). Obesity is further subdivided into the following classes: class I (BMI = 30–34.9 kg/m^2), class II (BMI = 35–39.9 kg/m^2), and class III (>40 kg/m^2). Obesity has not appeared in the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (DSM) previously. Although it was considered for inclusion in *DSM-5* (Devlin, 2007), ultimately it was not included. In this chapter we will consider what is known about the etiology of obesity and review conceptualizations for psychiatric disorders. We will then consider research regarding current nosological views of obesity to assess any benefit of classifying it as an eating disorder (ED).

Obesity Epidemiology and Risk Factors

Prevalence and Demographics

The prevalence of obesity in adults in the United States increased from 14.5% in the late 1970s to 30.5% in 1999–2000 (Flegal, Carroll, Kuczmarski, & Johnson, 1998; Flegal, Carroll, Ogden, & Johnson, 2002). The prevalence has stabilized for most groups since that time. The most recent data, collected in 2011–12, show a prevalence of 34.9% for adults overall, with significantly higher estimates of obesity among women, non-Hispanic Black adults, and middle-aged persons (40–59 years; Ogden, Carroll, Kit, & Flegal , 2014).

Generally, obesity is thought to result from a combination of reduced activity and increased calorie intake, creating an energy surplus, which is then stored as adipose tissue. But how does this actually occur? What are the factors that have contributed to an increase in the prevalence of obesity in the population?

Although there is no single, recognized cause of obesity, there are identified risk factors. There is a genetic predisposition to developing obesity, with environmental factors playing an

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important role in pathogenesis. However, the genetic factors predisposing to obesity are not universally evident, and the constellation of environmental factors varies between individuals.

Genetic Risk Factors

Based on data from twin studies, genetic factors account for almost 65% of variation in body weight (Segal, Feng, McGuire, Allison, & Miller, 2009). The first gene identified in the etiology of obesity was that for leptin in the mid-1990s. Leptin is a hormone produced by adipose tissue that signals satiety and reduces food intake by binding to its receptors in the central nervous system (CNS). Mutations in the leptin gene (or in the leptin receptor gene) that render leptin unable to bind to its receptor are associated with extreme obesity in animal models and in humans. These mutations are exceedingly rare and do not explain the cause of obesity in most cases. Since the identification of leptin, the advent and availability of genome-wide association studies have led to identification of multiple genes associated with obesity. However, the identified genes account for a small percentage of the variation in body weight. One study identified variations in 18 genes associated with obesity that accounted for less than 4% of variation in body weight (Speliotes et al., 2010). Thus, as yet, we do not have a clear understanding of the specific genetic sources of the proportion of individual differences in body weight that is attributable to heredity, nor of which genes these are and how they function to create and sustain an increase in body weight.

Environmental Risk Factors

Epidemiologists have identified multiple environmental factors that are correlated with increasing BMI in the population. These risk factors include increased television watching (Nguyen & El-Serag, 2010; see also Chapters 29 & 45), which seems to be mediated through an increase in snacking that accompanies TV viewing rather than a reduction in activity level (Jackson, Djafarian, Stewart, & Speakman, 2009). Researchers and public health experts also regularly cite the availability of cheap, high-calorie but low-nutrient-dense foods (and the relatively low availability of fresh foods) as contributing factors (Lovasi, Hutson, Guerra, & Neckerman, 2009; see also Chapter 49). Physical activity levels, which can depend on the quality of local parks and personal safety issues, may also contribute to obesity (Gordon-Larsen, Nelson, Page, & Popkin, 2006; Nelson, Gordon-Larsen, Song, & Popkin, 2006). Additionally, there are many medications associated with weight gain (see Chapter 59), including atypical antipsychotics, some antidepressants (especially tricyclic antidepressants and atypical antidepressants like mirtazapine), blood pressure medications (such as alpha- and beta-adrenergic blockers), antidiabetic medications, some antiepileptic medications (e.g., valproic acid, carbamazepine), and chronic oral corticosteroids (Malone, 2005).

Despite identified risk factors, many people with environmental and genetic risk factors *do not* develop obesity, and individuals can develop obesity *without* these risk factors. Additionally, treatments targeting these risk factors have not demonstrated significant, sustained weight reduction for most people. This has led researchers to consider the role of brain function in behaviors related to eating and activity, with neurobiology representing an increasingly active area of interest. In other words, are there individual, neurologically based responses to environmental risk factors and cues that predispose some people to obesity? Our understanding of

the role the brain plays in body weight has grown with the availability of neuroimaging. Below, we review current information about neurological processes related to eating behavior, and what is known about their role in obesity.

Eating Behavior

There are several eating behaviors that may be related to weight status. Rate of eating has been assessed in children, both in lab studies and by self-report (Berkowitz et al., 2010; Murakami, Miyake, Sasaki, Tanaka, & Arakawa, 2012). Berkowitz and colleagues (2010) examined the eating behavior of 4-year-olds, 32 of whom were born to overweight and obese mothers and 29 born to lean mothers, in a feeding lab. Rate of eating, as measured by mouthfuls per minute, was associated with increased odds of obesity, higher body fat, and increased skinfolds at the age of 6 years, suggesting that fast eating rate may be a behavioral marker for the development of obesity in children. Further, a Japanese study by Murakami et al. (2012) surveyed almost 16,000 children and 8,000 adolescents regarding their eating rate and weight status. There was an increased risk of obesity for those reporting relatively fast or very fast eating; the odds ratios, respectively, were 2.8 and 4.5 among male children, 2.7 and 5.7 among female children, and 2.3 and 3.8 in male adolescents. No significant risk was associated with eating rate for obesity among female adolescents. As a descriptive feature of binge eating is rapid consumption of food, this is an interesting behavior to assess prospectively, given evidence of its early onset in childhood.

Eating in the absence of hunger is another behavioral descriptor of binge eating that has been linked to overweight status. This variable is assessed in a feeding lab by presenting a meal to children and asking them to eat until they are full. A short time later, they are presented with a buffet of snack foods that they are free to eat. As reviewed by French, Epstein, Jeffery, Blundell, and Wardle (2012), studies consistently find that overweight children eat more in the absence of hunger than do normal weight children. Although there have been few prospective studies, children tend to show increased risk of weight gain with greater eating in the absence of hunger. This eating behavior may indicate problems with lack of satiety as well as increased enjoyment of food, both of which are independently related to increased weight status in cross-sectional studies of children (French et al., 2012).

Finally, food preferences as influenced by the family of origin and/or by one's food environment may also profoundly impact one's eating behaviors. These may include preference and cravings for high-fat and highly processed food items; frequent consumption of fast food, take-out foods, or meals at restaurants; and low intake of fruits and vegetables. These eating styles may be influenced by cultural factors, socioeconomic status, time available for food preparation, and modeling of unhealthy cooking in the family (see Chapter 23). One study of preschool children found that obese children with at least one obese caregiver, as compared to healthy weight children without any obese caregivers, were less likely to have fresh vegetables, more likely to have a television in the child's room, and less likely to own exercise equipment, as determined by in-home visits (Boles, Scharf, Filigno, Saelens, & Stark, 2013). Additionally, while eating meals as a family has been linked to lower levels of disordered eating, the quality of meals from a nutritional standpoint influences weight. In a study of adolescent African Americans and their families, those whose caregivers prepared foods with less healthy cooking methods as compared to those preparing meals with healthy cooking methods were at increased risk for overweight and obesity (Kramer et al., 2012).

Review of Hormonal and Neurological Regulation of Appetite and Eating

Appetite is regulated by a complex interaction between neurological and hormonal systems, and the interplay between these and environmental factors. One way to conceptualize obesity is a failure of the homeostatic system that regulates energy intake, energy expenditure, and thus weight. Multiple hormones and peptides play a role in the homeostatic system, including peripherally produced satiety signals and signals that promote hunger and eating.

Satiety signals are released from the gastrointestinal (GI) tract and bind at the vagus nerve or in the CNS. These signals involve cholecystokinin, serotonin (see Chapter 30), peptide-YY, glutamate, and glucagon-like peptide-1 (Faulconbridge & Hayes, 2011). Other signals are released from the pancreas (e.g., insulin, glucagon), adipose tissue (e.g., leptin, adiponectin), and the GI tract (e.g., ghrelin) and act directly on the CNS, either to promote eating or signal satiety. Areas of the brain involved in regulating homeostatic energy balance include the hypothalamus and the nucleus tractus solitarius (Faulconbridge & Hayes, 2011).

Is obesity caused, then, by a disruption in the homeostatic system regulating eating and weight? Some imaging studies have shown sluggish homeostatic responses, suggested by the absence of an inhibitory response in the hypothalamus after eating in obese men compared to lean men (Carnell, Gibson, Benson, Ochner, & Gelieber, 2012). In other words, the hypothalamus, believed to be central to maintenance of body weight, shows less inhibition after eating for obese compared with lean participants. Additional imaging studies comparing obese participants with those who were once obese but are now normal weight also demonstrate the absence of an inhibitory response in brain regions involved in homeostasis in both obese and previously obese participants compared with lean individuals (Cornier et al., 2009). Studies comparing lean with obese individuals are limited to yielding data that describe associations, not information about causality.

This gut-CNS system works to regulate energy balance as one aspect of appetite and weight regulation. Factors other than energy needs contribute to appetite and eating behavior, and in some cases may contribute to weight gain. These include the rewarding aspects of food. Neuroimaging has also been utilized to examine the potential role of neurological circuitry and neurotransmitters related to reward in obesity; this topic will be reviewed later in the chapter.

What is a Psychiatric Disorder?

Given what we know about obesity, it is important to consider how this may or may not fit as an ED, which is a form of psychiatric disorder. The conceptualization of psychiatric disorders has changed over the past century. The first *DSM* was published in 1952, less than 10 years after the end of World War II (American Psychiatric Association, 1952). The classifications were based on the theory that all psychological neuroses and conditions were reactions to conflict between the psyche and the environment (First, 2010). Precise descriptions were not provided in this original document, as it was thought that no one could know what the underlying pathology really was that caused the observable symptoms (Wilson, 1993). In 1968, *DSM-II* was published with similar diagnostics as in the first *DSM*, but the nomenclature was updated to share terminology with the *International Statistical Classification of Diseases and Related Health Problems* (*ICD-8*; American Psychiatric Association, 1968; First, 2010).

In 1980 the *DSM-III* introduced a radical paradigm shift to theoretical descriptive diagnoses, as compared to psychodynamically, etiologically based diagnoses, in order to increase diagnostic reliability and produce effective treatment options, such as the use of lithium for bipolar disorder (American Psychiatric Association, 1980; First, 2010; Regier, 2007). Thus, discrete, categorically based criteria were derived for a variety of syndromes, with the hope that disorders defined in this way could be better targeted by pharmaceutical and therapy treatments, similar to medical classification systems. These changes were also aimed at decreasing the stigma associated with mental disorders. The number of categories increased, as did the number of comorbidities, but there were still difficulties with its ease of use in clinical settings, given that patients are unique and heterogeneous. Some of these concerns were addressed with revisions in *DSM-III-R*; for example, diagnostic hierarchies were largely dropped, allowing for the diagnosis of several disorders at once (American Psychiatric Association, 1987; First, 2010; Regier, 2007).

DSM-IV was released in 1994 (American Psychiatric Association, 1994). Notably, the impact of functional impairment or distress was introduced. Other changes were based on extensive literature reviews, and were made only if there was compelling evidence to do so. Therefore, no sweeping paradigm changes were made between *DSM-III* and *DSM-IV*, or even with *DSM-IV-Text Revision* (*DSM-IV-TR*; American Psychiatric Association, 2000). Complaints grew, however, including overuse of the "Not Otherwise Specified" categories, which has been problematic for the EDs in particular (Regier, Kuhl, Narrow, & Kupfer, 2012; see also Chapters 4 & 11). Over half of persons seeking treatment under the *DSM-IV* system typically fell within the Eating Disorder Not Otherwise Specified (ED-NOS) category (Eddy, Celio, Hoste, Herzog, & Le Grange, 2008; Fairburn & Bohn, 2005).

The later editions of *DSM* were based on expert consensus. As research continued to advance our knowledge of the genetics and biological basis of psychological functioning, the desire to base diagnosis on these etiological underpinnings grew. With this knowledge, more overlap across disorders was recognized, such as the success of selective serotonin reuptake inhibitors for mood, anxiety, and EDs (First, 2010; see also Chapters 15, 54, & 59). Thus, the directive for *DSM-5* (American Psychiatric Association, 2013) was to try to move beyond a categorical approach to diagnosis toward a continuum approach based on pathophysiological data. Putatively, this would reduce the number of comorbid diagnoses and improve diagnostic validity (Regier et al., 2012). However, the call for such sweeping change was ahead of the scientific data, as the understanding of genetics, neuroimaging studies, developmental models, and basic science, among other topics, still contain large gaps that will not likely be filled even in the coming decade (see Chapters 1, 64, & 67). The field remains a great distance from establishing sensitive and specific pathophysiological tests for identifying psychiatric disorders.

Switching from a category-based diagnostic system to a continuum-based system would invalidate our knowledge of treatment efficacy for any given disorder and cause great disruption in treatment delivery and insurance-based reimbursements. For example, in a spectrum approach, EDs may be grouped as disorders of low weight or disorders of purging or disorders of binge eating, with overlap allowed among categories. There may also be an obsessive-compulsive spectrum of disorders that could include AN, or an addictions spectrum disorder that could include food addiction and obesity. Short of such a major shift, *DSM-5* (2013) includes dimensional ratings of severity for a set of symptom domains that are transdiagnostic, but not diagnostic continua.

