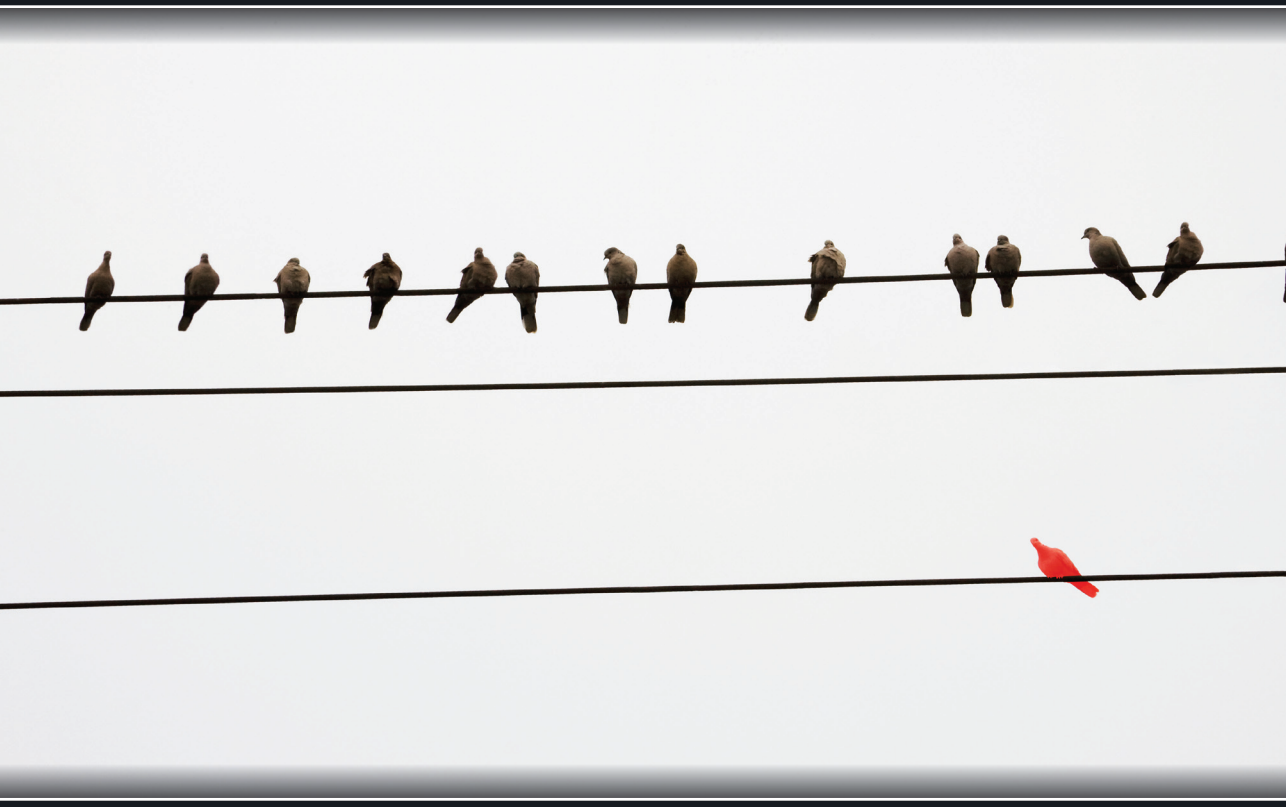

Edited by
Justin W. Weeks

THE WILEY BLACKWELL HANDBOOK OF *Social Anxiety Disorder*

WILEY Blackwell

The Wiley Blackwell Handbook of Social Anxiety Disorder

The Wiley Blackwell Handbook of Social Anxiety Disorder

Edited by

Justin W. Weeks
Center for Evaluation and Treatment of Anxiety (CETA)
Ohio University

WILEY Blackwell

This edition first published 2014

Registered Office
John Wiley & Sons Ltd, The Atrium, Southern Gate, Chichester, West Sussex, PO19 8SQ, UK

Editorial Offices
350 Main Street, Malden, MA 02148-5020, USA
9600 Garsington Road, Oxford, OX4 2DQ, UK
The Atrium, Southern Gate, Chichester, West Sussex, PO19 8SQ, UK

For details of our global editorial offices, for customer services, and for information about how to apply for permission to reuse the copyright material in this book please see our website at www.wiley.com/wiley-blackwell.

Library of Congress Cataloging-in-Publication Data

The Wiley Blackwell handbook of social anxiety disorder / edited by Justin W. Weeks.
pages cm
Includes bibliographical references and index.
ISBN 978-1-119-96860-3 (cloth)
1. Social phobia–Handbooks, manuals, etc. 2. Anxiety–Handbooks, manuals, etc.
I. Weeks, Justin W., editor of compilation. II. Title: Handbook of social anxiety disorder.
RC552.S62W55 2014
616.85′225–dc23

2013036258

A catalogue record for this book is available from the British Library.

Cover image: © Diane Macdonald / Getty Images
Cover design by Cyan Design

Set in 10/12.5pt Galliard by Aptara Inc., New Delhi, India

1 2014

To my parents, for setting me on the scholarly path,
and for their continued inspiration.

John and Jeanne Weeks

Contents

Notes on the Contributors xi

Part I Theoretical Overview: Social Anxiety Disorder

1 Cognitive-Behavioral Models of Social Anxiety Disorder 3
Judy Wong, Elizabeth A. Gordon, and Richard G. Heimberg

2 Evolutionary Models: Practical and Conceptual Utility for the Treatment and Study of Social Anxiety Disorder 24
Paul Gilbert

3 Genetic Factors in Social Anxiety Disorder 53
Murray B. Stein and Joel Gelernter

4 The Social Neuroscience of Social Anxiety Disorder 67
Supriya Syal and Dan J. Stein

5 The Pathophysiology of Social Anxiety 90
Wieke de Vente, Mirjana Majdandžić, and Susan Bögels

6 Personality: Understanding the Socially Anxious Temperament 111
Cheri A. Levinson, Simona C. Kaplan, and Thomas L. Rodebaugh

7 Behavioral Inhibition: A Discrete Precursor to Social Anxiety Disorder? 133
Dina R. Hirshfeld-Becker, Jamie A. Micco, Christine H. Wang, and Aude Henin

8 Relational Processes in Social Anxiety Disorder 159
Lynn E. Alden, Marci J. Regambal, and Leili Plasencia

Part II Variability Within Social Anxiety Disorder

9 Social Anxiety Disorder in Children and Adolescents 181
Thomas H. Ollendick, Kristy E. Benoit, and Amie E. Grills-Taquechel

10 Comorbidity: Social Anxiety Disorder and Psychiatric Comorbidity are not Shy to Co-Occur 201
Derek D. Szafranski, Alexander M. Talkovsky, Samantha G. Farris, and Peter J. Norton

11 Diversity Considerations in the Assessment and Treatment of Social Anxiety Disorder 223
Peter C. Meidlinger and Debra A. Hope

12 Heterogeneity Within Social Anxiety Disorder 247
Megan E. Spokas and LeeAnn Cardaciotto

Part III Optimizing Assessment Approaches: How to Best Target Social Anxiety Symptoms

13 Clinical Interviews: Empirical Overview and Procedural Recommendations 271
Daniel W. McNeil and Laura L. Quentin

14 Self-Report Assessment: The Status of the Field and Room for Improvement 292
Katya C. Fernandez, Marilyn L. Piccirillo, and Thomas L. Rodebaugh

Part IV Symptomological Manifestations

15 Cognitive Biases among Individuals with Social Anxiety 323
Shari A. Steinman, Eugenia I. Gorlin, and Bethany A. Teachman

16 Behavioral Deviations: Surface Features of Social Anxiety and What They Reveal 344
Wolf-Gero Lange, Mike Rinck, and Eni S. Becker

17 Examining the Controversy Surrounding Social Skills in Social Anxiety Disorder: The State of the Literature 366
Brent W. Schneider and Cynthia L. Turk

Part V Broadening the Scope of Social Anxiety Disorder: Areas Warranting Enhanced Empirical Attention

18 Translational Research in Social Anxiety: Summary of Newest Developments and Future Directions 391
Angela Fang and Stefan G. Hofmann

19 Positivity Impairment as a Broad-Based Feature of Social Anxiety 409
Eva Gilboa-Schechtman, Iris Shachar, and Yair Sahar

20 Fear of Positive Evaluation: The Neglected Fear Domain in Social Anxiety 433
Justin W. Weeks and Ashley N. Howell

21 The Neuroendocrinology of Social Anxiety Disorder 454
Gail A. Alvares and Adam J. Guastella

Part VI Treatment

22 Cognitive-Behavioral Therapy for Social Anxiety Disorder: The State of the Science 477
Dina Gordon, Judy Wong, and Richard G. Heimberg

23 Cognitive-Behavioral Therapy for Social Anxiety Disorder: Applying the Approach 498
Karen Rowa, Irena Milosevic, and Martin M. Antony

24 Pharmacological Treatment for Social Anxiety Disorder 521
Franklin R. Schneier, Laura B. Bragdon, Carlos Blanco, and Michael R. Liebowitz

25 Dual Diagnosis Cases: Treating Comorbid Social Anxiety Disorder and Substance Abuse or Dependence 547
Julia D. Buckner

26 Internet-Delivered Treatments for Social Anxiety Disorder 569
Gerhard Andersson, Per Carlbring, and Tomas Furmark

27 Acceptance and Mindfulness-Based Therapies for Social Anxiety Disorder: Current Findings and Future Directions 588
James D. Herbert, Marina Gershkovich, and Evan M. Forman

Index 609

Notes on the Contributors

Lynn E. Alden is a Professor of Psychology at the University of British Columbia and a former president of the Society for Interpersonal Theory and Research. Her research addresses the interplay of cognitive and interpersonal processes in the anxiety disorders. She has published numerous papers on social anxiety disorder and, with Ray Crozier, has published several volumes on shyness and social anxiety.

Gail A. Alvares is a PhD candidate at the Brain & Mind Research Institute, University of Sydney. Her research explores the effects of stress and anxiety on habit formation using translational models of decision making.

Gerhard Andersson, PhD, is a Professor in Clinical Psychology at Linköping University, Sweden. He has published 9 books and over 280 peer-reviewed papers on various topics such as depression, anxiety disorders, and somatic problems such as tinnitus and chronic pain. He is the group leader of a research group specializing in internet-based treatment.

Martin M. Antony, PhD, is a Professor in the Department of Psychology at Ryerson University, in Toronto. He has published 28 books and more than 175 scientific articles and chapters, mostly in the areas of anxiety disorders (e.g., social anxiety disorder, phobias, obsessive–compulsive disorder, panic disorder), perfectionism, and their treatment. He is a past president of the Canadian Psychological Association, and is a fellow of both the American and Canadian Psychological Associations.

Professor Eni S. Becker is chair of Clinical Psychology and the director of the research program "Experimental psychopathology and treatment" of the Behavioural Science Institute at the Radboud University Nijmegen. She is also chair of Nij-CARE (Nijmegen Centre of Anxiety research and Expertise). Her research interests are cognitive processes in anxiety and depression, approach and avoidance behaviour as well as cognitive bias modification.

Kristy E. Benoit is a pre-doctoral intern at Western Psychiatric Institute and Clinic, University of Pittsburgh Medical School, Pittsburgh, Pennsylvania. She is completing her doctoral studies at Virginia Tech and obtained her undergraduate degree from Harvard University. The author of several papers and chapters, her interests center upon experimental models of child psychopathologies and, in particular, the intergenerational transmission of anxiety from parents to their children. Social learning/social cognitive theory informs her work.

Carlos Blanco, MD, PhD, is a Professor of Clinical Psychiatry at the New York State Psychiatric Institute/Columbia University. The focus of his research is the epidemiology and treatment of mood, anxiety and addictive disorders. His research has been supported by NIH, the New York State Psychiatric Institute and several private foundations.

Susan Bögels is a Professor in Developmental Psychopathology at the Research Institute of Child Development and Education, University of Amsterdam, and director of the academic treatment center for parents and children UvA-Minds. Her current research interests concern the intergenerational transmission of anxiety, with a particular interest in the role of fathers; treatment of anxiety disorders in children; and mindfulness in families.

Laura B. Bragdon is a research coordinator at The Anxiety Disorders Center at Hartford Hospital's Institute of Living. She received her MA degree in clinical psychology from Columbia University in 2011. Most recently she coauthored the article "The evidence-based pharmacotherapy of social anxiety disorder." (*International Journal of Neuropsychopharmacology*, 21, 1–15. E-pub ahead of print).

Julia D. Buckner, PhD, is a licensed clinical psychologist and assistant professor in the Department of Psychology at Louisiana State University. She directs the LSU Anxiety & Addictive Behaviors Research Laboratory and Clinic. Her research efforts aim to understand factors that contribute to the co-occurrence of anxiety and problematic substance use. She also works to improve treatment outcomes for patients with anxiety disorders, with particular focus on patients with co-occurring substance use.

LeeAnn Cardaciotto, PhD, is an Assistant Professor at La Salle University in Philadelphia. Her research interests include the examination of an acceptance-based behavioral model of social anxiety disorder (Herbert & Cardaciotto, 2005), as well as the role of shame and self-compassion in the etiology and maintenance of social anxiety.

Per Carlbring is a Professor in Clinical Psychology at the Department of Psychology, Stockholm University, Stockholm, Sweden. He has conducted several studies on internet-based treatment for anxiety and mood disorders and has also done research on internet-based assessments and experiments.

Wieke de Vente is an Assistant Professor at the Research Institute of Child Development and Education, University of Amsterdam. Her research focuses on

psychophysiological aspects of psychopathology. Previously, she participated in a longitudinal study about lifestyle and chronic diseases and conducted a trial on the effectiveness of cognitive-behavioral treatment for work-related stress. Currently, she is involved in longitudinal studies about the early development of social anxiety, including psychophysiological development, and reducing stress during pregnancy through biofeedback.

Angela Fang, MA, is a doctoral student in the clinical psychology program at Boston University. Angela is currently conducting her dissertation entitled, "Effect of oxytocin on pro-social behavior in social anxiety disorder." Her research interests primarily involve examining the neurobiological and information processing mechanisms underlying anxiety disorders, particularly social anxiety disorder. She is also interested in examining the relationship between social anxiety disorder and related disorders such as body dysmorphic disorder.

Samantha G. Farris, BA, is a clinical psychology doctoral student at the University of Houston. She received her BA in psychology from Rutgers University, while working at the Center of Alcohol Studies. After completing her degree, Ms. Farris worked as a research coordinator at the Center for the Treatment and Study of Anxiety, at the University of Pennsylvania. Her research primarily addresses the interrelations between emotional and stress vulnerabilities and substance use disorders.

Katya C. Fernandez, MA, is a graduate student at Washington University in St. Louis. Her research focus is the development of enhanced assessment techniques for mood and anxiety. This research consists of augmenting traditional self-report with more powerful assessment modalities such as informant report and longitudinal assessment, and in developing novel assessment systems that are more easily accessible in research and clinical contexts.

Evan M. Forman, PhD, is Associate Professor and Director of Graduate Studies at Drexel University. Research interests include the development and evaluation of acceptance-based behavioral interventions for health behavior change (especially obesity) as well as mood and anxiety disorders; neurocognitive contributors to, and interventions for, overeating; and remote treatment.

Tomas Furmark is a Professor in Clinical Psychology at Uppsala University, Sweden. He has been a part of the Swedish Research team on internet-based treatment of social anxiety (SOFIE) since the start in 2003 and he is also doing neuroimaging research in anxiety disorders.

Joel Gelernter, MD, is Foundations Fund Professor of Psychiatry and Professor of Genetics and of Neurobiology at Yale University where he is Director, Division of Human Genetics (Psychiatry). He is also staff psychiatrist at the VA Connecticut Healthcare Center in West Haven, CT. Dr. Gelernter has been conducting genomic research in the areas of substance use disorders and anxiety disorders for the past 25 years.

Marina Gershkovich is a doctoral candidate in clinical psychology at Drexel University in Philadelphia. Current research interests include internet-based interventions for mood and anxiety disorders, and mindfulness and acceptance-based treatments.

Paul Gilbert, PhD, FBPsS, OBE, is a Professor of Clinical Psychology at the University of Derby and Consultant Clinical Psychologist at the Derbyshire Health Care Foundation Trust. He has researched evolutionary approaches to psychopathology for nearly 40 years with a special focus on shame and the treatment of shame-based difficulties in a variety of disorders, for which compassion focused therapy was developed. In 2006, he established the Compassionate Mind Foundation (www.compassionatemind.co.uk).

Eva Gilboa-Schechtman is an Associate Professor at Bar-Ilan University in Israel. Her research is focused on the understanding and treatment of anxiety disorders and depression. She is interested in the ways nonverbal cues and social rebuffs combine to maintain social anxiety, depression, and low self-esteem. She is the author of numerous publications on social anxiety, PTSD, and depression. She has coauthored a book on PTSD for adolescents, and co-edited books on CBT for emotional disorders.

Dina Gordon, MA, is a clinical psychology doctoral candidate at Temple University working under the mentorship of Dr. Richard Heimberg. She was trained in cognitive-behavioral therapy for adults with social anxiety disorder at the Adult Anxiety Clinic of Temple. Her research interests lie in the nature and treatment of anxiety disorders, specifically, in the roles of emotion regulation, mindfulness, distress tolerance, and experiential avoidance in the anxiety cycle and as moderators of treatment outcome.

Elizabeth A. Gordon, MA, is a clinical psychology doctoral candidate at Temple University working under the mentorship of Dr. Richard Heimberg. Her primary research interests include interpersonal processes among those who suffer with high social anxiety, such as communication within intimate relationships. She is also interested in how evolutionary and social psychological models can inform conceptualization and treatment of anxiety disorders and other mental health problems.

Eugenia I. Gorlin is a doctoral candidate at the University of Virginia. She received her MA from the University of Virginia and BS from Tufts University. Her research focuses on the way relatively controlled, strategic cognitive processes interact with more automatic processes in the course of both adaptive and maladaptive emotion regulation, and how the nature of these interactive processes can inform psychological treatment.

Amie E. Grills-Taquechel is an Assistant Professor of Counseling Psychology in the School of Education at Boston University. She has published in the areas of childhood anxiety and related disorders, trauma, and evidence-based assessment and treatment of children for the past 10 years. She has also given numerous conference presentations, secured grants, and received prestigious awards, including selection as a 2011 American Psychological Society Rising Star.

Adam J. Guastella is an Associate Professor, clinical psychologist, and principal research fellow at the Brain & Mind Research Institute, University of Sydney. His research focuses on using translational models to improve social function in disorders of social deficit and to develop novel methods to reduce anxiety.

Richard G. Heimberg, PhD, is the Director of the Adult Anxiety Clinic of Temple University. He is well known for his efforts to develop cognitive-behavioral treatments for social anxiety and examine their efficacy in comparison to (or in combination with) medication treatments. He has published 11 books and more than 350 papers on this and related topics. His research has been supported by a number of grants from the National Institute of Mental Health.

Aude Henin, PhD, is the Co-Director of the Child Cognitive Behavior Therapy Program at the Massachusetts General Hospital (MGH); and an assistant professor of psychology in the Department of Psychiatry at the Harvard Medical School. Her research focuses on the development and evaluation of cognitive-behavioral therapy protocols to assist with symptom management, skills development, and improved functional autonomy in transition age youth with mood or autistic disorders.

James D. Herbert, PhD, is Professor and Head of the Department of Psychology at Drexel University. Current research interests include acceptance-based behavior therapies, anxiety disorders, telepsychology, and the promotion of evidence-based practice in mental health.

Dina R. Hirshfeld-Becker, PhD, is the Co-Director of the Child Cognitive Behavior Therapy Program at the Massachusetts General Hospital (MGH); and is Associate Professor of Psychology in the Department of Psychiatry at the Harvard Medical School. Her research focuses on risk factors for psychiatric disorders in children at risk, and early intervention for children with anxiety disorders.

Stefan G. Hofmann, PhD, is a Professor of Psychology at the Department of Psychology at Boston University where he directs the Psychotherapy and Emotion Research Laboratory. His research focuses on the mechanisms of treatment change, translating discoveries from neuroscience into clinical applications, emotions, and cultural expressions of psychopathology. He is President of ABCT and IACP, Editor of Cognitive Therapy and Research, and Associate Editor of Journal of Consulting and Clinical Psychology.

For more information, see: http://www.bostonanxiety.org/

Debra A. Hope, PhD, is a Professor of Psychology at the University of Nebraska-Lincoln and Clinical Director of the Weibling Project. Her primary research interests are in treatment of anxiety disorders, particularly social anxiety, and the mental health impacts of discrimination.

Simona C. Kaplan received a BA from Washington University in St. Louis. She currently works at the Eating Disorders Research Unit at Columbia University.

As a research assistant at Washington University's Anxiety and Psychotherapy laboratory, Simona studied social anxiety and personality, with an emphasis on predictive interactive relationships between factors and lower-order facets of personality.

Ashley N. Howell, MS, is a clinical psychology doctoral candidate at Ohio University working under the mentorship of Dr. Justin Weeks. Her primary research interests include the roles of culture and cognition regarding etiological and maintenance factors for social anxiety disorder. Additional research interests include the objective assessment and analysis of social anxiety symptoms.

Wolf-Gero Lange is Assistant Professor at Radboud University Nijmegen (The Netherlands) and visiting scholar at University of Cologne (Germany). Originally, he was trained in biological psychology, but has since specialized in the experimental exploration of disrupted cognitive and behavioral aspects of social anxiety. In detail, his research addresses cognitive biases (in the processing of facial expressions), automatic behaviors such as impulsive approach and avoidance, prosocial behaviors such as mimicry, and subtle behaviors such as body sway and interpersonal space in social interactions.

Cheri A. Levinson, MA, is a graduate student at Washington University in St. Louis. Cheri's current research interests focus on the comorbidity between social anxiety disorder and eating disorders. Cheri is also interested in the relationship between social anxiety and peer victimization, personality, and health.

Michael R. Liebowitz, MD, is a Professor of Clinical Psychiatry at the College of Physicians and Surgeons of Columbia University and is a former Director of the Anxiety Disorders Clinic, and Research Psychiatrist at, the New York State Psychiatric Institute. Dr. Liebowitz is also Managing Director of The Medical Research Network, LLC, a private clinical trials site in NYC, and is the developer and copyright holder of the Liebowitz Social Anxiety Scale.

Mirjana Majdandžić is a researcher at the Research Institute of Child Development and Education, University of Amsterdam. Her research focuses on (observations of) temperament and parenting behavior. She has conducted a longitudinal observational study on temperament and parenting behavior in families with two children. Currently, she is involved in longitudinal studies about the early development of social anxiety, in which she focuses on behavioral inhibition and parenting behavior, in particular on challenging parenting behavior of fathers.

Daniel W. McNeil is an Eberly Distinguished Professor at West Virginia University. His work is based on an experimental psychopathology approach, and has included a focus across the continuum of social anxiety, broadly considered, including Social Anxiety Disorder, Social Phobia, public speaking phobia, and shyness. More broadly, he is interested in the relation between anxiety and pain, particularly in the domain of Health Psychology. Dr. McNeil directs the Anxiety, Psychophysiology, and Pain Research Laboratory.

Peter C. Meidlinger, MA, is a graduate student in the clinical psychology training program at the University of Nebraska-Lincoln. His primary research interests are minority stress processes among sexual minorities and the treatment of anxiety disorders.

Jamie A. Micco, PhD, is a staff psychologist in the Child Cognitive Behavior Therapy Program at the Massachusetts General Hospital (MGH); and is an Instructor of Psychology in the Department of Psychiatry at the Harvard Medical School. Her research focuses on cognitive bias retraining interventions for adolescent depression, and on the study of interpretation and attention biases in children at risk for anxiety disorders.

Irena Milosevic, PhD, is a post-doctoral fellow at the Anxiety Treatment and Research Centre and in the Consultation-Liaison Service at St. Joseph's Healthcare in Hamilton, Ontario. Her research has investigated the role of safety behavior in cognitive-behavioral treatment (CBT) for anxiety disorders and the effectiveness of integrated CBT for comorbid anxiety, mood, and substance use disorders. She has published scientific articles and presented extensively at research conferences on these and related topics.

Peter J. Norton, PhD, is an Associate Professor of Psychology at the University of Houston, and Director of the University of Houston Anxiety Disorder Clinic. Dr. Norton has also received early career awards and research grants from the US National Institute of Mental Health, National Institute of Drug Abuse, Anxiety Disorders Association of America, and American Psychological Association, for his work on studying and treating anxiety.

Thomas H. Ollendick is University Distinguished Professor of Psychology and Director of the Child Study Center at Virginia Tech. He is the author or coauthor of several research publications, book chapters, and books. The recipient of several NIMH grant awards, his clinical and research interests range from the study of diverse forms of child psychopathology to the assessment, treatment, and prevention of these child disorders from a social learning/social cognitive theory perspective.

Marilyn L. Piccirillo is an undergraduate student pursuing her Bachelor of Arts at Washington University in St. Louis. As a research assistant at Washington University's Anxiety and Psychotherapy Laboratory, Marilyn studied the relationship between positive affect and social comparison in social interactions and is primarily interested in the relationship between anxiety and urbanicity (the urban or rural nature of an individual's environment). She will continue her study of anxiety as a graduate student at Temple University.

Leili Plasencia is a PhD student in Clinical Psychology at the University of British Columbia. Her research addresses the role of safety-seeking behaviors in the anxiety disorders and the way in which anxiety disorders affect the individual's sense of self.

Laura L. Quentin is a doctoral student in Clinical Psychology at West Virginia University. Her research interests are in anxiety, fear, pain, and Health Psychology. She is involved in two grant-funded projects focusing on anxiety, pain, and depression. Her work on these projects includes administration of structured interviews.

Marci J. Regambal is a PhD student in Clinical Psychology at the University of British Columbia. She has coauthored book chapters on interpersonal processes in the anxiety disorders and social anxiety disorder and the self. Her primary research interests are trauma and posttraumatic stress disorder.

Mike Rinck is Associate Professor at Radboud University Nijmegen (The Netherlands) and Adjunct Professor at Ruhr-University Bochum (Germany). He was originally trained as a cognitive psychologist, and his research topics cover a wide range of cognitive processes and mental disorders within the field of Experimental Psychopathology. His most recent research addresses automatic approach-avoidance tendencies and cognitive bias modification techniques, mainly in anxiety disorders, depression, and addictions.

Thomas L. Rodebaugh, PhD, is an Associate Professor of Psychology at Washington University in St. Louis. He researches anxiety, anxiety disorders, and their assessment and treatment. In particular, he is interested in social anxiety and its interpersonal consequences and correlates. He also has focused on refining assessment of anxiety and applying appropriate statistical models to aid understanding of anxiety.

Karen Rowa, PhD, is an assistant professor in the Department of Psychiatry and Behavioral Neurosciences at McMaster University and a Psychologist at the Anxiety Treatment and Research Centre at St. Joseph's Healthcare in Hamilton, Ontario. She has published two books and numerous scientific articles and chapters in the areas of social anxiety disorder, obsessive–compulsive disorder, and their treatment.

Yair Sahar is a PhD student in Clinical Psychology at Bar-Ilan University. His primary research interests are in the processing of emotional facial expressions in social anxiety. He is also interested in attentional modification paradigms and their implementation in anxiety disorders.

Brent W. Schneider, MA, received his master's degree in psychology with an emphasis in clinical skills from Washburn University in 2012. He completed his master's degree internship placement at the Kansas City Center for Anxiety Treatment. In addition to his research and clinical interests in anxiety, he is interested in family satisfaction following a child's disclosure of his or her sexual orientation.

Franklin R. Schneier, MD, is a Professor of Clinical Psychiatry at Columbia University College of Physicians and Surgeons, and Research Scientist in the Anxiety Disorders Clinic at New York State Psychiatric Institute. He is a member of the Scientific Advisory Board of the Anxiety Disorders Association of America. Dr. Schneier's research has focused on the diagnosis, etiology, and treatment of social anxiety disorder, generalized anxiety disorder, and posttraumatic stress disorder.

Iris Shachar is a PhD student in Clinical Psychology at Bar-Ilan University. Her primary research interests are in social anxiety in adolescents, especially in the way socially anxious youth use technologically mediated social communication.

Megan E. Spokas, PhD, is an Assistant Professor at La Salle University in Philadelphia. Her research interests include the etiological origins of social anxiety disorder, and improving cognitive-behavioral treatments for anxiety disorders.

Dan J. Stein is Professor and Chair of the Department of Psychiatry at the University of Cape Town, Director of the Medical Research Council Unit on Anxiety Disorders, and Visiting Professor of Psychiatry at Mt. Sinai Medical School in New York. He is interested in the psychobiology and management of anxiety, obsessive–compulsive, and traumatic and stress disorders.

Murray B. Stein, MD, MPH, is a Professor of Psychiatry and Family and Preventive Medicine at the University of California San Diego. He has been conducting clinical and translational research in the area of anxiety disorders for the past 25 years.

Shari A. Steinman is a doctoral candidate at the University of Virginia. She received her MA from the University of Virginia and BS from Washington University in St. Louis. Her research focuses on how individuals think differently when anxious. In particular, she is interested in how cognitive biases play a role in the onset, maintenance, and treatment of anxiety problems.

Supriya Syal is a post-doctoral fellow at the University of Toronto where she is conducting research on the social modulation of cognitive behavior. Supriya followed her early training in Biochemistry and Neuroscience with a PhD in psychology at Cornell University in New York and post-doctoral work at the University of Cape Town. She is interested in social-affective regulation of complex behavior in particular, and evolution and cognitive neuroscience in general.

Derek D. Szafranski, MA, is a fourth year graduate student in clinical psychology at the University of Houston. Derek received his BS in psychology from Western Michigan University and his MA in psychology from the University of the Pacific. He specializes in anxiety treatment outcome research, and PSTD and concomitant problems among combat Veterans.

Alexander M. Talkovsky, BA, is a second year graduate student in clinical psychology at the University of Houston. Alex received his BA in psychology from Washington University in St. Louis. He specializes in the study and treatment of anxiety.

Bethany A. Teachman is an Associate Professor and the Director of Clinical Training at the University of Virginia in the Department of Psychology. She received her PhD from Yale University and BA from the University of British Columbia. Her research focuses on biases in cognitive processing that contribute to emotion dysregulation, such as anxiety and related disorders, with a particular interest in investigating how automatic cognitive processes can be modified.

Cynthia L. Turk, PhD, received her doctoral degree in clinical psychology from Oklahoma State University after completing her internship at the University of Mississippi/Department of Veterans Affairs Medical Centers Psychology Residency Consortium. She completed a post-doctoral fellowship at the Adult Anxiety Clinic at Temple University. She is currently Professor of Psychology and Director of the Anxiety Clinic at Washburn University. She has 60 professional publications and 85 conference presentations.

Christine H. Wang, BA, is a Clinical Research Coordinator in the Child Cognitive Behavior Therapy Program at the Massachusetts General Hospital. Her research interests focus on familial risk factors for the development of internalizing and externalizing disorders in children and adolescents and interventions for such disorders in various populations.

Justin W. Weeks, PhD, is the Director of the Center for Evaluation and Treatment of Anxiety (CETA) at Ohio University. He is the author of a number of scientific publications and book chapters—the majority of these publications focus specifically on social anxiety and social anxiety disorder. Dr. Weeks is well known for his work on fear of positive evaluation as a proposed core element of social anxiety disorder, as well as on the enhancement of objective assessment of social anxiety symptoms.

Judy Wong, MA, is a clinical psychology doctoral candidate at Temple University working under the mentorship of Dr. Richard Heimberg. Her primary research interests include the influence of culture, ethnic minority status, and immigration on mental health. Specifically, she is interested in looking at attitudes and factors associated with culture and ethnicity that influence treatment-seeking behavior. She is also interested in examining cultural competence in therapy.

I

Theoretical Overview

Social Anxiety Disorder

1

Cognitive-Behavioral Models of Social Anxiety Disorder

Judy Wong, Elizabeth A. Gordon, and Richard G. Heimberg

Adult Anxiety Clinic, Temple University, USA

Cognitive-Behavioral Models of Social Anxiety Disorder

Since its recognition as a mental disorder in the *Diagnostic and Statistical Manual of Mental Disorders*, third edition (American Psychiatric Association, 1980), social anxiety disorder (SAD, also known as social phobia) has received increasing attention in the field of psychology as a complex, debilitating disorder that, left untreated, is often unremitting. In the last few decades, many theorists have contributed significantly to our understanding of this disorder, subsequently informing approaches to treatment. In this chapter, we review and compare aspects of the two preeminent cognitive behavioral models of SAD, as well as more recently proposed models of SAD.

Clark and Wells (1995): A Cognitive Model of SAD

Clark and Wells (1995) put forth a cognitive[1] model of SAD to explain why exposure to feared situations alone was not enough to extinguish fear in socially anxious individuals. According to their model, SAD develops as a result of an interaction between innate behavioral predispositions and life experiences, leading individuals to perceive the social world as a dangerous one which they have little ability to navigate. A core feature of this model, derived from self-presentational models described below, is "a strong desire to convey a particular favorable impression of oneself to others and marked insecurity about one's ability to do so" (p. 69). These beliefs contribute to the sense that the person with SAD is at substantial risk of behaving in an inept and unacceptable fashion and that such behavior will have catastrophic consequences involving loss of status, loss of value, or rejection. The following is a brief overview of the model—a discussion of the empirical support for specific aspects of the model is beyond the scope of this chapter, but interested readers are referred to reviews of research by Clark and Wells (1995) and Clark (2001).

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

Dysfunctional Processes

Clark and Wells (1995) describe the dysfunctional pattern of social anxiety as being comprised of four interactive processes. The first process begins when people with SAD enter a feared situation and judge that they may be in danger of being negatively evaluated. They then turn their attention inward and use interoceptive information as the main source of feedback about their performance. Often, their internal experiences appear to provide confirmation of their social ineffectiveness, which is believed to be obvious to those around them (e.g., "I feel nervous, therefore everyone must realize I am nervous"). Compounding this negative self-perception, people with SAD often imagine themselves as others see them (the "observer perspective"), though these images are likely to be quite distorted. Clark and Wells refer to this attentional inward bias and distorted images as a *processing of the self as a social object*, and this is the putative reason why exposure alone to feared situations is insufficient to reduce social anxiety. They write:

> Clinically, the importance of this processing bias is that it prevents social phobics from getting maximum benefit from their everyday experience with social situations or from the exposure exercises used in behavior therapy treatment programs. When in feared social situations, social phobics process the negative feelings generated by their fear of the situation, but they do not check out what is really happening. (p. 72)

The second dysfunctional process relates to behaviors that socially anxious individuals engage in to prevent negative evaluation by others. Clark and Wells (1995) refer to these behaviors as *safety behaviors.* For instance, a person concerned with others noticing his profuse sweating may wear an extra layer of dark clothing. Ironically, safety behaviors often make the feared behavior or outcome more likely to occur: the extra layer of clothing may cause the person to sweat more. Safety behaviors also serve to maintain anxiety because they prevent the person from experiencing unambiguous, disconfirming evidence of their negative beliefs about feared consequences. So, although the feared outcome may not have occurred (e.g., people did not express disgust about the person's sweating), the person with SAD may attribute this to the fact that he or she engaged in this safety behavior.

The third dysfunctional process described by Clark and Wells (1995) is that individuals with SAD often overestimate how negatively others evaluate their performance and predict the consequences of social failures to be far worse than is realistic. As a result of these cognitive distortions, they are hypervigilant in monitoring their behavior and performance, which may further impair their ability to fully engage in social interactions. Real performance deficits may result, which could lead to others perceiving them to be socially unskilled, aloof, or unfriendly.

The final dysfunctional process delineated by Clark and Wells (1995) occurs either before or after a social situation is encountered. Prior to engaging in a social event, many individuals with SAD frequently experience a period of anticipatory anxiety in which previous negative experiences are recalled, and expectations of failure and images of the self performing poorly are evoked. This can lead to complete avoidance of the

situation. However, if the situation is not avoided, anticipatory anxiety can lead the person to enter the situation with a self-focused processing mode and reduced capacity for noticing positive reactions from others. Following a social interaction, people with SAD frequently review their performance in detail (referred to by Clark and Wells, p. 74, as a "postmortem" review or "post-event processing"), often recalling events and their outcomes to have been more negative than they really were, as their perceptions are colored by their attentional biases and cognitive distortions. Ultimately, this helps maintain negative self-schemas and increases the likelihood that the person will avoid feared situations in the future.

Rapee and Heimberg (1997): A Cognitive-Behavioral Model of SAD

Along with Clark and Wells' (1995) and Rapee and Heimberg's (1997) model is the other most widely cited and applied model of SAD in the literature. According to Rapee and Heimberg, social anxiety exists along a continuum, with individuals with SAD representing the higher end of the continuum. Similarly, the degree of dysfunctional patterns can be represented along a continuum. Thus, according to the model, the difference between those with SAD and those without is "the extent to which [individuals with SAD] appraise cues as predictive of threat and the extent of threat predicted by a given cue" (Rapee & Heimberg, 1997, p. 751).

A number of different factors are thought to influence the development of dysfunctional processes, which in turn lead to the development of SAD. A genetic tendency toward preferential attention to threat may be one factor, which interacts with early childhood family environment and/or other experiences (e.g., being teased or bullied) to create a perception of the social world as being dangerous and unforgiving. Consequently, a defining characteristic among those with SAD is the assumption that others are likely to evaluate them negatively. Additionally, individuals with SAD attach fundamental importance to being accepted by others. The result is a set of expectations and goals that the person feels unable to reach, accompanied by predictions of very negative consequences of this failure. The discrepancy between the mental representations of the self as seen by others and others' perceived expectations, according to Rapee and Heimberg (1997), lies at the heart of SAD. Below, we provide an overview of the model, including its recent update (Heimberg, Brozovich, & Rapee, 2010). As with the Clark–Wells model, a discussion of the empirical support for the Rapee–Heimberg model is beyond the scope of this chapter. Interested readers are referred to the original theoretical articles for reviews of empirical research; see also Roth and Heimberg (2001) and Turk, Lerner, Heimberg, and Rapee (2001).

Dysfunctional Processes

In this model, "social situations" are defined broadly and may include situations in which no social interaction actually occurs, as the presence of a *perceived* threat may be enough to evoke anxiety. Thus, the stranger walking down the street may become

an audience for and potential judge of the socially anxious person's appearance and behavior. For individuals with SAD, the prospect of an audience activates a mental representation of the self as they imagine they are perceived by that audience. This mental representation of the self is a distorted image that is shaped by a number of inputs. Rapee and Heimberg (1997) proposed that individuals form a "baseline image" (p. 745) that may be derived from past experiences and actual images of the self as seen by an audience (e.g., from mirrors or photographs) and which is consistent with negative self-schemas and core beliefs. It is modified in any given situation by internal (i.e., interoceptive) and external feedback. For instance, sensations of warmth may cause the person to imagine herself to be blushing noticeably, or a passing and ambiguous comment by another person in a group interaction may lead the person to think she has said something contrary to group opinion, and she thus imagines that she "looks stupid."

According to the model, one reason this mental representation of the self as seen by the audience is distorted is that individuals with SAD have a bias toward attending to external cues in the social environment that signal threat or negative evaluation. This orientation to threat is consistent with other anxiety disorders. However, Rapee and Heimberg (1997) also hypothesized that individuals with SAD also preferentially allocate attentional resources to monitoring and adjusting the mental representation of the self as perceived by the audience. This is in addition to the attentional resources needed to engage in the social task at hand. Consequently, social performance suffers as attentional resources are taxed, and the poor performance only serves to confirm negative mental representations of the self (e.g., that one is socially unskilled, awkward, etc.).

The model proposes that a key dysfunctional process is the comparison of the mental representation of the self with the perceived expectations of the audience. Socially anxious individuals typically believe that others hold extremely high standards for their performance, and the greater the perceived failure to live up to this standard, the greater the likelihood of negative evaluation, and the greater the anxiety. Socially anxious individuals anticipate the cost of such failure to be high, and this anticipation activates behavioral, cognitive, and physical symptoms of anxiety, which feed back into the mental representation of the self as seen by the audience in a most deflating way, renewing the vicious cycle, which continues until the situation comes to a natural end or is terminated by the anxious person. It is therefore not surprising that socially anxious individuals often engage in avoidance or escape from feared situations, as it seemingly provides respite from this cycle. However, behavioral avoidance becomes yet another source of shame and frustration and contributes to an increasingly negative mental representation of the self as seen by the audience.

In 2010, Heimberg et al. published an updated version of the model to incorporate knowledge from new findings about the processes that occur in SAD. For instance, a growing body of research has shown that individuals with SAD frequently engage in negative self-imagery (e.g., Hackman, Surawy, & Clark, 1998). In addition, compared with non-anxious individuals, the images of socially anxious individuals are often from the observer's perspective (Hackman et al., 1998). These findings are consistent with the theory that those with SAD formulate a mental representation of

the self as seen by the audience. The updated model highlights the role of negative imagery in influencing the mental representation of the self, and ultimately serving to maintain SAD.

A significant change to the model addresses what is thought to be the core fear in SAD, typically characterized as a fear of negative evaluation. However, recent research suggests that socially anxious individuals fear *any* evaluation, whether it is negative or positive (e.g., Weeks, Heimberg, Rodebaugh, & Norton, 2008; see **Chapter 20** of this volume). Fear of positive evaluation (FPE) may arise when successful social performance activates the belief that others will expect continued success in future social interactions, but the person may doubt his or her ability to meet these increased expectations. However, the construct of FPE is derived from an evolutionary model of SAD, which posits that socially anxious individuals work to maintain their (low) social status by not drawing attention to themselves (Gilbert, 2001; see **Chapter 2** of this volume). In this way, they do not risk losing status, nor will they have to engage in conflict with more powerful others to defend any elevated social status they may have achieved. The update to the Rapee–Heimberg model reflects this line of thinking, and the model now posits that those with SAD fear and attend to cues of evaluation, regardless of valence.

Lastly, another significant addition to the Rapee–Heimberg model is the inclusion of post-event processing (PEP) as a maintaining factor of SAD. As discussed by Clark and Wells (1995), PEP refers to the phenomenon of a person's review and recall of a situation after it has occurred. Often, the recall is biased and distorted, which then fuels fear and avoidance of future situations. PEP can therefore be conceptualized as the ongoing process that links the experience of one social situation to the next.

Comparisons Between the Models

As acknowledged by both teams of researchers, there is substantial common ground between the two models, with more points of agreement than difference. Both models highlight the excessive application of attentional resources to identifying threat cues, maladaptive avoidance behaviors, and the dysfunctional cognitions held by socially anxious individuals. These dysfunctional cognitions include distorted mental representations of the self as seen by others, unrealistic standards of performance, and unrealistically negative expectations of the consequences of a discrepancy between the two. According to both models, a lack of social skills is not a fundamental or universal difficulty among individuals with SAD. Rather, they suggest that social skills may be intact in socially anxious people, but anxiety, negative cognitions, or avoidance/safety behaviors may impede social interaction and give the appearance of social skill deficits (see **Chapter 17** in this volume for further discussion of social skills deficits in SAD).

A primary but subtle difference distinguishes the two models, and it concerns the nature of attentional focus that occurs among individuals with SAD. Clark and Wells (1995) assert that the core attentional bias in SAD is the person's shift to monitoring

internal cues, which prevents the person from attending to the actual reactions from others:

> Instead of observing other people more closely in order to gain clues about what they think about him or her, the social phobic appears to turn attention inwards, notice how he or she feels, and then automatically assume that this information is relevant to others' evaluation. (p. 71)

In contrast, Rapee and Heimberg (1997) emphasize that, although there is an increase in self-focused attention with increased anxiety, attention is directed externally in search of threat cues:

> [S]ocial threat takes the form of potential negative evaluation from others. Thus, individuals with social phobia will scan the environment for any signs of impending negative evaluation, will detect such signs rapidly, and will have difficulty disengaging attention from them. (p. 746)

Clark (2001) asserts that processing of external social cues *does* occur—and is negatively biased—but that this processing is reduced due to the direction of the person's attention toward internal cues. In contrast, Rapee and Heimberg describe a more interactive relationship between self-monitoring of internal cues and monitoring of the environment for external threat (Schultz & Heimberg, 2008)—persons with SAD essentially vacillate between searching for threat in the external environment and "looking" internally to evaluate the resources that they can marshal to defend against the threat.

In addition, the two models differ in the degree to which safety behaviors are featured as a core dysfunction in SAD. Rapee and Heimberg (1997) recognize in their model that socially anxious individuals are likely to engage in subtle avoidance behaviors aimed at reducing negative outcomes (e.g., joining a group conversation but remaining at the periphery), otherwise known as safety behaviors. Safety behaviors are not described as necessarily more problematic than overt avoidance in the Rapee–Heimberg model. In contrast, safety behaviors are seen as a core problem in the Clark–Wells model and are featured prominently in the illustrated diagram of the model (as revised by Clark, 2001).

Unsurprisingly, these differences are evident in the treatments associated with each theoretical model. In the treatment based on the Clark–Wells model, a central strategy is to help clients identify their safety behaviors and to compare their experiences using them and dropping them (Clark, 2001; Clark & Wells, 1995). In the second phase of treatment, clients are encouraged to shift to an external focus of attention while also dropping safety behaviors. As with other cognitive-behavioral treatments, behavioral exposures are coupled with cognitive restructuring to challenge distorted thinking and predictions of negative outcomes.

The basic cognitive-behavioral tenets of the treatment associated with the Rapee–Heimberg model are similar (Hope, Heimberg, & Turk, 2010). However, treatment is aimed at training socially anxious individuals to direct their attention away from the mental representation of the self and from indicators of evaluation in the

environment. Instead, clients are taught to attend to the task at hand and to indicators of non-negative reactions from the audience. In treatment, clients are also taught to avoid avoidance (both overt and subtle) so that they gather evidence contrary to their negative, automatic thoughts. Coupled with challenges to thinking errors, mental representations of the self as seen by the audience become more realistic, thereby reducing anxiety.

More Recent Cognitive-Behavioral Models of SAD

Hofmann (2007): Cognitive Factors that Maintain SAD

Hofmann (2007) argues that most cognitive-behavioral approaches to SAD draw too heavily from general cognitive-behavioral models and are not disorder-specific; he proposes what he describes as a "comprehensive maintenance model of SAD" (p. 196). He describes his model as similar to that of Clark and Wells (1995), but with some distinctions. Given the overlap of this model with the models previously discussed, we focus on points of departure.

The cycle of social anxiety, according to Hofmann (2007), begins with the person's perception that social standards of performance are high and that he or she is unable to meet them. Consequently, socially anxious individuals are motivated to keep performance expectations low, and one strategy may be to purposefully fail so that others' expectations of them do not increase (a phenomenon described above as FPE). This perception of one's inability to meet expectations exists in tandem with a deficiency in setting, defining, and achieving social goals. This component of Hofmann's model draws in part from the work of Leary and colleagues (Leary & Kowalski, 1995; also discussed below), who posits that the goal for most socially anxious individuals is to make a particular impression. However, according to Hofmann's model, socially anxious individuals have difficulty clearly defining their goals beyond this overarching one. Furthermore, they have trouble planning and implementing actions that are compatible with goal attainment. The perception of high standards and doubt that they can achieve them—fueled in part by a deficiency in defining achievable goals—results in increased apprehension as they enter social situations.

As in Rapee and Heimberg (1997), attention is thought to be directed at both self-monitoring and toward detecting external threat cues when a person enters a social situation. Like Clark and Wells (1995), however, Hofmann (2007) emphasizes the heightened self-focused attention that occurs in SAD. The perception of social threat is thought to evoke a number of dysfunctional processes that increase the expectation of negative outcome. Similar to other cognitive-behavioral models, Hofmann discusses the role of negative self-perceptions and the high estimated social cost of performing poorly. Hofmann adds that individuals with SAD are likely to perceive themselves to have low control over their emotions, causing them to fear the experience of anxiety, particularly when they believe that others will witness their loss of control. Regarding social skills, Hofmann agrees with Clark and Wells (1995) and Rapee and Heimberg (1997) in hypothesizing that the majority of socially anxious individuals have intact social skills; however, they perceive themselves to have poor

social skills. The combination of negative self-perceptions, high estimated social cost, low perceived emotional control, and perceived poor social skills leads to anxiety and increased expectation of failure. This, in turn, leads to behavioral avoidance and the use the safety behaviors, which prevent individuals from correcting their maladaptive thoughts. Finally, Hofmann agrees that following a social event or interaction, PEP serves to maintain SAD.

In summary, Hofmann's (2007) model overlaps with many aspects of the Clark–Wells and Rapee–Heimberg models but includes components distinct from those models, such as emphasizing the roles of social goal delineation and perceived emotional control. Hofmann's model also explicitly highlights the role of perceived poor social skills, arguing that social skills training is less crucial for most patients than improving their perception of their social skills.

Moscovitch (2009): The Proposed Core Fear in SAD

Moscovitch (2009) proposed a new cognitive-behavioral model intended to facilitate case conceptualization and treatment of SAD. Moscovitch contended that other cognitive-behavioral models and associated exposure-based treatments failed to conceptualize patient's core fears accurately. Moscovitch argued that orienting exposure-based treatments around feared social situations does not consider the precise feared stimulus idiosyncratic to each individual.

Moscovitch reminds us that exposure treatment is based on the principle that fear develops from past learning experiences in which a stimulus becomes associated with a dangerous or horrific outcome. In SAD, Moscovitch argues that many cognitive-behavioral models rely on the premise that the feared stimulus is negative evaluation, embarrassment, or an inability to convey a particular social impression. However, Moscovitch emphasizes that negative evaluation or embarrassment is the feared *consequence*, not the feared *stimulus* for those with high social anxiety. Rather, the true feared stimuli in SAD are *"characteristics of the self that one perceives as being deficient or contrary to perceived societal expectations"* (Moscovitch, 2009, p. 125, italics in original). He argues that self-attributes *themselves*, rather than the feared social situations, are the "most direct and sensible targets for exposure" (Moscovitch, 2009, p. 130).

According to Moscovitch's model, there are four dimensions of feared, self-relevant stimuli most salient for those with SAD. These include (1) perceived flaws in social skills and behaviors ("I will have nothing to say"); (2) perceived flaws in concealing visible signs of anxiety ("my hands will shake"); (3) perceived flaws in physical appearance ("I am ugly"); and (4) characterological flaws ("I am stupid"). He recommends that clinicians identify which of these dimensions are most salient to the individual and use that knowledge to guide a functional analysis, identifying relevant feared triggers and contexts, feared consequences, and fear-related safety behaviors.

With respect to exposures, Moscovitch suggests a shift in focus from situational exposure to dimension-specific exposure. Exposures should be framed as a chance to reveal feared aspects of oneself to others. He explains that patients should be encouraged to reveal their "authentic, non-concealed selves to others in the service of testing feared social and interpersonal consequences" (Moscovitch, 2009, p. 130). Additional clinical recommendations of this model include testing patients' inflated estimated

costs of violating perceived social norms and challenging their misperceptions of how critical audience observers actually are.

At a fundamental level, this model shares with many other cognitive-behavioral models the premise that SAD is based upon distorted, negative views of the self. It also highlights basic principles present in other models, such as the roles that avoidance and safety behaviors play in maintaining social anxiety and the related importance of eliminating concealment strategies for treatment to be effective. Heimberg (2009) questions the usefulness of making a distinction between feared stimuli and feared consequences, given that they seem to be highly confounded. Heimberg also challenges the idea that existing cognitive-behavioral treatments for SAD are not sufficiently tailored to individual clients.

Subtle differences exist, however. In comparison to Clark and Wells' (1995) model, which emphasizes that the socially anxious individual fears that he or she will *behave* in a socially inept fashion, Moscovitch's model emphasizes much broader feared self-dimensions (including those that encompass major characterological traits). Furthermore, whereas Clark and Wells (1995) assert that negative self-schema are activated only at certain times, negative schemata in Moscovitch's model are thought to be more stable. Finally, Moscovitch asserts that the internal focus on symptoms of anxiety proposed in Clark and Wells' model is important only to a subset of individuals with SAD.

Stopa (2009): The Importance of the Self in Understanding SAD

Like Moscovitch (2009) and Stopa (2009) asserts that understanding the role of the self is integral to understanding SAD. However, she disagrees with Moscovitch's idea that a perception of the self can be a feared stimulus and conceptualizes the self quite differently, drawing on social psychological theories about *multiple self-representations* (e.g., Conway & Pleydell-Pearce, 2000; Markus & Nurius, 1986). Although people have access to multiple self-representations, only a subset is retrieved at a given time. For individuals with SAD, self-representations that are comprised of negative and distorted images are cued for retrieval during social situations or in thinking about them. Thus, according to this conceptualization of SAD, therapy should aim to change the ease with which more positive self-representations are retrieved. Stopa also discusses how the discrepancy between a socially anxious individual's conceptualization of the ideal self versus his or her perception of the actual self may be a significant source of anxiety.

Stopa (2009) points out that all current cognitive-behavioral models (and not just Moscovitch's) emphasize the importance of the self, though the models vary in how central the self is compared to other processes. Clark and Wells (1995), Rapee and Heimberg (1997), and Hofmann (2007) all discuss the importance of some form of mental representation of the self as a factor that maintains SAD. She argues, however, that current cognitive-behavioral models do not capture the full complexity of the self. Stopa (2009) states that three broad categories can be used to think about the self: content, process, and structure. Content of the self refers to information about the self and the way in which that information is represented (e.g., verbal statements or imagery). When cognitive-behavioral models refer to mental representations of the self, they are referring to content. Process refers to how attention is allocated to

self-relevant information and also includes the strategies used to monitor and evaluate the self. Thus, the attentional biases highlighted in cognitive-behavioral models are a reference to self-related processes. Stopa (2009) argues that the third category, self-structure, is overlooked in current models. Self-structure refers to the way self-knowledge is organized, which impacts the ease with which different aspects of self-knowledge are accessed. Stopa argues that understanding self-structure may lead us to better understand how cognitive-behavioral therapy works to reduce social anxiety. She cites Brewin's (2006) retrieval competition hypothesis and Brewin's argument that "cognitive therapy does not change the contents of self-knowledge; instead, it helps create preferential access to more positive and functional knowledge about the self by inhibiting access to negative information" (Stopa, 2009, p. 49).

Other Models of SAD

Self-Presentation Model of SAD

Developed to explain normal experiences of discomfort in social situations, the original self-presentation model proposed that people experience social anxiety when they are motivated to make a particular impression on others yet doubt their ability to do so (Leary & Kowalski, 1995; Schlenker & Leary, 1982). Such scenarios may be common to healthy and impaired individuals alike; however, those who suffer with SAD experience these problems regularly and with greater intensity (Leary & Kowalski, 1995). They also experience more distress and interference because of it. A variety of temperamental, learning, and other factors can increase the risk of developing chronic and interfering social anxiety.

A refinement and extension of the model (Leary, 2001, 2010) specifies that self-presentational concerns are most likely to cause anxiety when people are concerned that important, close, or valued others will *devalue* their relationships with them. This extension highlights *sociometer theory*, the notion that people monitor their social environment on an ongoing and automatic basis for potential threats to their value to important others. Social anxiety serves as an early warning system to alert people when their relationships are in danger and to motivate them to take the necessary reparative action. This process is intrinsically linked to the original self-presentational theory, as the impression one makes on others directly contributes to the others' valuation of the relationship. Leary further specifies that people are generally concerned about making a good impression (or avoiding being devalued) based on their performance in four domains, including competence, physical attractiveness, conforming to group norms and ethics, and being socially skilled or desirable (Leary, 2001, 2010).

Key to the self-presentation model (and incorporated in the models presented by Hofmann, 2007; Moscovitch, 2009; and the Heimberg et al., 2010 update of the Rapee–Heimberg model) is the assertion that socially anxious individuals do not fear negative evaluation *per se*. The self-presentation model emphasizes that people experience social anxiety when they risk not making a particular impression as determined by specific social goals. Although, in most cases, people wish to make positive impressions on others (and fear negative evaluation if they do not), this is not always the case. Typically, someone may wish to make a particular kind of positive impression to

garner someone's affection, to win an election, or achieve some other goal. However, people may also wish to make negative impressions to inspire fear, leverage power, or escape an unwanted responsibility. Social anxiety may be experienced if one senses that he or she is making a positive impression, if that is not the particular type of impression desired (Leary & Kowalski, 1995; Schlenker & Leary, 1982).

By focusing on the strategic dilemma faced by individuals with SAD, the self-presentation model can provide a clear rationale for the cognitive, interpersonal, and behavioral diversity that characterizes socially anxious persons. This is because people may arrive at a discrepancy between their desired impression and the one that they feel they can make via several possible routes. Some people may have overly perfectionist standards or an excessively high need to please others. These individuals may hold reasonably positive impressions of themselves, but, because they sense that perfection is required for acceptance, they may experience chronic social anxiety nonetheless. Others may have poor social skills and have learned from experience that they have difficulty making the impression on others that they wish. Still others may have poor self-esteem and view themselves as deficient. Although these different groups of people may all experience social anxiety, they differ with respect to the etiology of their disorder, as well as the core cognitive and behavioral aspects of the disorder.

The self-presentation model of SAD shares some commonalities with cognitive-behavioral models discussed in this chapter, with some important distinctions. Like many other models, it emphasizes that fear is elicited when one risks scrutiny by others. Further, like evolutionary models discussed below and elsewhere in this volume (Gilbert, 2001; also see **Chapter 2** of this volume), it asserts that being somewhat sensitive to the impression one is making is adaptive, as humans depend on stable social relationships for their well-being and survival. Similarly, experiencing negative and motivating emotions when failing to make a good impression is viewed as adaptive. Leary (2010, p. 478) states that, "People who are never socially anxious do not work to regulate others' perceptions and evaluations of them and, as a result, tend to behave in ways that offend and alienate others." Hence, the experience of social anxiety is placed within a broader context of our species' need to belong, something that is intrinsically adaptive and necessary (Baumeister & Leary, 1995).

One of the most prominent differences between this and other models is that it does not posit that those with SAD are necessarily characterized by negative self-schemata or a fear of personal deficiencies; rather, social anxiety is a strategic problem that can result from a variety of beliefs and perceptions related to perfectionism, dependency, or negative self-image. It emphasizes that socially anxious individuals may be most concerned with failing to live up to the expectations of others, rather than believing that they have failed to live up to their own. As such, the model suggests that one affected person may benefit from therapy that targets a reduction of unrealistic standards, whereas another may benefit from therapy that addresses core beliefs about the self as inept or defective.

Reinforcement Sensitivity Theory

Kimbrel's (2008; Kimbrel, Nelson-Gray, & Mitchell, 2012) model of SAD is an extension of the revised Reinforcement Sensitivity Theory (RST; Gray & McNaughton, 2000), a biologically based theory of personality and psychopathology. Kimbrel

integrates a wide range of personality, biological, environmental, and cognitive factors into a unified model of SAD and links the functioning of three brain subsystems to the cognitive and behavioral processes addressed in other models of SAD.

RST explains behavioral tendencies as stemming from three major brain subsystems—the behavioral inhibition system (BIS), behavioral approach system (BAS), and fight-flight-freeze system (FFFS). Whereas the BAS underlies reward-seeking behavior and impulsivity, the FFFS motivates avoidance and escape behavior in response to dangerous or frightening stimuli. Finally, the BIS serves as the defensive approach subsystem, whose primary objective is to resolve conflicts involving approach–avoidance goals. This is accomplished by inhibiting behavior and increasing arousal and attention toward threatening cues. Not surprisingly, the BIS is heavily involved in the emotions of anxiety and the personality trait of neuroticism, both of which are associated with SAD (Gray & McNaughton, 2000).