So where does obesity fall in this historical account of diagnostics? As reviewed above, obesity has been viewed most often as a medical disorder. What differentiates this state or condition

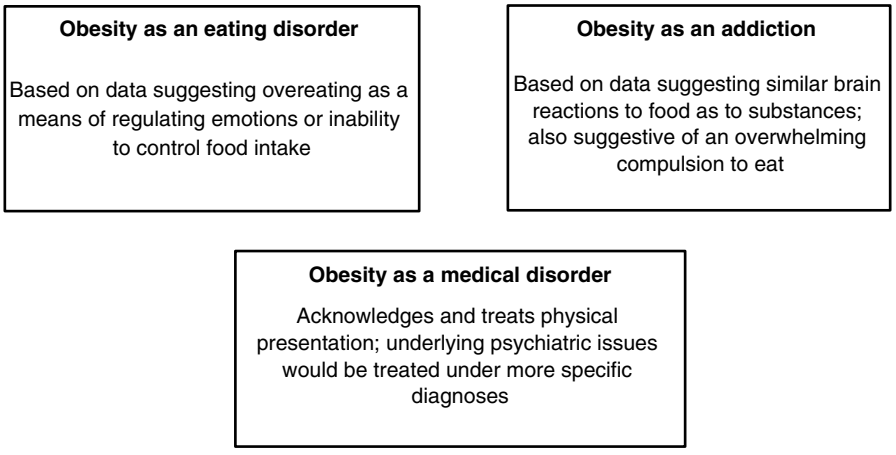

Figure 65.1 Proposed conceptualizations of obesity.

from, say, hypertension or diabetes, are the behavioral components of eating and physical activity (or lack thereof) that are so central to its development. Practically speaking, these behaviors also contribute to the development of hypertension and diabetes, but no one is proposing to include those disorders in the *DSM*. Obesity has components of both EDs and substance use disorders (SUDs), as addressed below, so the advent of a spectrum or continuum diagnostic model might better encompass obesity as a psychiatric disorder than the categorical system currently employed. These possibilities are depicted in Figure 65.1.

Obesity as an Eating Disorder

The existing ED definitions are based on categorical descriptions comprised of symptoms and behaviors (see Chapters 1–4, 8–11, & 13). These include criteria such as binge eating, overvaluation of weight and shape, and purging. It is fairly clear when these thoughts or behaviors are present during the course of an ED. So, turning to obesity, what universal symptoms could be associated with high body weight? Currently, there are no behavioral, emotional, or cognitive features in the definition of obesity. Devlin (2007) posits that Western cultures have a moralistic view of obesity that places responsibility for weight status on the individual. He points to two problems with this view. First, as reviewed above, obesity is influenced by several factors, such as genetics and the food environment. Second, persons with obesity may maintain their weight—they do not have to be in a constant state of energy imbalance. To the contrary, they could be eating quantities consistent with weight maintenance or loss, much as persons of normal weight may eat for extended periods of time while still maintaining an "abnormal" BMI.

Given these points, diagnostic criteria for obesity would be difficult to define. Eating patterns and behaviors of persons in this weight category vary *between* individuals at any given time and *within* individuals across time. Therefore, static criteria, such as a certain number of kilograms gained per week, would not be of much use. A criterion such as "consumption of fast food more than three times per week" would also not be useful, as persons of any weight may engage in this behavior, and many persons with obesity may never eat at fast-food restaurants.

A requirement of most psychiatric disorders is the presence of distress or impairment in functioning (*DSM-5*, 2013). Studies of mood and quality of life suggest that depression increases and quality of life is worsened with increasing BMI (Ul-Haq, Mackay, Fenwick, & Pell, 2013). Examples of this from clinical experience often include difficulties fitting into booths in restaurants or rides at amusement parks, and having to purchase two tickets when traveling by airplane. Further, some studies suggest that the relationship between BMI and health-related quality of life is mediated by other factors such as body image (Cox et al., 2011; see also Chapters 21 & 22), binge eating (Ranzenhofer et al., 2012), and employment (Lund et al., 2011). The effects on weight-related quality of life are most pronounced at stage III obesity, as is increased risk of mortality (Ul-Haq et al., 2013). Additionally, for some people who are obese, it is the substantial societal bias against them that creates, or at least contributes to, distress or impairment in the obese individual, rather than being a particular weight itself (Puhl & Brownell, 2001; see also Chapter 41). In these cases, classifying obesity as a psychiatric illness is reminiscent of including homosexuality in earlier *DSM* iterations. Finally, distress or impairment in functioning could be measured by including obesity-related medical comorbidities (e.g., diabetes mellitus, heart disease, some cancers, or obstructive sleep apnea), or by disability status due to weight-related limitations (e.g., lack of mobility, inability to perform one's duties). Again, these comorbidities may be present in persons of normal weight, and not present in persons of extreme weight, making them nonspecific diagnostic criteria.

Eating Disorders Related to Obesity

There are two categories of disordered eating—binge eating disorder (BED; see Chapters 4 & 10) and night eating syndrome (NES; see Chapters 4 & 11)—that are often associated with obesity. Stunkard, Grace, and Wolff (1955) first noted the latter association in their paper describing NES among patients seeking treatment for their obesity. Four years later Stunkard (1959) described the pattern of "binge eating syndrome" among a similar group of patients. For many years the attention of researchers turned toward describing AN and BN more rigorously, but, in the 1990s, as the prevalence of obesity rose in the general population, researchers started examining specific phenotypes of obesity that could possibly be targeted and modified, including BED (Spitzer et al., 1992, 1993) and NES (Birketvedt et al., 1999; Stunkard et al., 1996).

Binge Eating Disorder

Prevalence of BED in the general population for the United States falls at 2.6% (Kessler et al., 2013; see also Chapters 5 & 10), increasing incrementally with weight. Among a larger, international sample of over 24,000 participants, people who were overweight (BMI = 25–29.9 kg/m^2) were 1.3 times more likely, and those who were extremely obese (BMI > 40 kg/m^2) were 6.6 times more likely than normal weight respondents to have a history of BED. BED is also linked prospectively with weight gain. Persons with BED were found to gain a mean of 4.3 kg (9.5 lb) in the year before presenting for treatment, with larger weight gains significantly related to severity of the binge eating (Barnes, Blomquist, & Grilo, 2011). BED was also linked to impairment in role functioning (i.e., work, home, social life, relationships) for just under half of respondents (Kessler et al., 2013). Finally, BED is associated with increased risk (odds ratios) for chronic neck and back pain (1.5), other chronic pain conditions (1.8), diabetes (2.4), hypertension (1.8), and chronic headaches (1.8) (Kessler et al., 2013).

Night Eating Syndrome

The prevalence of NES also increases with weight, ranging from 1.1 to 5.8% in the general population and community studies (de Zwaan, Müller, Allison, Brähler, & Hilbert, 2014; Lamerz et al., 2005; Rand, Macgregor, & Stunkard, 1997; Striegel-Moore et al., 2005; Tholin et al., 2009; see also Chapter 11), 6 to 16% in persons seeking outpatient weight loss treatment (Adami, Campostano, Marinari, Ravera, & Scopinaro, 2002; Calugi, Dalle Grave, & Marchesini, 2009; Gluck, Geliebter, & Satov, 2001; Stunkard et al., 1996), and 8 to 55% among those seeking bariatric surgery (Allison et al., 2006; Latner, Wetzler, Goodman, & Glinski, 2004; Mitchell et al., 2014; Powers, Perez, Boyd, & Rosemurgy, 1999; Rand et al., 1997). Tholin and colleagues (2009) showed an increased risk for obesity (2.5 times for men and 2.8 times for women) among participants who screened positive for night eating in the Swedish Twin Registry STAGE cohort. Additionally, among a sample of outpatient psychiatric patients, those meeting criteria for NES were five times more likely to be obese than patients without NES (Lundgren et al., 2006).

NES status also predicts weight gain prospectively. Andersen, Stunkard, Sørensen, Petersen, and Heitmann (2004) reported that women who endorsed night eating in the Danish MONICA study cohort gained 5.2 kg (11.5 lb) more over 6 years than women without night eating; this trend was not shown for men. Gluck, Venti, Salbe, and Krakoff (2008) studied participants in an inpatient study who ate ad libitum during a 3-day monitoring period. Those who ate after 11:00 p.m. gained a mean of 6.2 kg (13.7 lb) as compared to 1.7 kg among those who did not eat after 11:00 p.m., measured 3.4 years later, on average.

Finally, night eating may exacerbate medical conditions such as diabetes mellitus. Morse, Ciechanowski, Katon, and Hirsch (2006) found that diabetic patients with evening hyperphagia were more likely to have hemoglobin A1c values greater than 7 (indicating consistently elevated blood sugar) and to have two or more diabetic complications. Hood, Reutrakul, and Crowley (2014) also recently showed that night eating was related to hemoglobin A1c values exceeding 7. Thus, these results from various populations and methodologies strongly suggest that NES may contribute to weight gain or, at the least, the maintenance of higher weight, in those who suffer from it compared with those who do not.

Eating Disorders and Emotion Regulation

For all of the EDs, there is evidence supporting an affect regulation/stress response model, which posits that disordered eating behavior functions to regulate emotions, especially negative emotional states (Devlin, 2007). For people with BED who, by definition regularly engage in overeating behavior, the model posits that they have difficulty tolerating emotions such as sadness, anxiety, and anger. These aversive internal states are temporarily but quickly relieved by binge eating, which reinforces binge eating as a coping skill (Telch, Agras, & Linehan, 2001). In other words, people with BED are conditioned to binge eat as a means of removing a negative stimulus (i.e., binge episodes are negatively reinforced). However, while there is a higher rate of obesity in populations with BED and NES, the majority of people whose weights are classified as obese do not regularly engage in binge- or night-eating behavior, and this ED model does not seem to apply for this population who are not overeating in response to aversive stimuli.

Reward Circuitry: Obesity as a Disorder of "Addiction"?

An alternative conceptualization of obesity as a psychiatric disorder would be as an "addiction," or the misuse of food as a SUD. Addiction models posit that eating is intrinsically rewarding, which may be on the same spectrum of conditioned responses as BED (i.e., eating is immediately rewarding). There are data supporting the hypothesis that people with obesity may be predisposed to such conditioning, and in fact may be somewhere on the spectrum between lean individuals and those who engage in binge eating behavior. Neuroimaging studies suggest that people with binge eating have increased responses to food cues in areas of the brain associated with reward, motor planning, and cognitive control; these responses are present but less intense in obese people who do not binge eat, who in turn have more pronounced responses than people whose weight is in the normal BMI range (Carnell et al., 2012).

Compared to normal-weight participants, obese participants show increased activation in brain regions (see Chapter 17) associated with reward processing (anterior cingulate and medial prefrontal cortex, ventral and dorsal striatum, insula, amygdala, orbitofrontal cortex [OFC], hippocampus, and ventral pallidum) in the presence of food cues in a fasting state (i.e., anticipatory activation), suggesting they have an increased anticipatory food reward. Those with obesity also show differences in a fed state, compared to normal-weight controls, in response to food cues: greater activation in lateral OFC, caudate, and anterior cingulate cortex. Other studies report reduced activation of the caudate in obese individuals in the fed state, and weaker activation may suggest lower actual reward (De Silva, Salem, Matthews, & Dhillon, 2012). Stice, Spoor, Bohon, Veldhuizen, and Small (2008) postulate that obese individuals have higher anticipated reward and reduced consummatory reward, which together contribute to overeating. Additionally, studies find an association between obesity and increased activation in response to visual, olfactory, and gustatory food cues in brain regions involved in reward, motivation, emotion, and memory (Carnell et al., 2012). Thus, some people may experience abnormal activity in brain reward circuits in response to food and eating, predisposing them to energy imbalance and obesity. This is similar to individuals with SUDs, who show increased activity in these circuits in response to psychoactive drugs (Dawe & Loxton, 2004).

Volkow, Wang, Tomasi, and Baler (2013) outline and summarize the evidence supporting a neurobiological overlap between obesity and addiction. They note the common involvement of dopamine circuitry in food intake and drug intake, and that the changes that occur when an individual progresses from drug use to abuse or dependence and associated compulsive behaviors are also evident in studies of obesity. Volkow et al. (2013) also note that obese subjects show decreased activity in frontal brain regions associated with executive function and cognitive control. This is a consistent finding in drug-addicted patient populations as well. Further, there is circuitry involved in motivation that is dysregulated in drug-addicted and obese populations. These changes are associated with enhanced motivation to seek drugs, despite the negative consequences of doing so. Volkow et al. further outline the abnormal functioning of the insula, a brain region related to interoceptive awareness and involved in the integration of homeostatic input with emotion and motivation (see Chapter 17). The insula is implicated in cravings for food, cigarettes, and cocaine, and appears to function differently in obese than in lean subjects.

However, as Ziauddeen and Fletcher (2013) suggest, there are limits to our current understanding of the neurobiology of obesity as a type of addiction. They identify shortcomings with using BMI alone as a marker of addiction, as there are people in the "normal" BMI range who may show patterns of behavior more readily described as food addiction than do some

people with a BMI exceeding 25 kg/m^2. Moreover, despite the great deal of enthusiastic attention paid to "food addiction," evaluations of the function of brain regions known to be associated with drug addiction frequently yield conflicting findings within and across studies of obese populations.

Diagnostic Considerations: Can Eating Be an Addiction?

DSM 5 (2013) changed the conceptualization of addictive disorders, with substance abuse and dependence being combined, and the section titled "Addiction and Related Disorders." This broadening allows room for other types of addictive behaviors seen clinically and discussed in the media, such as gambling, Internet, and sex. However, a review by Moreno and Tandon (2011) found that no consensual definition of "food addiction" exists. Several details would need to reach consensus for this discussion to continue, such as evaluating weight versus calories; *how* versus the type of foods one eats; and the temporal pattern of food intake (Moreno & Tandon, 2011). Finally, we should consider how closely overeating parallels the *DSM-5* addictions criteria.

There are several concepts that match between SUD and "food addiction." First, they are both maladaptive patterns of behavior that tend to be associated with clinically significant impairment or distress. SUD requires use in physically hazardous situations; while there is no direct connection, perhaps eating in the presence of obesity-related medical comorbidities would fit. SUD also requires substance use resulting in failure to fulfill major role obligations; eating does not typically interfere to this extent (Moreno & Tandon, 2011).

More problems arise as we examine the role of tolerance. Tolerance in SUD is seen in the need for an increasing amount of substance to achieve the desired effect, and a diminished effect with continued use of the same substance. While persons with BED may increase the amount they eat over time, this is generally not the effect seen with overeating. In particular, there is no diminished effect (Moreno & Tandon, 2011). In fact, as Wilson (2010) states, children crave and eat more sweets than adults do. This pattern is the opposite of that described for tolerance in substances. If there were a true sugar addiction, we would see that people needed more and more, say, lollipops, over time, to get a desired reduction in cravings. The concept of withdrawal also remains problematic, as there is no clear parallel between a withdrawal state from substances and that from craved foods. Although some patients describe distress and continued cravings, this experience is not well defined, nor do we understand how common it is.