According to this model, SAD is promoted (in both a distal and proximal sense) by high levels of FFFS and BIS sensitivity and, in some cases, lower levels of BAS sensitivity. The feared stimulus in social anxiety—*social engagement*—may be both rewarding and dangerous. Hence, both reward-seeking (BAS) and danger-avoiding (FFFS) behavioral systems come into play, along with the BIS, whose mission is to reconcile the two (Corr, 2002).

The distal component of the model explains that an individual's genetic inheritance may promote high BIS and FFFS sensitivity, which manifest in the early temperamental trait known as *behavioral inhibition* (BI). Infants with high BI experience increased arousal and anxiety in response to novel social situations and are at higher risk of developing SAD than their low-BI counterparts (Kagan, Reznick, & Snidman, 1987; Kagan, Snidman, Kahn, & Towsley, 2007; see **Chapter 7** of this volume). Subsequently, *sensitizing social experiences* (e.g., being teased by peers during childhood) may increase FFFS sensitivity by strengthening synaptic connections in the amygdala (Rosen & Schulkin, 1998), increasing the risk of developing SAD. In addition, generally stressful life experiences, such as being abused or separated from one's parents in childhood, may damage the hippocampus and other neural substrates of the BIS, leading to exaggerated stress and fear responses (and also increasing risk of developing SAD).

Other distal factors may be protective rather than harmful. Gaining exposure to habituating social experiences—such as attending frequent play dates in childhood—may result in decreased sensitivity of the BIS and FFFS, reducing the risk of developing SAD. Further, having high BAS sensitivity—typically associated with high levels of extraversion—is another protective factor incorporated into this model. The model stresses that common starting points can lead to multiple outcomes (i.e., not all infants with BI later go on to develop SAD) and there are multiple pathways to developing SAD (i.e., even those who do not manifest BI in infancy may go on to develop SAD if exposed to other risk factors).

The model's proximal components highlight the role of cognitive and behavioral factors. When someone with high BIS and FFFS sensitivity encounters actual or potential social situations, negative beliefs and expectancies about social situations emerge, along with increased inhibition and arousal. Further, attentional biases for threatening cues and memory biases for threat-relevant information are activated. The result is increased perception of threat, which fosters social anxiety and avoidance, and

sometimes poor performance. In turn, avoidance and feedback about one's negative social performance reinforce high levels of BIS and FFFS sensitivity. The individual now risks developing functional impairment and life interference associated with a clinical diagnosis of SAD.

Because the cognitive and behavioral components of this model overlap significantly with the cognitive-behavioral models discussed earlier (Clark & Wells, 1995; Hofmann, 2007; Moscovitch, 2009; Rapee & Heimberg, 1997), one may conclude that RST is simply an extension of cognitive-behavioral models. That is, the FFFS/BIS/BAS components may be seen as modular extensions that merely increase the primacy of cognitions and behaviors, which more centrally drive social anxiety. A key distinction, however, is that RST proposes that the information-processing biases seen in individuals with SAD are the "direct result of the hypersensitivity in the BIS and FFFS" (Kimbrel, 2008, p. 605). This distinction has important implications for intervention.

Cognitive-behavioral models emphasize that negative core beliefs about the self drive social anxiety, and as such, it is helpful to target such beliefs directly in therapy, via cognitive restructuring. In contrast, the RST model suggests that exposure therapy can lead directly to habituation of the BIS and FFFS, which will lead to a reduction of cognitive biases without the need for direct cognitive intervention. This premise is supported by some research showing that patients treated with exposure therapy alone experience reduced cognitive biases similar to those produced by cognitive restructuring interventions (e.g., Mattick, Peters, & Clarke, 1989). Moreover, medications that are able to reduce excitability in the BIS and FFFS circuits—such as selective serotonin reuptake inhibitors and benzodiazepines—have been shown to reduce cognitive biases (Harmer, Shelley, Cowen, & Goodwin, 2004; Otto & Safren, 2001).

Interpersonal Model of SAD

The interpersonal model of SAD emphasizes that social anxiety is, at its very core, an interpersonal disorder (Alden & Taylor, 2010; see **Chapter 8** of this volume). It posits that early social experiences, together with innate biological factors, foster certain beliefs about the self and others—termed *relational schema*—that increase negative expectations when interacting with others. In turn, these negative expectations lead to self-protective behaviors that typically involve some sort of avoidance or concealment of anxiety symptoms. This unwelcoming strategy disrupts healthy social exchanges and the formation of close relationships (Alden & Bieling, 1998; Plasencia, Alden, & Taylor, 2011). Ultimately, negative expectations about socializing are only strengthened when the individual fails to connect with others and to establish meaningful relationships. The social isolation and impairment in close relationships characteristic of those with SAD is understood to be one of the most painful and significant consequences of the disorder and one that only serves to perpetuate anxiety even further (Alden & Taylor, 2010).

Because the interpersonal model of SAD is discussed extensively in **Chapter 8**, we do not discuss it here in detail. However, we note that it is quite compatible with other cognitive-behavioral models of SAD, although it differs in emphasis. For example, whereas all models discussed in the present chapter highlight the roles that

negative beliefs about the self (e.g., "I am incompetent") and others (e.g., "they are excessively critical") play in the etiology and maintenance of SAD, other cognitive-behavioral models emphasize the intrapsychic nature of these "core beliefs," whereas interpersonal models highlight their relational nature. There is also a distinction in terms of emphasis. For the other cognitive-behavioral models, cognitive biases and negative core beliefs are a central driver of the maintaining processes of social anxiety, whereas for interpersonal models, interpersonal functioning fulfills this role.

The way that safety behaviors are incorporated into each model illustrates this distinction. Both interpersonal and other cognitive-behavioral models emphasize that individuals with SAD engage in strategies, such as avoiding eye contact or sharing little about the self, designed to avoid scrutiny and minimize evaluation by others. Moreover, both suggest that such behaviors serve only to exacerbate disordered processes in the long term. However, these models propose a different explanation as to why this is the case. Most cognitive-behavioral models posit that safety behaviors, like other forms of avoidance, prevent individuals from habituating to anxiety and disconfirming their beliefs about the risk of socializing more freely (Clark & Wells, 1995; Rapee & Heimberg, 1997; Wells et al., 1995). The interpersonal model, in contrast, focuses on how such safety behaviors disrupt the formation of relationships and the ability to connect meaningfully with others (Alden, 2001; Alden & Bieling, 1998).

Evolutionary/Psychobiological Models

Evolutionary or psychobiological models of social anxiety (e.g., Gilbert, 2001; see **Chapter 2** of this volume) start with the argument that humans are a social species who have needed to manage close affiliations within a group over time to survive, thrive, and reproduce. Group dynamics for social primates have often been characterized by some degree of hierarchy and competition, although cooperative behavior exists as well. Social anxiety is seen as an adaptive behavioral strategy when the individual sees a social situation as competitive and doubts his or her ability to compete successfully for a high-ranking position (Gilbert, 2001). If one cannot compete for a top position in the hierarchy, the next best thing is to avoid rejection or harm and to maintain one's current position. Hence, when an individual is threatened with the risk of losing social status, a host of submissive cognitions and behaviors are engaged to appease higher-ranked individuals. By keeping a low and non-aggressive profile, the individual may be able to maintain important connections without suffering loss of status.

The model posits that those with chronic and interfering social anxiety are particularly likely to see social situations as competitive and to hold negative beliefs about the self, such that they doubt their own ability to compete effectively for a high-ranking position. As a result, these individuals are most likely to adopt a submissive posture when socializing with others. Indeed, social anxiety is associated with lower perceived social self-ranking and submissive behaviors such as avoiding direct eye contact, being overly apologetic, and making few direct statements (Antony, Rowa, Liss, Swallow, & Swinson, 2005; Leary, 1983; Weeks, Rodebaugh, Heimberg, Norton, & Jakatdar, 2009). Further, social threats lead to increased submissive behavior among those with high social anxiety but not others (Weeks, Heimberg, & Heuer, 2011).

Many of the thoughts and behaviors described in evolutionary models of SAD parallel those described in cognitive-behavioral models. For example, both kinds of models discuss the presence of cognitions that characterize the self as unable, inept, and ineffectual. Further, the submissive behaviors described in psychobiological models overlap with the avoidance and safety behaviors discussed in cognitive-behavioral and interpersonal models alike. A key distinction, however, is that these models differ in the function these behaviors are proposed to serve. Whereas behaviors such as making poor eye contact and saying very little are thought to represent social skills deficits in some models and serve the function of anxiety reduction in others, the same behavior is seen as strategic within evolutionary models. That is, it serves the specific purpose of placating higher-ups and deescalating competition. Overall, for psychobiological models, there is a primacy placed on concepts related to social comparison and hierarchy, and further, on understanding the potentially adaptive functions of these inherited behavioral systems. In contrast, other models emphasize that underlying beliefs, automatic thoughts, and ongoing behaviors drive the cycle.

Looking Across the Models

Etiology and Developmental Perspectives

One of the main goals driving psychopathology research, and the development of theoretical models of different disorders, is to increase our understanding of human behavior, and another is to inform treatment. Etiological explanations for the development of psychopathology address both goals and have the potential to inform preventive interventions to curb the development of a disorder in high-risk individuals. Furthermore, etiological considerations could lead to differentiating subgroups among those with the same disorder (Leary, 2001). For instance, a socially anxious individual whose onset of SAD was preceded by adolescent bullying experiences may look different from someone who exhibited severe social anxiety as a very young child. However, not all theoretical models of SAD emphasize etiological explanations to the same degree.

The primary etiological question for cognitive behavioral theorists is *why* and *how* cognitive biases develop. Relative to Clark and Wells (1995) and Rapee and Heimberg (1997) paid more attention to etiological considerations when describing their model. Rapee and Heimberg (1997) speculated that SAD develops through an interaction of genetic predisposition and childhood environmental factors (e.g., parenting practices and modeling of behavior). For instance, a person who goes on to develop SAD may be more genetically predisposed toward an inhibited approach to the world (i.e., biased toward threat cues). In addition, the person could receive messages from caregivers that other people's opinions matter greatly, leading to the development of beliefs that negative evaluation is highly probable and undesirable. Recent attention is also being given to the role of emotion dysregulation in SAD (e.g., Heimberg et al., 2010); it is likely that future etiological explanations from a cognitive-behavioral framework will address how emotion dysregulatory patterns develop.

Among the models reviewed, the model based on RST (Kimbrel, 2008) emphasizes etiological factors most heavily. A strength of this model is that it provides clear hypotheses regarding the interaction between genetic and environmental factors that lead to the development of SAD in some individuals. Another strength is that it offers suggestions about ways to intervene with at-risk individuals before they develop SAD.

Closely related to the question of etiology is whether theoretical models of SAD developed on the basis of research with adults apply to children and adolescents, a question that has received little examination. Hodson, McManus, Clark, and Doll (2008) examined whether the Clark–Wells model could be applied to young people. They measured levels of social anxiety, social safety behaviors, social cognitions, post-event processing, self-focused attention, and depression in 171 students between the ages of 11 and 14 years. Individuals were categorized into high, middle, or low social anxiety groups, based on their scores on the social anxiety measure. The high social anxiety group scored significantly higher on the five variables associated with the Clark–Wells model, compared with the middle and low social anxiety groups. Although the five variables predicted both depression and social anxiety, they were more strongly predictive of social anxiety. Results support the applicability of the model to young people with SAD. Further research is needed to examine how this model and others map onto childhood SAD and whether SAD treatments developed for adults can be translated for use among children and adolescents. Moreover, integrating the adult and child research literatures will help everyone better understand SAD. For more on social anxiety in children and adolescents, see **Chapter 9** of this volume.

Discrepancy as the Key

Across most models, a core feature proposed to drive SAD is a discrepancy between a person's perception of the demands of the social world and his or her perceived ability to meet those demands. In Leary's (2001, 2010) self-presentational model and the cognitive-behavioral models of Clark and Wells (1995) and Hofmann (2007), the discrepancy is between the individual's desire to live up to other people's standards and the perception that he or she is unable to do so. Stated slightly differently, Rapee and Heimberg (1997) posit that the discrepancy is between perceptions of others' expectations and the person's mental representation of the self as seen by the audience. Moscovitch (2009) similarly sees the core difficulty in SAD as the fear that the self is deficient in meeting societal expectations. Lastly, evolutionary models (Gilbert, 2001) describe a tendency to see social situations as competitive and a related discrepancy between beliefs about what is necessary to compete for high social status and one's perceived ability to do so.

Social Anxiety: An Adaptive and Normative Process?

Underlying any theoretical approach to SAD is an assumption about whether social anxiety is a normative process. A closely related question is whether we conceptualize social anxiety as existing on a continuum. The theories we have reviewed vary in the extent to which they address this question. Rapee and Heimberg (1997) addressed

it directly in their discussion of the distinction between shyness, SAD, and avoidant personality disorder. They take the position that the three labels represent differing degrees of evaluative concern, with shyness representing the low to middle range of the continuum, SAD the middle to upper range, and avoidant personality disorder the upper to extreme end. Thus, social anxiety is viewed as something that mostly everyone experiences, though to differing degrees. From a treatment standpoint, presenting social anxiety as normative helps patients understand that eliminating anxiety (a commonly expressed goal among patients) is not feasible. Talking about social anxiety in this way can also reduce self-stigma about the diagnosis and perhaps foster acceptance of this perceived "flaw." Another implication is that our existing models and treatments can be applied to those diagnosed with avoidant personality disorder.

Perhaps a more provocative argument than the one discussed above is the assertion that social anxiety is, at the low end of the continuum, an *adaptive* process, as suggested by the evolutionary model (Gilbert, 2001). As mentioned earlier, the self-presentation model (Leary, 2001, 2010) also asserts that concern about others' evaluation of us aids in the development and maintenance of stable relationships. The importance of considering social anxiety as adaptive is that it pushes us to consider the function of social anxiety. With SAD patients, we can then help them delineate when social anxiety is adaptive and when it is maladaptive in their lives.

SAD: An Intrapersonal and Interpersonal Disorder

Cognitive-behavioral models of SAD are largely intrapersonal ones. However, many individuals with SAD experience interpersonal dysfunction. They have fewer friends and dating partners (Rodebaugh, 2009; Wenzel, 2002; Whisman, Sheldon, & Goering, 2000) and are less likely to get married than individuals with other anxiety disorders (Schneier, Johnson, Hornig, Liebowitz, & Weissman, 1992). They are also more likely to report perceived friendship impairment (Rodebaugh, 2009) and reduced quality of their romantic relationships (Sparrevohn & Rapee, 2009). Interpersonal impairment is not surprising, given that the disorder is defined by how a person perceives and acts in response to other people; the avoidance of social situations would make it more difficult to establish and maintain relationships. Of the models reviewed in this chapter, the self-presentation model (Leary, 2001, 2010) and interpersonal model (Alden & Taylor, 2010) are the ones that examine the disorder from the lens of how socially anxious individuals relate to others. All SAD models describe dysfunctional processes that can occur when socially anxious people interact with those they are closer to as well as with complete strangers. However, the self-presentation and interpersonal models of SAD have a greater focus on relationships with important others and on impairment in the development of deeper, meaningful connections with others. A strength of both models is that they better integrate our understanding of dysfunctional intrapersonal and interpersonal processes in SAD. For example, although the authors of these models do not use this particular language, their models provide a framework for discussing cognitive biases and distortions about relationships.

Summary and Future Directions

The review of the current models of SAD reveals that our thinking and understanding of the disorder has come a long way since it was formally recognized in 1980. However, further refinement of our theories is, of course, always warranted so that our models can remain fruitful for research. We have discussed several theoretical models and examined the similarities and differences among them. The next step would be to continue to evaluate the empirical support for these models, as well as test aspects of these models that differ from each other.

Note

1. Clark and Wells (1995) refer to their model as "cognitive," whereas we refer to all models described in this chapter as "cognitive behavioral." This may seem like a bit of semantics, but we think it is important to recognize explicitly that both cognitive and behavioral processes play a major role in all of these conceptual models.

References

Alden, L. E. (2001). Interpersonal perspectives on social phobia. In R. Crozier & L. E. Alden (Eds.), *The international handbook of social anxiety: Concepts, research and intervention relating to the self and shyness* (pp. 381–404). Chichester, UK: John Wiley & Sons, Ltd.

Alden, L. E., & Bieling, P. (1998). Interpersonal consequences of the pursuit of safety. *Behaviour Research and Therapy, 36*, 53–64. doi:10.1016/S0005-7967(97)00072-7

Alden, L. E., & Taylor, C. T. (2010). Interpersonal processes in social anxiety disorder. In J. G. Beck (Ed.), *Interpersonal processes in the anxiety disorders* (pp. 125–152). Washington, DC: American Psychological Association.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

Antony, M. M., Rowa, K., Liss, A., Swallow, S. R., & Swinson, R. R. (2005). Social comparison processes in social phobia. *Behavior Therapy, 36*, 65–75. doi:10.1016/S0005-7894(05)80055-3

Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychological Bulletin, 117*, 497–529. doi:10.1037/0033-2909.117.3.497

Brewin, C. R. (2006). Understanding cognitive behaviour therapy: A retrieval competition account. *Behaviour Research and Therapy, 44*, 765–784. doi:10.1016/j.brat.2006.02.005

Clark, D. M. (2001). A cognitive perspective on social phobia. In W. R. Crozier & L. E. Alden (Eds.), *International handbook of social anxiety: Concepts, research and intervention relating to the self and shyness* (pp. 405–430). Chichester, UK: John Wiley & Sons, Ltd.

Clark, D. M., & Wells, A. (1995). A cognitive model of social phobia. In R. Heimberg, M. Liebowitz, D. A. Hope, & F. R. Schneier (Eds.), *Social phobia: Diagnosis, assessment, and treatment* (pp. 69–93). New York, NY: Guilford Press.

Conway, M. A., & Pleydell-Pearce, C. W. (2000). The construction of autobiographical memories in the self-memory system. *Psychological Review, 107*, 261–288. doi:10.1037//0033-295X.107.2.261

Corr, P. J. (2002). J.A. Gray's reinforcement sensitivity theory: Tests of the joint subsystems hypothesis of anxiety and impulsivity. *Personality and Individual Differences, 33*, 511–532. doi:10.1016/S0191-8869(01)00170-2

Gilbert, P. (2001). Evolution and social anxiety: The role of attraction, social competition, and social hierarchies. *Psychiatric Clinics of North America, 24*, 723–751. doi:10.1016/S0193-953X(05)70260-4

Gray, J. A., & McNaughton, N. (2000). *The neuropsychology of anxiety: An enquiry into the functions of the septo-hippocampal system* (2nd ed.). Oxford, UK: Oxford University Press.

Hackmann, A., Surawy, C., & Clark, D. M. (1998). Seeing yourself through others' eyes: A study of spontaneously occurring images in social phobia. *Behavioral and Cognitive Psychotherapy, 26*, 3–12. doi:10.1017/S1352465898000022

Harmer, C. J., Shelley, N. C., Cowen, P. J., & Goodwin, G. M. (2004). Increased positive versus negative affective perception and memory in healthy volunteers following selective serotonin and norepinephrine reuptake inhibition. *American Journal of Psychiatry, 161*, 1256–1263. doi:10.1176/appi.ajp.161.7.1256

Heimberg, R. G. (2009). A new model to facilitate individualized case conceptualization and treatment of social phobia: An examination and reaction to Moscovitch's model. *Cognitive and Behavioral Practice, 16*, 135–141. doi:10.1016/j.cbpra.2008.09.004

Heimberg, R. G., Brozovich, F. A., & Rapee, R. M. (2010). A cognitive behavioral model of social anxiety disorder: Update and extension. In S. G. Hofmann & P. M. DiBartolo (Eds.), *Social anxiety: Clinical, developmental, and social perspectives* (2nd ed., pp. 395–422). New York, NY: Academic Press. doi:10.1016/B978-0-12-375096-9.00015-8

Hodson, K. J., McManus, F. V., Clark, D. M., & Doll, H. (2008). Can Clark and Wells' (1995) cognitive model of social phobia be applied to young people? *Behavioural and Cognitive Psychotherapy, 36*, 449–461. doi:10.1017/S1352465808004487

Hofmann, S. G. (2007). Cognitive factors that maintain social anxiety disorder: A comprehensive model and its treatment implications. *Cognitive Behaviour Therapy, 36*, 193–209. doi:10.1080/16506070701421313

Hope, D. A., Heimberg, R. G., & Turk, C. L. (2010). *Managing social anxiety: A cognitive-behavioral therapy approach* (*Client workbook*; 2nd ed.). New York, NY: Oxford University Press.

Kagan, J., Reznick, J. S., & Snidman, N. (1987). The physiology and psychology of behavioral inhibition in children. *Child Development, 58*, 1459–1473. doi:10.2307/1130685

Kagan, J., Snidman, N., Kahn, V., & Towsley, S. (2007). The preservation of two infant temperaments into adolescence. *Monographs of the Society for Research in Child Development, 72*, 1–75, 76–91. doi:10.1111/j.1540-5834.2007.00436.x

Kimbrel, N. A. (2008). A model of the development and maintenance of generalized social phobia. *Clinical Psychology Review, 28*, 592–612. doi:10.1016/j.cpr.2007.08.003

Kimbrel, N. A., Nelson-Gray, R. O., & Mitchell, J. T. (2012). BIS, BAS, and bias: The role of personality and cognitive bias in social anxiety. *Personality and Individual Differences, 52*, 395–400. doi:10.1016/j.paid.2011.10.041

Leary, M. R. (1983). *Understanding social anxiety: Social, personality, and clinical perspectives.* Beverly Hills, CA: Sage.

Leary, M. R. (2001). Social anxiety as an early warning system: A refinement and extension of the self-presentational theory of social anxiety. In S. G. Hofman & P. M. DiBartolo (Eds.), *From social anxiety to social phobia: Multiple perspectives* (pp. 321–334). New York, NY: Allyn & Bacon.

Leary, M. R. (2010). Social anxiety as an early warning system: A refinement and extension of the self-presentation theory of social anxiety. In S. G. Hofmann & P. M. DiBartolo (Eds.),

Social anxiety: Clinical, developmental, and social perspectives (2nd ed., pp. 471–486). New York, NY: Academic Press.

Leary, M. R., & Kowalski, R. M. (1995). The self-presentation model of social phobia. In R. G. Heimberg, M. R. Liebowitz, D. A. Hope, & F. R. Schneier (Eds.), *Social phobia: Diagnosis, assessment, and treatment* (pp. 94–112). New York, NY: Guilford Press.

Marcus, H., & Nurius, P. (1986). Possible selves. *American Psychologist, 41*, 954–969.

Mattick, R. P., Peters, L., & Clarke, J. C. (1989). Exposure and cognitive restructuring for social phobia: A controlled study. *Behavior Therapy, 20*, 3–23. doi:10.1016/S0005-7894(89)80115-7

Moscovitch, D. A. (2009). What is the core fear in social phobia? A new model to facilitate individualized case conceptualization and treatment. *Cognitive and Behavioral Practice, 16*, 123–134. doi:10.1016/j.cbpra.2008.04.002

Otto, M. W., & Safren, S. A. (2001). Mechanisms of action in the treatment of social phobia. In S. G. Hofman & P. M. DiBartolo (Eds.), *From social anxiety to social phobia: Multiple perspectives* (pp. 391–409). Needham Heights, MA: Allyn & Bacon.

Plasencia, M. L., Alden, L. E., & Taylor, C. T. (2011). Differential effects of safety behaviour subtypes in social anxiety disorder. *Behaviour Research and Therapy, 49*, 665–675. doi:10.1016/j.brat.2011.07.005

Rapee, R. M., & Heimberg, R. G. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy, 35*, 741–756. doi:10.1016/S0005-7967(97)00022-3

Rodebaugh, T. L. (2009). Social phobia and perceived friendship quality. *Journal of Anxiety Disorders, 23*, 872–878. doi:10.1016/j.janxdis.2009.05.001

Rosen, J. B., & Schulkin, J. (1998). From normal fear to pathological anxiety. *Psychological Review, 105*, 325–350. doi:10.1037/0033-295X.105.2.325

Roth, D. A., & Heimberg, R. G. (2001). Cognitive-behavioral models of social anxiety disorder. *Psychiatric Clinics of North America, 24*, 753–771. doi:10.1016/S0193-953X(05)70261-6

Schlenker, B. R., & Leary, M. R. (1982). Social anxiety and self-presentation: A conceptualization model. *Psychological Bulletin, 92*, 641–669. doi:10.1037/0033-2909.92.3.641

Schneier, F. R., Johnson, J., Hornig, C. D., Liebowitz, M. R., & Weissman, M. M. (1992). Social phobia: Comorbidity and morbidity in an epidemiologic sample. *Archives of General Psychiatry, 49*, 282–288. doi:10.1001/archpsyc.1992.01820040034004

Schultz, L. T., & Heimberg, R. G. (2008). Attentional focus in social anxiety disorder: Potential for interactive processes. *Clinical Psychology Review, 28*, 1206–1221. doi:10.1016.j.cpr.2008.04.003

Sparrevohn, R. M., & Rapee, R. M. (2009). Self-disclosure, emotional expression and intimacy within romantic relationships of people with social phobia. *Behaviour Research and Therapy, 47*, 1074–1078. doi:10.1016/j.brat.2009.07.016

Stopa, L. (2009). Why is the self important in understanding and treatment social phobia? *Cognitive Behaviour Therapy, 38*, 48–54. doi:10.1080/16506070902980737

Turk, C. L., Lerner, J., Heimberg, R. G., & Rapee, R. M. (2001). An integrated cognitive-behavioral model of social anxiety. In S. G. Hofmann & P. M. DiBartolo (Eds.), *From social anxiety to social phobia: Multiple perspectives* (pp. 281–303). Needham Heights, MA: Allyn & Bacon.

Weeks, J. W., Heimberg, R. G., & Heuer, R. (2011). Exploring the role of behavioral submissiveness in social anxiety. *Journal of Social and Clinical Psychology, 30*, 217–249. doi:10.1521/jscp.2011.30.3.217

Weeks, J. W., Heimberg, R. G., Rodebaugh, T. L., & Norton, P. J. (2008). Exploring the relationship between fear of positive evaluation and social anxiety. *Journal of Anxiety Disorders, 22*, 386–400. doi:10.1016/j.janxdis.2007.04.009

Weeks, J. W., Rodebaugh, T. L., Heimberg, R. G., Norton, P. J., & Jakatdar, T. A. (2009). "To avoid evaluation, withdraw": Fears of evaluation and depressive cognitions lead to social anxiety and submissive withdrawal. *Cognitive Therapy and Research, 33*, 375–389. doi:10.1007/s10608-008-9203-0

Wells, A., Clark, D. M., Salkovskis, P., Ludgate, J., Hackmann, A., & Gelder, M. (1995). Social phobia: The role of in-situation safety behaviors in maintaining anxiety and negative beliefs. *Behavior Therapy, 26*, 153–161. doi:10.1016/S0005-7894(05)80088-7

Wenzel, A. (2002). Characteristics of close relationships in individuals with social phobia: A preliminary comparison with nonanxious individuals. In J. H. Harvey & A. Wenzel (Eds.), *A clinician's guide to maintaining and enhancing close relationships* (pp. 199–213). Mahwah, NJ: Lawrence Erlbaum Associates.

Whisman, M., Sheldon, C., & Goering, P. (2000). Psychiatric disorders and dissatisfaction with social relationships: Does type of relationship matter? *Journal of Abnormal Psychology, 109*, 803–808. doi:10.1037/0021-843X.109.4.803

2

Evolutionary Models

Practical and Conceptual Utility for the Treatment and Study of Social Anxiety Disorder

Paul Gilbert

University of Derby, UK

The evolutionary approach to social anxiety, or indeed to any mental health problem, outlines *general mechanisms and processes* that contribute to their vulnerability, onset, maintenance, and recovery; but also the *specific mechanisms and processes* for any particular disorder or problem (Baron-Cohen, 1997; Brune et al., 2012; Gilbert, 1998a). This chapter will explore these two dimensions, beginning with how evolution has created brains that are easily tipped into psychopathological (especially anxiety) states.

I should mention at the outset that although social anxiety may be classified as a set of specific disorders, including specific social phobia(s), generalized social anxiety disorder (i.e., SAD), and avoidant personality disorder, social anxiety accompanies many forms of mental health difficulties including depression, psychosis, eating disorders, and substance misuse (Schneier, Johnson, Hornig, Liebowitz, & Weissman, 1992). So, in this chapter, I address social anxiety as a broad dimension of functioning, as well as a specific disorder.

General Evolutionary Processes in Social Anxiety

It is now well recognized that humans like other animals are not born into the world as blank slates, but come with various predispositions and biases for learning. When certain threats have existed over a long period of time, and are relatively regularly encountered, then adaptation to them can arise in the form of attentional and response biases (Marks, 1987). These biases increase the frequency by which fears and phobias are acquired, partly because some "threats" are attention-grabbing, and fear acquisition and response operates on a "better safe than sorry" principle (Gilbert, 1998b; Marks, 1987; Marks & Nesse, 1997; Tobena, Marks, & Dar, 1999). So, for

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

example, fear of spiders, snakes, and signs of disease are much more prevalent than fear of cars or electricity even though the latter will kill more people in the modern age. Jung referred to this as "archetypal disposition" and Seligman as "preparedness" (Seligman, 1971).

Old and New Brain Mechanisms

In addition to built-in attention and response biases, about 2 million years ago our human ancestors began to get smart and evolved a range of cognitive abilities that enabled them to: understand the relationships between things, imagine, anticipate, plan, ruminate, and communicate complex information beyond grunts and shrieks. Not only was use of language and symbols important for communication, but they also played fundamental roles in the evolution of thinking and reasoning itself. These new competencies offered fantastic advantages in the struggle for survival, but they also have a very serious downside. For example, imagine a zebra running away from a lion and eventually the lion gets exhausted and wanders off. Fairly shortly after, the zebra will calm down and return to its group and grazing. Humans, on the other hand, could stay somewhat "traumatized" and start imagining what *could have happened if . . . they had been caught*; imagine being eaten alive; leaving the children unattended and so on. So unlike other animals, it is possible for us to live in an inner world of thoughts and fantasies (Saplosky, 2004; Wells, 2009). Importantly, these "inner simulations and creations" have powerful physiological effects that can contribute to a range of health problems (Saplosky, 2004). If we lay in bed and imagine hot sexual fantasies this reveals very quickly the power of fantasy and imagination on stimulating some very *specific cell clusters* in our brain (in this case the pituitary) and setting off a cascade of hormones and other physiological processes. The way and content of the reasoning, fantasies, imaginary scripts, and scenes we create are physiologically powerful, and orientate attention and response systems. So for any disorder, including SAD, attention to the kinds of simulations and fantasies individuals are creating and running in their minds is important, and also provides opportunities for intervention (Wells, 2009; Wild, 2009).

So, we have these general mechanisms that are rooted in biological systems. They can give rise to biases in threat processing that can feed into other general competencies for imagination, anticipation, and planning. These can help us to anticipate and deal with threats before they arise; but on the downside, they can hold us in an inner world of threat activation, where we are running *better safe than sorry* simulations in our minds (Gilbert, 1993; Gilbert & Choden, 2013).

A third factor which arises from recent evolution, and is especially important for social anxiety, is a sense of self. Chimpanzees have degrees of self-awareness and mentalizing ability that enable them to understand the impact of their social signals on others—and conceal them. Cheney, Seyfarth, and Smuts (1986) offer an interesting example:

> In a captive group of chimpanzees two adult males Nicki and Luit were engaged in a prolonged struggle for dominance. During one fight Nicki was driven into a tree. As Luit sat at the bottom of the tree, he nervously "fear grinned". He then turned away

> from Nicki, put a hand over his mouth and pressed his lips together to hide a sign of submission. Only after the third attempt when Luit succeeded in wiping the grin from his face did he once again turn to face Nicki. (p. 1364)

The capacity to monitor how one exists for others and how others will interpret one's displays offers important advantages in social interactions. However, the way in which humans imagine how they exist for others is complex, and can be negatively biased in all kinds of ways due to: projection (e.g., one's father was critical, so one always feels that older males will be critical or dismissive of the self); assuming others judge one as (harshly) you might judge yourself; assuming that others notice things that they do not (e.g., see hands trembling, or hear voice as shaky), selective attention (only noticing negative feedback and not positive feedback). All these processes can contribute to various safety behaviors including overly monitoring one's communication and displays to others (Wells, 2009).

Gilbert (1997, 1989) referred to this monitoring system as social attention–holding power (SAHP)—evolved attentional and cognitive mechanisms for monitoring and evaluating how others are judging, feeling, and thinking about oneself. When our SAHP is positively tuned, we focus on positive qualities of self and the degree to which we feel valued, appreciated, or liked by others. Individuals who are threat-focused are more attentive to the negative qualities of self, forms of self-criticism, their "less attractive characteristics," and potential unfavorable evaluation from others. These are not independent processes, and can be operating in the same context of display. For example, for a job interview we might be monitoring both positive and negative judgments of our behavior to the changing textures of the interview. Independently, and linked to earlier work on social anxiety and impression management (Schlenker & Leary, 1982), Leary (2004) derived a similar idea, though less linked to evolutionary theory, and called it a *sociometer*—a self-evaluative process mechanism that is socially focused, and which is concerned with the impressions that one is creating in the minds of others, and self-presentation.

Linked to this *monitoring of how we exist for others* is also a capacity to monitor and judge how we exist for ourselves; to have a sense of oneself as an individual, a separate being, and to be able to form judgments and have feelings about being a separate self. Leary (2004) called this the *Curse of the Self* because our ability to have an objective sense of self that we can judge, like or dislike, approve or criticize can be a source for personal pride but also shame and sense of inferiority compared with others (Gilbert, 1998c; Gilbert et al., 2007). Insults to our sense of self can stir all kind of defenses; no monkey ever got angry or depressed because another monkey pointed out that they were fat, ugly, or lazy. In addition, in objectifying and evaluating the self we can become self-critical and even self-hating (Gilbert et al., 2010). Social anxiety is significantly associated with self-criticism (Cox, Fleet, & Stein, 2004).

Social Mentalities

Social anxiety also needs to be located in basic social motivational systems and the organization and co-regulation of motivation-focused strategies. Animals need to pursue different motivations and biosocial goals (Gilbert, 1989). So, for example,

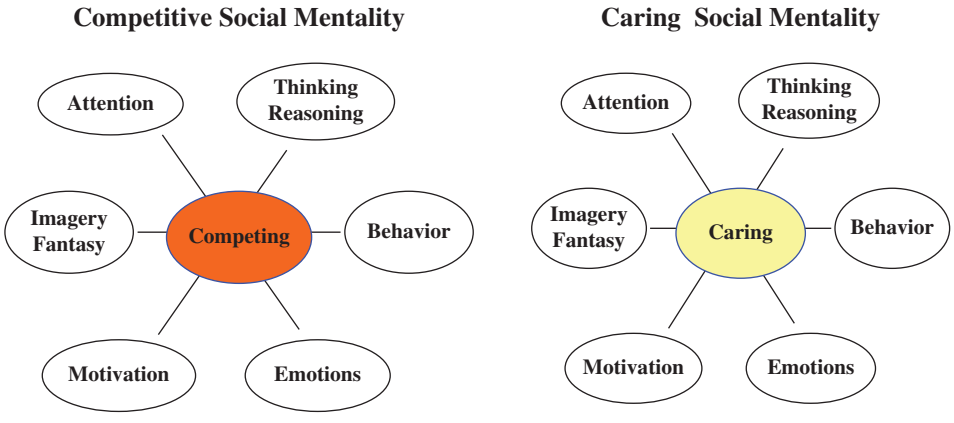

Figure 2.1 Different social mentalities. From Gilbert 2010 © Paul Gilbert.

there are different motivations to compete for resources, seek out sexual partners and opportunities, develop caring attachments to offspring, and develop alliances and friendships. Different brain systems are involved in these different pursuits (Gilbert, 1989; Panksepp, 1998). Different motives direct attention, thinking, emotion, and behavior in different ways, and this is especially important when we think about social motivation. Gilbert coined the term *social mentality* to indicate that social motives have organizing properties on a whole range of psychological mechanisms (Gilbert, 1989, 2005). So, for example, imagine person "A" in the environment. For various reasons, this individual might trigger in us: a hostile-competitive orientation to them; a desire to be friendly and cooperative with them; be sexual; be relatively indifferent; or a desire to be caring, compassionate, and helpful. The social mentality that is triggered will organize many aspects of our minds as outlined in Figure 2.1.

How basic social motives and social mentalities operate in certain conditions is key to the evolutionary approach. Hence, in this approach, social anxiety cannot be understood only in terms of single processes such as cognitive, behavioral, emotional, or motivational processes, but must instead be understood in terms of *the interactions* between these systems that guide individuals to attend, think, process, and behave in certain ways in certain contexts to perform role-focused behaviors.

A social mentality is linked to capacities for developing dynamic, reciprocal role relationships, and the ability to navigate the relationship moment by moment over time. For example, cooperative-affiliative or friendship-seeking mentalities require individuals to be: sensitive to the signals of others who could provide friendship (e.g., similar values and personality styles, nonverbal communication such as smiles, a sense of trustworthiness); able to engage those individuals in ways that stimulate them to be friendly to the self; and monitor the interactions for "friendliness or likability" over time. These capacities for dynamic, reciprocal, moment-by-moment monitoring, and adapting to interactional flow, serve to maintain and develop relationships in certain ways. In the person's absence we might be thinking about them and what we might do together when we meet again. So, a social mentality focuses on the processing of reciprocal sequences of interactions—both moment-by-moment and as interactional

simulations (plans and scenes) in one's mind. This requires individuals to coordinate attention, thinking, emotion, and behaviors in ongoing relational sequences. So, social mentalities, like motives, are not static but dynamic organizing processes in our minds.

There are of course many potential social mentalities, but at least five main classes of social behaviors can be identified that suggest particular motivations and specific competencies (Gilbert, 1989, 1992, 2005). These are (1) *Care giving*: the motives and abilities to be sensitive to the needs of others, and provide what is needed for their protection, well-being, and development. This is important in parent–child relationships but also in any caring relationship; (2) *Care seeking/receiving*: the abilities to recognize the need for, and seek out, help and support from others when needed and to be able to respond appropriately (e.g., by calming down) when it is present/provided; (3) *Cooperative*: cooperative relationships are complex because they can be based on affiliative friendships and involve sharing, mutual caring, and appreciation of each other. However, they can also be strategic whereby individuals recognize that they will achieve better outcomes if they work together and share the benefits of their efforts—even if they do not like each other. Generally, it is the sense of and processing of "we-ness" (us-focused) rather than "I-ness" (me-focused); (4) *Competitive and social ranking*: it is focused on social rank and position, which then give access to resources. So, it focuses on who is more or less "powerful or able than oneself," and how to behave with them so as to maximize chances of increasing access to/control over resources while simultaneously minimizing the social risk of trying to do so. A fifth (5) relates to sexual behavior.

In the unfolding of interactions, these motivational systems and social mentalities are undergoing a constant process of selection and blending. It is within social tasks that problems like social anxiety arise. For example, socially anxious individuals tend to be orientated by a rank-focused view of social relationships (I-ness and me-focused) where they must impress others or risk rejection; and where (submissive-like) safety behaviors and social avoidance are enacted, rather than approach and friendship-formation behaviors (Gilbert, 2001; Gilbert & Trower, 2001). One of the arguments that will be suggested is that socially anxious individuals have problems in appropriately managing cooperative and competitive social mentalities, feeling safe in certain social environments, and being able to experience and engage in affiliative rather than rank-linked (submissive) behaviors (Gilbert & Trower, 2001).

Development: There is increasing evidence that basic motivation and emotional systems are influenced by genes; with some individuals being more affiliative than others, some more fearful and dispositionally shy, others more extraverted or aggressive. However, many genetic dispositions are strongly influenced by contextual and environmental factors, such that phenotypic outcomes are not always predictable (Belsky & Pluess, 2009; Boyce & Ellis, 2005). Early emotional learning, conditioning, and emotional memory play key roles in the ease with which mentalities mature and develop. For example, in a major review of environmental factors linked to parenting style and social anxiety, Brook and Schmidt (2008) point out that "practices of control, overprotection, rejection, neglect, lack of warmth or affection, anxious parenting, insensitivity, restrictiveness, social isolation, criticism, shame tactics, behavioral rigidity and concern with the opinions of others" (p. 126) are all associated with social anxiety (but of course, they are with many other conditions, too). In addition,

they note other key early influences such as traumas and abuse, cultural factors, and variations in gender sensitivities to such influences. Again, all of these are not specific to social anxiety, but are general vulnerability factors. They are backgrounds that are shaping and entraining phenotypes into developing strategies for potentially socially hostile environments (Boyce & Ellis, 2005). Also important is the study of attachment dynamics in social anxiety. For example, disorganized attachment is linked to unresolved traumas and threats *in the parent* who are then not able to provide coherent emotional regulation parenting, or to create learning contexts where others are not experienced as safe and soothing but as potentially threatening (Liotti, 2011).

In contrast, individuals who come from primarily affiliative backgrounds are not only more socially confident but are likely to orientate themselves to others in affiliative ways (Mikulincer & Shaver, 2007). This is important because an affiliative orientation to others, in contrast to a fearful-avoidant or submissive/appeasing orientation, is crucial to a whole range of emotion regulation processes (Cozolino, 2007). It is these aspects to keep in mind when considering the socially anxious person's ability to generate, engage with, feel affiliative, and be responsive to affiliative interactions that provide soothing and safeness (Insel, 2010; Porges, 2007).

Conflicts: Many of our motivational and emotional systems can easily come into conflict. One of the sources of conflict is of course fear of the outcome of allowing one motivation to dominate. This was basically Freud's view, that the fear of recognizing certain emotions and motives rendered them unconscious. This was developed in a more cognitive-emotional approach with the work of the ego analyst Karen Horney (1945/1992)—who is well worth reading today, especially for social anxiety. More recently, however, psychologists are focused on what is called *experiential avoidance* rather than (say) unconscious repression (Hayes, Strosahl, & Wilson, 2004). In social mentality theory, experiential avoidance also suggests internal conflicts. For example, in social conflict situations, socially anxious people may be familiar with their anxiety but less familiar with, and less able to process and manage, anger and assertiveness—which are thus avoided (feared). In a recent study, Breen and Kashdan (2011) found that students with elevated social anxiety tended to suppress anger that they experienced with social rejection compared to lower socially anxious students, suggesting that anger is indeed difficult and perhaps feared in social anxiety. However, anger can be an important source for assertiveness in competitive situations, or even in bringing individuals back into a cooperative and respectful relationship with each other; so, not being able to tolerate and process anger could cause problems with confident assertiveness.

We are of course all familiar with the distinction in conflicts between what we feel we want to do and what we actually do. Sometimes of course we all behave quite submissively and appeasing (e.g., to our bosses, to avoid getting sacked) even if we do not want to at an interpersonal level (e.g., we do not like him/her). Provided we feel this is a voluntary choice for social presentation reasons, we may not be anxious in doing so. But what if it is involuntary?; if we find ourselves automatically being frightened and submissive in situations that we would rather be assertive? The ability to manage these conflicts is important. Some people not only struggle with inner conflicts (fight, flight, submit) but may even be frightened of them (Gilbert, 2000, 2012). And sometimes, conflicts can be internal with conflicting emotions,

such as having aggressive thoughts or fantasies about loved others; or having envious thoughts and fantasies in social situations. Envious and social comparative (competitive mentality–linked) feelings toward confident others have not been explored in social anxiety, but I have had a few clients acknowledge that these can arise and interfere with their self-presentation efforts (e.g., "look at her, she looks so slim and attractive and finds it so easy to be easy with people and tell jokes—why can't I be more like that?"). And sometimes the envy is anger-oriented (e.g., "she looks confident and friendly but I bet underneath she's not nice, or is a shallow person!"). Envious social comparison can mean that socially anxious people put the comparison "bar too high," and believe that they have to be like the most confident or popular (rather than comparing themselves to "average Joe" who is quite happy in social contexts).

So the key questions are: what social mentalities are triggered and prominent in the minds of people with social anxiety? How might we help them to develop and blend social mentalities that are more conducive to affiliative forms of social relating, which can downregulate threat in social situations and create feelings of safeness (Gilbert, 2009; Insel, 2010)?

Evolutionary approaches to social anxiety point toward in-built threat biases; in-built motivational biases; processing competencies such as imagination, reasoning, and mentalizing; competencies for processing how we might exist in the minds of others and a sense of an objective self; and, importantly, the ability to feel safe in affiliative contexts and operate affiliatively. In what follows, we will now explore how these are played out in specific patterns of motivational systems and social mentalities in social anxiety.

Emotions: An Evolutionary, Functional Analysis of Emotion Systems

Social anxiety is clearly linked to emotional difficulties in social domains. It is useful therefore to take an evolutionary functional analysis look at emotions, because it is the interactions with certain kinds of emotions (especially those evolved for operating in social contexts) that can give clues to some of the problems in social anxiety. Perhaps one of the best-known evolution and neuroscience functional analytic models of emotion is that of Panksepp (1998, 2010). He distinguishes various functional emotional systems as: (1) a seeking system which is basically linked to drives to go out and achieve things necessary for survival; (2) an anger/rage system that is triggered when motives and drives are blocked; (3) a fear system that is triggered when the animal is under threat of harm or loss; (4) a sexuality/lust system that is orientated to specific targets with specific behavioral outputs; (5) a care and maternal nurturance system; (6) a grief system for attachment loss, that is linked to protest–despair; and (7) a play system that is linked to joyfulness.

A different model and classification of evolved, functional emotion systems, and one which we use in compassion focused therapy (Gilbert, 2010), is based on the major review of affiliative emotion and its link to threat and drive emotions by Depue and

Morrone-Strupinsky (2005). They focus on distinguishing affiliative positive emotion from other types of positive emotion. If we link this to the recognition that we also have a core threat system (LeDoux, 1998) then we can think about the relationship between three types of emotion regulation systems—their functions and triggers. These are:

- *Threat and self-protection focused systems*: designed to attract attention to, and tune in to detect and respond to, threats. There is a menu of threat-based emotions such as anger, anxiety, and disgust; and a menu of defensive behaviors such as fight, flight, submission, freeze, etc. Defenses involving submissive behavior are only ever used in social situations. In social anxiety, threat is from social cues and monitoring one's own performance as it may appear to others (Gilbert, 2001; Schlenker & Leary, 1982). In general, threat-based defenses are also designed to be rapidly activated and to turn off positive affect and interest (e.g., upon spotting a lion or an aggressive bully in the jungle, one needs to lose interest in lunch or sunbathing and run).
- *Drive, seeking, and acquisition focused system*: designed to attract attention to advantageous resources, and experience driving pleasure in pursuing and securing them. This system is particularly important in competitive behavior. The socially anxious tend to overuse the system, feeling that they have to impress, interest or even excite others to be accepted.
- *Contentment, soothing, and affiliative focused system*: designed to enable states of quiescence and peacefulness when individuals are no longer threat-focused or seeking out and competing for resources. Over evolutionary time, this system of contentment/calming has been adapted for some of the functions of affiliative behavior (Depue & Morrone-Strupinsky, 2005). The ability *to feel* socially safe without any pressure to compete or impress seems to be problematic for socially anxious people. There is increasing evidence that this is linked to disruption in specific neurophysiological systems that promote affiliation, such as oxytocin and endorphins (Dunbar, 2010; Insel, 2010).

We can depict these three interacting systems as three circles (see Figure 2.2). Put simply, socially anxious people can get caught in the interaction between drive and threat systems, partly because they come to social relationships as a competitive endeavor as opposed to an affiliative one (Gilbert, 2001; Gilbert & Trower, 1990, 2001). We will explore the importance of stimulating affiliative emotions and affiliative social mentalities after exploring the nature of competitive threat in social anxiety.

Competition and the Dynamics of Evolutionary Change

If social anxiety is linked to the competitive mentalities, then the dynamics of human social competition are important. For the most part, evolution progresses through selection, wherein those features or mechanisms that are best adapted to environmental contexts/niches are passed to the next generation. The exact features can vary, from

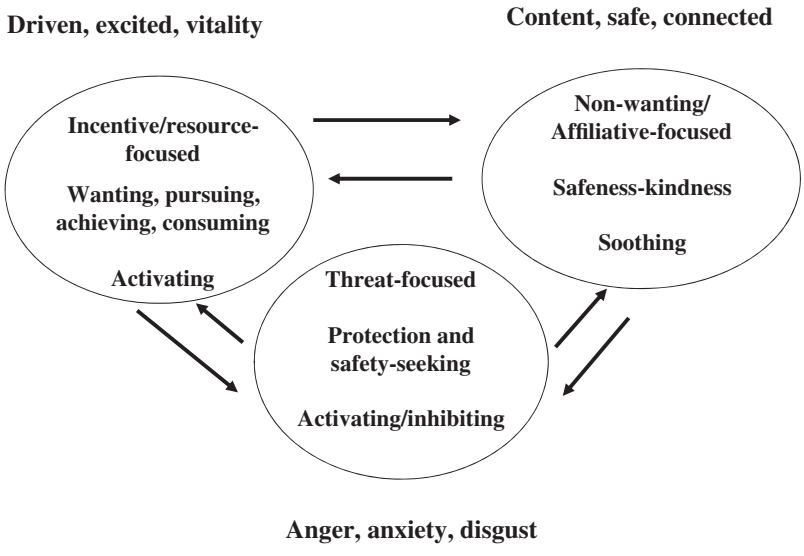

Figure 2.2 Three types of affect regulation system. Reproduced with permission from Gilbert (2009; Diagram 1, p. 24). Copyright 2009 Constable & Robinson Ltd.

physical characteristics such as the long trunk of the elephant, through to psychological characteristics such as attachment behavior and friendship formation and ability to compete with others (Dunbar & Barrett, 2008). Although evolution is believed to take place over the long term, recent evidence suggests that quite rapid evolution can also take place, even in psychological competencies (Bolhuis, Brown, Richardson, & Laland, 2011); this may have been the case in the dynamics of social competition, with humans shifting to competition for attractiveness (Barkow, 1989).

For obvious reasons, social anxiety is about the perception of social threat and, of course, conspecifics and social relationships are clearly potential sources of threat. This is because individuals compete and fight over resources, and also where some individuals are selected as sexual partners and allies while others are not. Injuries, being excluded from access to valuable resources, rejection/shunning/ignoring, and social exclusion are all major threats to social animals. So there are two major forms of social threats:

(1) Direct: whereby one or a number of individuals threaten to (or do) drive away, harm, injure or even kill another member(s).
(2) Indirect: whereby an individual is unable to elicit sufficient investment, support or interest from other individuals to develop cooperative and supportive relationships.

These two are not mutually exclusive. This raises two issues about competitive behaviors:

(1) What are the main evolved strategies used in competitive interactions?
(2) What are the main types of resources which humans compete over?

Resource Competition, Group Living, and Social Hierarchies

There are different ways in which different species have evolved to deal with the conflicts inherent in life. For the most part, *territorial* animals deal with conflicts by spacing. For example, polar bears and tigers are relatively solitary and come together with other single individuals for breeding. Once mating has taken place, individuals separate again and eventually the mother will give birth on her own, and care for the young for a short time before they grow sufficiently to also disperse. Conflicts over territories and access to sexual partners can sometimes result in quite serious injuries, but in general, inter- and intrasexual aggressions are regulated by territorial distance—staying apart.

But most mammals are actually *group living* and so require quite different ways to deal with inherent competitive conflicts and tensions—and the threat-based fight/flight system (Porges, 2007). Group living conflicts are not just over food or sexual opportunities, but actually the very advantages of living in groups themselves—which are many. They reduce the risk of any one individual being subject to predation (i.e., physical place in a group can be important); groups can act as an early warning signal (e.g., one bird takes flight, so the flock takes flight); and groups congregate in areas of resources and they provide access to breeding partners. But what is also crucial is that groups provide the context for the evolution of genuinely social behavior whereby individuals interact, get to know each other, and may come to help and support each other. That mutuality becomes a key survival resource and, of course, is therefore open to evolutionary selective pressures (Gilbert, 1989; Warneken, Chen, & Tomasello, 2006). The constant interaction with other individuals has given rise to the evolution of social competencies for understanding complex social interactions and communications, and developing complex reciprocal relating patterns. In primates, it has been group living and social interactions that have been key to the evolution of social intelligence (Dunbar, 2010) and (along with parent–child attachment mechanisms) offered the contexts for the evolution of mentalizing, empathy, and higher forms of caring (e.g., compassion; see Gilbert, 2009; Liotti & Gilbert, 2011).

Nonetheless, relationships are constantly in the process of moving between caring/cooperative and competitive dynamics (i.e., stimulating different social mentalities), and so, compared with species that live isolated lives, group living mammals have completely different ways of organizing social conflict. Social hierarchies are one solution to conflicts in close living individuals.

Competition and the Psychologies of Social Rank and Subordination

In all species, there are different forms of competition. The two most common are display competition and conflict competition. Display competition is common in intersexual competition where individuals attract each other; those who have the best displays or are regarded as the most attractive are the most successful. The most obvious example is of course bird plumage. In humans, (sexual) attractiveness arises

from signals of health, youth, confidence, and altruism. This is competition where another individual (i.e., potential sexual partner) or a larger audience are making decisions to select and engage with an individual(s) in some way. Those with certain characteristics tend to be more commonly chosen—which has sometimes been referred to as survival of the prettiest or most attractive (Etcoff, 1999).

Conflict competition has different forms, where there are winners and losers, and individuals are attempting to inhibit or disrupt the behavior of competitors. The way in which others' readiness for, and ability to engage in, conflict is detected and managed is important for all those involved in conflicts. Without individuals being able to "size each other up" and make judgments of whether conflicts are winnable, there could be constant fights which risk injury and even death to both parties. In fact, we now know that many animals are able to make judgments as to the value of the resource they are in conflict over and chances of making a successful bid for that resource.

One of the mechanisms for regulating conflict is, of course, submissive behavior. Submissive behaviors evolved as fundamental social behaviors which facilitated control over aggression and promoted social cohesion. As MacLean (1990) points out:

> . . . .Ethologists have made it popularly known . . . that a passive response (a submissive display) to an aggressive display may make it possible under most circumstances to avoid unnecessary, and sometimes mortal, conflict. Hence it could be argued that the *submissive display is the most important of all displays* because without it numerous individuals might not survive. (italics added, p. 235)

Therefore, one of the most stable social contexts in which evolution has occurred was within groups that are hierarchically organized and where displays of deference and subordinate status are the currency for regulating conflict. These hierarchies partly choreograph not only conflicts (and how individuals respond to conflicts) but also displays, general confidence, explorative behavior, and the degree of *attention* and cooperative investment members pay each other. So, for example, many primate hierarchies are also linked to alliance formation and alliance building, especially through grooming (Dunbar, 2010). Those higher up the hierarchy of attention obtain more deference and grooming than those lower in the hierarchy (Chance, 1984). Studies have also shown that status affects how humans monitor social threat, and people with lower status show more threat-focused attention with activation in the medial frontal cortex (Boksem, Kostermans, Milivojevic, & De Cremer, 2012).

It is useful to keep in mind that status and social rank, and their link to social anxiety and general social wariness/attentional orientation, are not something limited to clinical versus nonclinical populations, but rather to general principles of social behavior. There is evidence from general populations that feeling inferior to others is associated with reduced assertive behavior and increased submissive behavior. However, while linked of course, submissive and assertive behaviors are not mirror images (Gilbert & Allan, 1994).

The importance of social rank in regard to confident behavior has been recognized by most commentators throughout history, and has been the focus of anthropologists

and sociologists for some time. For example, Scott (1990) quotes from Hochschild (1983) on the advantages of status and high(er) social rank in humans:

> . . . to have high status is to have a stronger claim on rewards, including emotional rewards. It is also to have greater access to the means of enforcing claims. The deferential behaviour of servants and women – the encouraging smiles, the attentive listening, the appreciative laughter, the comments of affirmation, or concern – comes to seem normal, even built into the personality rather than inherent in the kinds of exchange that low-status people enter into. (p. 28)

And he goes on to point out that:

> More of the public life of subordinates than of the dominant is devoted to 'command' performances. The change in the posture, demeanour, and apparent activity of an office work force when the supervisor suddenly appears is an obvious case. The supervisor, though she too is constrained, can typically be more relaxed about her manner, less on guard, for it is the supervisor, after all who sets the tone of the encounter. Power means not having to act or, more accurately, the capacity to be more negligent and casual about any single performance. (p. 29)

Gilbert, Allan, Ball, and Bradshaw (1996) showed that although it is sometimes thought that confidence (and the overconfidence or *warm glow* effect) is linked to depression, in fact it is most strongly linked to judgments of status and social comparison; that is, those who rate themselves as more inferior and submissive do not show an overconfidence effect when estimating their likely results on an intelligence task.

So, social status judgments orientate attention and confidence estimates. What about the defenses? There are in fact a variety of defenses which evolved to regulate conflict, and activation of these defenses is commonly involved in a variety of mental health problems including social anxiety (Gilbert, 2000). Submissive behavior is, however, not just manifest at the point of conflict, but can also be linked to *strategies of living* whereby subordinates are: wary of dominant individuals (i.e., threat-attentive), generally more socially timid, and less exploitative. Subordinates are also different from more dominant animals on a range of physiological and neurophysiological processes (Sapolsky, 2004). If social anxiety is linked to issues of low rank and fears of more powerful others, then we would anticipate that socially anxious people would see themselves as inferior, endorse submissive behavioral defenses, and perceive others as viewing them as relatively low in rank, too. In fact, there is good evidence that this is the case in both clinical and nonclinical populations (Gilbert, 2000). Moreover, objective indices echo these endorsements of submissiveness; two submissive behavioral defenses that are seen in nonhuman primates (reduced, collapsed posture [i.e., *body collapse*]; and its auditory equivalent, vocal pitch elevation) are exhibited by socially anxious human males when competing for the positive attention of a female peer (in evolutionary terms, a potential mate) (see Weeks, Heimberg, & Heuer, 2011).

Status evaluations are also associated with anger expression. Anger inhibition is related to social rank, with subordinates inhibiting anger expression to more powerful others (Fournier, Moskowitz, & Zuroff, 2002). Erwin, Heimberg, Schneier, and Liebowitz (2003) studied a group of 234 socially anxious patients, and found that

social anxiety was related to increased depression and anger, and poorer anger regulations skills compared to controls. Moreover, problem with anger was a predictor of outcome in cognitive-behavior therapy. Breen and Kashdan (2011) used an imaginary rejection task, and found that social anxiety was associated with increased avoidance of anger, as well as anger suppression. One must be cautious here, however, because anger expression could be related to the rank of the target. For example, socially anxious people may be more likely to express their anger and frustration at home, to children, or work subordinates. In a recent case, the wife of a socially anxious man told how he was often quite placid but could become verbally aggressive at home and certainly in the car with other drivers who he would shout at (presumably knowing they could not hear him). She felt behind his submissive anxious style was a lot of anger.