From a treatment perspective, there are several issues with considering obesity, or overeating, an addiction. Cognitive-behavioral therapy (CBT) is a treatment that challenges patients to increase the flexibility of their eating and to eat regularly throughout the day to increase control over their eating (see Chapter 56). It does not suggest that patients remove trigger foods or certain food groups from their diets altogether. As CBT is considered an effective treatment for bulimia nervosa (BN) and BED, the addictions treatment model of avoiding particular foods flies in the face of the evidence that individuals with impulsive or compulsive eating can, in fact, successfully incorporate a wide variety of foods in their diet with treatment (Wilson, 2010).

Therefore, while the brain circuitry may be quite similar between persons with SUD and those with BED or overeating issues, there are many differences between the two that call for further research before overeating is considered an addiction. This is not to say that

consideration of calorically dense foods with little nutritional value should not, perhaps, be studied more closely regarding their impact on those who are most susceptible to overeating, as well as on the general population. Gearhardt and Brownell (2013) propose optimizing policies regarding the availability of such "addictive" foods or their taxation, much as cigarettes and alcohol have been regulated to minimize their impact on those with addiction issues and on society more generally. Some such initiatives (e.g., taxing sugared soft drinks) have been defeated in cities such as New York and Philadelphia thus far, while food labeling regarding sugar and fat content has improved.

The Potential Impact of Inclusion of Obesity as an Eating Disorder

Along with the scientific arguments, we must also consider the psychological impact of designating obesity as an ED. The benefits of designating a condition a "psychiatric disorder" should outweigh the cons. Obesity already carries a significant burden of stigma in our society, with some researchers suggesting that "obese persons are the last acceptable targets of discrimination" (Puhl & Brownell, 2001, p. 788). Many advocates for those with mental illnesses, including EDs, have battled back against stigma. AN and obesity are similar in that they are "visible" disorders, as opposed to BN or, say, generalized anxiety disorder. Persons suffering from the former disorders are much more likely to be stereotyped or stigmatized without any interpersonal interactions even occurring. The benefits of respect and of access to care and effective treatment should supersede the increase, or at least solidification, of stigma that would likely occur with labeling obesity as a mental disorder.

In the case of AN, the benefits have seemingly outweighed the cons, as weight restoration is only part of the picture, and, usually, longer-term psychiatric intervention is needed for successful treatment (see Chapter 64). In the case of obesity, as a general category, it seems unlikely that designation as a mental disorder would improve our approaches to care. Weight management requires long-term attention and care, much like caring for diabetes, but such long-term management does not necessarily have to take the form of psychiatric treatment. Treating those with BED, NES, or possibly "food addiction" (as the phenomenon is studied further), yields benefits, but as discussed above, lumping all persons with a BMI above 30 kg/m^2 into a single disorder would result in a widely heterogeneous category. Such a grouping would not likely improve care, and depending on the wording of the diagnostic criteria, may capture persons who were not distressed or limited in their functioning in any significant way. This remains a significant detraction for including obesity as an ED.

Conclusions and Future Directions

DSM categories are not phenotypes. Persons are complicated and eat for a myriad of reasons, particularly in cultures where food is abundant. Persons also gain weight for many reasons, including genetic predisposition, one's food environment, access to physical activity, cultural food preferences, access to low-calorie foods, external stressors, and emotional dysregulation, to name a few. In this chapter we have considered obesity in the context of the models described in Figure 65.1. With a shift in directives for mental health research to examine psychiatric phenomena along continua, as opposed to categories with checklists of symptoms, research will continue to examine the underlying genetic, physiological, behavioral, and psychosocial data

for symptoms that cut across categories—for example, substance and sugar addiction or general overeating and emotion regulation. Advancement of brain imaging and genetics studies could be fruitful as our approaches become cheaper and more widely accessible, but it will be important to replicate findings both in identifying patterns and traits *within* those who are obese, and *across* symptom groupings among those who suffer with various addictions or EDs. Better understanding of the experience of extreme food cravings and identification of "food withdrawal" would also be helpful from a more descriptive or behavioral perspective to define the food addiction phenotype. We look forward to such data to generate the most fruitful conceptualization of obesity so as to promote optimal physical *and* mental health.

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Social and Financial Policy in the United States

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Eating disorders (EDs) are arguably some of the most misunderstood of all mental health disorders. There are a multitude of myths and misconceptions about EDs that lead to confusion, lack of support for people who struggle with EDs, and woefully inadequate attention from elected officials and policy-makers.

It is widely understood by those affected by EDs—patients, families, treatment providers, researchers, and education/prevention experts—that EDs are serious, life-threatening illnesses that can lead to a lifetime of suffering or death if left untreated (Eating Disorders Coalition for Research, Policy & Action [EDC], 2013b; see also Chapters 12, 14, 52, & 55). It is estimated that over 14 million Americans struggle with EDs (Hudson, Hiripi, Pope, & Kessler, 2007; Kjelsås, Bjørnstrøm, & Götestam, 2004; see also Chapter 5). Experts agree that EDs must be addressed as both physical and psychological disorders and that treatment is often prolonged due to the complex nature of the illnesses.

This chapter discusses the social and financial costs associated with EDs and reviews some of the past and current efforts designed to address mental health and EDs as public policy issues in the United States. The chapter closes with a discussion of what the future might hold to ensure that every person who suffers from an ED has access to the quality care available and that the care is adequately covered.

Cost of Illness

Treatment can involve several levels of care over time and severe cases can take years to fully treat (see Chapter 50). The Cost of Illness (COI) can be extremely high and the Health-Related Quality of Life (HRQoL) can be low over time, yet far too few people with EDs receive the treatment they need. Quality of Life is a concept that helps define an individual's overall well-being, and typically includes variables such as employment, physical and mental health, education, social connectedness, and leisure time. HRQoL usually relates to how a person's well-being is impacted by a disease (Centers for Disease Control [CDC], n.d.).

The Wiley Handbook of Eating Disorders, First Edition. Edited by Linda Smolak and Michael P. Levine.

Historically, insurance companies have failed to authorize treatment for EDs for a variety of reasons, such as a pre-existing condition clause, no or minimal mental health coverage, restrictions on ambulatory, hospital, and residential level of care, and unenrolling students who require time off due to health reasons. The cost of untreated EDs can be astronomical, and often families end up picking up the costs, which can lead to severe financial stress and even financial ruin.

Typically, the COI related to EDs encompasses direct costs paid by health insurance, such as medical services, pharmaceutical costs, nonmedical costs, and out-of-pocket costs to families. While the results of COI studies vary due to the year conducted and source of data, direct costs are substantial, ranging from $2,291 to $8,042 (2008 US$) per patient per annum (Stuhldreher et al., 2012). Costs for higher levels of care can be much greater, yet these levels of care are accessed by many fewer people. In a 2006 survey of residential ED treatment, Frisch, Herzog, and Franko (2006) estimated the mean residential length of stay was 83 days, with an average cost per day of $956 or cost per stay of $79,348.

Additionally, indirect costs should be considered. These include losses of productivity in the workplace for both individual and family, as well as the losses associated with premature death. While families experience these excruciating losses, the costs go uncalculated. Indirect costs have ranged from $1,528 to $4,455 (2008 US$) per patient per annum (Krauth, Buser, & Vogel, 2002; Stuhldreher et al., 2012). More study is needed to fully understand the substantial indirect costs of these illnesses.

What are the True "Costs" of Eating Disorders?

There is no doubt that EDs are costly. The cost to treat an ED is substantial, the cost to the individual patient and their family is significant, and the cost to society is considerable. And, while these costs are high, the cost of life lost to a treatable illness from which recovery is possible is incalculable. Consider these words from the diary of Anna Westin (2000):

> My life is worthless right now. Saying goodbye to such an unfriendly place can't be as hard as believing in it. And, essentially my spirit has fled already

Anna struggled with anorexia nervosa (AN). She wrote these words just days before she died. An ED cost Anna her life, the cost to her family was unimaginable with nearly unbearable grief, and the cost to society was the loss of a highly intelligent young woman who would have made great contributions in her chosen field and to society had she lived. We would argue that the total cost of Anna's illness is impossible to calculate and the cost of treatment, had it been authorized, would have been minimal compared to the cost incurred by not treating her illness.

Attempts to calculate the COI help to assess the economic burden associated with EDs. Some of the most interesting work uses a comprehensive description of costs using a "societal perspective" rather than a third-party payer perspective (Stuhldreher et al., 2012). Use of a societal perspective that includes direct costs (e.g., medical expenses), nonmedical costs (e.g., transportation and social services), and indirect costs (e.g., loss of productivity due to illness, absence from work, and premature death) gives a more complete picture of actual costs. Citing a methodologically rigorous German study by Krauth and colleagues, published in 2002, Stuhldreher et al. (2012) note that "the indirect costs exceed the costs of treatment many times over" (p. 487). Yet, how do you put a price on a life?

> Andrea was trained in opera, loved the theater, and was an avid dancer. She was working towards a degree in international business and politics and looked forward to saving the world. (Doris Smelzer, Andrea's mother, 2001)

Could Andrea have really saved the world? We will never know; she died when she was 19 years old after struggling with bulimia nervosa (BN) for 1 year.

What is the cost to employers when an employee suffers from an ED?

> I knew we would benefit from Heather's zeal, talent, organization, and courage. We spent one and a half years working for gender justice. Heather made the world better not just for each of us, but for countless people that none of us will ever know. (Kelly, 2001)

Heather died of a heart attack at age 27 after an 11-year struggle with BN and AN. Heather, Anna, and Andrea, and untold others, have died prematurely from EDs.

Disease Disparity

Eating disorders may be the most misunderstood and underfunded of all mental health conditions. For example, in 2011 the National Institutes of Health (NIH) spent $27 million to study EDs compared to $169 million on autism, and $264 million for schizophrenia, even though the death rate of AN is two times higher than it is for autism and schizophrenia (EDC, n.d., 2013a). Steinhausen (2008) found that, when accounting for all other causes of death, individuals with AN are 18 times more likely to die than their peers without EDs, and the risk of suicide is increased dramatically. These illnesses are costing society in lives lost.

As emphasized previously, EDs are extremely costly overall. They are expensive to treat, they are life-threatening and deadly, other psychological issues and physical complications often accompany them, and the impact on sufferers and caregivers is sizable. The true cost includes healthcare costs and the significant loss in occupational, societal, and economic achievement of people who struggle, especially if treatment is denied due to limited benefits or disagreement on medical necessity of treatment between the treatment team and the insurance reviewer.

What Can Be Done?

In spite of these disturbing facts, EDs are all too often minimized or dismissed by the general public, insurance companies, and policy-makers. For example, examination of ED-related stories in the news media demonstrates EDs are often viewed as an issue primarily impacting celebrities, females, and Whites, despite the data that EDs impact individuals across age, race, gender, and socioeconomic status (O'Hara & Clegg Smith, 2007; see also Chapters 25, 36, & 37). O'Hara and Clegg Smith (2007) found that 48% of ED-related articles in news media were published in Arts and Entertainment sections, versus only 13% in Health sections. Etiology and treatment were also frequently misrepresented. One of the most common etiology attributions described in news articles was "parental influence," with 48% of articles including this as part of etiology and only 1% of articles including genetics (see Chapter 28) or biology (see Chapter 17) as part of a description of etiology (O'Hara & Clegg Smith, 2007).

The public also demonstrates difficulty in considering EDs as a legitimate illness. In their work exploring public perception of biogenetic contributions to etiology of EDs, Angermeyer and colleagues (2013) found only 30% of their sample believed that AN had a brain-based etiology, and 25% believed the same of BN. More striking even, were the findings that 31% believed AN was the result of "weak will," while 40% believed the same of BN (Angermeyer et al., 2013). This kind of stigma is a considerable barrier to care, even for those accessing care. Becker and colleagues found that 59% of a sample with experience in ED treatment experienced perceived stigma within their social network (Becker, Hadley Arrindell, Perloe, Fay, & Striegel-Moore, 2010).

In the past decade there has been a deliberate and concerted effort on the part of patients, caregivers, professionals, and advocates to change these perceptions and address EDs as serious public health concerns that require attention from policy-makers and elected officials. Why is the tide so difficult to turn? These concerns are not new: "Advocates are emphatic about the need for adequate funding for treatment of eating disorders, not only because the suffering caused by eating disorders is well documented but also because treatment interventions of proven efficacy are available" (Striegel-Moore, Leslie, Petrill, Garvin, & Rosenheck, 2000, p. 382). This is as true today as it was 14 years ago, despite advances in mental health parity (Substance Abuse and Mental Health Services Administration [SAMHSA], 2012).

Yet, why is it that thousands of people who have the potential to achieve great things die as a result of EDs because of barriers to care that could have saved their lives? There is effective treatment for EDs (see Chapters 56–62) and when people have access to the care they need, the risk of long-term complications, chronicity, and overall costs can be significantly reduced (Simon, Schmidt, & Pilling, 2005). Yet, it is estimated that only 1 in 10 people with EDs receive treatment (Striegel-Moore et al., 2000). Recent estimates of general mental health care access indicate that only 38% of adults with mental illness access care (SAMHSA, 2012). It is critical to understand the life-threatening barriers to care.

Obstacles Related to Access to Care

People with EDs often face nearly insurmountable obstacles when trying to purchase an insurance policy or, if a policy is granted, getting adequate coverage within the policy. Pre-existing condition exclusions and specific exclusion of ED treatment in policies present significant obstacles to treatment. Insurance plans have historically excluded ED treatment along with interventions that would be considered elective and non-life-threatening such as therapeutic recreational camping, treatment for caffeine-related disorders, and elective cosmetic surgery (EDC, 2012). These obstacles can have devastating effects, including an increased risk of a chronic condition, long-term disability, and even death. Insurance has historically often been denied based on a pre-existing condition, ineligibility due to age, and minimal scope of service.

Fortunately, this particular barrier is being addressed by recent policy changes set forth in the Affordable Care Act (ACA, 2010). The ACA includes a provision that insurance coverage cannot be denied based on health status or pre-existing conditions. This single action alone should significantly improve access to insurance coverage for people with EDs. Under the ACA, starting in 2014, all insurance plans must include coverage for at least these 10 categories of Essential Health Benefits (EHB): ambulatory patient services; emergency services; hospitalization; maternity and newborn care; *mental health and substance use disorder services, including behavioral health treatment*; prescription drugs; rehabilitative and habilitative services and devices; laboratory services; preventive and wellness services and chronic disease

management; and pediatric services, including oral and vision care (HealthCare.gov, n.d.a). Inclusion of mental health in the EHB should provide for improved access to care for the treatment of EDs. Whether it does or not remains to be seen. Health care exchanges, the marketplace for the new plans including all EHB categories, rolled out in late 2013 (HealthCare.gov, n.d.b). Consumers and professionals await determination of specific inclusion or exclusion of EDs treatment in these plans.

Despite the improvements of elimination of pre-existing condition clauses and inclusion of mental health in the EHB, there are other common situations in which ED treatment is denied that may not be impacted by the changes in health policy. Treatment denial can occur when an insurance company's benefit determination is that the client does not meet medical necessity criteria, despite contradictory clinical recommendation from treating clinicians. Treatment denial can also occur after treatment is initially authorized. A determination to not authorize further treatment can be made based on a benefit decision judgment that the patient has not made enough progress to warrant additional treatment. Other times, when progress is being made, the patient and his or her family are told that they have made so much progress that treatment is no longer medically necessary. A survey conducted by the Eating Disorders Coalition for Research, Policy & Action (EDC, 2011) indicated that most providers believe that early discharge mandated by health insurance put their patients with AN into life-threatening situations, and 100% of providers who were surveyed believe that some of their patients suffer relapses due to limitations of managed care.

It is abhorrent that insurance companies are allowed to arbitrarily determine who gets treatment and for how long, and possibly even who lives and who dies. A young survivor of AN spoke at a 2011 EDC Congressional Briefing about her battle with the disease. She told how she was able to access treatment but her best friend, who had a different insurance policy, was not able to get authorization to the care she needed. Ultimately, one woman lived and one woman died. The survivor summed it up this way: "It's not fair that the insurance cards we carried around with us ultimately decided which one of us lived and died" (Espel, 2011, p. 3).

In navigating the system of healthcare, obstacles also exist that are related to variable criteria for coverage across insurance plans. At times, these criteria are not transparent and are difficult, if not impossible, to meet. Additionally, there is often a lack of coordination between reviewers involved in the insurance authorization process, short timeframes for ongoing review and authorization of care, and "carve outs" (i.e., exclusion) of ED care from the insurance policy (Sigman, 1996; Silber, 1994; Silber & Robb, 2002). These issues have persisted for two decades.

Additionally, even when a patient is asking for treatment, strong feelings of guilt about the high cost and the burden on the family may result in avoidance in making and keeping appointments and in getting adequate care over the course of the illness (Silber, 1994). Time in treatment groups is spent on processing fears related to insurance issues that result in early discharge, rather than on delivery of treatment modalities. Anna Westin expressed this guilt when she told her little sister that she felt that her family would be better off if she were dead because she was a burden. Anna died by suicide on February 17, 2000.

Public Policy, Parity, the Legal System, and Eating Disorders

Is there an end in sight? Are we any closer to ensuring that people who struggle with EDs have access to treatment? In a survey of 109 ED treatment providers across the United States, despite the passage of the ACA in 2010, 98.1% felt that it will take further legislative action to

ensure people with EDs have access to care (EDC, 2011). Broad public policies designed to address mental health issues through legislation have historically been inadequate and ineffective, especially in attaining parity for treatment of EDs.

Federal Policy

Barry, Huskamp, and Goldman (2010) conducted an extensive search into the history of federal mental health and addiction parity issues. They outline a steady decline in, and more stringent day and visit limits on, mental health care over the last half-century. President Kennedy first tried to achieve mental health parity in 1961 when he required the Federal Employees Health Benefits Program (FEHBP), the health insurer for federal employees, to cover mental health conditions on a par with medical and surgical conditions. However, in 1975 and again in 1981, these protections were scaled back and insurance companies were allowed to treat mental health disorders and medical/surgical disorders differently. As a result mental health coverage diminished over time.

Beginning in the 1990s, consumers and advocates began framing the lack of mental health parity as a civil rights issue, and insurance companies were accused of discrimination for denying treatment based on diagnosis. In 1996, Senators Paul Wellstone (Democrat, Minnesota) and Pete Domenici (Republican, New Mexico), both of whom had family members with mental illness, introduced a federal bill that would require insurance companies to cover mental health treatment. The Mental Health Parity Act (MHPA) of 1996 was groundbreaking legislation; however, once it was passed it proved to be inadequate in many ways. The 1996 bill did little to ensure mental health parity for the vast majority of Americans; Senator Wellstone often expressed his frustration that the bill was flawed but maintained hope that it was a step in the right direction (P. Wellstone, personal communication, 2000).

One of the main issues in the MHPA of 1996 was that it covered mental illnesses as defined by individual plans. At the time, most insurance plans offered coverage for what they determined were "biologically based illness," such as schizophrenia, bipolar disorder, and major depressive disorder. Diagnoses that insurance plans determined to be "non-biologically based," such as post-traumatic stress disorder (PTSD) and EDs, were not covered. Today, scientific consensus clearly shows that EDs are and should be considered a biologically based illness (Klump, Bulik, Kaye, Treasure, & Tyson, 2009).

The 1996 MHPA was a sunset bill that expired in 2001. There was a significant amount of determination and activity to pass a more comprehensive version of mental health parity that began soon after the original bill was passed. Then tragedy struck; the Democratic champion of the bill, Senator Paul Wellstone, was killed in a plane crash in 2002. After the Senator's death there was concern that the fight for mental health parity would end. But in 2007, Representative Patrick Kennedy (Democrat, Rhode Island) and Representative Jim Ramstad (Republican, Minnesota) introduced the Paul Wellstone and Pete Domenici Mental Health Parity and Addiction Equity Act (MHPAEA). Getting enough support to pass the new and improved parity bill was a long and, at times, frustrating uphill battle. There was fierce debate between Members of the House of Representatives and Members of the Senate about the language in the bill. The House version of MHPAEA offered full parity for any mental health disorder, whereas the Senate language was similar to the MHPA of 1996, which allowed insurance companies to determine what diagnoses to cover.

Ultimately, the final version that passed was based on the Senate provisions and has unfortunately resulted in more of the same practices and limitations. There remain significant

challenges in getting insurance companies to authorize treatment for EDs, especially at the higher levels of care like partial hospital and residential care.

Both the Mental Health Parity Act of 1996 and the Mental Health Parity and Addiction Equity Act of 2008 were celebrated for improving access to mental health care for millions of Americans. Passing these bills was monumental in that they addressed some of the major roadblocks for people who have certain types of mental health diagnoses. However, neither bill has had a significant impact on improving access to care for people struggling with EDs. Additionally, even now, nearly 5 years after the passage of the bill, the final regulations on MPHEA have not yet been released.

Without the final rules in place to provide more direction on proper implementation, many people with EDs are still being denied the mental health care to which they are entitled under parity. Without these rules, parity has not been fully implemented. The law is not fully functional, and the spirit and intent of the law are being ignored.

The problems with accessing ED care that still exist—despite parity being the law—show up in multiple ways: arbitrary limits on treatment, lack of transparency in medical decision-making criteria, denial of benefits in the middle of treatment, and unequal application of authorization requirements. Legal judgments have been made in some of these situations and receive significant media attention (Brush, 2009).

Yet another stumbling block to parity is unequal application of authorization requirements. Authorization is the process by which insurance companies issue approval for care. Significant disparity exists between application of authorization requirements for physical and mental health treatments. Imagine an individual who needs to restore 15 lb (6.8 kg) to get to a minimally acceptable body weight, at which point he or she may be considered appropriate for transfer to a lower level of care. At the community treatment standard of 2–3 lb (0.9–1.4 kg) per week (Academy for Eating Disorders, 2011; Lund et al., 2009), this level of weight restoration will take an extended period of time (5–7 weeks). Some insurance companies require treating clinicians to call in for continuing reviews on the patient every 2–3 days. Equal application of authorization would have a clinician treating a woman just going in to labor calling the insurance company for authorization for care every few minutes to see if they can continue to deliver the baby. This is highly unlikely in the physical health treatment world.

Additionally, there has been considerable elimination of coverage for particular levels of care, arbitrarily limiting the scope of treatment available in mental health. The 2013 EDC Survey of Benchmark Plans (EDC, 2013d) outlines that this sort of limitation in the scope of treatment is particularly applied to residential ED care, a level of care that is part of the American Psychiatric Association's (2006) clinical standard of care in the community. In a letter to the Secretary of the U.S. Department of Health and Human Services, members of Congress expressed concern regarding limitations on scope of coverage (EDC, 2013c). As outlined in this letter, specific levels of care are increasingly showing up on lists of exclusions because certain services are thought to have no "medical analogy." This means that plans refuse to cover a mental health service because insurance companies say there are no similar or analogous services on the medical side.

While these refusals may be related to confusion about the kind of treatment delivered and the equivalency issues, there is more to the issue. It is clear that there remains a question regarding the legitimacy of EDs as real illnesses (see Chapter 1), despite clear evidence that EDs are complex, brain-based illnesses with strong biological and genetic underpinnings (see Chapters 17, 28, 30, & 67). Exclusion for ED treatment is often sandwiched between procedures and activities that are denoted as elective or medically unnecessary. For an illness with

the highest mortality rate of any mental health illness (Birmingham, Su, Hlynsky, Goldner, & Gao, 2005; see also Chapters 14 & 55), an illness that has established treatment guidelines that are the standard of care across a field of experts (American Psychiatric Association, 2006), treatment for EDs should not qualify as elective.

The "improved" mental health parity bill, passed in 2008, allows insurance companies to determine what diagnoses to cover, applies only to plans that offer mental health coverage, and is full of loopholes that have allowed insurance companies to use the law as justification to deny ED coverage and/or severely limit what treatment is covered. There is ongoing debate about scope of services and continuum of care and, as noted previously, at the time of this writing the final rules for implementation of the 2008 MHPAEA have not been signed into law. Strong final rules that take these access issues into consideration would improve the process and access to care dramatically.

State Level Policy

During the 1970s and the 1980s, efforts to improve private insurance benefits were also happening at the state level. Most states enacted their own mental health parity laws, but state laws varied a great deal in strength, with only 18 states establishing minimum benefits for mental health treatment. Moreover, only a minority of state mental health parity laws require insurance companies to include AN or BN in their benefits package. Out of 50 states, only 10 require that all health plans cover treatment for AN and BN, 18 have limited coverage, and 23 have no coverage for EDs named in the law. And, to this date, no state law requires coverage for Eating Disorders not Otherwise Specified (the other specified and unspecified EDs in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders*; *DSM-5*) and Binge Eating Disorder (BED) (State Refor(u)m, 2013).

Is Legislative Action the Answer?

It is clear that in order to address the multitude of issues facing the ED community, political advocacy specific to EDs is necessary. Legislative efforts designed to address the ongoing lack of mental health parity and resultant discrimination against people with mental health diagnoses, including EDs, have to be established. In addition, efforts to increase funding for research into EDs and to improve education and prevention efforts are a necessity (see Chapter 48).

History has proven that policy-makers will not give adequate attention to ED issues without intentional intervention by advocates and other interested parties. As a result of this realization, a group of concerned professionals representing five ED organizations gathered in 2000 to form a grassroots advocacy organization that would bring the issues to Washington, DC. The result of that meeting was the birth of a federal advocacy organization called the Eating Disorders Coalition for Research, Policy & Action (EDC). The EDC has been working to advance the federal recognition of EDs as a public health priority and now has 40 member organizations and hundreds of individual members working together.

Prior to the formation of the EDC there was no coordinated effort to address EDs as a public policy issue at the federal level. The failure of MHPA and MHPAEA to significantly improve access to treatment for people with EDs, and the slow, arduous process of change in Washington, DC, prompted support for a comprehensive ED bill. Consequently, hundreds of advocates, patients, family members, treatment professionals, and researchers, with support

from key Members of Congress, worked to draft legislation that addressed many important issues that had yet to be addressed by policy-makers in Washington, DC. The EDC reached out to stakeholders across the United States and asked them what they would want included in a federal bill that would address EDs. After two national conferences and months of hard work the Federal Response to Eliminate Eating Disorders Act (FREED Act) was drafted. The FREED Act was first introduced in the House of Representatives in 2009 and was reintroduced in 2011 and again in 2013 (H.R. 2101, 2013).

There are three distinct sections in the FREED Act: education/prevention; treatment; and research. Once passed, the FREED Act will do what MHPA and MHPAEA have failed to do: improve access to care, allocate more research dollars to EDs, and enhance education and prevention programs.

Healthcare Reform

The delivery of health services, including mental health services, across the United States is changing, due in part to the Patient Protection and Affordable Care Act, which was passed and signed into law in 2010 (Affordable Care Act, 2010). Commonly referred to as the ACA, this law expands mental health coverage in a number of ways. First, it builds on the Mental Health Parity and Addiction Equity Act of 2008 by extending mental health benefits to people enrolled in individual and small group insurance plans who generally lack these benefits, thereby expanding parity to millions of Americans whose coverage has not been in compliance with Mental Health Parity (MHP) requirements. In addition, insurance exchanges at the state level will be based on a benchmark plan in the state, which must include coverage for the EHB.

However, in a survey conducted by the EDC (2013d) to discover what each state has to offer in terms of mental health coverage, it became apparent that there is wide disparity between states in expected mental health and substance abuse services. The EDC survey of all 50 states plus the District of Columbia found 27 adopted benchmark plans that severely limit mental health services, whereas, in contrast, 24 have strong mental health coverage. Few states name EDs as a covered benefit, and there is speculation that insurance providers will continue to limit coverage of ED treatment, particularly intensive and residential care.

Unfortunately, even in states that have strong parity laws and have had some success in implementing laws to protect people struggling with mental health issues, there are significant numbers of patients who are denied access to ED treatment. It was predicted early on that many of the issues would eventually be settled in court. Patients, families, and professionals across the United States have countless stories to tell about wrongful denials and unfair practices by insurance companies.

Court Involvement

There have been some successes in court that are drawing attention to the problem of insurance companies wrongfully denying ED treatment. In 2000, the Attorney General of Minnesota brought a lawsuit against Blue Cross Blue Shield of Minnesota for denying, delaying, and withholding mental health treatment, including ED treatment, to people in Minnesota. Blue Cross Blue Shield of Minnesota agreed to send all denials of care through a settlement-established, independent review panel of judges, and to focus on improved mental health coverage for all members. Thus, the result of that lawsuit was an out-of-court settlement that

greatly improved access to all forms of mental health treatment, including treatment for EDs. There are examples of class action lawsuits that have resulted in settlements and directives to provide ED treatment, including a case in New Jersey that was settled in 2008 (Gottlieb, 2008).