One area that would also benefit from this kind of evolutionary analysis is recent important work showing that anxious people show elevated startle reflexes *in safe environments,* and that startle reflex is an important predictor of anxiety disorder onset (Craske et al., 2012). This is especially interesting since a fair percentage (5 of 16) of their sample were social phobics. Not only does this work address the issue about the relationship between feeling safe versus feeling threatened, with a recognition that they may be regulated by different systems, but it raises the issue of whether low-rank individuals (or those seeing themselves in involuntary subordinate positions; e.g., see Gilbert, 1992, 1993) can afford to feel safe. Indeed, clinically we sometimes find that some patients are frightened when feeling safe. For example, "Sally" recorded memories of playing happily in the garden and feeling safe with friends when her mother (who was an alcoholic) would suddenly appear in a rage and beat her. "Feeling safe" she said "is the last thing you want to do because that's when things hit you out of the blue—you should always stay on guard." Hence, the mechanisms that "set" safeness regulation might be rank-sensitive. Keeping in mind the three emotion regulation systems mentioned above (see Figure 2.2), it is possible that the processes by which safeness systems are set/tuned—that is, toned up or toned down, sensitized or desensitized—may be different for different anxiety disorders, and that social anxiety may possibly be linked to impaired abilities to "feel safe" in social environments, and rather, quick to feel threatened in benign environments. Also, what sets the conditions for feeling safe (setting of the systems) might be quite different from what sets the conditions for the ease of triggering threat or anxiety. Once these are seen as interacting but different emotion regulation systems, new ways of thinking about the interactions and co-regulation of the systems open up.

Subordination, Self-Blame, and Self-Criticism

Social status also affects attributions for causality of social ruptures. For example, in pre-scientific cultures, individuals have attributed powerful forces to "Gods" who they must then placate and win over—be this by sacrifice (e.g., of virgins) or carrying out the (assumed) will of the Gods, or other means. However, if in the following year there appears to be no support from the God, or things actually get worse, then individuals typically blame themselves (e.g., "What have we done to upset you?") and will intensify their appeasing and submissive behaviors. When individuals are confronted by

frightening, powerful others they need to self-monitor to avoid stimulating the anger of the other. Self-blame can avoid showing anger to the more dominant. Andrews and Brewin (1990) found that women living with abusive males often blamed themselves for the violence, but changed their attributional style when they moved away (i.e., when it was safe to do so). So sometimes it may be safer to blame oneself because this will inhibit one's assertiveness or anger especially if any outward expression could be dangerous (i.e., the dominant could powerfully retaliate and escalate). So, self-monitoring and self-blaming can be defensive strategies when having to seek work with or be in proximity to powerful others (Scott, 1990).

Some years ago, Forrest and Hokanson (1975) showed that the self-criticism, self-blaming, and self-attacking responses to a social interaction confederate were typical of depressed people; and were ways that they tried to regulate arousal in themselves, and tension within the interaction. Nondepressed individuals, however, were much more assertive. The same study has not been done for social anxiety, but it is reasonable to assume that such a study would yield similar results. Evidence that self-blame goes with feeling inferior was also found by Gilbert and Miles (2000). In their study, blaming oneself for criticisms and put-downs by others was highly associated with shame and seeing the self as inferior, whereas blaming others was associated with seeing the self as (relatively) superior and low in shame. Thus, there is a strong link between self-blame, seeing the self as inferior, and submissive behavior.

Using data from a large national comorbidity study in the United States, Cox et al. (2004) found that *self-criticism* was highly elevated in social phobia, and noted that "These levels were significantly greater compared to those observed in another anxiety disorder (panic disorder), the pure speaking subtype of social phobia, and cases of major depression alone" (p. 227). Trower, Sherling, Beech, Horrop, and Gilbert (1998) asked socially anxious and nonsocially anxious students to engage in a conversation with a lecturer while being videotaped. The lecturer was part of the study and was (unbeknown to the students) instructed to break conversational rules, such as butting in and changing the subject. On viewing the videotape, socially anxious students blamed themselves for the problems in the conversation while nonsocially anxious students blamed the lecturer.

Hence, some elements of negative self-evaluation, elevated self-monitoring in relation to others, and tendencies to self-blame in conflict situations may be fuelled by nonconscious submissive strategies to cope with potentially hostile, rejecting others. So it is possible that subordinate strategies, once activated in individuals, will then organize attentional, emotional, cognitive, and self-processing systems to facilitate efficient subordinate strategies—which include anxious attention to others, a highly internal self-monitoring attentional orientation, social inhibition, and anger suppression.

Social Anxiety and Paranoia

Strictly speaking, there are many general forms of *social* anxiety, including separation and rejection anxiety. One form of social anxiety is obviously *paranoid anxiety,* where individuals become fearful of the malevolent intent of others. In fact, the most "social"

form of anxiety in animals is paranoia, insomuch as it is the potential for harm/injury from others that is the source of the anxiety (Gilbert, 2001). The relationship between dimensions of paranoid anxiety and social anxiety is very poorly researched, even though most clinicians will be aware that some socially anxious people are very suspicious and distrusting of others. Many of the elements we have discussed above for social anxiety also appear true for paranoid ideation, which is associated with feelings of inferiority, submissive behavior, and self-criticism. For example, in a mixed clinical population ($N = 71$), Gilbert, Boxall, Cheung, Tuffield, and Irons (2005) found that paranoid ideation was highly correlated with social phobia and submissive behavior. Paranoid ideation has also been associated with self-criticism (especially self-hatred) in nonclinical (Mills, Gilbert, Bellew, McEwan, & Gale, 2007) and clinical populations (Hutton, Kelly, Lowens, Taylor, & Tai, 2013). Given that there are different forms and functions of self-criticism, to date these differences have not been explored in social anxiety. It is possible, for example, that nonparanoid socially anxious people tend to be more self-critical about their abilities to perform rather than self-hating.

Using different measures of (negative) self-evaluation (i.e., internal shame; see Cook, 1996) and negative beliefs about how one was viewed by others (i.e., external shame; Goss, Gilbert, & Allan, 1994), Matos, Pinto-Gouveia, and Gilbert (2013) found that shame memories tended to be more traumatic and central for people who endorsed more paranoid ideation. Via multiple regressions, external shame (beliefs of being looked down on by others) was a better predictor of paranoid ideation than internal shame. In a way, that is not so surprising since external shame (thinking that others look down on you and see you in a poor light) is the basis of paranoia. In contrast, social anxiety was more associated with internalized negative thoughts about oneself.

It has also been suggested that paranoid anxiety is closely linked to "out group" anxiety—this is linked to a fear that others are ganging up against the self. In more severe forms of paranoia, it is always groups (e.g., the police, the mafia) rather than single individuals that people fear. Also, they have no wish to form any affiliative associations with them. In contrast, social anxiety is more in-group fears to do with one's ability to compete for positive (being liked and valued) attention that is the issue (Gilbert et al., 2005). Paranoid anxieties are not really about "competing to be seen as desirable and attractive." To date though there has been no clear research to evaluate these distinctions.

Low Rank and Positive Evaluation

So you would think then that socially anxious people would be pleased to get positive attention. The problem is that it depends on what kind of positive attention, and it could be a double-edged sword because it also puts you in the limelight, heightens self-consciousness, and increases rank-based threat. Could subordinate strategies also impact on positive evaluation and positive emotion? Depressed individuals obviously experience low positive emotion, and this is linked with feeling subordinate, defeated, and entrapped (Gilbert, Allan, Brough, Melley, & Miles, 2002; see Taylor, Gooding, Wood, & Tarrier, 2011, for a review). Gilbert (1992) suggested that socially anxious

and depressed people share many overlapping features but that depressed people experienced more defeat and a sense of entrapment.

Weeks, Rodebaugh, Heimberg, Norton, and Jakatdar (2009) proposed a cognitive model of social-anxiety-related submission based upon psycho-evolutionary accounts of social anxiety and depression. They found that fears of evaluation and depressive cognitions loaded onto a single latent higher-order submissive cognitions factor. So, symptoms associated with social anxiety and depression may both have been adaptive functions for coping with social threats in the ancestral environment, and the cognitive symptoms associated with these disorders may function together as part of a social-anxiety-related submission mechanism. What is more interesting, submissive cognitions were found to mediate the relationship between social comparison and self-reported submissive behaviors (Weeks et al., 2009).

Another parallel with depression is that recent research has suggested that socially anxious people have low levels of positive emotion, especially in social situations. In a recent major review, Kashdan, Weeks, and Savostyanova (2011) found that socially anxious people were less orientated to positive events, experienced less positive events (especially social events), and were often fearful of positive emotion in certain contexts. This work links to increasing interest in the fears of positive emotion (e.g., people worry that if they are happy and relaxed something can hit them "out of the blue") (Gilbert et al., 2011).

In a series of studies, Weeks and colleagues have been exploring the fears of positive evaluation (e.g., Weeks, Heimberg, & Rodebaugh, 2008; Weeks, Jakatdar, & Heimberg, 2010; see also **Chapter 20**). They have shown that socially anxious people can be frightened of positive evaluation by others. One reason may be because it puts people in the spotlight on the one hand and also creates an image of self that they may not be able to defend or maintain—consistent with psycho-evolutionary theory, individuals who perceive themselves as ranking socially lower than others would be motivated to avoid giving such a positive impression that they would be viewed as a threat by other members of the group (i.e., to avoid an upward shift in a social hierarchy) (see Weeks & Howell, 2012). There are many ways in which these processes can be understood.

Kashdan (2007) Kashdan and colleagues (2011) utilized an emotion regulation model to explain the difficulties in positive emotion and positive evaluation. An evolutionary model can add to this. For example, if social anxiety is mapping onto mechanisms for subordinate living, then the above findings would not be surprising. Subordinates need to be very wary of explorative and confident behavior, or of making claims on resources, in case they miss threats from more powerful others or stimulate hostile interest (i.e., envious attacks). Imagine subordinates finding food and showing pleasure—this will quickly be conveyed to others who may well come and steal it; or showing sexual interest may alert the attentions of a more dominant/powerful competitor. So, part of the "strategy for subordinate living" in competitive contexts is to live a more threat-focused and less-positive/joy-focused life. There is also evidence that subordination is associated with downgrading of dopamine$_2$ receptors—a neurotransmitter associated with positive affect and drive (Grant et al., 1998). This may account for some of the low positive affect associated with these difficulties. Keep in mind too that we are arguing that these are dimensions of rank regulation that can

operate throughout various forms of mental health difficulties and *are not* limited to social anxiety, but *are* core to social anxiety.

Social Attractiveness and the Importance of Affiliative Psychology

So far we have explored the basic mechanisms of attention, and cognitive and behavioral regulation, but there is a complicated twist to this relating to exactly *what* humans are competing for. Here, there are big differences between humans and other primates, and these differences may help us to understand social anxiety in new ways.

First, human social status and rank have many dimensions. People can rank and compare themselves on physical attractiveness, intelligence, athletic ability, confidence, creativity, and so on—dimensions that attract positive interest (Barkow, 1989; Gilbert, 1989; Gilbert, Price, & Allan, 1995). For the most part, self-esteem is linked to having skills and talents that one believes others value (Santor & Walker, 1999). Recently, Anderson, Willer, Kilduff, and Brown (2012) found that individuals can rank themselves in terms of power influence and leadership, but also in terms of respect. Importantly, those who feel they do not have much to contribute to a group may prefer middle-to-low "power/leadership" status, whereas respect is always sought. We can also make a distinction between what peoples' basic motivations for rank are. It is not the case that all individuals are seeking high rank but more often *to avoid low rank*—that is, seeking to avoid inferiority—and its consequences (e.g., rejection or marginalization). Competition and striving to avoid inferiority are not the same as general up-rank competition (Gilbert et al., 2007)—but, it is still striving, and in that sense is linked to the competitive/drive system. The idea that human competition became focused on influencing the minds of others positively in one's favor has been around for a long time, reaching back to George Herbert Mead's concepts of the *looking glass self*. It was further developed by Barkow (1989) in his discussion of *competition for prestige and reputation*. Competition, then, becomes focused on the impact you have in the minds of other people. This is why we can see social intelligence becoming hugely important as an evolved competency.

Whether we call it prestige (Barkow, 1989), respect (Anderson et al., 2012), or attractiveness/desirability (Gilbert, 1997; Gilbert et al., 1995), humans now compete to create positive emotions/images in the minds of others such that other individuals (e.g., sexual partners, potential friends, bosses) and indeed, groups too, value, engage, and choose to invest in the relationship. In fact, this is not a new idea. Schlenker and Leary (1982) developed a theory of social anxiety concerning a heightened motivation to create good impressions in the minds of others, and that anxiety was a common consequence of doubts or failures in the ability to do so. Leary (2004) went on to develop his theory of social anxiety based on impression management—although he did not address the evolutionary competitive dynamics of this.

If we are successful, there are many advantages to being liked and valued. Others are unlikely to attack you; they will support you and develop reciprocal relationships of helpfulness; you are likely to share positive affect with them rather than negative affect; and the positive feedback you get in relationships has a major impact on your

physiological regulation including cortisol, immune system function, and oxytocin. So, the socially anxious fear missing out on these advantages because they will appear unattractive; appear fearful, boring, incompetent, and not really worth engaging or investing in. So (in the competitive mentality), in trying to increase one's social position (or feel included and avoid risk of rejection), one is focused on one's individuality (or own group), social comparison, and acting to: trying to control/obtain resources for oneself; drawing attention to oneself; and trying to impress others or make a "bid" for their attention, interest, approval, and investment. However, there is also the avoidance of *losing* rank in the process due to performing poorly—making a bid or challenge that causes unfavorable attention, and then being worse off.

We should keep in mind that the social audience *is a judging audience,* and will be wanting to form relationships with others who offer some benefits for them. Thus, we are attracted to the more physically attractive, confident, and humorous (Etcoff, 1999). So, it is true that we are less attracted to and want less to associate with the slow, the fearful, and the boring. The more we judge ourselves to be like this, the more anxious we are likely to be, especially in cultures like the West that stress competitiveness over collectivism and co-operation.

Paradoxically then, social anxiety can be understood in the context of the feeling that one needs to strive and compete for social places in the minds of others and for *affiliative relationships* but feeling unable to do so; if they do attract attention to themselves, it will not be positive but rather negative, because they will perform badly. This leads to increased social wariness and submissiveness as defenses—but with the recognition that submissive defenses such as avoidance of eye gaze, inhibited speech outputs are not attractive. This may be another distinction between social anxiety and paranoid anxiety. Social anxiety is the fear of not having qualities or displays that can attract others; whereas paranoid anxiety is much less focused on positive displays and attracting, with far more interest in protecting oneself from the malevolence of others and keeping others away.

Shame, Social (Un)Attractiveness, and Social Anxiety

The fear of being the unattractive and unattracting self takes us into the domain of shame. The abilities to monitor and evaluate how we think we exist in the minds of others, and also to monitor and evaluate our own behaviors and displays, are key to the experience of shame (Gilbert, 1998c; Tracy, Robins, & Tangney, 2007). Indeed, both shame and social anxiety are self-conscious emotions, and it is surprising that these literatures have not been better linked together (Gilbert, Allan, & Pehl, 1994; Gilbert & Trower, 2001). There are many ways in which we can define shame but generally it is associated and a range of different processes:

(1) Negative self-judgments such as seeing oneself as inferior, inadequate, and/or useless. This has also being labeled as *internal or internalized shame* (Cook, 1996; Gilbert, 1998c; Gilbert et al., 2007). The shame measure of Andrews, Qian, and Valentine (2002) focuses on negative self-judgments linked to: one's behavior, one's character, and one's body. The Test of Self Conscious Affect, a well-used

American measure of shame, focuses on negative self-judgments and self-labeling in particular contexts (Tangney & Dearing, 2002). Gilbert suggested that internal shame is linked to self-criticism, and distinguished between different types and functions of self-criticism. For example, some individuals are critical because they feel they can do better, let themselves down, and try to avoid mistakes; whereas others are self-critical because they dislike or hate the self.

(2) Negative judgments by others, also referred to as *external shame* (Gilbert, 1998c), is linked to ideas that others hold negative judgments about the self and see the self as inferior or lacking in some way. As a measure, it was developed from the internal shame scale of Cook (1996) by Goss et al. (1994). These studies found that internal and external shame are very highly correlated.

(3) In an evolutionary model, shame is a marker or "alerter" of potential for social damage via social put-down or rejection (Gilbert, 1998c; Gilbert et al., 2007). The behavioral and emotional defenses to shame are linked to those of social threats, but depend on a range of factors such as previous history, social context, and social dominance. For example, when some "dominant" individuals are shamed, they become angry and aggressive, whereas others become anxious and avoidant.

(4) There is now good research showing that shame is one of the most powerful physiological stimulators of the threat system, especially the experience of others being critical (Dickerson, Gruenewald, & Kemeny, 2004).

Clearly, all of these dimensions have important and useful things to say about social anxiety too. However, more work needs to be done to see the subtle differences between social anxiety and shame-proneness (Gilbert et al., 1994). Sometimes put-downs and rank-threat do not stimulate submissive behavior, but aggression. So, social anxiety might only link to submissive defenses of the competitive mentality. Is it possible to have social anxiety without some sense of shame?

Emotion Regulation and Affiliation

One more twist! If we follow the three circle model of affect regulation as given in Figure 2.2, then we need to think about emotion systems as mutually co-regulating. Until recently, anxiety disorders were explored primarily within the context of threat-processing and threat emotions. More recently, however, it has become clear that threat emotions are regulated through positive, and especially affiliative, emotion and relationships. To understand this, it is useful to revisit the idea of there being two fundamentally different (although highly overlapping) types of positive emotion, with different neurophysiological systems, mediators, and functions (Depue & Morrone-Strupinsky, 2005).

The first is basically linked to drives, and is associated with feelings of excitement, pleasure, and anticipation of reward. If you become a multimillionaire on the Euro lottery, you are likely to get a buzz of excitement and sympathetic arousal. You will develop energized mood, racing thoughts, find it difficult to settle, and would certainly find sleeping difficult for a few days! Depue and Morrone-Strupinsky (2005)

suggested this is also the system that is involved in seeking dominance and competing for resources, including competitive display behavior. Clearly, in our relationships, we often want to be seen as exciting and fun to be with rather than as boring or flat.

However, while these positive emotions are important in the development of affiliative relationships, there is another key positive social emotion which is quite different and is associated with slowing down, contentment, and calming (Depue & Morrone-Strupinsky, 2005). Here, one is neither seeking resources nor responding to threat; one is not seeking to be threatening, exciting, or "impressive," but rather to create a sense of safeness, connectedness, peacefulness, and calmness in the relationship. In fact, one of the most important adaptations to have taken place in group living is physiological adaptations which allow animals to come together without stimulating fight and flight—that is, they can feel safe with each other (Gilbert, 1989; Insel, 2010; Porges, 2007).

The mechanisms by which social relationships have *calming* effects have been gradually revealed by neuroscience. This type of positive effect, associated with well-being, is associated with parasympathetic arousal, calm mind, and ease of sleeping. These types of positive emotion seem to be linked to the endorphins and also a hormone called oxytocin (Depue & Morrone-Strupinsky, 2005; see also **Chapter 21** in this volume for an extensive discussion). They are basically affiliative emotions that are linked to liking and helping (Insel, 2010). Oxytocin plays a crucial role in the parent–infant attachment relationships, but also in affiliative relating in general; in particular, to feeling safe, trusting others, and how we process potentially threatening social information (MacDonald & MacDonald, 2010). When individuals are stressed or distressed, their ability to turn to others for support, which calms them, is central to coping with stress (Cacioppo & Patrick, 2008). Kelly, Zuroff, Leybman, and Gilbert (2012) found that a measure of general social safeness and capacities for feeling connected to others was a better predictor of vulnerability to psychopathology than negative affect, positive affect, or needs for social support. So, it is possible that the general day-to-day tone of the affiliative (especially endorphin–oxytocin) system plays a role in the vulnerability to conditions like social anxiety.

Treatment Implications

There are many good psychological treatments now for social anxiety. While some interventions have involved exposure and skills training, there is increasing focus on the development of emotion regulation; and affect–acceptance, mindfulness, and self-compassion (Roemer & Orsillo, 2012). In the evolutionary model, however, part of the focus might be on developing the cooperative and affiliative mentalities, and helping individuals shift out of competitive mentalities (Gilbert & Trower, 2001). One way of doing this is by helping individuals to focus on developing genuine compassion, interest, and concern to be helpful and supportive of others. In fact, there is increasing evidence that focusing on, and practicing, compassion (especially compassion for others) has a range of physiological and emotional effects (e.g., see Hoffmann, Grossman, & Hinton, 2011). Compassion training has also been shown to produce important neurophysiological changes (Klimecki, Leiberg, Lamm, & Singer,

2013). This could be helpful, because most theories of social anxiety suggest that people are far more self-focused, monitoring of social comparison (i.e., judging others as superior or inferior, and judging whether the self is creating negative images in the minds of others), while at the same time believing that one has to be exciting and impress other people to avoid being ignored or shamed (Gilbert, 2001; Gilbert & Trower 1990, 2001; Schlenker & Leary, 1982).

In contrast, affiliative psychology focuses attention on the other person, taking a genuine interest in them, wanting to be helpful and supportive (rather than impress them). In fact, the kinds of social mentalities utilized to navigate our social relations, and how they texture self-identity in those relations, have a major bearing on a whole range of mental health indices. For example, Crocker and Canevello (2008) compared compassionate self-goals to what they called *ego self-identity goals*. Compassionate self-goals were related to wanting to be helpful to others, and to wanting not to be hurtful. In contrast, ego self-identity goals were self-focused (such as wanting to be "recognized," and avoid making mistakes or being shamed). Compassion self-identity goals were associated with better and more supportive social relationships and well-being, whereas self-image-focus goals were defensive and associated with poorer social relationships, confusion, and less positive affect. It is likely (although there is no evidence to date) that ego-focused self-identity goals would be quite prominent in socially anxious people.

Now, of course, a "care for others" can at times be a type of submissiveness. In our department, we are looking at what we call "submissive compassion" where the main focus is to be kind to others "in order to be liked and avoid being rejected." Preliminary data suggest that this type of "caring" is linked to depression and anxiety, whereas genuine caring is not (see also McEwan, Gilbert, & Duarte, 2012). The problem with submissive caring, or "controlled caring," is that it does not really provide opportunities for developing genuine empathy and compassion motivation. This is because caring becomes more of a safety strategy (to ensure being liked and avoiding rejection, or feeling that oneself is a good person), rather than based on cultivating emotional processing systems that underpin empathic caring.

Developing Compassion

But what happens if you deliberately set out to cultivate a compassionate and caring social mentality by providing practices that focus on trying to understand the minds of others and take a caring interest in them—that is, to develop the *compassionate mind* (Gilbert, 2009)?

In fact there is increasing evidence that training individuals in developing feelings of kindness and affiliation *for* others (sometimes in meditation practice) influences well-being, coping, and feelings of social connectedness (Fredrickson, Cohn, Coffey, Pek, Finkel, 2008; Hofmann et al., 2011; Hutcherson, Seppala, & Gross, 2008), and affects neurophysiological systems, especially the frontal cortex (Lutz, Brefczynski-Lewis, Johnstone, & Davidson, 2008). Many of these practices are based on *loving kindness meditations*, but it is important to consider that the capacities to mentalize and develop genuine empathy for others (Fonagy, Gergely, Jurist, & Target, 2002) have

to be important competencies for compassion, too. To date, there is no clear evidence that compassion cultivation and compassionate mind training will help socially anxious people, but there is a lot of indirect evidence that it might. For example, it could help socially anxious people to switch out of their competitive mentality; it will help them focus away from trying to stimulate positive emotions based on excitement and drive, and focus instead on affiliative relating and ways of calming and soothing, feeling safe in being with others because one has a caring interest in others.

In compassion-focused therapy (Gilbert, 2010), various methods and meditative techniques are utilized to help the person imagine *becoming the compassionate self* with wisdom, a sense of authority/confidence; and focusing on the motivation to be empathic, mentalize, and be committed to the well-being of others. This is not so dissimilar to Mahayana Tibetan compassion practices, which focus on *Bodhichitta*—the seeking of enlightenment to become a compassionate being, for the purpose of helping others (Leighton, 2003).

There is evidence that the practice of imagining one's "best possible self" is related to emotional change such as increased optimism (Meevissen, Peters, & Alberts, 2011; Peters, Flink, Boersma, & Linton, 2010). Similarly, practicing positive self-imagery by way of recalling a time when one felt relaxed and positive was related to higher levels of self-esteem and reduced anxiety in response to anxiety-provoking vignettes in socially anxious people (e.g., meeting your partner's parents for the first time) (Stopa, Brown, & Hirsch, 2012).

Compassion is also a two-way street to the extent that we can also be open to the compassion received from others. Recent research has suggested that many indicators of mental health difficulties are associated with fears of being open and responsive to the compassion of others. Keep in mind, however, that when one begins to experience affiliative emotions, this can also generate difficult emotions related to past disappointments in affiliation (being let down or even abused in close relationships), and ongoing feelings of loneliness or separation. So, the first steps are to be open to the kindness of others—but this is not always easy for people, and even kindness can be experienced as a threat (Rockliff, Gilbert, McEwan, Lightman, & Glover, 2008; Rockliff et al., 2011).

Self-Compassion

We have seen that socially anxious people are also self-critical, and are poor at self-reassurance or self-soothing. Self-compassion, however, is conducive to well-being (Neff, Kirkpatrick, & Rude, 2007). In fact, there are now a number of therapies that are specifically focused on developing self-compassion (Gilbert, 2009), including for anxiety disorders (Roemer & Orsillo, 2012). The obvious problem with self-criticism is that it will be constantly stimulating threat and does not facilitate a calming or soothing quality on anxiety. Teaching people self-compassion, and helping to generate self-affiliative emotion, is therefore one antidote to self-criticism because it involves developing a new emotion-based relationship with oneself. In addition, self-compassion can begin to address problems with shame (Gilbert, 2010). This

is important, because shame and fear of being shamed are major barriers to the development of affiliative open relationships.

Conclusions

The chapter began by recognizing that social competition is a source of threat for many animals. Consequently, various defense systems for coping with conflict competition have evolved, including abilities to monitor, track, and make judgments about oneself in relation to others; taking submissive positions if one sees oneself as inferior; and having attentional sensitivity to those who are up-rank. There is good evidence that socially anxious people are indeed caught up in these defensive strategies.

However, the dynamics of human competition are different to those of other animals. While other animals may be worried about attracting the attention of a more dominant (potentially hostile) individual, humans are more worried about being rejected, marginalized, and/or deemed boring because one is judged unattractive, uninteresting, or incompetent. So the socially anxious individual is competing hard to try to be included, wanted, and valued, which puts them under enormous pressure to try to impress others. So, rather than engaging relationships in a friendly and affiliative orientation, they come with a competitive mentality. In doing so, they are immediately linked to the defenses of competitive behavior. Providing socially anxious people with accurate feedback and reducing over-monitoring and safety behaviors have proved helpful. One additional source of help for socially anxious people is to ensure that, whatever intervention is used, they can experience the affiliative emotions as well, so compassion focusing on self and other might be one way to do this (Gilbert, 2010).

Acknowledgment

The author expresses his sincere gratitude to Dr. J. W. Weeks for his extensive guidance, suggestions, editorial advice, and input.

References

Anderson, C., Willer, R., Kilduff, G. J., & Brown, C. E. (2012). The origins of deference: When do people prefer lower status? *Journal of Personality and Social Psychology, 102*, 1077–1088. doi:10.1037/a0027409

Andrews, B., & Brewin, C. R. (1990). Attributions of blame for marital violence: A study of antecedents and consequences. *Journal of Family and Marriage, 52*, 757–767.

Andrews, B., Qian, M., & Valentine, J. (2002). Predicting depressive symptoms with a new measure of shame: The experience of shame scale. *The British Journal of Clinical Psychology, 41*, 29–33.

Barkow, J. H. (1989). *Darwin, sex and status: Biological approaches to mind and culture*. Toronto, ON: University of Toronto Press.

Baron-Cohen, S. (Ed.) (1997). *The maladaptive mind: Classic readings in evolutionary psychology*. London, UK: Psychology Press.

Belsky, J., & Pluess, M. (2009). Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin, 135*, 885–908. doi:10.1037/a0017376

Boksem, M. A. S., Kostermans, E., Milivojevic, B., & De Cremer, D. (2012). Social status determines how we monitor and evaluate our performance. *Social Cognitive and Affective Neuroscience, 7*, 304–313. doi:10.1093/scan/nsr010

Bolhuis, J. J., Brown, G. R., Richardson, R. C., & Laland, R. N. (2011). Darwin in mind: New opportunities for evolutionary psychology. *PLOS: Biology, 9*, 1–8. doi:10.1371/journal.pbio.1001109

Boyce, W. T., & Ellis, B. J. (2005). Biological sensitively to context: An evolutionary-developmental theory of the origins and functions of stress reactivity. *Development and Psychopathology, 17*, 271–302.

Breen, W. E., & Kashdan, T. B. (2011). Anger suppression after imagined rejection among individuals with social anxiety. *Journal of Anxiety Disorders, 25*, 879–887. doi:10.1016/j.janxdis.2011.04.009

Brook, C. A., & Schmidt, L. A. (2008). Social anxiety disorder: A review of environmental risk factors. *Neuropsychiatric Disease and Treatment, 4*, 123–143.

Brune, M., Belsky, J., Fabrega, H., Feierman, J. R., Gilbert, P., Glantz, K., . . . Wilson, D. R. (2012). The crisis of psychiatry – Insights and prospects from evolutionary theory. *World Psychiatry, 11*, 55–57.

Cacioppo, J. T., & Patrick, W. (2008). *Loneliness: Human nature and the need for social connection*. New York, NY: W. W. Norton & Company.

Chance, M. R. A. (1984). Biological systems synthesis of mentality and the nature of the two modes of mental operation: Hedonic and agonic. *Man-Environment Systems, 14*, 143–157.

Cheney, D., Seyfarth, R., & Smuts, B. (1986). Social relationships and social cognition in nonhuman primates. *Science, 234*, 1361–1365.

Cook, D. R. (1996). Empirical studies of shame and guilt: The internalized shame scale. In D. L. Nathanson (Ed.), *Knowing feeling: Affect, script and psychotherapy* (pp. 132–165). New York, NY: W. W. Norton & Company.

Cox, B. J., Fleet, C., & Stein, M. (2004). Self-criticism and social phobia in the US national comorbidity survey. *Journal of Affective Disorders, 82*, 227–234.

Cozolino, L. (2007). *The neuroscience of human relationships: Attachment and the developing brain*. New York, NY: W. W. Norton & Company.

Craske, M. G., Wolitzky-Taylor, K. B., Mineka, S., Zinbarg, R., Waters, A. M., Vrshek-Schallhorn, S., . . . Ornitz, E. (2012). Elevated responding to safe conditions as a specific risk factor for anxiety versus depressive disorders: Evidence from a longitudinal investigation. *Journal of Abnormal Psychology, 121*, 315–324.

Crocker, J., & Canevello, A. (2008). Creating and undermining social support in communal relationships: The role of compassionate and self-image goals. *Journal of Personality and Social Psychology, 95*, 555–575.

Depue, R. A., & Morrone-Strupinsky, J. V. (2005). A neurobehavioral model of affiliative bonding. *Behavioral and Brain Sciences, 28*, 313–395.

Dickerson, S. S., Gruenewald, T. L., & Kemeny, M. E. (2004). When the social self is threatened: Shame, physiology, and health. *Journal of Personality, 72*, 1192–1216.

Dunbar, R. I. M. (2010). The social role of touch in humans and primates: Behavioural function and neurobiological mechanisms. *Neuroscience and Biobehavioral Reviews, 34*, 260–268. doi:10.1016/j.neubiorev.2008.07.001

Dunbar, R. I. M., & Barrett, L. (2008). *The Oxford handbook of evolutionary psychology*. Oxford, UK: Oxford University Press.

Erwin, B., Heimberg, R. G., Schneier, F., & Liebowitz, M. R. (2003). Anger experience and expression in social anxiety disorder: Pretreatment profile and predictors of attrition and response to cognitive-behavioral treatment. *Behavior Therapy, 34*, 331–350.

Etcoff, N. (1999). *Survival of the prettiest: The science of beauty*. New York, NY: Doubleday.

Fonagy, P., Gergely, G., Jurist, E. L., & Target, M. (2002). *Affect regulation, mentalization, and the development of the self*. London, UK: Other Press.

Forrest, M. S., & Hokanson, J. E. (1975). Depression and autonomic arousal reduction accompanying self-punitive behavior. *Journal of Abnormal Psychology, 84*, 346–357.

Fournier, M. A., Moskowitz, D. S., & Zuroff, D. C. (2002). Social rank strategies in hierarchical relationships. *Journal of Personality and Social Psychology, 83*(2), 425–433.

Fredrickson, B. L., Cohn, M. A., Coffey, K. A., Pek, J., & Finkel. S. A. (2008). Open hearts build lives: Positive emotions, induced through loving-kindness meditation, build consequential personal resources. *Journal of Personality and Social Psychology, 95*, 1045–1062.

Gilbert, P. (1989). *Human nature and suffering*. Hove, UK: Lawrence Erlbaum Associates.

Gilbert, P. (1992). *Depression: The evolution of powerlessness*. Hove, UK: Lawrence Erlbaum.

Gilbert, P. (1993). Defence and safety: Their function in social behaviour and psychopathology. *British Journal of Clinical Psychology, 32*, 131–153.

Gilbert, P. (1997). The evolution of social attractiveness and its role in shame, humiliation, guilt and therapy. *British Journal of Medical Psychology, 70*, 113–147.

Gilbert, P. (1998a). Evolutionary psychopathology: Why isn't the mind better designed than it is? *British Journal of Medical Psychology, 71*, 353–373.

Gilbert, P. (1998b). The evolved basis and adaptive functions of cognitive distortions. *British Journal of Medical Psychology, 71*, 447–463.

Gilbert, P. (1998c). What is shame? Some core issues and controversies. In P. Gilbert & B. Andrews (Eds.), *Shame: Interpersonal behavior, psychopathology and culture* (pp. 3–36). New York, NY: Oxford University Press.

Gilbert, P. (2000). Varieties of submissive behaviour: Their evolution and role in depression. In L. Sloman & P. Gilbert (Eds.), *Subordination and defeat. An evolutionary approach to mood disorders* (pp. 3–46). Hillsadale, NJ: Lawrence Erlbaum.

Gilbert, P. (2001). Evolution and social anxiety: The role of social competition and social hierarchies. *Psychiatric Clinics of North America, 24*, 723–751.

Gilbert, P. (2005). Social mentalities: A biopsychosocial and evolutionary reflection on social relationships. In M. Baldwin (Ed.), *Interpersonal cognition* (pp. 299–333). New York, NY: Guilford.

Gilbert, P. (2009). *The compassionate mind*. London, UK: Constable & Robinson Ltd.

Gilbert, P. (2010). *Compassion focused therapy: The CBT distinctive features series*. London, UK: Routledge.

Gilbert, P. (2012). Compassion focused therapy. In W. Dryden (Ed.), *Cognitive behaviour therapies* (pp. 140–165). London, UK: Sage.

Gilbert, P., & Allan, S. (1994). Assertiveness, submissive behavior, and social comparison. *British Journal of Clinical Psychology, 33*, 295–306.

Gilbert, P., Allan, S., Ball, L., & Bradshaw, Z. (1996). Overconfidence and personal evaluations of social rank. *British Journal of Medical Psychology, 69*, 59–68.

Gilbert, P., Allan, S., Brough, S., Melley, S., & Miles, J. (2002). Anhedonia and positive affect: Relationship to social rank, defeat and entrapment. *Journal of Affective Disorders, 71*, 141–151.

Gilbert, P., Allan, S., & Pehl, J. (1994). The phenomenology of shame and guilt: An empirical investigation. *British Journal of Medical Psychology, 67*, 23–36.

Gilbert, P., Boxall, M., Cheung, M., Tuffield, V., & Irons, C. (2005). The relation of paranoid thinking and social anxiety in a mixed clinical population. *Clinical Psychology and Psychotherapy, 12*, 124–133.

Gilbert, P., Broomhead, C., Irons, C., McEwan, K., Bellew, R., Mills, A., Gale, C. (2007). Development of a striving to avoid inferiority scale development and its relationship to depression, anxiety and stress. *British Journal of Social Psychology, 46*, 633–648.

Gilbert, P., & Choden (2013). *Mindful compassion*. London, UK: Constable-Robinson.

Gilbert, P., Irons, C., McEwan, K., Bhundia, R., Christie, R, & Broomhead, C. (2010). Self-harm in a mixed clinical population: The roles of shame, forms and functions of self-criticism and social rank. *British Journal of Clinical Psychology, 49*, 563–576.

Gilbert, P., McEwan, K., Gibbons, L., Chotai, S., Duarte, J., & Matos, M. (2011). Fears of compassion and happiness in relation to alexithymia, mindfulness and self-criticism. *Psychology and Psychotherapy, 84*, 239–255. doi:10.1348/147608310×526511

Gilbert, P., & Miles, J. N. V. (2000). Sensitivity to put-down: Its relationship to perceptions of shame, social anxiety, depression, anger and self-other blame. *Personality and Individual Differences, 29*, 757–774.

Gilbert, P., Price, J. S., & Allan, S. (1995). Social comparison, social attractiveness and evolution: How might they be related? *New Ideas in Psychology, 13*, 149–165.

Gilbert, P., & Trower, P. (1990). The evolution and manifestation of social anxiety. In W. R. Crozier (Ed.), *Shyness and embarrassment: Perspectives from social psychology*. Cambridge, UK: Cambridge University Press.

Gilbert, P., & Trower, P. (2001). Evolution and process in social anxiety. In W. R. Crozier & L. E. Alden (Eds.), *International handbook of social anxiety: Concepts, research and interventions relating to the self and shyness* (pp. 259–279). Chichester, UK: John Wiley & Sons, Ltd.

Goss, K., Gilbert, P., & Allan, S. (1994). An exploration of shame measures: I. The "Other as Shamer Scale". *Personality and Individual Differences, 17*, 713–717.

Grant, K. A., Shively, C. A., Nader, M. S., Ehrenkaufer, R. L., Line, S. W., Morton, T. E.,... Mach, R. H. (1998). Effects of social status on striatal dopamine D_2 receptor binding characteristics in cynomologus monkeys assessed with positron emission tomography. *Synapse, 29*, 80–83.

Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (2004). *Acceptance and commitment therapy: An experiential approach to behavior change*. New York, NY: Guilford.

Hochschild, A. R. (1983). *The managed heart: Commercialization of human feeling*. Berkeley: University of California Press.

Hoffmann, S. G., Grossman, P., & Hinton, D. E. (2011). Loving-kindness and compassion meditation: Potential for psychological intervention. *Clinical Psychology Review, 13*, 1126–1132.

Horney, K. (1945/1992). *Our inner conflicts: A constructivist theory of neuroses*. New York, NY: W. W. Norton & Company.

Hutcherson, C. A., Seppala, E. M., & Gross, J. J. (2008). Loving-kindness meditation increases social connectedness. *Emotion, 8*(5), 720–724.

Hutton, P., Kelly, J., Lowens, I., Taylor, P. J., & Tai, S. (2013). Self-attacking and self-reassurance in persecutory delusions: A comparison of healthy, depressed and paranoid individuals. *Psychiatry Research, 205*, 127–136.

Insel, T. R. (2010). The challenge of translation in social neuroscience: A review of oxytocin, vasopressin, and affiliative behavior. *Neuron, 65*, 768–779.

Kashdan, T. B. (2007). Social anxiety spectrum and diminished positive experiences: Theoretical synthesis and meta-analysis. *Clinical Psychology Review, 27*, 348–365.

Kashdan, T. B., Weeks, J. W., & Savostyanova, A. A. (2011). Whether, how, and when social anxiety shapes positive experiences and events: A self-regulatory framework and treatment implications. *Clinical Psychology Review, 31*, 786–799.

Kelly, A. C., Zuroff, D. C., Leybman, M. J., & Gilbert, P. (2012). Social safeness, received social support, and maladjustment: Testing a tripartite model of affect regulation. *Cognitive Therapy and Research* (published online). doi:10.1007/s10608-011-9432-5

Klimecki, O. M., Leiberg, S., Lamm, C., & Singer, T. (2013). Functional neural plasticity and associated changes in positive affect after compassion training. *Cerebral Cortex, 23*, 1552–1561. doi:10.1093/cercor/bhs142

Leary, M. R. (2004). *The curse of the self: Self-awareness, egotism and the quality of human life*. New York, NY: Oxford University.

LeDoux, J. (1998). *The emotional brain*. London, UK: Weidenfeld and Nicolson.

Leighton, T.D. (2003). *Faces of compassion: Classic Bodhisattva archetypes and their modern expression*. Somerville, MA: Wisdom Publications.

Liotti, G. (2011). Attachment disorganization and the controlling strategies: An illustration of the contributions of attachment theory to developmental psychopathology and to psychotherapy integration. *Journal of Psychotherapy Integration, 21*, 232–252. doi:10.1037/a0025422

Liotti, G., & Gilbert, P. (2011). Mentalizing, motivations and social mentalities: Theoretical considerations and implications for psychotherapy. *Psychology and Psychotherapy, 84*, 9–25.

Lutz, A., Brefczynski-Lewis, J., Johnstone, T., & Davidson, R. J. (2008). Regulation of the neural circuitry of emotion by compassion meditation: Effects of the meditative expertise. *Public Library of Science, 3*, 1–5.

MacDonald, K., & MacDonald, T. M. (2010). The peptide that binds: A systematic review of oxytocin and its prosocial effects in humans. *Harvard Review of Psychiatry, 18*, 1–21.

MacLean, P. D. (1990). *The triune brain in evolution*. New York, NY: Plenum Press.

Marks, I. M. (1987). *Fears, phobias, and rituals: Panic, anxiety and their disorders*. Oxford, UK: Oxford University Press.

Marks, I. M., & Nesse, R. M. (1997). Fears and fitness: An evolutionary analysis of anxiety disorders. In S. Baron Cohen (Ed.), *The maladaptive mind: Classic readings in evolutionary psychology* (pp. 57–72). London, UK: Psychology Press.

Matos, M., Pinto-Gouveia, J., & Gilbert, P. (2013). The effect of shame and shame memories on paranoid ideation and social anxiety. *Clinical Psychology and Psychotherapy, 20*, 334–349. doi:10.1002/cpp.1766

McEwan, K., Gilbert, P., & Duarte, J. (2012). An exploration of competitiveness and caring in relation to psychopathology. *British Journal of Clinical Psychology, 51*, 19–36. doi:10.1111/j.2044-8260.2011.02010.x

Meevissen, Y. M. C., Peters, M. L., & Alberts, H. J. E. M. (2011). Become more optimistic by imagining a best possible self: Effects of a two week intervention. *Journal of Behavior Therapy and Experimental Psychiatry, 42*, 371–378.

Mikulincer, M., & Shaver, P. R. (2007). *Attachment in adulthood: Structure, dynamics, and change*. New York, NY: Guilford.

Mills, A., Gilbert, P., Bellew, R., McEwan, K., & Gale, C. (2007). Paranoid beliefs and self-criticism in students. *Clinical Psychology and Psychotherapy, 14*, 385–364.

Neff, K. D., Kirkpatrick, K., & Rude, S. S. (2007). Self-compassion and its link to adaptive psychological functioning. *Journal of Research in Personality, 41*, 139–154.

Panksepp, J. (1998). *Affective neuroscience*. New York, NY: Oxford University Press.

Panksepp, J. (2010). Affective neuroscience of the emotional brain-mind: Evolutionary perspectives and implications for understanding depression. *Dialogues in Clinical Neuroscience, 12*, 383–399. Retrieved from www.dialogues-cns.org

Peters, M. L., Flink, I. K., Boersma, K., & Linton, S. J. (2010). Manipulating optimism: Can imagining a best possible self be used to increase positive future expectancies? *The Journal of Positive Psychology, 5*, 204–211.

Porges, S. W. (2007). The polyvagal perspective. *Biological Psychology, 74*, 116–143. doi:10.1016/j.biopsycho.2006.06.009

Rockliff, H., Gilbert, P., McEwan, K., Lightman, S., & Glover, D. (2008). A pilot exploration of heart rate variability and salivary cortisol responses to compassion-focused imagery. *Journal of Clinical Neuropsychiatry, 5*, 132–139.

Rockliff, H., Karl, A., McEwan, K., Gilbert, J., Matos, M., & Gilbert, P. (2011). Effects of intranasal oxytocin on compassion focused imagery. *Emotion, 11*, 1388–1396. doi:10.1037/a0023861

Roemer, L., & Orsillo, S. M. (2012). Anxiety disorders: Acceptance, compassion and wisdom. In C. K. Germer & R. D. Siegel (Eds.), *Wisdom and compassion in psychotherapy: Deepening mindfulness in clinical practice* (pp. 234–248). New York, NY: Guilford.

Santor, D. A., & Walker, J. (1999). Garnering the interests of others: Mediating the effects among physical attractiveness, self-worth, and dominance. *British Journal of Social Psychology, 38*, 461–477.

Sapolsky, R. M. (2004). *Why zebras don't get ulcers.* St. Martin's Press.

Schlenker, B. R., & Leary, M. R. (1982). Social anxiety and self-presentation: A conceptualization and model. *Psychological Bulletin, 92*, 641–669.

Schneier, F. R., Johnson, J., Hornig, C. D., Liebowitz, M. R., & Weissman, M. M. (1992). Social phobia: Comordity and morbidity in an epidemiolgic sample. *Archives of General Psychiatry, 49*, 282–288.

Scott, J. (1990). *Domination and the arts of resistance.* New Haven, CT: Yale University Press.

Seligman, M. E. P. (1971). Phobias and preparedness. *Behaviour Therapy, 2*, 307–320.

Stopa, L., Brown, M. A., & Hirsch, C. R. (2012). The effects of repeated imagery practice on self-concept, anxiety and performance in socially anxious participants. *Journal of Experimental Psychopathology, 3*, 223–242.

Tangney, J. P., & Dearing, R. L. (2002). *Shame and guilt.* New York, NY: Guilford.

Taylor, P., Gooding, P., Wood, A. N., & Tarrier, N. (2011). The role of defeat and entrapment in depression, anxiety and suicide. *Psychological Bulletin, 137*, 391–420. doi:10.1037/a0022935

Tobena, A., Marks, I., & Dar, R. (1999). Advantages of bias and prejudice: An exploration of their neurocognitive templates. *Neuroscience and Behavioral Reviews, 23*, 1047–1058.

Tracy, J. L., Robins, R. W., & Tangney, J. P. (Eds.) (2007). *The self-conscious emotions: Theory and research.* New York, NY: Guilford.

Trower, P., Sherling, G., Beech, J., Harrop, C., & Gilbert, P. (1998). The socially anxious perspective in face to face interaction: An experimental comparison. *Clinical Psychology and Psychotherapy: An International Journal of Theory and Practice, 5*, 155–166.

Warneken, F., Chen, F., & Tomasello, M. (2006). Co-operative activities in young children and chimpanzees. *Child Development, 77*, 640–663.

Weeks, J. W., Heimberg, R. G., & Heuer, R. (2011). Exploring the role of submissiveness in social anxiety: Testing an evolutionary model of social anxiety disorder. *Journal of Social and Clinical Psychology, 30*, 217–249.

Weeks, J. W., Heimberg, R. G., & Rodebaugh, T. L. (2008). The Fear of Positive Evaluation Scale (FPES): Assessing a proposed cognitive component of social anxiety disorder. *Journal of Anxiety Disorders, 22*, 44–55.

Weeks, J. W., & Howell, A. N. (2012). The bivalent fear of evaluation model of social anxiety: Further integrating findings on fears of positive and negative evaluation. *Cognitive Behaviour Therapy, 41*, 83–95.

Weeks, J. W., Jakatdar, T. A., & Heimberg, R. G. (2010). Comparing and contrasting fears of positive and negative evaluation as facets of social anxiety. *Journal of Social and Clinical Psychology, 29*, 68–94.

Weeks, J. W., Rodebaugh, T. L., Heimberg, R. G., Norton, P. J., & Jakatdar, T. A. (2009). "To avoid evaluation, withdraw": Fears of evaluation and depressive cognitions lead to social anxiety and submissive withdrawal. *Cognitive Therapy and Research, 33*, 375–389.

Wells, A. (2009). *Metacognitive therapy anxiety and depression*. New York, NY: Guilford.

Wild, J. (2009). Imagery and the self in social phobia. In L. Stopa (Ed.), *Imagery and the threatened and self: Perspectives on mental imagery and the self in cognitive therapy* (pp. 94–111). London, UK: Routledge.

3

Genetic Factors in Social Anxiety Disorder

Murray B. Stein[1] and Joel Gelernter[2]

[1] *University of California San Diego, USA*
[2] *Yale University School of Medicine, USA*

Overview

Social anxiety disorder (SAD; i.e., social phobia) is characterized by extreme concern about negative evaluation by others, and consequent fear and avoidance of situations in which such social evaluation might occur (Stein & Stein, 2008). At first blush, it might seem strange to consider that there could be a genetic basis for what, essentially, is a belief system and a set of accompanying reactions. But SAD is one of several, frequently comorbid mental disorders (e.g., panic disorder, major depression) that do seem to be familial and heritable (Gelernter & Stein, 2009). Moreover, SAD can, to some extent, be viewed as an extreme concoction of personality traits, such as shyness and neuroticism, that have themselves been shown to have genetic bases (Bienvenu, Hettema, Neale, Prescott, & Kendler, 2007; Flint, 2004). Taken together, these observations have led over the past 15 years to a search for the genetic basis of SAD and related traits (Stein & Gelernter, 2010). This search, although still in a rather perfunctory stage, has begun to reveal aspects of the genetic architecture of SAD.

In this chapter, we refer to family and twin data that support the notion of there being a heritable component to SAD. We review linkage studies (of which there are few) and association studies (of which there are relatively more) in SAD and related traits, emphasizing areas of convergent or replicated findings (of which there are very few). We also discuss several approaches to understanding a genetic basis for SAD that rely on the assessment of neurocognitive indices (e.g., attentional biases; regional cerebral blood flow)—so-called intermediate phenotypes or, as we will refer to them here, endophenotypes—thought to underlie the disorder, in part, biologically. We conclude with a discussion of future directions for research into the genetic, and finally, also the genomic, basis for SAD.

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

Family and Twin Evidence for Genetic Factors in SAD

SAD does have a familial basis, which is clearest for the generalized subtype of the disorder, which characterizes those individuals with multiple, pervasive social fears, that is, not those with exclusively public-speaking fears (Stein, Chartier, Hazen, et al., 1998). (Note that the authors of the Diagnostic and Statistical Manual of mental disorders [DSM-5] have chosen to do away with a description of the "generalized" subtype and instead to define its converse, a "public-speaking" subtype [Bogels et al., 2010]). A family study also showed that several social anxiety-related traits, such as social interactional anxiety, have a familial basis (Stein, Chartier, Lizak, & Jang, 2001). These kinds of findings suggest that an underlying endophenotype(s) may be what is familial (and, possibly, genetic), and not necessarily the DSM disorder of SAD, *per se*.

Twin studies, in which higher phenotypic correlations in monozygotic (i.e., "identical") than dizygotic (i.e., "non-identical") twin pairs is evidence of a heritable component, strongly suggest that SAD has a partially genetic basis (Kendler, Myers, Prescott, & Neale, 2001). The twin studies generally suggest that the contribution of genes to the excess similarity (or co-occurrence) in social anxiety of monozygotic over dizygotic pairs is modest (in the range of 20–40%), on par with many other common mental disorders. This is also true when social-anxiety-related symptoms such as *behavioral inhibition* (Smith et al., 2012; Warren, Schmitz, & Emde, 1999) or a core cognitive construct of SAD, the *fear of negative evaluation* (Stein, Jang, & Livesley, 2002; Weeks et al., 2005), are assessed in twin studies, and other genetically informative family-based studies. Twin studies also tend to find that SAD, panic disorder, and agoraphobia have in common a genetic factor that explains a moderate portion of the variance in these disorders (e.g., Mosing et al., 2009). This has prompted the search not only for gene alleles that specifically increase risk for SAD but also for these frequently co-occurring disorders.

Genetic Linkage Studies in SAD

Genetic linkage is the tendency of genes that are located close to each other on a chromosome to be inherited together during cellular meiosis. Genes nearer to each other are less likely to be separated onto different chromatids during chromosomal crossover, and are therefore considered genetically *linked* (Strachan, 2010). The *LOD score* (logarithm [base 10] of odds) is a statistical test used for linkage analysis, used to determine the likelihood of linkage between a trait (e.g., social anxiety) and a given genetic marker. Positive LOD scores suggest the presence of linkage, whereas negative LOD scores indicate that linkage is less likely.

In earlier work, we were able to provide evidence excluding linkage of the generalized subtype of SAD to the serotonin transporter promoter, serotonin$_{2A}$ receptor, and to a series of dopamine (DA) receptor genes in a particular set of families (Kennedy et al., 2001; Stein, Chartier, Kozak, et al., 1998). These studies, we now know, were underpowered to detect anything other than genes with major effects. The presence

of such gene effects is now known to be a very unlikely scenario, given our increased understanding of genetic contributions to complex traits like anxiety disorders (Colhoun, McKeigue, & Davey, 2003; Smoller, Block, & Young, 2009). We subsequently conducted a genome-wide linkage study of SAD with analysis at 10 cM (centiMorgan) resolution in a set of extended pedigrees (approximately 160 individuals) (Gelernter, Page, Stein, & Woods, 2004). A nonparametric LOD score of 3.41 was observed on chromosome 16 at position 62.3 cM ($p = .0003$) within a 53.3 cM region that spans from position 40.6 to 93.9, suggesting possible linkage within this region. A possible candidate gene mapped in this region is *SLC6A2*, the norepinephrine transporter protein locus (protein product, NET1), which maps within the region of interest, but not at the linkage peak itself.

Additional studies are needed to replicate these findings and to enable fine-mapping that would help confirm the identity of potential susceptibility genes within this region. Interestingly, a recent study tested the association of 29 single-nucleotide polymorphisms (SNPs) in *SLC6A2* in a case–control sample of 449 patients with panic disorder and 279 ethnically matched controls (Buttenschon et al., 2011). The investigators found seven SNPs located within the 5′ end of the gene significantly associated with panic disorder. These findings point to the possibility that panic disorder and SAD, which are highly comorbid and share many clinical features (e.g., see **Chapter 10**), may have common susceptibility genes, and that future work focused on an expanded anxiety phenotype may be fruitful. It is noteworthy that the families we used for our earlier SAD linkage study (Stein, Chartier, Kozak, et al., 1998) had originally been recruited on the basis of multiple individuals affected with panic disorder.

A similar conclusion was reached by investigators who reanalyzed their panic disorder linkage data by considering a broader phenotype (that included phobic disorders, among them SAD) (Logue et al., 2012). These investigators used a *fuzzy-clustering method* (Kaabi et al., 2006) to report that this emphasis on a broader phobic-anxiety phenotype led them to detect evidence of linkage on several chromosomes (e.g., 4q21 and 7p) that had previously been implicated by Kaabi et al. (2006) using fuzzy clustering to evaluate anxiety disorder linkage, and other investigators looking at panic disorder. Although it is hard to know, based on these data, to what extent these loci may be directly relevant to SAD (in terms of shared genetic liability), the findings do point to the likely merits of expanding our phenotypic scope beyond specific DSM disorders to include, for example, highly related and overlapping disorders simultaneously, such as panic disorder, SAD, and possibly other phobic disorders.

This approach, that is, expanding the phenotype under consideration to better accommodate the complexity of these anxiety-related phenotypes, has recently been adopted by other anxiety disorders investigators. Turning to multiply-affected families in a panic disorder linkage study, the researchers reanalyzed their data using 10 additional literature-based panic and phobia-related phenotypes (Fyer et al., 2012). Their highest LOD score, on chromosome 13 (D13S793, 76 cM), was for the phenotype that included specific or social phobia. The authors conclude that these data, though limited by relatively low power, suggest a region on chromosome 13 as a potential site for further exploration in relation to the risk for specific phobias and SAD.

It should be apparent from the review of the above studies that, with the barely possible exception of *NET1* on chromosome 16, a coherent story has yet to emerge,

in large part due to the paucity of studies and the low power for the studies that have been conducted. For the past decade, most investigators have turned to the use of techniques thought to be better suited to the detection of gene variants contributing to variation in complex traits. In large part, linkage studies have been overtaken by association studies for this purpose.

Genetic Association Studies in SAD and Related Traits

There are now hundreds of published association studies looking at anxiety disorders or anxiety-related traits. Panic disorder has been the most extensively evaluated phenotype in such studies (Maron, Hettema, & Shlik, 2010; Schumacher et al., 2011), whereas far fewer have focused on SAD (Hamilton, 2009). As for linkage, few studies can be considered to be adequately powered. In this section, we will review the published association study literature in SAD and related traits.

Social Anxiety Disorder

Finnish researchers took a cross-species approach to identify genes that regulate anxiety-like behavior, using inbred mouse strains that differed in their innate anxiety levels as a model system. Having earlier identified 17 genes with expression levels that correlated with anxiety behavior across the studied strains (Hovatta et al., 2005), they reasoned that these genes would be strong candidates for anxiety in humans. Accordingly, using a sample from a population-based Finnish cohort, they tested a total of 208 SNPs for association in their 13 known human homologs as candidate genes for human anxiety disorders (Donner et al., 2008). They found that specific alleles and haplotypes of 6 of the 13 genes showed nominal evidence for association ($p \leq .01$). The strongest evidence for association with SAD was found for *ALAD* (delta-aminolevulinate dehydratase) ($p = .0009$).

In a follow-up to that study, the investigators tested for association between anxiety disorders and circadian system genes, noting that circadian disturbances (seen as sleep problems) were common in persons with anxiety disorders (Sipila et al., 2010). They analyzed 131 SNPs from 13 circadian clock-related genes in the aforementioned Finnish sample consisting of 321 persons with anxiety disorders and 653 healthy controls. They found SNPs in two genes that showed nominal evidence of association to SAD: in *ARNTL2* rs2306073 ($p = .0099$) and in *DRD2* rs7131056 ($p = .0084$).

Most recently, investigators evaluated variation in the *SIRT1* gene in relation to anxiety disorders. The motivation for this study came from observations that sirtuin regulates anxiety and exploratory drive in mice via effects on the gene that encodes monoamine oxidase A (*MAO-A*), resulting in a reduction in brain serotonin levels (Libert et al., 2011). The human component of this study involved the genotyping of 14 SNPs across the *SIRT1* gene in a random sample of 3420 Swiss men and women for whom DSM-IV diagnoses were available. They found significant association between SNPs in *SIRT1* and panic disorder (rs12778366 and rs10997870), one of which was also significantly associated with SAD (rs12778366). These findings provide a

very interesting avenue for future therapeutics involving sirtuin drugs that cross the blood–brain barrier.

Blushing

Investigators included 62 patients with SAD and 62 age- and sex-matched healthy controls to study the influence of a variant in the serotonin transporter gene (*SLC6A4*) promoter region (5HTTLPR, and rs25531) on tendency to blush (Domschke et al., 2009; see **Chapter 5** for in-depth discussion of the link between SAD and blushing). They found that the less functionally active 5-HTTLPR genotypes were significantly associated with increased blushing propensity in patients with SAD as compared with controls. The relatively small sample size notwithstanding, the authors suggested that their findings might indicate a potential role of functional serotonin transporter gene variation in blushing propensity. The authors also concluded that this study warranted replication and encouraged genetic analyses of further intermediate phenotypes of SAD.