In a recent case heard in California, *Harlick v. Blue Shield*, attorney Lisa Kantor successfully argued that Jean Harlick's insurance company should have covered her client's residential treatment. In this case, Blue Shield of California initially approved residential treatment for Ms. Harlick but later rescinded, stating that the plan did not cover residential treatment for mental health disorders. California has a strong parity law that states that all medically necessary treatment for severe mental illness has to be covered and EDs are included in the definition of severe mental illness. The case was originally lost but, upon appeal, the decision was reversed; ultimately, the case was settled based in part on the following argument written by the Ninth Circuit Court:

> Blue Shield's argument lacks support in common sense. Some medically necessary treatments for severe mental illness have no analogue in treatments for physical illnesses. For example, it makes no sense in a case such as Harlick's to pay for time in a skilled Nursing Facility – which cannot effectively treat her anorexia nervosa – but not pay for time in a residential treatment facility that specializes in treating eating disorders. (*Harlick v. Blue Shield of California*, 2012, p. 2)

Conclusions and Future Directions

In summary, in the United States, treatment providers and patients and their families routinely cite a number of problems that interfere with accessing and sustaining treatment for eating-disordered patients. These problems and obstacles include:

- lack of uniform criteria for coverage;
- lack of uniform criteria for levels of care;
- insufficient reimbursement for specialized care;
- inability to navigate the appeals process and other barriers constructed by insurers to make appeals cumbersome and confusing;
- severe limitations on both inpatient and outpatient services;
- exclusions for intensive and residential services;
- limited research documenting efficacy of treatment.

Future research must examine not only treatment efficacy, but also the cost-effectiveness of broad inclusion of ED treatment in healthcare coverage.

Fortunately, there are more voices now contributing to the strength and imperative nature of the concerns. Numerous advocacy-focused organizations such as the EDC, the Emily Program Foundation, and the Residential Eating Disorders Consortium use their considerable influence to bring ED issues to policy-makers in Washington, DC. There are other organizations and individuals that are focused on changing policy one state at a time. All of these groups have put their energy into designing campaigns to help the general public and elected officials better understand EDs and the needs of people who are affected by them.

ED patients, families, treatment providers, researchers, advocates, and the health insurance industry have to work together to address the multitude of issues described in this chapter.

Patients must become their own advocates and they need the necessary tools to fight wrongful denials of insurance claims by their insurance companies. Families need support and guidance in order to be advocates for their daughters and sons who are struggling with an ED. Treatment providers must do what they can to advocate for the appropriate and necessary treatment their patients require. The public must be educated to better understand EDs and insurance issues. Researchers must conduct studies that document the efficacy of treatment and how providing the necessary care results in far lower costs overall. Advocates must continue to take these issues to state capitols and the federal government and to demand attention and legislation that will solve the problems. And, finally, the health insurance industry must evaluate its policies and practices that have been detrimental and even deadly for ED patients.

Taken together, social, legislative, judicial, and financial policy, coupled with advocacy related to ED patients' access to quality care, will improve the experience of people struggling with EDs and of their families. Advocacy efforts must continue if we are to remedy the decades of challenges, frustrations, loss, and despair experienced by people with EDs and their families. In the words attributed to Margaret Mead, "Never doubt that a small group of thoughtful, committed citizens can change the world; indeed, it's the only thing that ever has" (Sommers & Dinneen, 1984, p. 158). As fully implemented healthcare reform and mental health parity unfolds, collectively we must strive to ensure the implications for people with EDs and their families will be positive and adequate.

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Toward an Integrated Biopsychosocial Model of Eating Disorders

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For at least 40 years theorists have argued that etiological models of eating disorders (EDs) should be multidimensional (Garfinkel & Garner, 1982). Indeed, there are many such models available, including recent variants presented by Kaye, Bailer, and Klabunde (2012) and Wertheim, Paxton, and Blaney (2009). Most theorists, researchers, and clinicians in the field endorse a multidimensional approach.

It is, then, a bit surprising that the two dominant paradigms in current ED theory emphasize almost opposite approaches to etiology. One, the biopsychiatric or biologically based mental illness (BBMI) approach, emphasizes genetic factors and brain functioning (Kaye et al., 2012; Klump, Bulik, Kaye, Treasure, & Tyson, 2009; Nunn, Lask, & Frampton, 2011; Strober & Johnson, 2012; see also Chapters 17, 28, & 30). In contrast, the sociocultural model focuses on cultural themes and practices that are transmitted through convergent social messages about body shape, weight, and the controllability or malleability of weight and shape (Stice, 1994; Thompson, Heinberg, Altabe, & Tantleff-Dunn, 1999; see also Chapters 21, 26, 29, & 31).

Certainly both types of theory routinely include other variables, most commonly personality or temperament characteristics (e.g., perfectionism, harm avoidance; see Chapter 32) and environmental stressors (e.g., poor parenting, sexual abuse, subcultures that enforce a drive for thinness and a fear of fat; see Chapters 34 & 35). Furthermore, while both have made contributions to our understanding of EDs, they also each have important weaknesses (Levine & Smolak, 2014). Yet each largely ignores or even disparages the major features and claims of the other model. How, then, can we reasonably hope to integrate the approaches into a truly multidimensional model? Indeed, is it even reasonable to try? Or are the two models actually opposing and irreconcilable scientific paradigms, like behaviorism and psychoanalysis?

The overarching goal of this chapter is to provide guideposts for a more inclusive multidimensional model of the development and maintenance of EDs. We do not intend to offer *a* model, even as an example. This is partly because we are trying to avoid diagrams or a sequence of paragraphs that will necessarily grant primacy to one variable or set of variables. In other words, we are attempting to begin to defuse at least some of the disagreements between the BBMI and sociocultural camps. We do, however, want to analyze the various components of

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an inclusive, integrative model. To that end we suggest that a model of ED needs to consider at least the following:

1 EDs have stages of development that need to be explained.
2 There are similarities across EDs as well as differences between ED and non-ED populations, particularly in terms of body image and eating behaviors.
3 Certain types of data are required to establish causal relationships.
4 Specific influences need to be distinguished from nonspecific influences.
5 We must account for gender, ethnicity, age, and cultural differences.
6 How separable are biological, environmental, sociocultural, and psychological influences?

Stages of a Disorder

The behaviors associated with anorexia nervosa (AN) and bulimia nervosa (BN) are extreme and unusual (Strober & Johnson, 2012; see also Chapters 8 & 9). It is unlikely that they suddenly appear overnight. Furthermore, there is a developmental path to all behaviors, whether they are normative, idiosyncratic, or pathological. Thus, there is a pathway to ED. Models should describe this development, establishing both causal and correlative factors. Research of various sorts (Connors, 1996; Garfinkel & Garner, 1982; Steiger & Bruce, 2007), including clinical experience, indicates that it is a virtual certainty that there will be multiple pathways, demonstrating the developmental psychopathology principle of equifinality (Cicchetti & Rogosch, 1996). In its current form, the BBMI model has little to say about these developmental pathways. Although there is a small amount of genetic developmental data (Klump, Burt, McGue, & Iacono, 2007; Klump et al., 2012; see also Chapter 28), there are no prospective neuroscience data. Without prospective data, the premorbid brain functioning and the pathway that brought the person to the brain associated with an ED (see Chapters 17 & 30) cannot be identified. On the other hand, although more data are needed, the sociocultural model does have data predicting the onset of disordered eating and EDs (The McKnight Investigators, 2003) as well as data from fairly young children (Dohnt & Tiggemann, 2006) elucidating the development of risk factors for EDs. However, no model has defined an entire developmental pathway.

At some point, a person decides to purge or to ingest only starvation levels of food. Some developmental process creates a vulnerability. Then there may need to be a "trigger" that moves the individual into a full-blown ED. For example, the pioneering writings of Bruch (1973) and Crisp (1983) suggested triggers related to indicators of maturation into adulthood. It may also be, as is the case for other severe forms of psychopathology (Ingram & Price, 2010), that there is some threshold level of cumulative risk factors or vulnerabilities. Once the risk factors or vulnerabilities have accumulated to that level, the pathological behaviors emerge, perhaps as a coping mechanism. A frequent critique of the sociocultural approach by BBMI theorists is that many women demonstrate body dissatisfaction and use weight control techniques, but only a small group develop ED (Levine & Smolak, 2014). Although a sociocultural model is theoretically capable of using multiplicative probabilities to explain how a high prevalence of risk factors leads to a low incidence and prevalence of ED (Levine & Smolak, 2014), neither the sociocultural nor the BBMI model has clearly identified how an ED is activated. That trigger, or shift in levels of vulnerabilities, or emerging convergence of vulnerabilities and risks needs to be explained.

EDs are treatable, but it is often difficult to achieve complete remission, and relapse is fairly common (see Chapters 12 & 55–62). Thus, it is important to distinguish factors that maintain or worsen the disorder from factors that cause the disorder. Whereas some maintenance variables may be similar to those that cause the disorder, others may be different. BBMI models suggest that neurochemistry is responsible for maintenance or exacerbation of eating disorders (see Chapters 17 & 30), while cognitive theorists implicate the social and self-generated rewards associated with the pathological attitudes and behaviors (see Chapters 18 & 56). There may be relationships between these maintaining factors and those that make a person more resistant to treatment or more likely to relapse. Or, again, the resistance/relapse influences may be different factors, including, perhaps, elements of the treatment protocol.

It may be that no single model can identify developmental factors, activating forces, maintenance influences, and treatment success variables. However, it is important that model designers identify which phase of the disorder they are seeking to explain in ways that can guide research. Recognizing the different phases may be an important way of integrating various models.

What Needs to be Explained

It is important to outline what an etiological model of EDs must explain (Nunn et al., 2011). We believe that one should begin by asking: what are the similarities across EDs that distinguish an ED from other phenomena, ranging from dieting to mood disorders? This question acknowledges the fundamental importance of explaining (a) characteristics that are unique to EDs, such as the self-starvation that marks AN or the binge-purge cycle that defines BN; and (b) the "transdiagnostic" similarities within the family of currently recognized EDs (Fairburn, 2008). Then there are those attributes, such as a rigid and ruminative thinking, high levels of harm avoidance, and dysregulation of the hypothalamic-pituitary-adrenal axis, which are associated with EDs but also with a number of other psychiatric disorders, such as depression and anxiety disorders (Jokinen & Nordström, 2009; Nyman et al., 2011; Olatunji, Naragon-Gainey, & Wolitzky-Taylor, 2013). We argue that specifying and explaining the hallmark ED characteristics must be the central focus of any etiological theory. We also want to emphasize that at this point we are not trying to identify causes; this section addresses only ED signs, symptoms, and correlates.

Body Image Disturbances

In both AN and BN—and their clinically significant variants that were previously part of "eating disorder not otherwise specified" (ED-NOS) and are now recognized as "other specified feeding and eating disorders" (OSFED)—but not Binge Eating Disorder (BED), body image disturbances are part of the current core diagnostic criteria (American Psychiatric Association, 2013; see also Chapter 1). In AN, this takes the form of (a) a distorted perception of the body as being fat when, in fact, it is emaciated; and (b) an extreme and unrealistic fear of becoming fat. In BN, the body image-related criteria are (a) body shape and weight unduly influence self-esteem and self-definition; and (b) compensatory behaviors (e.g., vomiting) are used to avoid becoming fat. These are defining features of the disorders. And the disorders are not as distinct as a classification system makes them appear: the disorders are concurrent in some people, and in others AN later gives way to BN (see Chapter 55).

In addition, body image disturbances, particularly in the form of body dissatisfaction or overconcern with weight and shape, are excellent predictors of the development of eating disturbances and disorders (Stice, 2002; The McKnight Investigators, 2003; see also Chapter 22). In fact, Stice (2002) argued that body dissatisfaction was the single best predictor of eating disturbances and disorders. This is consistent with the finding that levels of weight and shape concerns are arguably the single best predictor of treatment failures, ranging from dropout to relapse (see Chapters 55 & 64). Clearly, then, body dissatisfaction and weight/shape concerns are more than simply markers of EDs. They are a crucial part of the core pathology of EDs and hence must be a central element of any model of AN or BN.

BED symptoms are less focused on body weight and shape (see Chapter 10). Instead, the criteria are eating-related. This important distinction between BED and AN or BN raises the likelihood that separate models will be needed for the disorders. Given this, the remainder of this chapter emphasizes AN and BN. The link between AN and BN is further underscored by the shifting between the disorders that is frequently noted, whereas crossover from AN or BN to BED is substantially less common (Strober & Johnson, 2012; see also Chapter 55).

Eating Disturbances

Again, eating disturbances are at the core of both AN and BN (American Psychiatric Association, 2013). In AN the eating disturbance is severe, relentless, and all-consuming dietary restriction, with caloric intake significantly below healthy levels long enough to produce dangerously low body weight. Indeed, this self-starvation can be sufficient to cause organ damage or organ failure, leading to death (see Chapters 8, 12, 14, & 52). Such levels of dieting may result in neurochemical changes that maintain the problematic eating. The restriction may also actually be negatively reinforcing in terms of arousal or anxiety management and avoidance of postprandial distress (Kaye, Wierenga, Bailer, Simmons, & Bischoff-Grethe, 2013), and/or positively reinforcing because restriction creates and sustains a sense of control and achievement, particularly for a person obsessed with weight and shape (see Chapter 18). Regardless of the cause, it does appear that severe caloric restriction is self-maintaining. Interference with this extreme dieting—or even the threat of refeeding—can result in withdrawal from treatment.

In BN the eating disturbance is binge eating followed by purging. Again, this is a potentially life-threatening set of behaviors, particularly self-induced vomiting as a form of purging. Physical damage to the cardiovascular or gastrointestinal systems presents a long-term threat, while aspirating vomit or hemorrhaging from the esophagus or stomach poses immediate danger (see Chapter 14). There are also various long-term and serious risks (e.g., to intestinal functioning) created by abuse of laxatives and diuretics. Clients do not typically show the high levels of resistance to ending binge eating and purging that those with AN demonstrate in relation to restriction. However, about one third of those with BN relapse after treatment (see Chapter 55), indicating that in a significant number of instances binge eating and purging likely fulfill several psychological functions and are difficult to give up permanently (Johnson & Connors, 1987).

Interestingly, calorie restriction, in the form of dieting and fasting, may play a role in the onset of BN. Several prospective studies (Neumark-Sztainer et al., 2006; Stice, Davis, Miller, & Marti, 2008; see also Chapter 24) have indicated that self-imposed dieting or fasting increases the risk of bulimic behaviors and symptoms. For some people, avoiding certain foods, especially foods of high caloric density, increases the reward value of those foods (Stice, Burger, & Yokum,

2013). Thus, explaining the decision to restrict calories may be a particularly important component of any model attempting to explain the developmental pathways to AN and BN.