Shyness

A polymorphism of the serotonin transporter promoter region (5HTTLPR), also mentioned above (see Blushing), was related to shyness in a sample of 98 Israeli children attending second grade (Arbelle et al., 2003). This polymorphism has been the focus of considerable prior scrutiny, with much of it focused on the risk for depression under stress (i.e., gene–environment interaction), although considerable controversy remains about the role of this gene (Karg, Burmeister, Shedden, & Sen, 2011; Risch et al., 2009). To the best of our knowledge, the association between 5HTTLPR and shyness has yet to be replicated.

Neuroticism

Neuroticism is a personality trait that is well recognized as a susceptibility factor for most anxiety disorders, including SAD (Kotov, Gamez, Schmidt, & Watson, 2010). Accordingly, a better understanding of the genetic bases for neuroticism could shed light on the contribution of those genes to SAD. There are, in fact, twin data directly addressing the question of the genetic relationship between neuroticism and SAD. In a large study of over 9000 twins, investigators evaluated self-reported neuroticism and several disorders for which neuroticism is a risk factor, including major depression, generalized anxiety disorder, panic disorder, specific phobias, and SAD (Hettema, Neale, Myers, Prescott, & Kendler, 2006). Using multivariate structural equation modeling, they determined how genetic and environmental factors shared by neuroticism and the aforementioned disorders might explain their comorbidity. They found that neuroticism and each of these disorders, including SAD, were moderately genetically correlated, meaning that they had genetic influences in common that contributed to their observed comorbidity. The authors concluded, as have others (Bienvenu, Wuyek, & Stein, 2010), that there is merit in searching for the common genetic

determinants of neuroticism and these disorders, providing a strong motivation for the work described below.

Many studies have examined the association between neuroticism and one or several variants in one or several candidate genes. Rather than review that voluminous literature here, we have decided to focus on studies that have examined neuroticism at the genome-wide level. In an early genome-wide association study (GWAS) of neuroticism, investigators performed pooled GWAS using deoxyribonucleic acid (DNA) pools from approximately 2000 persons selected (from among a much larger cohort of over 88,000 people from southwest England) to be at the extremes (i.e., either high or low) on neuroticism (Shifman et al., 2008). In their second stage, they then attempted to replicate the most significant SNPs in several other samples. They were unable to find SNPs that replicated across the samples, which they attributed to low power, and called for much larger studies to be conducted in the future.

In another study, investigators accessed DNA from 1227 healthy US adults in the National Institute of Mental Health genetics repository in the first stage of a two-stage GWAS of neuroticism (van den Oord et al., 2008). The most promising SNPs from the first stage were then taken forward into a German sample of 1880 healthy individuals. In aggregate, the most promising results were several SNPs in the gene *MAMDC1* but, as the authors pointed out, effect sizes were very small. More recently, a GWAS of 3792 individuals from a genetically isolated population from Sardinia, Italy, found a nonsignificant association of neuroticism with SNAP25 (rs362584; point $P = 5 \times 10^{-5}$). While interesting, this failed to achieve significance at the genome-wide level (Terracciano, Sanna, et al., 2010), and such findings have rarely been replicated. Overall, the genome-wide studies conducted to date in neuroticism, though underpowered, were nonetheless able to detect some possible gene effects, though these were not replicated across studies and the effects were very small (e.g., explaining ~1% of variance in the trait). This (i.e., the small effect size, not the lack of replication) is now an almost invariant finding in GWAS of mental disorders and other complex traits (Manolio et al., 2009).

Extroversion

Although neuroticism has been, by far, the best studied of the personality traits in the context of mood and anxiety disorders, another personality trait—extraversion—is also of considerable interest (Stein & Bienvenu, 2004). In a general population sample in which personality traits and mental disorders were assessed, researchers found that low extroversion (in addition to high neuroticism) characterized individuals with SAD (or agoraphobia) (Bienvenu et al., 2001). These observations suggest that attention to extroversion, as a complement to neuroticism, is likely to inform the study of the genetics of SAD (see also **Chapter 6** for expanded discussion of personality factors in SAD). Using a population-based sample of 7000 twins, the authors calculated the genetic relationship between neuroticism, extroversion, and SAD (Bienvenu et al., 2007). Genetic factors that influenced individual variation in extroversion and neuroticism appeared to account entirely for the genetic risk for SAD, therein highlighting the value of examining the molecular genetic determinants of both extroversion and neuroticism to SAD.

Several studies have taken a genome-wide approach to the study of extroversion (or its converse, introversion). In a GWAS of personality traits mentioned above, extroversion was found to be associated with a widely studied Val66Met (rs6265) SNP in the gene that codes for brain-derived neurotrophic factor (*BDNF*), a regulator of synaptic plasticity (Terracciano, Sanna, et al., 2010). The investigators subsequently examined the association between *BDNF* and personality traits in samples from Sardinia ($n = 1560$) and Baltimore ($n = 1131$) (Terracciano, Tanaka, et al., 2010). Consistent with their aforementioned GWAS results, they found an association for Val66Met (in the same direction as their previous study, where Met carriers [i.e., those with one or two copies of the methionine [MET] coding version of the allele] were more introverted). They also found, in a larger Sardinian sample ($n = 2333$), a significant interaction between Val66Met and 5HTTLPR, wherein 5-HTTLPR LL carriers scored lower on neuroticism in the presence of the *BDNF* Val variant, but scored higher on neuroticism in the presence of the *BDNF* Met variant. The authors proposed that their findings not only supported an association between the *BDNF* Met variant and introversion, but further suggest an interaction between *BDNF* and 5HTTLPR to influence neuroticism. To the best of our knowledge these findings have yet to be replicated, but they do suggest that a possible contribution of both genes, and their interaction, to risk for SAD and related traits should be further studied.

A meta-analysis of GWAS of personality traits was recently published (de Moor et al., 2012). Although the study was large by mental health standards, having included 17,375 adults, no genome-wide significant associations were detected for either neuroticism or extroversion. Consequently, at this writing, studies of personality traits have not contributed genes of potential relevance to SAD other than those noted above. Nevertheless, we believe that further study of genetic determinants of extroversion will be of value for the understanding of risk for SAD.

Behavioral Inhibition and Selective Mutism

Behavioral inhibition (BI) refers to a temperamental predisposition to withdrawal, avoidance, fear of the unfamiliar, and hyperarousal of the sympathetic nervous system (Hirshfeld-Becker et al., 2008; see also **Chapter 7**). Chronically high levels of BI are associated with SAD by adolescence (Essex, Klein, Slattery, Goldsmith, & Kalin, 2010). BI may represent an "intermediate phenotype" for panic and phobic disorders, including SAD (Smoller et al., 2009). In an initial study, researchers conducted family-based association analyses of BI in families of 72 high BI children, with 4 candidate genes identified using mouse models of BI (Smoller et al., 2001). The investigators observed modest evidence of association ($p = .05$) between BI and the glutamic acid decarboxylase gene (65 kDa isoform) that encodes an enzyme involved in GABA synthesis, making it a physiologically plausible candidate gene for BI. In a follow-up study, the investigators genotyped a marker tightly linked to the corticotrophin-releasing hormone (CRH) locus in 85 families of children who underwent laboratory-based behavioral assessments of BI (Smoller et al., 2003). Using family-based association analyses, they observed an inverse association between an allele of the CRH-linked

locus and BI ($p = .015$). In a subsequent family-based study, investigators found a strong association between childhood BI and several SNPs spanning *RGS2* (haplotype $P = 3 \times 10^{-5}$; odds ratio = 2.99 in complete trios) (Smoller et al., 2008). Of interest in that study, variation in this gene (i.e., more copies of the same alleles associated with BI) was also associated in healthy adults with increased amygdala reactivity to an emotional faces task, a replicated neuroimaging characteristic of persons with BI in childhood (Schwartz, Wright, Shin, Kagan, & Rauch, 2003) and SAD in adulthood (Ball et al., 2012; Battaglia et al., 2012; Phan et al., 2009; Stein, Goldin, Sareen, Zorrilla, & Brown, 2002).

Selective mutism is a disorder of childhood wherein the child fails to speak in nearly all social situations, yet has normal language development as evidenced by speech with familiar people (typically the parents). This disorder is increasingly being considered an early-onset, severe variant of SAD (Bogels et al., 2010; Carbone et al., 2010; Cohan, Price, & Stein, 2006). Motivated by the observations of high rates of SAD in persons with autism spectrum disorders (Kuusikko et al., 2008; White, Oswald, Ollendick, & Scahill, 2009), we evaluated the association of SNPs in *CNTNAP2* with selective mutism in a family-based study of trios (i.e., child and both parents) (Stein et al., 2011). We found a significant association between several *CNTNAP2* SNPs, and a haplotype containing these SNPs, with selective mutism. To the best of our knowledge, this is the first and only genetic study of selective mutism. In a sample of healthy young adults systematically assessed for their retrospective ratings of BI in childhood, we also found an association between reports of high BI and variation in this same SNP (and in the same direction of effect) of *CNTNAP2*. Given its association with selective mutism and with BI, we believe that *CNTNAP2*, a gene known to be important in language development, should be further studied as a risk factor for SAD and related conditions.

Conclusions and Future Directions

It should be apparent from the research literature summarized here that we still know very little about the specific genetic determinants of SAD. What is clear is that SAD does have a familial and a genetic basis but, like many other mental disorders, it is a complex, heterogenous entity, strongly influenced by environmental factors as well, whose structure is not well characterized by DSM criteria alone. Twin studies emphasize that common genetic factors cut across a range of highly comorbid disorders (e.g., SAD, panic disorder, possibly other phobic disorders), and strongly support an experimental approach that embraces the inclusion of all of these disorders in case–control studies, rather than limiting inclusion to persons with (or without) SAD.

There are other areas of research that may prove fruitful in understanding the genetic bases for SAD and, importantly, the neurobiological consequences of those genetic factors. One such area is the study of individuals with Williams syndrome (WS). Individuals with WS, caused by hemizygous deletion of a portion of 7q11.23, have a behavioral phenotype that is in some ways the converse of social anxiety (Morris, 2010). Such individuals are extremely (and often inappropriately) friendly, outgoing, empathetic, and talkative, to the point that they approach new acquaintances as if

they were longstanding close friends. WS has recently been shown to be associated with differences in insular structure and, in particular, functional connections with other brain loci such as the amygdala (Jabbi et al., 2012) that would suggest that abnormalities in these neural systems should be more closely evaluated in SAD.

The further development of animal models of SAD may point to candidate genes worth studying in humans. A recent example from animal work—though not portrayed by the investigators as a model of SAD—pertains to the finding of chronic social aversion and anxiety in rodents exposed to repeated aggression (Barik et al., 2013). In those mice, it was shown that glucocorticoid receptor activation in dopaminoceptive (but not dopamine-releasing) neurons promoted social aversion, and that this behavior could be reversed by the acute inhibition of dopamine-releasing neurons. Whereas the perils of extrapolating from rodent behavior to human behavior and symptoms is obvious, these kinds of studies can point investigators in new directions with regard to investigating candidate genes, and candidate neural systems, relevant to the understanding of SAD.

GWAS have been extremely valuable in identifying risk genes for other complex traits, including neuropsychiatric traits (Psychiatric GWAS Consortium, 2013), and have come to be viewed as a necessary step in understanding the genetic architecture of such disorders. GWAS, however, require samples larger than those used for most studies of SAD or related traits to date. In addition to facilitating the collection of very large sample sizes (ideally 10,000 and more) by enabling the inclusion of multiple genetically related disorders, this strategy has the considerable advantage of letting the genome tell us about the ideal structure of our diagnostic criteria, rather than the other way around. We hope that funding agencies, seeing the potential for discoveries that can drive novel therapeutics, will see fit to invest in the study of genetic determinants of SAD and related disorders. With that funding, investigators will have the opportunity to conduct adequately powered GWAS and, more importantly, to move into the territory of next-generation sequencing studies, where the likelihood is that rare structural genomic variants will be discovered (Alkan, Coe, & Eichler, 2011; Kiezun et al., 2012) that will completely redefine our understanding of SAD and related conditions.

References

Alkan, C., Coe, B. P., & Eichler, E. E. (2011). Genome structural variation discovery and genotyping. *Nature Reviews. Genetics, 12*(5), 363–376. doi:10.1038/nrg2958

Arbelle, S., Benjamin, J., Golin, M., Kremer, I., Belmaker, R. H., & Ebstein, R. P. (2003). Relation of shyness in grade school children to the genotype for the long form of the serotonin transporter promoter region polymorphism. *American Journal of Psychiatry, 160*(4), 671–676.

Ball, T. M., Sullivan, S., Flagan, T., Hitchcock, C. A., Simmons, A., Paulus, M. P., & Stein, M. B. (2012). Selective effects of social anxiety, anxiety sensitivity, and negative affectivity on the neural bases of emotional face processing. *Neuroimage, 59*(2), 1879–1887.

Barik, J., Marti, F., Morel, C., Fernandez, S. P., Lanteri, C., Godeheu, G., . . . Tronche, F. (2013). Chronic stress triggers social aversion via glucocorticoid receptor in dopaminoceptive neurons. *Science, 339*(6117), 332–335. doi:10.1126/science.1226767

Battaglia, M., Zanoni, A., Taddei, M., Giorda, R., Bertoletti, E., Lampis, V., . . . Tettamanti, M. (2012). Cerebral responses to emotional expressions and the development of social anxiety disorder: A preliminary longitudinal study. *Depression and Anxiety, 29*(1), 54–61. doi:10.1002/da.20896

Bienvenu, O. J., Hettema, J. M., Neale, M. C., Prescott, C. A., & Kendler, K. S. (2007). Low extraversion and high neuroticism as indices of genetic and environmental risk for social phobia, agoraphobia, and animal phobia. *American Journal of Psychiatry, 164*(11), 1714–1721.

Bienvenu, O. J., Nestadt, G., Samuels, J. F., Howard, W. T., Costa, P. T., Jr., & Eaton, W. W. (2001). Phobic, panic, and major depressive disorders and the five-factor model of personality. *Journal of Nervous and Mental Disease, 189*, 154–161.

Bienvenu, O. J., Wuyek, L. A., & Stein, M. B. (2010). Anxiety disorders diagnosis: Some history and controversies. *Current Topics in Behavioral Neurosciences, 2*, 3–19.

Bogels, S. M., Alden, L., Beidel, D. C., Clark, L. A., Pine, D. S., Stein, M. B., & Voncken, M. (2010). Social anxiety disorder: Questions and answers for the DSM-V. *Depression and Anxiety, 27*(2), 168–189. doi:10.1002/da.20670

Buttenschon, H. N., Kristensen, A. S., Buch, H. N., Andersen, J. H., Bonde, J. P., Grynderup, M., & Mors, O. (2011). The norepinephrine transporter gene is a candidate gene for panic disorder. *Journal of Neural Transmission, 118*(6), 969–976. doi:10.1007/s00702-011-0624-7

Carbone, D., Schmidt, L. A., Cunningham, C. C., McHolm, A. E., Edison, S., St, P. J., & Boyle, M. H. (2010). Behavioral and socio-emotional functioning in children with selective mutism: A comparison with anxious and typically developing children across multiple informants. *Journal of Abnormal Child Psychology, 38*(8), 1057–1067. doi:10.1007/s10802-010-9425-y

Cohan, S. L., Price, J. M., & Stein, M. B. (2006). Suffering in silence: Why a developmental psychopathology perspective on selective mutism is needed. *Journal of Developmental and Behavioral Pediatrics, 27*(4), 341–355.

Colhoun, H. M., McKeigue, P. M., & Davey, S. G. (2003). Problems of reporting genetic associations with complex outcomes. *Lancet, 361*(9360), 865–872.

de Moor, M. H., Costa, P. T., Terracciano, A., Krueger, R. F., de Geus, E. J., Toshiko, T., . . . Boomsma, D. I. (2012). Meta-analysis of genome-wide association studies for personality. *Molecular Psychiatry, 17*(3), 337–349. doi:10.1038/mp.2010.128

Domschke, K., Stevens, S., Beck, B., Baffa, A., Hohoff, C., Deckert, J., & Gerlach, A. L. (2009). Blushing propensity in social anxiety disorder: Influence of serotonin transporter gene variation. *Journal of Neural Transmission, 116*(6), 663–666. doi:10.1007/s00702-008-0090-z

Donner, J., Pirkola, S., Silander, K., Kananen, L., Terwilliger, J. D., Lonnqvist, J., . . . Hovatta, I. (2008). An association analysis of murine anxiety genes in humans implicates novel candidate genes for anxiety disorders. *Biological Psychiatry, 64*(8), 672–680. doi:S0006-3223(08)00691-4 pii;10.1016/j.biopsych.2008.06.002

Essex, M. J., Klein, M. H., Slattery, M. J., Goldsmith, H. H., & Kalin, N. H. (2010). Early risk factors and developmental pathways to chronic high inhibition and social anxiety disorder in adolescence. *American Journal of Psychiatry, 167*(1), 40–46. doi:appi.ajp.2009.07010051 pii;10.1176/appi.ajp.2009.07010051

Flint, J. (2004). The genetic basis of neuroticism. *Neuroscience and Biobehavioral Reviews, 28*(3), 307–316.

Fyer, A. J., Costa, R., Haghighi, F., Logue, M. W., Knowles, J. A., Weissman, M. M., . . . Hamilton, S. P. (2012). Linkage analysis of alternative anxiety phenotypes in multiply

affected panic disorder families. *Psychiatric Genetics, 22*(3), 123–129. doi:10.1097/YPG.0b013e328353956a

Gelernter, J., Page, G. P., Stein, M. B., & Woods, S. W. (2004). Genome-wide linkage scan for loci predisposing to social phobia: Evidence for a chromosome 16 risk locus. *American Journal of Psychiatry, 161*(1), 59–66.

Gelernter, J., & Stein, M. B. (2009). Heritability and genetics of anxiety disorders. In M. M. Antony & M. B. Stein (Eds.), *Handbook of anxiety disorders* (pp. 87–96). New York, NY: Oxford University Press.

Hamilton, S. P. (2009). Linkage and association studies of anxiety disorders. *Depression and Anxiety, 26*(11), 976–983. doi:10.1002/da.20615

Hettema, J. M., Neale, M. C., Myers, J. M., Prescott, C. A., & Kendler, K. S. (2006). A population-based twin study of the relationship between neuroticism and internalizing disorders. *American Journal of Psychiatry, 163*(5), 857–864.

Hirshfeld-Becker, D. R., Micco, J., Henin, A., Bloomfield, A., Biederman, J., & Rosenbaum, J. F. (2008). Behavioral inhibition. *Depression and Anxiety, 25*, 357–367.

Hovatta, I., Tennant, R. S., Helton, R., Marr, R. A., Singer, O., Redwine, J. M., . . . Barlow, C. (2005). Glyoxalase 1 and glutathione reductase 1 regulate anxiety in mice. *Nature, 438*(7068), 662–666.

Jabbi, M., Kippenhan, J. S., Kohn, P., Marenco, S., Mervis, C. B., Morris, C. A., . . . Berman, K. F. (2012). The Williams syndrome chromosome 7q11.23 hemideletion confers hypersocial, anxious personality coupled with altered insula structure and function. *Proceedings of the National Academy of Sciences of the United States of America, 109*(14), E860–E866. doi:10.1073/pnas.1114774109

Kaabi, B., Gelernter, J., Woods, S. W., Goddard, A., Page, G. P., & Elston, R. C. (2006). Genome scan for loci predisposing to anxiety disorders using a novel multivariate approach: Strong evidence for a chromosome 4 risk locus. *American Journal of Human Genetics, 78*(4), 543–553.

Karg, K., Burmeister, M., Shedden, K., & Sen, S. (2011). The serotonin transporter promoter variant (5-HTTLPR), stress, and depression meta-analysis revisited: Evidence of genetic moderation. *Archives of General Psychiatry, 68*(5), 444–454. doi:archgenpsychiatry.2010.189 pii;10.1001/archgenpsychiatry.2010.189

Kendler, K. S., Myers, J., Prescott, C. A., & Neale, M. C. (2001). The genetic epidemiology of irrational fears and phobias in men. *Archives of General Psychiatry, 58*, 257–265.

Kennedy, J. L., Neves-Pereira, M., King, N., Lizak, M. V., Basile, V. S., Chartier, M. J., & Stein, M. B. (2001). Dopamine system genes not linked to social phobia. *Psychiatric Genetics, 11*(4), 213–217.

Kiezun, A., Garimella, K., Do, R., Stitziel, N. O., Neale, B. M., McLaren, P. J., . . . Sunyaev, S. R. (2012). Exome sequencing and the genetic basis of complex traits. *Nature Genetics, 44*(6), 623–630. doi:10.1038/ng.2303

Kotov, R., Gamez, W., Schmidt, F., & Watson, D. (2010). Linking "big" personality traits to anxiety, depressive, and substance use disorders: A meta-analysis. *Psychological Bulletin, 136*(5), 768–821. doi:10.1037/a0020327

Kuusikko, S., Pollock-Wurman, R., Jussila, K., Carter, A. S., Mattila, M. L., Ebeling, H., . . . Moilanen, I. (2008). Social anxiety in high-functioning children and adolescents with Autism and Asperger syndrome. *Journal of Autism and Developmental Disorders, 38*(9), 1697–1709.

Libert, S., Pointer, K., Bell, E. L., Das, A., Cohen, D. E., Asara, J. M., . . . Guarente, L. (2011). SIRT1 activates MAO-A in the brain to mediate anxiety and exploratory drive. *Cell, 147*(7), 1459–1472. doi:10.1016/j.cell.2011.10.054

Logue, M. W., Bauver, S. R., Knowles, J. A., Gameroff, M. J., Weissman, M. M., Crowe, R. R., . . . Hamilton, S. P. (2012). Multivariate analysis of anxiety disorders yields further evidence of linkage to chromosomes 4q21 and 7p in panic disorder families. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 159B*(3), 274–280. doi:10.1002/ajmg.b.32024

Manolio, T. A., Collins, F. S., Cox, N. J., Goldstein, D. B., Hindorff, L. A., Hunter, D. J., . . . Visscher, P. M. (2009). Finding the missing heritability of complex diseases. *Nature, 461*(7265), 747–753. doi:10.1038/nature08494

Maron, E., Hettema, J. M., & Shlik, J. (2010). Advances in molecular genetics of panic disorder. *Molecular Psychiatry, 15*(7), 681–701. doi:10.1038/mp.2009.145

Morris, C. A. (2010). The behavioral phenotype of Williams syndrome: A recognizable pattern of neurodevelopment. *American Journal of Medical Genetics. Part C, Seminars in Medical Genetics, 154C*(4), 427–431. doi:10.1002/ajmg.c.30286

Mosing, M. A., Gordon, S. D., Medland, S. E., Statham, D. J., Nelson, E. C., Heath, A. C., . . . Wray, N. R. (2009). Genetic and environmental influences on the co-morbidity between depression, panic disorder, agoraphobia, and social phobia: A twin study. *Depression and Anxiety, 26*(11), 1004–1011. doi:10.1002/da.20611

Phan, K. L., Orlichenko, A., Boyd, E., Angstadt, M., Coccaro, E. F., Liberzon, I., & Arfanakis, K. (2009). Preliminary evidence of white matter abnormality in the uncinate fasciculus in generalized social anxiety disorder. *Biological Psychiatry, 66*(7), 691–694. doi:S0006-3223(09)00295-9 pii;10.1016/j.biopsych.2009.02.028

Psychiatric GWAS Consortium. (2013). Identification of risk loci with shared effects on five major psychiatric disorders: A genome-wide analysis. *Lancet, 381*, 1371–1379. doi:10.1016/S0140-6736(12)62129-1

Risch, N., Herrell, R., Lehner, T., Liang, K. Y., Eaves, L., Hoh, J., . . . Merikangas, K. R. (2009). Interaction between the serotonin transporter gene (5-HTTLPR), stressful life events, and risk of depression: A meta-analysis. *Journal of the American Medical Association, 301*(23), 2462–2471. doi:301/23/2462 pii;10.1001/jama.2009.878

Schumacher, J., Kristensen, A. S., Wendland, J. R., Nothen, M. M., Mors, O., & McMahon, F. J. (2011). The genetics of panic disorder. *Journal of Medical Genetics, 48*(6), 361–368. doi:10.1136/jmg.2010.086876

Schwartz, C. E., Wright, C. I., Shin, L. M., Kagan, J., & Rauch, S. L. (2003). Inhibited and uninhibited infants "Grown Up": Adult amygdalar response to novelty. *Science, 300*, 1952–1953.

Shifman, S., Bhomra, A., Smiley, S., Wray, N. R., James, M. R., Martin, N. G., . . . Flint, J. (2008). A whole genome association study of neuroticism using DNA pooling. *Molecular Psychiatry, 13*(3), 302–312.

Sipila, T., Kananen, L., Greco, D., Donner, J., Silander, K., Terwilliger, J. D., . . . Hovatta, I. (2010). An association analysis of circadian genes in anxiety disorders. *Biological Psychiatry, 67*(12), 1163–1170. doi:S0006-3223(09)01448-6 pii;10.1016/j.biopsych.2009.12.011 doi

Smith, A. K., Rhee, S. H., Corley, R. P., Friedman, N. P., Hewitt, J. K., & Robinson, J. L. (2012). The magnitude of genetic and environmental influences on parental and observational measures of behavioral inhibition and shyness in toddlerhood. *Behavioral Genetics, 42*(5), 764–777. doi:10.1007/s10519-012-9551-0

Smoller, J. W., Block, S. R., & Young, M. M. (2009). Genetics of anxiety disorders: The complex road from DSM to DNA. *Depression and Anxiety, 26*(11), 965–975. doi:10.1002/da.20623

Smoller, J. W., Paulus, M. P., Fagerness, J. A., Purcell, S., Yamaki, L. H., Hirshfeld-Becker, D., . . . Stein, M. B. (2008). Influence of RGS2 on anxiety-related temperament, personality, and brain function. *Archives of General Psychiatry, 65*(3), 298–308.

Smoller, J. W., Rosenbaum, J. F., Biederman, J., Kennedy, J., Dai, D., Racette, S. R., . . . Slaugenhaupt, S. A. (2003). Association of a genetic marker at the corticotropin-releasing hormone locus with behavioral inhibition. *Biological Psychiatry, 54*(12), 1376–1381.

Smoller, J. W., Rosenbaum, J. F., Biederman, J., Susswein, L. S., Kennedy, J., Kagan, J., . . . Slaugenhaupt, S. A. (2001). Genetic association analysis of behavioral inhibition using candidate loci from mouse models. *American Journal of Medical Genetics (Neuropsychiatric Genetics), 105*, 226–235.

Stein, M. B., & Bienvenu, O. J. (2004). Diagnostic classification of anxiety disorders: DSM-V and beyond. In D. S. Charney & E. J. Nestler (Eds.), *The neurobiology of mental illness* (2nd ed., Vol. *2*, pp. 525–534). New York, NY: Oxford University Press.

Stein, M. B., Chartier, M. J., Hazen, A. L., Kozak, M. V., Tancer, M. E., Lander, S., . . . Walker, J. R. (1998). A direct-interview family study of generalized social phobia. *American Journal of Psychiatry, 155*(1), 90–97.

Stein, M. B., Chartier, M. J., Kozak, M. V., Hazen, A. L., King, N., & Kennedy, J. L. (1998). Genetic linkage to the serotonin transporter and 5HT2A receptor excluded in generalized social phobia. *Psychiatry Research, 81*, 283–291.

Stein, M. B., Chartier, M. J., Lizak, M. V., & Jang, K. L. (2001). Familial aggregation of anxiety-related quantitative traits in generalized social phobia: Clues to understanding "disorder" heritability? *American Journal of Medical Genetics (Neuropsychiatric Genetics), 105*, 79–83.

Stein, M. B., & Gelernter, J. (2010). Genetic basis of social anxiety disorder. In S. G. Hofmann & P. M. Di Bartolo (Eds.), *Social anxiety: Clinical, developmental, and social perspectives*, (2nd ed., pp. 313–319). San Diego, CA: Academic Press.

Stein, M. B., Goldin, P. R., Sareen, J., Zorrilla, L. T., & Brown, G. G. (2002). Increased amygdala activation to angry and contemptuous faces in generalized social phobia. *Archives of General Psychiatry, 59*(11), 1027–1034.

Stein, M. B., Jang, K. L., & Livesley, W. J. (2002). Heritability of social-anxiety related concerns and personality characteristics: A twin study. *Journal of Nervous and Mental Disease, 190*(4), 219–224.

Stein, M. B., & Stein, D. J. (2008). Social anxiety disorder. *Lancet, 371*(9618), 1115–1125.

Stein, M. B., Yang, B. Z., Chavira, D. A., Hitchcock, C. A., Sung, S. C., Shipon-Blum, E., & Gelernter, J. (2011). A common genetic variant in the neurexin superfamily member CNTNAP2 is associated with increased risk for selective mutism and social anxiety-related traits. *Biological Psychiatry, 69*(9), 825–831. doi:10.1016/j.biopsych.2010.11.008

Strachan, T. R. A. (2010). *Human molecular genetics* (4th ed.). Taylor & Francis.