Other Characteristics

Most models of EDs focus on factors other than the body image and eating disturbances. For example, genetic models often emphasize genetically mediated variables such as temperament, including harm avoidance, while neuroscience perspectives describe neurochemical and hormonal differences between those diagnosed with ED and other women (Kaye et al., 2012, 2013; Striegel-Moore & Bulik, 2007; Strober & Johnson, 2012). Sociocultural models may highlight individual difference variables, including high levels of negative affect or a tendency to engage in social comparison (Stice, 2001; Thompson et al., 1999). Table 67.1 lists and briefly describes some of the more commonly cited differences between clinical EDs and

Table 67.1 Characteristics of eating disorders (EDs), beyond body image and eating patterns, that theories should consider.

Variable	*Comment*
Cognition	Weight and shape schema may determine how people interpret messages and experiences. Information processing biases re food and weight. Rigid, absolute thinking concerning body
Comorbidity	Most people with ED diagnoses also have other diagnoses. Depression and anxiety disorders are particularly common
Culture	The nature, type, and frequency of ED appears to vary across cultures. Cultural research also indicates that the introduction of American media or body image values may increase ED
Ethnicity	In the United States Blacks may experience fewer restricting EDs but not be "protected" against other forms. Other U.S. ethnic differences have not been consistently documented
Gender	Women experience EDs, particularly AN and BN, at higher rates than men. These gaps may be narrowing
Genetics	Women with first-degree relatives suffering from an ED are more likely to develop ED than those without ED in their families. Twin research indicates a moderate to strong heritability
Neurochemistry	Women in the acute phase of ED have different levels of several neurotransmitters in the serotonin, dopamine, and GABA systems compared to those not suffering from ED. Some of these differences continue even after recovery
Personality	Biopsychiatric proponents tend to emphasize "temperament" characteristics that they argue are genetically based. These include harm avoidance and negative affect. Theorists from a variety of perspectives have focused on perfectionism. Sociocultural theorists often discuss individual characteristics including self-esteem, and a tendency to engage in social comparison
Trauma	Sexual abuse is generally accepted as a nonspecific influence on the development of BN. Other forms of sexual violence, ranging from harassment to rape, have been shown to be at least correlates. Other forms of abuse have received less attention but may also be relevant

control samples. Note that these factors represent differences, which are essentially correlations, and not necessarily causal relationships.

It is also noteworthy that some reviews focus very heavily on personality or cognitive factors that appear to be part of the clinical presentation of EDs (Nunn et al., 2011; Strober & Johnson, 2012). Such factors are often important in treatment, and rigorous research may eventually establish their relevance to the etiology of EDs (Levine & Smolak, 2014; Smolak, 2012). But the core components of EDs, those with important predictive abilities for the onset of EDs as well as treatment success, are the body image and eating problems. While a model should include other constructs, there must be a clear emphasis on the body image and eating dysfunctions.

Data and Establishing Causality

In order to establish causality, especially in a relatively young scientific field such as EDs, caution and good science require a network of data interpreted within theory. It is entirely possible that every element of the biopsychiatric and sociocultural theories is correct; modern physics makes it clear that the same set of phenomena looks and appears to act very differently, depending on the scientist's perspective (Greene, 2003). It is probable that many of the elements of each are indeed factual. But we need data in order to establish which ones are correct. As indicated by the logical positivism, empiricism, and emphasis on falsification that have long defined the Western scientific model (Popper, 1959), these data need to be of a particular type.

Experimental data continue to be the gold standard of scientific research. Certainly, ethics render some types of experimental studies impossible. However, imaginative use of convergent data from treatment and prevention interventions, as well as case-controlled retrospective studies that incorporate psychiatric controls (Jacobi & Fittig, 2010), may provide at least a partial solution. Furthermore, research must, at the very least, convincingly demonstrate that a putative cause predates the onset of a symptom or disorder (Kraemer et al., 1997). Prospective data are essential. Furthermore, it is critical that data collection begin in early childhood in order to fully track the developmental precursors of ED. While there are both experimental and prospective data for the sociocultural model (Smolak, 2012; Stice, 2002; Wertheim et al., 2009; see also Chapters 26, 29, & 31), there are virtually none for neurobiological theories and scant prospective data for genetic approaches (Klump et al., 2012). To be explicit, there are no experimental data for the BBMI approach, unless one counts Stice et al.'s (2013) data showing that fasting affects brain functioning. However, since BBMI theorists often minimize the role of body image or dieting (because of their "normative" nature), these data do not appear to be part of this model per se.

Neuropsychiatric researchers and theorists sometimes argue that evaluating brain functioning during and after an ED provides information about causality (Kaye et al., 2013). The idea is that a neurochemical difference between ED and control women that exists only during the acute phase of the illness (i.e., that disappears after recovery) is an outcome of the illness per se. On the other hand, if the difference continues postrecovery (i.e., when the person is restored to a normal state and normal functioning), it likely predated the illness and may be a causal contributor. Thus, the acute phase of the illness is viewed as analogous to a "natural experiment." However, by definition, a "natural experiment" is not substantially better controlled and informative than any correlational study.

Furthermore, developmental neuroscientists have long established that any experience can and does alter neurological structure and functioning (Cichetti & Curtis, 2006). Given the

duration, severity, and physiological effects of both AN and BN, there is little doubt that the illnesses can result in long-term and, perhaps, permanent alterations in the brain. Again, only prospective research can identify precursors to EDs. Without either, or preferably both, experimental or prospective evidence, it is inappropriate to claim a "causal" factor has been identified. At best, one can claim to have uncovered a correlate. Of course, correlations are subject to the possibility of confounding variables influencing or even determining the correlation.

The possible or even likely impact of a third variable raises two other points regarding methodology. First, studies attempting to identify contributing or causal factors in the development and maintenance of EDs need to control for at least the most likely third variables. Studies that measure only one predictor variable cannot do this. Second, replication is crucial. When findings are difficult to replicate, it raises the possibility that some third variable created the relationship. For example, it is possible that some of the brain-ED correlation is actually attributable to a comorbid disorder, a factor that is only sometimes considered through inclusion of either psychiatric controls or covariance analysis.

Specific versus Nonspecific Influences

We need to distinguish specific influences on EDs from nonspecific influences on various forms of psychopathology (Connors, 1996). Nonspecific risk factors may certainly be important; indeed, they may be crucial and, particularly in interaction with specific factors, even causal. However, by definition, and as established through research across multiple outcomes (Ingram & Price, 2010), nonspecific factors are not likely to result in the unique behaviors and attitudes that are the hallmark of EDs. Thus, when identifying risk factors, it is important to delineate what their roles are likely to be in EDs and what types of behaviors they may or may not explain. The roles should include direct, indirect, interactional, and transactional pathways.

There are several factors that contribute to numerous forms of psychopathology, including EDs. For example, sexual abuse has been related to ED, depression, anxiety disorders, and personality disorders (Smolak, 2012; Thompson & Wonderlich, 2004; see also Chapter 34). In almost all instances it is important to address the abuse experiences in therapy, independently of the diagnosis. Yet, that will not typically relieve the ED symptoms. And some treatments that are effective with other diagnoses, such as cognitive-behavioral therapy (CBT), are most successful with EDs if they are adapted specifically to EDs (see Chapter 56). In fact, Fairburn, Cooper, and Shafran (2008) emphasize that the comparatively effective clinical intervention known as enhanced cognitive behavior therapy (CBT-E) "is a treatment for eating disorder psychopathology, rather than an eating disorder diagnosis" (p. 23). And pharmacological treatments that help reduce anxiety or depression symptoms have proven to be of limited value with EDs (see Chapter 59). Addressing the risk factors and maladaptive psychological processes that are specific to body image and eating dysfunction is most likely to reduce ED symptomology. It is noteworthy that arguably the most effective treatment and prevention techniques are rooted in cognitive theory and target specific beliefs about weight and shape (Fairburn et al., 2008; see also Chapters 18 & 56).

Much of the research based on the sociocultural model focuses on factors that influence body image and weight control techniques. Internalization of the thin ideal has been a particular emphasis. As noted earlier, body image is a particularly powerful predictor of the onset of EDs as well as of treatment success. From the BBMI perspective, there certainly has been discussion of specific genetic factors related to body image or disordered eating (Klump et al., 2012).

However, no loci have been identified despite large-scale genome-wide studies (Verweij et al., 2010). Furthermore, the BBMI model has frequently emphasized personality factors that are not specific to ED over ED-specific behaviors (Levine & Smolak, 2014).

Comorbid conditions are also important here. Somewhere between 60% and 80% of women diagnosed with EDs have one or more comorbid conditions (Hudson, Hiripi, Pope, & Kessler, 2007; see also Chapters 15 & 54). Any research, then, needs to consider how the depression, anxiety, personality disorders, or other psychiatric conditions might mediate or moderate the relationship between the variable under investigation and EDs. While this is especially important with nonspecific risk factors, it holds with specific risk factors too. For example, body dissatisfaction is probably related to both depression and EDs, at least among women (Stice, Hayward, Cameron, Killen, & Taylor, 2000).

Gender, Ethnicity, and Cultural Differences

We must account for the gender, ethnicity, and cultural differences (see Chapters 23, 25, & 27). This principle reminds us, regardless of paradigmatic or theoretical orientation, that it is inadequate to begin a model with a claim that all a model need do is explain why females (or Whites or people living in Western cultures) develop EDs. Gender is not a fixed risk factor. Researchers and theorists need to explain how and why females are more susceptible. The same is true for ethnic, cultural, and sexuality differences.

Feminist theory likely represents the most intensive and complete interrogation of gender and sexuality differences in EDs (Bartky, 1990; Bordo, 1993; Smolak & Murnen, 2004; see also Chapters 19 & 27). Sociocultural perspectives also give some consideration to these issues, though most of the specific theories (Stice, 1994, 2001; Thompson et al., 1999) give little precise attention to them. Nonetheless, these theories do imply that anything that influences the cultural messages involved in thin-ideal internalization will be important. On the other hand, BBMI models only rarely consider gender explicitly, instead assuming that women are more likely to develop EDs (Kaye et al., 2013). These theorists have given little or no attention to ethnicity or cultural differences, either in their theorizing or their research samples.

Biological versus Environmental versus Sociocultural versus Psychological: How Separable Are They?

Some theorists seem inclined to argue that a particular set of variables has primacy over the others. For example, Strober and Johnson (2012) suggest that genetic, neurochemical, and genetically mediated personality variables are more important than sociocultural variables in determining ED. Certainly we all want to generate the most parsimonious model possible. However, the data are clear that sociocultural variables play a role in focusing women on body shape, body management, and management of appetites, including eating. Similarly, it is evident that women whose EDs are active show neurochemical differences from those whose EDs are remitted or controls. Furthermore, women are undoubtedly more affected than men. All of these, and other, facts should be included in any model.

As we have emphasized throughout this chapter, this means that a variety of potentially explanatory factors will need to be considered. It is the nature of the way that we do science, and perhaps of human cognition, that we like to categorize risk factors as, for example,

biological or social. The current preference for flow-chart type of models (e.g., as in Nunn et al., 2011, or Wertheim et al., 2009), charting a path for our understanding, and perhaps for a path analysis, encourages us to select a starting point. Yet, we should keep in mind that these truly are heuristic conveniences, rather than accurate reflections of reality.

Let's assume for a moment that we begin a model with the infant brain. We prefer this over the construct of a "genetic predisposition" as a starting point because it is more measurable. Even the brain of a newborn reflects the effects of genetic endowment, prenatal environment, and perinatal complications or difficulties. If we were able to measure harm avoidance in a newborn, which we cannot, we already would be unable to specify whether a high level was attributable to genetics or perinatal complications. Similarly, by the time we look at a young infant, we would be unable to tell whether negative affect, which can be effectively measured during the first 6 months, is primarily rooted in genetics, exposure to prenatal stress or drugs, or postnatal parenting, or some reciprocal interaction of factors. Developmental psychologists have long argued that it is futile and misleading to try to separate such influences (Reese & Overton, 1970).

The development of an ED, as with all development, occurs within a context. As Bronfenbrenner (1979) first argued over 30 years ago, characteristics of the individual (understood at multiple levels of analysis, including the neurophysiological), the immediate environment, the environment influencing other people who in turn influence the individual, and cultural values are all in constant reciprocal interaction in producing behavior and development (see also Markus & Kitayama, 2010). As these relationships vary over time, it is important to consider the timing of an influence. Among the possibilities included in timing effects are a synchrony of events, periods of particularly high neuroplasticity, and transitions where there are limited social supports available (Smolak & Levine, 1996). All of these examples may be times when someone is more vulnerable to social messages or environmental trauma.

Conclusions and Future Directions

There are several different models of EDs—psychodynamic, cognitive-behavioral, feminist, sociocultural, and biopsychiatric (see Chapters 17–21). Given its evidence base, the cognitive-behavioral model remains very influential in therapeutic interventions for BN and BED, while the sociocultural and biopsychiatric approaches currently dominate risk factor research. In many ways, the latter two models would appear to be incompatible. Few sociocultural theorists give attention to neurobiology or genetics, and some biopsychiatric theorists actually dismiss sociocultural influences as central to ED (Levine & Smolak, 2014). Yet, there are some attempts to integrate the perspectives, both theoretically (Striegel-Moore & Bulik, 2007) and in research (Suisman et al., 2012), suggesting that both make important and complementary contributions. The goal of this chapter was to offer some suggestions as to what an integration of these two, or any other theories, into a multidimensional, biopsychosocial theory, should take into account.

Our focus has been on AN and BN, substantially because of their shared symptoms. Specifically, both feature negative or distorted body image and eating dysfunction in their symptomology. It is important to reiterate that these are not only symptoms but also predictors of the onset and recovery from EDs. On both a theoretical and applications level, it is crucial that theories emphasize disordered body image and eating. Of course, these are not the only characteristics of people with clinical EDs; differences between ED and control samples

in terms of cognitive, personality, neurochemical, and genetic factors, as well as psychosocial and environmental stressors, may be important to explain also. With this fundamental principle in mind, we offer the following as critical principles and directions for further EDs research:

1 If there are no experimental and no prospective data for a variable, it should not be discussed as causal. Gathering prospective data, particularly if data collection begins prior to the onset of any symptoms, is difficult on many levels. But such data are absolutely crucial. Because EDs, especially AN, are rare, procuring a large enough sample may well require collaboration. Nevertheless, this may be a good starting point for integration of sociocultural and biopsychiatric models. Identifying high-risk samples through genetic studies (in terms of familial vulnerability) and high-risk cultural groups (such as gymnasts or dancers) might make it easier to end up with enough symptomatic participants to actually draw conclusions.
2 Researchers should quit considering gender as a "fixed" variable**.** Feminist theorists argue that most gender differences are rooted in culturally defined experiential differences. Even physiological sex differences, such as hormonal levels, are affected by experiences. These experiences, ranging from toys that encourage investment in appearance (Dittmar, Halliwell, & Ive, 2006), to clothing that emphasizes girls' sexiness (Goodin, Van Denberg, Murnen, & Smolak, 2011), to sexual violence (Piran & Teall, 2012), need to be defined and examined as causal factors in the development of EDs and as possible mediators and moderators of other variables. Similar arguments can be made about ethnicity and culture. Gender, ethnic, cultural, and age group differences may help to elucidate universal and group-specific contributors to ED.
3 The interactive and cumulative roles of cognitive, psychological, neurochemical, genetic, social, and cultural factors need to be given more attention. Too many studies look only at media or only genetics, for example. Certainly, it is not realistic to think that all things can be examined in a single study. But we need to expend more energy combining levels of influence and analysis, thereby testing new relationships.
4 Factors such as body image (particularly weight and shape concerns) that appear to influence all stages of EDs (onset, maintenance, recovery) deserve particular attention. In the case of body image, understanding what moves someone from "normative discontent" to pathology or what distinguishes the two continues to be a particularly pressing issue.
5 We should always remember that the principal goal of our research is to find ways to prevent and treat EDs. Certainly, there is some value to just understanding the disorders, including precise descriptions of symptom development and transformation over time. For example, recognizing the role of trauma has led to changes in therapy protocols. But if a causal factor truly is "fixed" and not amenable to intervention it is less deserving of research attention than "variable" factors that can be more readily addressed in prevention and treatment. It is noteworthy that genetic and neurochemical influences should probably not be thought of as fixed, as they are consistently affected by environmental factors.

We believe that attention to these principles will enable researchers, who inevitably hold one or more of a variety of perspectives, to clarify the model(s) from which they are working while abandoning as futile the tendency to presume, and then seek validation for, the proposition that one category of factors (e.g., "biological") is primary. Borrowing a principle that has been central in developmental psychology for well over 40 years (Reese & Overton, 1970), we need

to insist that the adjective "biopsychosocial" be indicative of a nonreductive, integrative approach that captures the complexity of the development, maintenance, and treatment of eating disorders.

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Planning for Future Research: 10 Critical Questions

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The ideas and research findings presented in these two volumes represent many features of the maturation and increasing sophistication of the eating disorders (EDs) field since the pioneering publications in English of the 1970s and 1980s highlighted in Section I. Looking across the previous 67 chapters, it is particularly exciting to see the field benefit from fruitful connections with various disciplines and subdisciplines whose depth is anchored in much longer, richer histories. To give a few examples, these volumes demonstrate the positive influence of (in alphabetical order):

- behavioral medicine, including stress and coping in ED sufferers and their caregivers, as well as the study of risk and protective factors within a broad biopsychosocial framework;
- cross-cultural psychiatry and psychology;
- developmental psychology and developmental psychopathology;
- medical anthropology;
- multivariate statistics, featuring potentially useful procedures such as taxometric analysis, latent class analysis, and multiple group analysis;
- neuroscience, including behavior genetics and the study of neurocircuitry;
- nutrition and food science;
- positive psychology, including the study of intrinsic motivation and mindfulness;
- psychology/psychiatry, with their focus on what is normal and what is pathological, including a system of classification;
- psychometrics, emphasizing the development of valid and reliable screening, research, and diagnostic tools;
- public health, including epidemiology, illness screening, healthcare utilization, prevention, and advocacy;
- women and gender studies, including feminist psychology and mass communications.

In order to advance and remain vital, the EDs field needs to embrace critical self-analysis of what it has chosen to emphasize, how it has construed and applied what has been putatively

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demonstrated to be true, and what has been overlooked and actively ignored. Each of the contributors to these volumes was asked to raise significant issues in current research that could guide the direction of future investigations. This concluding chapter poses 10 critically important questions that we believe reflect broad themes worthy of attention in future theory and research. We encourage all readers to consider and assert what they believe are the most pressing directions for ED research, especially those that integrate at least three of the important themes.

Question 1: Have We Correctly Classified and Distinguished the Eating Disorders?

Categorical-prototypical thinking about the EDs is likely to retain its hegemony in the relevant fields represented in these volumes. From the standpoint of political influence in psychiatry and psychology, in healthcare coverage, and in guidance of research, a syndrome qualifies as an ED if the American Psychiatric Association decides that it does.

Of course, such decisions are far from arbitrary. The ED working group for the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*; American Psychiatric Association, 2013), published during the writing of these chapters, integrated expert consensus, reliability field trials, coordination with the forthcoming (in 2015) 11th edition of the *International Classification of Diseases* (*ICD-11*), and input from multiple sources in regard to feasibility and clinical utility (Wildes & Marcus, 2013). Nevertheless, the chapters in Sections I (Definitions and History) and III (Diagnosis) of Volume 1 point to the ongoing need for rigorous evaluation of the construct validity of each of the six diagnoses subsumed by the ED category in *DSM-5* (in order of presentation): avoidant/restrictive food intake disorder (ARFID); anorexia nervosa (AN, restricting and binge-eating/purging subtypes); bulimia nervosa (BN); binge eating disorder (BED); other specified feeding or eating disorder (OSFED); and unspecified feeding or eating disorder (USFED). Future research should use large samples from multisite collaborations to explore the emergence, replicability, and validity (e.g., value in directing treatment) of these or similar categorical distinctions (e.g., the Broad Categories of Diagnosis proposed by Walsh & Sysko, 2009) in adults, and, as important, in adolescents and children.

Hence, 35 years following publication of the revolutionary *DSM-III* (American Psychiatric Association, 1980), there remains a pressing need to investigate whether the *DSM*-designated ED diagnoses offer useful distinctions with respect to etiology, epidemiology, course, and treatment responsiveness. In this regard an important issue is to what extent these categories apply meaningfully to people who are not White females, 14 years or older, and from Western or industrialized countries.

Another key issue noted in a number of chapters is the significant variability within each of the recognized EDs, across the disorders within the superordinate EDs category, and over time—as evidenced in fluctuations in severity and in diagnostic crossover. This heterogeneity raises the important research question of whether the construct validity of the six prototypical diagnostic categories can be increased by supplemental evaluation of pertinent dimensions. Candidates for a dimensional profile that are discussed in several of the chapters include potentially significant psychological (e.g., overconcern with weight and shape; fear of fat) and/or neurobiological variables (impulsivity; cognitive rigidity; or negative valence systems as postulated by the National Institute of Mental Health [NIMH], 2011, in its Research Domain Criteria Matrix). Specification, assessment, and validation of candidate dimensions are

connected to the challenge of determining whether those basic dimensions are continuous with the range of normal attitudes and behaviors seen in populations at large. Note that, in conceptualizing and researching distinctions in the development and course of diagnostic categories, the important phenomena of diagnostic stability and diagnostic crossover are particularly deserving of further investigation.

Question 2: How Many People Develop Eating Disorders and Who is At Risk?

A strikingly high percentage of the chapters in these two volumes cite Hudson, Hiripi, Pope, and Kessler's (2007) prevalence findings from the National Comorbidity Survey Replication. This attests to the critical importance of good epidemiological data. Such data are scarce for a number of reasons, such as the cost in money, time, and effort to gather accurate data about relatively rare disorders from large and representative community samples. Nevertheless, such data should be a priority because they inform theory, cross-cultural studies, our understanding of correlates, risk and protective factors, and prevention. Some of the correlates traditionally assessed in epidemiological research with larger and representative samples, such as age, race or ethnicity, immigrant status, social class, gender, and geographic location, will undoubtedly be important in improving multifactorial etiological models. This, in turn, can help guide treatment and prevention.

Cross-cultural epidemiological research is needed to understand better the conditions that facilitate or inhibit an increased incidence of EDs. This has the potential to improve treatment and prevention and to focus models of etiology by providing data that show how risk and resilience are defined differently and operate differently within different contexts. Cross-cultural epidemiological research also has the potential to improve our thinking about assessment methodology, as we wrestle with aspects of translating the key features of EDs into different cultural and assessment settings.

It is an important theme of several chapters that epidemiological research is also needed to facilitate our conceptualization and assessment of "subclinical" eating problems, sometimes known as "disordered eating" (DE). Further research on DE is needed because it is important for:

- improving screening and early identification, followed by referral to either indicated (targeted) prevention or treatment;
- evaluating the extent to which, and for whom, DE is a developmental precursor to ED;
- clarifying the trajectory of disorder followed by partial remission;
- understanding how variables such as negative body image, calorie-restrictive dieting, self-objectification, and loss of control over eating may be connected to other health issues (e.g., depression, substance abuse, sexually transmitted diseases, overweight and obesity) of importance at the population level.

Question 3: What are We to Do with Comorbidity?

It is well established that people suffering from an ED are at greater risk for other psychological disorders. Cognitive rigidity, rejection sensitivity, negative affect, experiential avoidance, impulsivity, and a genetic vulnerability to stress over-reactivity are examples of processes and

characteristics that contribute to the causes and maintenance of a wide variety of psychological disorders (Mansell, Harvey, Watkins, & Shafran, 2009). In addition to mandating that a complete assessment and diagnosis of EDs also consider potentially comorbid disorders, comorbidity has two significant implications for further research.

First, ED theory, assessment, and research should consider nonspecific risk factors as clues to understanding comorbidity. Instead of dismissing these risk factors as irrelevant to the ED enterprise, they should be embraced as creating, maintaining, and strengthening dimensions that may form a core of psychopathology. Many of the chapters in Volume 1, Section V (Risk and Protective Factors and Correlates) raise such possibilities. These investigations will be particularly important in developing and guiding treatment.

The second important implication of comorbidity is closely related to the first. Although it may seem obvious and therefore trivial, it bears repeating that EDs qua EDs have unique features in relation to other disorders while sharing a variety of features with various other disorders. Many of the chapters in Volume 1 that consider risk and protective factors demonstrate that the more clearly a factor is related to the body image and eating components of EDs, the more likely the factor is specific to EDs. Yet, many variables that are prospectively and consistently related to EDs are also prospectively and consistently related to other forms of psychopathology, that is, are nonspecific risk factors. Therefore, our causal models and the research derived from them need to be clear as to whether variable X is contributing to the unique features of the ED in question, to psychopathology in general, or to both. Similarly, future studies of etiology and of therapy need to take into account how comorbid problems might mediate or moderate the relationship between the variable(s) under investigation, such as trauma or the effects of a particular therapy, and ED(s) as the outcome.

Question 4: What Standards of Evidence and Argument Will Help Us Determine What Causes Eating Disorders?

The question "what are the causes of EDs?" is so important and so complex that it will undoubtedly continue to be a stimulus for both reviews and controversy. Future research in the field of EDs would benefit from a two-step approach to capitalizing on different perspectives. The first step is a move toward general agreement on what needs to be explained, in light of well-established findings from epidemiological research and clinical descriptions. The second and equally important step concerns the standards of evidence that should be adopted in accepting or rejecting a variable as a causal risk, protective, or therapeutic factor.

Examination of consistencies across a number of the chapters in the present volumes points to consensus that a causal model of EDs—as full-fledged EDs, not chronic dieting, DE, or a very negative body image—needs to account for at least the following:

1 EDs have stages of development. This is true in the onset of EDs, as well as the recovery from EDs, as several of the treatment and therapy chapters in Volume 2, Sections III and IV, describe.
2 The presence of similarities across EDs that are not evident in other disorders. These similarities include a damaging overinvestment in weight and shape and substantial disturbances in eating. These are the symptoms that are specific to EDs. It is likely that risk factors specific to EDs will be associated with these specific components.

3 ED-specific influences need to be distinguished from nonspecific influences constituting vulnerability/risk for psychological disorders in general (see Question 2).
4 There are important distinctions in EDs as a function of gender, ethnicity, age, and cultural differences.
5 The inseparability (and not just the interaction) of biological, environmental, sociocultural, and psychological (i.e., biopsychosocial) influences.

It is noteworthy that the therapy chapters, as well as many of the prevention chapters (Volume 2, Section II), also seem to agree on the research methods (e.g., the randomized controlled trial with follow-up) necessary to yield data whose replication strongly supports the claim that an intervention causes the desired outcome. This reinforces the contention of several chapters throughout the volumes that it would be beneficial for the field to establish a similar consensus as to what standards of evidence (i.e., best practices) are necessary to establish a causal explanation for EDs as a developmental outcome. It is now a truism that there are no necessary and sufficient causes of any ED; rather, there are risk and protective factors (Volume 1, Sections IV & V; see next question). For the sake of brevity, readers are referred to Kraemer et al.'s (1997) detailed and cogent analysis of the systematic and convergent evidence necessary to distinguish ambiguous correlates of a condition from fixed markers, variable risk factors, and causal risk factors. This is an excellent starting point for future research on etiology.

Researchers need to continue to develop valid and reliable tools for assessing risk factors and outcomes. Simply translating measures or establishing internal consistency in a new sample does not validate a measure for use with a different gender, age, ethnic, or cultural group. One of the reasons that there are so many conflicting data discussed through this book is that various measures with varying levels of validity have been used.

Question 5: What are the Developmental Pathways for Eating Disorders?

As is evident from many chapters in both volumes, the move away from thinking in terms of "causes" means that, with respect to epidemiology, development, and prevention, the etiological issues must be framed in terms of multiple risk and protective factors that operate in multiple pathways toward and away from both psychopathology in general and EDs in particular. Multifactorial models, including moderators and mediators, will be needed. Indeed, some of the factors currently commonly conceptualized as risk or protective factors may ultimately be shown to moderate or mediate relationships between other risk factors and EDs. There is not likely to be a single developmental pathway to EDs. This is particularly evident among those whose ED onsets much later or much earlier than the norm. It is also evident in the differences among the EDs. But even among a relatively homogeneous group, such as White American adolescent girls, there is likely to be heterogeneity in the combination of factors that resulted in their ED.

In the future, understanding the interplay of risk and protective factors in the development (and/or maintenance) of EDs will require careful and, in our opinion, increased attention, not just to demonstrating the influence of such variables, but also to organizing them within multifactorial theories. The sections on risk and protective factors, special groups (Volume 1, Section VI), and, of course, theoretical perspectives all emphasize the contributions of theory-based research. Section IV in Volume 1 demonstrates the contributions of several important

theories. These chapters also point to new components of existing theories, as well as emerging theories that might be particularly valuable in guiding future research. The field of EDs needs theoretical developments in epidemiology, developmental psychopathology, and cross-cultural research in order to harness the power of a hypothetico-deductive methodology for understanding risk and resilience.

Consistent with the Kraemer et al. (1997) risk factor standards endorsed above, Sections IV through VI of Volume 1 underscore the important roles of longitudinal and experimental designs in testing theories. Of course, it is sometimes difficult to use these designs for examining certain risk factors, such as childhood sexual abuse. Furthermore, sometimes there is not yet sufficient justification to invest in prospective research. In these instances, case-controlled retrospective risk factor assessment may be helpful. However, given the need to distinguish ED-specific from ED-nonspecific influences, retrospective risk factor assessment must include at least one comparable psychiatric control group representing at least one of the disorders (notably depression, anxiety, and substance abuse) comorbid with EDs.

Question 6: How Can We Become More Developmental in Investigating the Development of Eating Disorders?

Both the risk factor chapters and the chapters focusing on children and adolescents clearly indicate that the foundation of many EDs is laid in childhood. This diathesis may derive from exposure to sociocultural pressures from family, peers, and media; it may be a genetically based personality vulnerability that is exacerbated by early experiences; or it may be a traumatic experience. Furthermore, epidemiological data outlined in Volume 1, Section II, demonstrate that females are at significantly greater risk for most EDs than males, and that AN frequently onsets in middle adolescence while BN commonly first appears in late adolescence. These findings suggest that developmental approaches may be an important part of understanding the etiology, maintenance, treatment, and prevention of EDs.

Of course a developmental approach means studying risk and protective factors as well as symptoms in children. As the risk factor chapters demonstrate, this has been done for many of the putative risk factors, particularly those associated with sociocultural models. Attention to biological factors in childhood and adolescence studies has been rarer, particularly for the neurobiological factors. There is a pressing need for such research.

Developmental psychologists adopt a lifespan approach. So we do not want to overlook the fact that EDs can onset later than is typical. We may find this is truer of some groups than others. For example, men may be more likely to show later onset than women are. It may also be the case that EDs other than AN and BN onset later (or earlier) than is typical of these two disorders. The process of the development of EDs that onset later in life also remains to be described. It is not even clear that all theoretical models apply equally well at all ages. While some of the chapters in Section VI (Special Groups) describe these differences, this is an area ripe for additional research.

Children, adolescents, and older adults also need to be included in more of the therapy studies. As the chapters in Volume 2, Sections III through V demonstrate, most of the research focuses on late adolescence and adulthood. We need more research examining what recovery looks like in children, adolescents, young adults, and older adults to better understand whether the process is similar across groups. Of course, which therapeutic approaches are most effective at different developmental stages is an open question.

A developmental approach is more than just using young samples. An ecological approach consistent with this emphasis is also seen in the chapters on culture and on special groups (Volume 1, Sections II, V, and VI), as well as in some of the prevention work described in Volume 2, Section II. Developmental psychologists have long abandoned the nature versus nurture debate for an approach that recognizes the inseparability and integration of the diverse factors involved in human development. A few sociocultural researchers and trauma-focused researchers have started this process in ED research but much more work is needed.

Question 7: What are the Crucial Issues Regarding the Role of the Family in the Development and Treatment of Eating Disorders?

The role of parents and other family members in the development and treatment of EDs is a salient and sometimes contentious matter. Le Grange, Lock, Loeb, and Nicholls' (2010) Academy for Eating Disorders (AED) position paper on "The Role of the Family in Eating Disorders" noted that the evidence to date did not support a causal role for the family in the development of EDs. However, several of the theory and risk factors chapters review data collected over the past 20–25 years that contradict that proposition. This is particularly true if, as is necessary (see Questions 5 and 6), we abandon the construct of one-to-one cause (e.g., family criticism of a child's weight and shape) and effect (e.g., development of BN) in favor of examining the impact of the family influences on known risk factors for EDs, such as weight and shape concerns and negative body image. Perhaps the only fact or principle on which everyone agrees is that no single person or factor is "responsible" for an ED, blame is counterproductive, and everyone who can has a responsibility to contribute positively to treatment and prevention. Whenever possible, families should be included in the recovery process "as a potential resource in therapy" (Le Grange et al., 2010, p. 2).

We agree with one of the principal conclusions in several of the chapters in Volume 1: as is true for all risk and protective factors, the research necessary to clarify the role of the family has yet to be done. Consequently, we encourage future theorists and researchers to do two things. First, keep in mind the aforementioned standards of evidence. There remains a need for well-designed longitudinal studies, with samples of sufficient size to detect meaningful prospective correlations, that examine family factors as putative risk or resilience factors, use (or develop) reliable and valid measures, and that differentiate between general psychopathological outcomes and specific ED outcomes. Retrospective studies with the proper psychiatric case controls can also contribute useful data in understanding negative and positive familial influences.

Second, in conceptualizing and assessing risk and resilience, it would be beneficial for researchers and clinicians to adopt a second principle in relation to family influences. In fact, this principle applies equally well to other sociocultural influences on the development, treatment, and prevention of EDs. Specifically, as is the case for peers, media, athletics, gender, ethnicity, or culture, there is a need to be precise in distinguishing, defining, and measuring dimensions of parental influence. These dimensions include observational learning (modeling), communication (e.g., criticism about eating, weight, and shape), and direct pressures to be thin or always in control. Looking across many chapters in Volume 1, it appears that researchers need to have modest expectations for the effect size of any individual risk factor. This means that in order to test risk-resilience theories of etiology (see Question 6), it will be important to examine the ways in which multiple influences, including family influences, might work together to promote or prevent the emergence of an ED.

Question 8: Can People Recover from an Eating Disorder?

A survey of the chapters in Volume 1, Sections I (Definitions and History) and III (Diagnosis), and Volume 2, Sections III (Issues in Treatment), IV (Therapeutic Approaches), and V (Controversies and Future Directions) indicates that, if given the opportunity, a significant percentage of people can indeed recover from an ED, even though success may require many years. That survey also reveals that the percentage of sufferers who recover varies across reviews and types of EDs, leading different experts to be more or less enthusiastic about complete recovery. Nevertheless, in general, recovery is possible even if the ED is severe, life threatening, and/or entrenched and chronic. This optimism is warranted even when recovery is defined as we believe it should be in future research. Specifically, recovery should be operationalized dimensionally and as a trajectory. At a minimum those dimensions should address:

- elimination of the ED and other symptoms (e.g., depression);
- restoration of weight and/or functional eating patterns;
- evidence of restored physical health;
- significantly improved attitudes about weight/shape and control;
- improved psychosocial and academic/occupational functioning.

For a minority of people recovery will be a lifelong status. They had an ED, now they are recovered, and they are likely to remain recovered for the rest of their lives. We need further research to determine, for each of the EDs, what aspects of our multidimensional definition of short-term recovery (e.g., eliminating undue influence of weight and shape) are most important in determining the developmental trajectory of sustained long-term recovery.

According to the therapy and outcome chapters, as well as several chapters in Volume 1, for many other people with an ED the course or outcome is better characterized as a trajectory of illness and of remission, that is, as a process that over time constitutes recovering. In this open-ended, dynamic, and variable process, there are longer and shorter remissions, interspersed with relapse (including diagnostic crossover) and significant improvements (partial remissions). Future research is needed to clarify some of the patterns across sufferers who are recovering, while investigating potential moderators of those patterns. Moderators emphasized throughout these volumes include ED subtype, developmental stage, gender, ethnicity, and cultural conceptions of illness, recovery, and caregiving. We agree with Strober and Lock that it should also be a clinical research priority for investigators to examine the following logical, but as yet not demonstrated hypothesis: earlier and more sustained remission predicts fewer and shorter periods of relapse later in the process, whereas, as is the case for mood disorders, partial remission following treatment predicts frequent recurrence or chronicity of disorder. This research would help clinicians and treatment teams to target the illness features and components of intervention with the greatest significance for recovery and resilience.

Question 9: What are the Best Ways to Treat Eating Disorders?

Examining the chapters on diagnosis, theory, and treatment reveals the many challenges to answering this question succinctly in ways that will be useful to people with EDs, clinicians, family members, and others (e.g., insurance companies in the United States). Researchers attempting to answer this critically important question must acknowledge (a) the heterogeneity

of EDs in terms of syndromes, comorbidity, psychopathology, age of onset, and cultural context; (b) the need to tailor treatment to both the severity of the disorder and the individual's position in the process that links the nature of their illness, preparation for recovery, and steps in recovery; (c) the value of evidence-based psychotherapy; and (d) the need for treatments whose efficacy is established in randomized controlled trials (RCTs) to be accessible and feasible for effective use in many different clinical settings by therapists with a wide range of clinical experience. Thus, all researchers must face some version of the time-honored question: "What is the best treatment protocol, including alternate interventions, for particular people at particular points in the development of their ED?"

In contrast to the present state of affairs for BN, BED, and other conditions characterized by binge eating, it would be difficult currently to provide an evidence-based answer to the question "What is the best or 'go to' treatment for anorexia nervosa in adults?" A set of evidence-based and humane practices is beginning to emerge for the minority of AN patients whose disorder is severe and enduring. Further research is needed to evaluate and elaborate the contention that for this "severe and enduring" group the process of recovering needs to be redefined to emphasize long-term maintenance of the patient-therapist relationship in order to effect small but achievable improvements in mood, function, harm avoidance, and in a sense of security, safety, respect, and self-compassion.

For the majority of AN patients, however, future research needs to continue developing and systematically evaluating a set of best practices for stepped care, including the type and role of inpatient, residential, and outpatient psychotherapy and pharmacotherapy. Several chapters in Volumes 1 and 2 suggest that clinical researchers should investigate the value of a flexible, eclectic approach to using family, psychodynamic, cognitive-behavioral, feminist, and interpersonal methods for addressing the ways in which extreme, idiosyncratic, and dysfunctional motives and meaning systems become attached to eating, weight, shape, and control. This endeavor will require further investigation of what truly distinguishes AN from BN and which AN features are most important in facilitating recovery.

Another pressing research question is to investigate the effectiveness of various, and perhaps newly developed, treatments for ethnic minority groups, men, and various age groups. Similarly, more research is needed to establish best-practice guidelines for treating those who have any combination of the following: difficulties in managing and expressing negative affect; high levels of comorbid psychopathology; the frequent experience of loss of control over eating. As is the case for AN, and likely for EDs in general, there is also a need for further research that clarifies when (in the therapeutic process), for whom (e.g., those with comorbid anxiety and depression, or impulsivity), and how the effective medications (e.g., antidepressants) are best used, such as when to begin with a combination, and whether understudied populations such as adolescents and males can benefit.

Question 10: Can Eating Disorders Be Prevented?

This is an important question. As documented in Volume 1, Section II (Epidemiology), millions of people worldwide are suffering from EDs, and there is absolutely no reason to believe that a medical model of detect and treat will significantly reduce that number, especially as new instances arise and as so few sufferers are seeking or getting adequate treatment. At present, the honest, evidence-based answer is two-fold: We do not know if

EDs can be prevented, but the chapters in Volume 2, Section II (Prevention) demonstrate clearly that there are several positive developments that support the potential of systematic prevention efforts.

The prevention chapters point to five significant areas for future prevention research. First, prevention outcome studies, in general, need to be framed by careful attention to methodology and standards of evidence. Second, along the same lines, there is a need for quantitative and qualitative analyses that facilitate an understanding of the processes by which the interventions are effective or ineffective for specific groups. Third, interventions that have been effective in selective and targeted prevention with older adolescent and young adult girls and women need to be adapted for other audiences (e.g., early adolescents boys and girls; male high-school athletes) and for those at lower risk and then subjected to rigorous evaluations.

Fourth, the Internet holds substantial promise in promoting prevention efforts that are widely available, cost-effective, respectful of individuals, and flexible. Future research needs to examine Internet-based prevention in order to clarify the processes of recruitment, engagement with the most effective program components, tailoring content to level of risk, ongoing monitoring of and feedback to participants, sustained positive changes, and referral and support for those whose risk is so great they probably have an ED.

Finally, work in developmental psychology, education, epidemiology, feminist community psychology, public health, and substance abuse prevention as well as many of the theoretical models described in Volume 1 converge to suggest that EDs are not simply an individual pathology. Rather, cultural milieus and social pressures, at both micro and macro levels, support the development and maintenance of EDs. Prevention programs have begun to investigate whether targeting the actual source of such sociocultural demands can reduce the onset or severity of EDs. Continued research in this area will not only help to identify effective prevention programs but also may help to clarify the role of sociocultural influences in the etiology of EDs.

Moving Forward

The research directions we are advocating, along with other research needs outlined in the chapter conclusions within this two-volume set, require considerable economic and human resources. That means that future research will also require support from—and therefore advocacy in relation to—many components of society, such as granting agencies, school systems, segments of the national government, mass media, medical and other healthcare education, nonprofit eating disorder organizations, and professional associations. A number of chapters in Sections II and V of Volume 2 detail ongoing political and cultural work to undo the undue influence of weight, shape, and disordered eating in Australia, Canada, Germany, Great Britain, Israel, and the United States.

It may seem that we are left with more questions than answers. Yet, the two volumes of this Handbook clearly demonstrate the substantial progress that has been made in understanding, treating, and preventing EDs. Scientific approaches will provide the data for us to continue to advance. Advocacy for funding and for recognition of the seriousness of EDs will also be crucial in moving the field forward and, ultimately, in helping us to achieve our shared goals of alleviating the great suffering associated with EDs.

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Abbreviations used: AN, anorexia nervosa; BED, binge eating disorder; BN, bulimia nervosa; ED, eating disorder; OSFED, other specified feeding or eating disorder; PTSD, posttraumatic stress disorder; UFED, unspecified feeding or eating disorder. Note that under the main entries 'anorexia nervosa' and 'bulimia nervosa' the entries are restricted to only major entries covering whole sections or chapters. In the main, only authors of historical or major importance are referenced. 'vs.' usually indicates differential diagnosis.

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