Terracciano, A., Sanna, S., Uda, M., Deiana, B., Usala, G., Busonero, F., . . . Costa, P. T., Jr. (2010). Genome-wide association scan for five major dimensions of personality. *Molecular Psychiatry, 15*(6), 647–656. doi:mp2008113 pii;10.1038/mp.2008.113

Terracciano, A., Tanaka, T., Sutin, A. R., Deiana, B., Balaci, L., Sanna, S., . . . Costa, P. T., Jr. (2010). BDNF Val66Met is associated with introversion and interacts with 5-HTTLPR to influence neuroticism. *Neuropsychopharmacology, 35*(5), 1083–1089. doi:npp2009213 pii;10.1038/npp.2009.213

van den Oord, E. J., Kuo, P. H., Hartmann, A. M., Webb, B. T., Moller, H. J., Hettema, J. M., . . . Rujescu, D. (2008). Genomewide association analysis followed by a replication

study implicates a novel candidate gene for neuroticism. *Archives of General Psychiatry, 65*(9), 1062–1071.

Warren, S. L., Schmitz, S., & Emde, R. N. (1999). Behavioral genetic analyses of self-reported anxiety at 7 years of age. *Journal of the American Academy of Child and Adolescent Psychiatry, 38*(11), 1403–1408.

Weeks, J. W., Heimberg, R. G., Fresco, D. M., Hart, T. A., Turk, C. L., Schneier, F. R., & Liebowitz, M. R. (2005). Empirical validation and psychometric evaluation of the Brief Fear of Negative Evaluation Scale in patients with social anxiety disorder. *Psychological Assessment, 17*(2), 179–190.

White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review, 29*(3), 216–229. doi:S0272-7358(09)00004-X pii;10.1016/j.cpr.2009.01.003

4

The Social Neuroscience of Social Anxiety Disorder

Supriya Syal[1,2] and Dan J. Stein[1]

[1] *University of Cape Town, South Africa*
[2] *University of Toronto, Canada*

I love mankind—it's people I can't stand.
—Charles M. Schulz, *Go Fly a Kite, Charlie Brown*

Introduction

Humans are extraordinarily social. Social behavior permeates all facets of human life and provides the context for most forms of human learning. From birth, human infants attend to social cues and seek out social interaction (Moll & Tomasello, 2007), behaviors that persist for the course of human lives. Social and cultural learning (i.e., our ability to interact with and learn from others of our own species) are widely appreciated as key contributors to the evolution of human intelligence (van Schaik & Burkart, 2011) and an influential theory of human evolution posits that the human brain evolved to facilitate social living (Dunbar, 1998). Recent evidence from non-human primates indicates that living in larger, more complex social groups changes structural and functional coupling across diverse neural circuits in the primate brain (Sallet et al., 2011), and social network size positively predicts differences in brain structure in humans as well (Bickart, Wright, Dautoff, Dickerson, & Barrett, 2011). Across species, social living is adaptive at many levels, directly impacting reproductive fitness (Silk, 2007), resistance to disease (Cohen, Doyle, Skoner, Rabin, & Gwaltney, 1997), decreased predation risk (Sterck, Watts, & van Schaik, 1997), and length of lifespan (Berkman, 1995; Ruan & Wu, 2008). Conversely, social isolation is a major risk factor for morbidity and mortality (House, Landis, & Umberson, 1988; Thomas, 2012), increased lifetime stress, and decreased lifetime health outcomes (Cacioppo, Hawkley, Norman, & Berntson, 2011). In short, humans seek out social interaction, and social interaction is good for us.

And yet, in the face of all of this evidence for human prosociality, social anxiety disorder (SAD) instills an abiding fear of social interaction in the people it afflicts.

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

People with SAD feel an intense and irrational fear of social situations, which may lead to avoidance of social situations altogether (Stein & Stein, 2008). In agreement with prior behavioral and theoretical work, neuroimaging data indicate that people with SAD feel a heightened fear of social disapproval (Guyer et al., 2008) and have an enhanced fear response to social stimuli such as faces, even when those stimuli contain no aversive content (Birbaumer et al., 1998). Conversely, people with high social anxiety also display an absence of (the normal) positive bias when viewing expressive faces (Moser, Huppert, Duval, & Simons, 2008), and report being less expressive of positive emotions in interpersonal relationships (Turk, Heimberg, & Luterek, 2005). Thus, it appears that individuals with SAD are predisposed to detecting threat rather than reward in social environments, even when no actual threat exists, but instead putative reward (derived through social interaction) does in fact exist. Considered through the lens of social neuroscience then, SAD can be conceptualized as an aberration of social behavior, and by proxy, the social brain.

Conceiving of social anxiety as a disorder of the social brain immediately raises the question of both the proximal and distal (evolutionary) mechanisms underlying the etiology of SAD. Proximal mechanisms may include environmental stressors such as early attachments issues, childhood trauma, or early averse social experiences that could lead to dysfunctional social patterning during development (Brumariu & Kerns, 2008; Kuo, Goldin, Werner, Heimberg, & Gross, 2011). Evolutionary perspectives have suggested that social anxiety results from: (a) aberrant activations of basic defense mechanisms for threat detection; and (b) aberrant displays of overtly submissive roles in social hierarchies, aimed at gathering approval from others, and based in exaggerated negative perceptions of the self that serve to maintain and perpetuate these submissive tendencies (Gilbert, 2001; see also **Chapter 2**)—that is, dysfunction of self-protective social regulation systems. Since these ideas remain embedded in the assumption that SAD is an aberration of systems that evolved to promote social living, they emphasize the importance of studying social anxiety from the perspective of integrative approaches to social behavior—or put differently, from the emerging field of social neuroscience.

The Scope of this Essay

In the first part of this chapter, we will review key functional magnetic resonance imaging (fMRI) and behavioral research findings on social brain function in SAD. This chapter will not review the corpus of non-fMRI work in SAD (e.g., positron emission tomography, electroencephelography, structural MRI, etc.; please see Freitas-Ferrari et al., 2010; Miskovic & Schmidt, 2012; Staugaard, 2010, for reviews). Given the ubiquity of human faces in human social interaction, a large part of currently available research data on SAD has addressed socioemotional face processing, with additional work targeting social stressors such as public-speaking, with a few notable exceptions (discussed below). However, because of the fundamentally social structure of the human ecological niche, a disorder of the social brain is likely to impinge upon numerous areas of human social behavior. The second section of this essay will both review recent social anxiety research that has explored additional dimensions of social

function, as well as emphasize research questions about SAD that have remained unanswered thus far. We hope to encourage researchers to draw from findings in social neuroscience that are pertinent to social anxiety, and use them to study the many facets of human social behavior that might be affected in SAD.

Part 1: Current Research on Social Neuroscience of SAD

Brain Imaging of Socioemotional Processing in SAD

As mentioned earlier, since social interaction typically involves face-to-face interactions with social others, studies of social affect and cognition involving face perception and processing have abounded in the study of SAD. A second line of research has focused on social provocation paradigms that involve public-speaking tasks. These have been used to study SAD because speaking in social contexts forms a behavior that is typically avoided by SAD patients. Data from these and other paradigms have highlighted aberrations in a network of interconnected regions involved in social-affective processing in SAD.

Amygdala Following up on behavioral work that reported a bias toward interpreting neutral faces as negative (Winton, Clark, & Edelmann, 1995), early functional neuroimaging studies found enhanced amygdala activation in response to neutral (nonaversive) faces in individuals with SAD (Birbaumer et al., 1998). Similarly, early behavioral work noted a memory bias for critical faces (Lundh & Ost, 1996), and subsequent neuroimaging work found increased amygdala activity in response to angry and contemptuous faces in individuals with SAD relative to control participants (Stein, Goldin, Sareen, Zorrilla, & Brown, 2002). Exaggerated amygdala response to negative facial affect has since been corroborated a number of times in SAD (Phan, Fitzgerald, & Nathan, 2006; Straube, Kolassa, Glauer, Mentzel, & Miltner, 2004), including a study that examined the processing of schematic faces (i.e., line drawings) (Evans, Wright, & Wedig, 2008). Moreover, the magnitude of amygdala activation to threatening faces is positively related to the severity of SAD (Phan et al., 2006), and successful treatment of SAD is associated with decreased blood flow to the amygdala during the performance of a public-speaking task (Furmark et al., 2002). Taken together, this body of work highlights the central role of the amygdala in the pathophysiology of SAD, in keeping with our understanding of the amygdala as a crucial component of neural networks involved in both threat detection and socioemotional processing.

Insula Also known as the "interoceptive cortex," the human insula integrates homeostatic as well as emotional information to create a mental image of one's physical state. The insula remains intimately linked to the processing of both physical and emotional pain in humans. Increased insular activation has been noted in SAD in a number of studies during the processing of multiple classes of social stimuli including both aversive faces (Amir et al., 2005; Etkin & Wager, 2007; Straube et al., 2004) and social transgressions (Blair et al., 2010). Insula activation in patients in

response to phobia-related linguistic cues has also been related to symptom severity of SAD (Schmidt, Mohr, Miltner, & Straube, 2010), and patients display improved symptoms and decreased regional blood flow to the insula after selective serotonin reuptake inhibitor (SSRI) treatment (Warwick, Carey, & Van der Linden, 2006). In humans, the insula in turn relays interoceptive information to the anterior cingulate cortex, which can then modulate the allocation of attentional resources (Weissman, Gopalakrishnan, & Hazlett, 2005). In addition to increased insula activation, individuals with SAD also show increased anterior cingulate activation (ACC) during the processing of disgusted faces (Amir et al., 2005) and decreased anterior insula–dorsal ACC coupling during the processing of fear relative to happy faces (Klumpp, Angstadt, & Phan, 2012). These findings tie in well with Paulus and Stein's (2006) *insular view of anxiety* whereby individuals who are prone to anxiety display enhanced detection of the difference between observed and expected physiological body states, which leads into anxious and avoidant behaviors.

Striatum Early single-photon emission computed tomography (SPECT) data indicated that SAD patients might display reduced dopaminergic function in the striatum (Schneier & Liebowitz, 2000; Tiihonen et al., 1997), consistent with animal models of SAD (Grant et al., 1998). To our knowledge, only one study has specifically probed striatal activity using fMRI in SAD (Sareen et al., 2007). This study recorded decreased activation of the caudate head (dorsal striatum) as well as the inferior parietal lobule in SAD patients, in the absence of any behavioral difference between the patient and control groups. The caudate is involved in the perception of reward in relation to action contingency which is a common feature of normative social interactions (Tricomi, Delgado, & Fiez, 2004; Wilms et al., 2010). These data, in conjunction with reduced dopaminergic activity in the striatum, as noted above, lend support to the idea that SAD patients might not associate reward with social interactions in the manner that healthy individuals do. In support of this, Beaton et al. (2008) reported that sociable individuals exhibit greater activation of the nucleus accumbens (primary reward region) in response to strange and familiar faces than shy adults. Indeed, in shy adults, early behavioral inhibition predicts subsequent greater amygdala responses to strange versus familiar faces (Schwartz, Wright, Shin, Kagan, & Rauch, 2003).

Fusiform cortex The fusiform cortex (FFC) is part of the social brain network that attends to biologically relevant information such as faces. Since the vast majority of studies of SAD have used human faces as social stimuli, studying activation of the FFC in SAD becomes of particular interest. Notably, both increased (Etkin & Wager, 2007; Mueller et al., 2008) and decreased (Gentili et al., 2008) activity in the FFC in response to emotional faces has been reported in SAD. Socially anxious (shy) adults display decreased activation of the fusiform face area during processing of strangers' neutral faces, but greater activation during processing of personally familiar faces, as compared with social adult controls. Notably, this decrease in FFA activation in response to strangers is paired with increased right amygdala activation in shy adults as compared with social adults (Beaton et al., 2009). Another study has noted that activation of the fusiform gyrus also influences the amygdala response to emotional faces in individuals with subclinically elevated social anxiety (Pujol et al., 2009). Decreased

fusiform activation might indicate SAD subjects' averting gaze or attention from anxiety-provoking social stimuli. Hypoactivation of the fusiform is also known to mark other putative disorders of social affect such as Asperger syndrome (Deeley et al., 2007) and other autism spectrum disorders (Critchley, 2000; Schultz et al., 2003). In healthy humans, in addition to face processing, fusiform activation has also been linked to chemosensory anxiety signals (Prehn-Kristensen et al., 2009) and increased trait anxiety (Denkova et al., 2010).

Prefrontal cortex Recent models of emotion regulation have emphasized the role of prefrontal cortex (PFC) and cingulate cortex in facilitating cognitive control of emotional responses, either through controlling attention to emotional stimuli or through top-down modulation of the meaning associated with those stimuli (Ochsner & Gross, 2005). In addition to hyper-reactivity of the ACC to aversive faces, people with SAD display enhanced activity of several regions of frontal cortex (Brodmann's area 10, 46, and 38) in response to fearful—relative to neutral—faces (Blair, Shaywitz, et al., 2008), and increased activation of PFC (medial (mPFC) and ventrolateral) in response to harsh faces (Stein et al., 2002). SAD patients also show increased activation in the mPFC (and amygdala) in response to negative social self-referential appraisals (Blair, Geraci, et al., 2008) and unintentional social transgressions (Blair et al., 2010). Research with healthy individuals has indicated that the mPFC is involved in representations of both self and others' mental states (Mitchell, Macrae, & Banaji, 2006). It is possible that aberrant activity in these regions in SAD, in concert with heightened amygdala activity, could reflect the maintenance of negative self-evaluation and/or the failure of cognitive reappraisal mechanisms that help healthy people regulate their emotions in response to (real or imagined) negative social affect more successfully.

In support of this, in a recent study of cognitive reappraisal of emotion in SAD, Goldin, Manber-Ball, Werner, Heimberg, and Gross (2009) found that while both SAD patients and control subjects displayed significant reduction of initial amygdala response during cognitive reappraisal (wherein participants are instructed to regulate their emotional reactivity), patients and controls were different in the temporal onset of cognitive control responses. Control participants had early activation of cognitive control, visual and language processing while SAD patients displayed later cognitive control, visual processing, and visceral brain responses during reappraisal of negative self-beliefs (Goldin, Manber-Ball, et al., 2009). These data also suggest that while patients with SAD are able to engage in cognitive reappraisal, it is a more effortful task for them as they require additional time to overcome the initial anxiety induced by negative self-beliefs.

Conversely, data from two studies of SAD seem to indicate the opposite effect for PFC activation. One of these used a public-speaking/social provocation task and reported decreased cortical (dorsal anterior cingulate/PFC) activity in SAD patients (Lorberbaum et al., 2004), while the other reported diminished mPFC activity during the performance of a sociocognitive task that involved attributing mental states to others (Sripada et al., 2009). However, the Lorberbaum et al. study noted significantly increased anxiety at rest (that could potentially preclude any further increase) in SAD patients. And, the Sripada et al. study used a *neuroeconomic trust game* design wherein the social face stimuli were obscured by colored ovals in order to obviate neural

responses to face. Thus, these task design constraints might help explain the disparity of findings with reference to increased activation of the mPFC noted in the SAD studies described above. An additional interpretation of the increased PFC activity noted in SAD is that it serves a compensatory function aimed at modulating hyperactivity of the amygdala. This brings up the possibility that different patients may display individual differences in the amount of compensatory activity they display—and decreased PFC activity might be an indication of the reduced regulation of the amygdala in SAD.

Additional areas of interest in SAD A number of studies in SAD have also noted deactivation of the visual cortices during public-speaking (Bell, Malizia, & Nutt 1999; Tillfors, 2001; Van Ameringen et al., 2004), leading to the suggestion that people with SAD deflect visual attention away from anxiety-provoking (social) stimuli under these task conditions. Indeed, 6 weeks of treatment with a NK1-agonist, which is associated with symptom improvement and decreased blood flow to the medial temporal lobe, also leads to significantly increased blood flow in the occipital cortex in SAD patients during a public-speaking task, which could potentially be related to improved visual attention/lesser visual avoidance following treatment (Furmark et al., 2005).

Some work has suggested that the orbitofrontal cortex (OFC) is also involved in the processing of social cues such as aversive prosody (Quadflieg, Mohr, Mentzel, Miltner, & Straube, 2008) and the processing of verbal threat-related stimuli (Schmidt et al., 2010), as well as aversive delay conditioning (Veit et al., 2002) in SAD. Additional regions of aberrant activity include the dorsolateral prefrontal cortex (DLPFC), the temporal poles, and the intraparietal sulcus (Etkin & Wager, 2007; Gentili et al., 2008).

Connectivity analyses Based on functional data that hint at impaired cross-brain network dynamics, recent work is starting to address spontaneous brain activity in SAD. Reduced resting-state functional connectivity between the amygdala and the medial OFC (Hahn et al., 2011), as well as compromised integrity of the white matter tracts that connect the amygdala with the medial OFC in patients with SAD (Phan et al., 2009), has been reported. Additionally, a recent study using temporal precedence cues to infer directed interactions also found decreased regulatory influences from several neocortical regions to the amygdala, and increased directed influence from the amygdala to the visual cortices during relaxed wakefulness (Liao et al., 2010).

Physiological and Behavioral Data

Although behavioral studies suggest that people with SAD display perseveration of emotional responses to social stimuli, possibly mediated by lower task-related and greater self-related attention, specific tests of visual attention biases towards social threat stimuli (e.g., see Bögels & Mansell, 2004; Staugaard, 2010) have had mixed results. While they highlight attentional allocation biases to social threat, these range along both vigilance and avoidance dimensions. On the one hand, it seems reasonable that people with social anxiety should avoid anxiety-causing stimulation, which leads to their not reappraising the negative expectations they hold about others' reactions

to them (Clark & Wells, 1995). Contrariwise, SAD might in fact potentiate attention to, and difficulty in disengaging from, threatening social stimuli, so as to evaluate the safety of any given environment (Rapee & Heimberg, 1997). Cognitive-affective information processing models combine these two approaches to propose a vigilance-avoidance pattern of reactivity in SAD that involves automatic allocation of attention toward potential threat, followed by inhibition and avoidance of the threat signals (Mogg & Bradley, 1998).

Similarly, prolonged autonomic responses (e.g., changes in heart rate or skin conductance) are also not consistent (see **Chapter 5** for a review), even though subjective measures indicate that SAD individuals feel more anxious and perceive greater physiological reactivity in themselves in comparison to controls. It is possible that increased self-focused attention causes SAD individuals to remain more aware of their physiological states, causing them to report greater physiological arousal than their nonanxious counterparts. Interpreted in the light of the original James–Lange theory of emotion (James, 1894; Lange, 1885), these data indicate that the perception of emotions in individuals with SAD (as well as control subjects) involves subjective interpretations of perceived physiological and emotional states, and remains accessible to top-down modulation, which may be impaired in SAD.

From an evolutionary perspective, the phenomenon of SAD raises the question of how best to optimize responses to social threat; too much attention leads to too many false alarms (Nesse, 2001) and related loss of resources, while too little attention may lead to excessive exposure to threat. A social brain perspective emphasizes the value of carefully measuring both the magnitude and the time course of: the avoidance of, attention to, and disengagement from social stimuli in order to fully understand the underlying proximal mechanisms.

Summary

In summary, individuals with elevated social anxiety (patients or subclinical subjects) show atypical activation of brain regions involved in social and emotional information processing. Data from both brain activation and behavioral paradigms provide some support for early-vigilant–late-avoidant responses in the production and maintenance of disordered social anxiety states. Enhanced amygdala response to socioemotional stimuli remains a hallmark characteristic of SAD and might correspond with initial vigilance toward threat. The evaluation of internal feeling states does not depend on the amygdala (Anderson & Phelps, 2002), but instead recruits higher-level cortical regions such as the insula, ACC, and mPFC. Given that the latter regions display aberrant activation in SAD as reviewed above, it is likely that these represent neural sites that mediate the perseveration of emotional responses that have been noted in SAD. Aberrant cortical activity noted in SAD in social performance tasks might reflect impairment of cognitive modulation and reappraisal of emotion by the PFC and ACC, and subsequent avoidance of threat signals via deactivation of visual cortices in individuals with SAD.

Increased self-directed attention and rumination in individuals with SAD might indicate enhanced attunement to physiological states in interoceptive and limbic

cortices, leading to increased feelings of subjective anxiety. Moreover, reduced dopaminergic function in the striatum and aberrant functioning of the OFC might lead to impairments in instrumental learning of the subjective reward value of social stimuli in SAD.

Taken together, these data indicate that aberrant socioemotional processing and attentional allocation in SAD are associated with network-wide deficits in prosocial behavior, and underscore the need to use research on social behavior to inform the study of social anxiety as an aberration of the social brain. Next, we will focus our attention on how social neuroscientific approaches to the study of social behavior can inform the study of social anxiety.

Part 2: Social Neuroscientific Directions for Future Research in SAD

The field of social neuroscience takes a multilevel integrative approach to the study of human behavior. In essence, social neuroscience is the study of interconnectedness of cultural and biological factors that mediate social living. It considers the effects of social context on nervous, endocrine, and immune systems; underlying genetic constraints or predispositions; and the control that these exert on social behavior in humans at both proximal and ultimate levels of analyses. Therefore, social neuroscientific approaches involve studying social behavior at multiple levels of organization (e.g., social stimuli that cause social anxiety also activate neuroendocrine stress responses that can have immune consequences); and consider interactions between systems that exist within humans (e.g., endocrine), and facets of the social world that humans live in (other humans!). Based in the assumption of continuity of evolutionary adaptations for social living, social neuroscientific approaches draw from translational findings across species in the service of a more nuanced appreciation of the intricate systems that create and sustain social life. The field of social neuroscience (and neuroscience in general) seeks to draw from such comparative approaches in order to better understand human social behavior within the context of human social evolution.

Presented below are some streams of research into social behavior that have been investigated in social neuroscience, and which provide promising avenues for future research in SAD.

Genetics of SAD

The heritability estimate for SAD is around 51% (Kendler, Karkowski, & Prescott, 1999). Variations in genes encoding components of serotonergic neurotransmission (5-HT) appear to display a relationship with SAD (Furmark, 2009; Furmark et al., 2008; Lochner et al., 2007; see also **Chapter 3** for expanded review of genetic factors in SAD). In the context of social neuroscientific findings, we wish to make the case for studying allelic variations in oxytocin and vasopressin receptor genes in individuals with SAD. Recent work has illustrated the role of two peptide neurohormones, oxytocin (OT) and arginine vasopressin (AVP), in regulating the response to social stressors in healthy humans (Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003; Thompson,

Gupta, Miller, Mills, & Orr, 2004), making them of great interest to the study of SAD. OT appears to modulate social cognition in a manner that promotes positive sociality, while centrally active AVP seems to be associated with increased vigilance and anxiety (Bos, Panksepp, & Bluthe, 2012). A comprehensive review of these and other findings is provided in **Chapter 21** on the neuroendocrinology of SAD—presented here is the argument for studying the genetics of the OT and vasopressin receptor genes in individuals with SAD.

Although further work is required to determine the relationship between single nucleotide polymorphisms (SNP) in the oxytocin receptor gene (OXTR1) and social behavior, at least 5 OXTR1 SNPs (of a total of 18) appear to be involved in social behavior, or are related to the risk for developing pathologies of social affect (Israel et al., 2009; Jacob et al., 2007; Lerer et al., 2007; Liu et al., 2010; Park et al., 2010; Rodrigues & Saslow, 2009; Thompson, Parker, Hallmayer, Waugh, & Gotlib, 2011; Tost et al., 2010; Wu et al., 2005; Yrigollen et al., 2008). Similarly, several studies have shown an association between variations of the arginine vasopressin receptor gene (AVPR1A) and other disorders of social affect, such as autism spectrum disorders (Kim et al., 2002; Wassink et al., 2004; Yirmiya et al., 2006). Notably, polymorphisms of AVPR1a have also been associated with human amygdala function (Meyer-Lindenberg et al., 2009), and microsatellites (repeat elements in the promoter region of a gene) of the AVPR1a have been linked to social behavior across species. Expansions of a complex microsatellite in a regulatory region of the vasopressin receptor encoding gene predict sociobehavioral traits in voles (Hammock & Young, 2005); furthermore, AVPR1a microsatellites are linked to social behavior regulation in humans also (Bachner-Melman et al., 2005). Both of these lines of data indicate that genetic profiling of OXTR1 and AVPR1a in individuals with SAD could serve to provide many exciting new avenues for research into the interplay between neurohormones and social behavior in SAD.

Immune Function in SAD

Multilevel integrative approaches in social neuroscience call for the examination of a given phenomenon employing all pertinent schools of study that could help to build a holistic understanding of underlying mechanisms. An example of such an approach is the study of how social environments exert far-reaching effects on health outcomes through modulation of the immune response. Adverse social interactions or social isolation (discussed below) can alter immune function through their effects on neuroendocrine activity and related gene expression, but this has received little attention in SAD research. Social isolation alters hypothalamic–pituitary–adrenocortical activity (Adam, Hawkley, Kudielka, & Cacioppo, 2006), and alters gene transcription specifically toward under-expression of genes involved in anti-inflammatory and over-expression of genes involved in pro-inflammatory, response elements (Bierhaus et al., 2003; Cole, Hawkley, Arevalo, & Cacioppo, 2011); these, in turn, lead to increased disease risk. Similarly, social conflict, interpersonal stress, and lack of social support have been associated with higher levels of biomarkers for stress (Miller, Rohleder, & Cole, 2009) that are associated with negative health outcomes such as cardiovascular disease, type II diabetes, arthritis, osteoporosis, and Alzheimer's disease (Ershler

& Keller, 2000; Kiecolt-Glaser, Gouin, & Hantsoo, 2010). Increased stress responsivity during social situations remains a hallmark of SAD and, in addition to the pro-inflammatory consequences noted above, psychosocial stress is associated with a host of neural aberrations, including aberrations in morphology as well as activation of emotion-processing regions such as the amygdala and hippocampus (Tottenham & Sheridan, 2009). These strong ties between social stress and the risk for disease underscore the need to study immune function in individuals with SAD. Based on the data on social isolation, one would predict a pro-inflammatory immune profile in individuals with severe social anxiety, highlighting the systemic nature of this disorder, as well as providing immediate therapeutic targets.

Social Isolation in Social Anxiety

A domain of social anxiety that has remained understudied is (hypothetical) increasing social isolation with increasing duration of the disorder. Increased avoidance of social situations in SAD likely leads to increasing social isolation in afflicted individuals. Research indicates that perceived social isolation is more closely related to the quality than the quantity of social interactions, which are experienced less positively by lonely individuals (Hawkley et al., 2008). Thus, it could be that dissatisfaction from even the decreased social interactions that SAD individuals do have serves to further a feeling of social isolation. Reflecting similarities to increased perceived anxiety in SAD patients compared with controls even in the absence of objective differences in arousal, lonely individuals report higher levels of perceived stress than nonlonely individuals even when the frequency and intensity of the stressors does not differ (Cacioppo & Hawkley, 2009; Cacioppo, Hawkley, & Berntson, 2003). Further, social isolation is also associated with a heightened attention to negative social information and avoidance of unpleasant social stimuli (Cacioppo, Norris, Decety, Monteleone, & Nusbaum, 2009) akin to behavioral phenotypes of people with SAD. SAD has been conceived of as a disorder that involves excessive fear of negative evaluations and rejection from others. Research on social rejection suggests that social rejection activates pain pathways (Eisenberger, Lieberman, & Williams, 2003). Increased activation of insular and cingulate cortices in response to social stimuli in SAD patients might indicate similar mechanisms at play, and bear greater investigation. Research is needed to investigate the relationships between duration and severity of social anxiety, the degree of social isolation, and how this impacts the behavior and physiology of SAD individuals.

Self-Reference, Self-Reappraisal, and Social Others in SAD

Recent work has begun to explore neural circuits underlying self-referential processing and self-regulation of negative emotions in SAD. Consistent with the idea that fear of negative evaluation is a core feature of SAD, patients show increased amygdala and mPFC responses to negative self-beliefs (Goldin, Manber-Ball, et al., 2009), as well as others' negative, positive, and neutral opinions about them (Blair et al., 2011), when compared with control subjects. Social neuroscience research with healthy participants indicates that the PFC exerts regulatory control on fear circuits originating in the amygdala by dampening their activity (Ochsner & Gross, 2007; Sotres-Bayon &

Quirk, 2010). Both the ventromedial and the dorsolateral aspects of the PFC have been associated with fear modulation, with the former being active in outcome-based forms of fear regulation, while the latter is involved in description-based strategic reappraisal of stimuli (Ochsner & Gross, 2007). A recent study that looked at cognitive-linguistic reappraisal strategies in SAD patients suggests that while reappraisal results in greater cognitive and attention regulation–related brain activation in controls, patients display disordered patterns of medial and dorsal PFC activity even during cognitive regulation of social threat (Goldin, Hakimi, & Gross, 2009). This effect occurs despite the fact that although patients self-report greater negative emotion in response to threat stimuli, they show *equivalent reduction* in negative emotion following cognitive regulation. In a related vein, studies of self-referential processing (which examine SAD reactivity to statements that refer to the self versus others) note that, in addition to heightened amygdala activity, SAD patients show increased mPFC activity in response to: self-referential criticisms (Blair, Geraci, et al., 2008); in anticipation of self-referential comments (Guyer et al., 2008); as well as in response to unintentional social transgressions (Blair et al., 2010). Overall then, these data seem to indicate that a pervasive fear of negative evaluation marks SAD patients, causing them to be overly sensitive to others' evaluations of them, and probably also to have greater difficulty regulating these feelings through cognitive reappraisal strategies.

How does this impact the quality of relationships that SAD patients maintain with close others? Social anxiety is associated with less assertiveness, and more avoidance of expressing emotion in relationships with friends, family, and romantic partners (Davila & Beck, 2002; Grant, Beck, & Farrow, 2007). However, research has also noted that while social anxiety is characterized by avoidant interpersonal styles, it is also associated with higher interpersonal dependence on close social (romantic) relationships (Darcy, Davila, & Beck, 2005). Studies with healthy participants show that the region involved in self-reflective processes—the mPFC—is activated when healthy individuals judge psychological traits or states of those close to them, such as their mother (Ruby & Decety, 2004), close friend (Ochsner et al., 2005), or someone similar to self (Mitchell, Banaji, & Macrae, 2005). Since self-referential processing is aberrant in SAD, would patients also be atypical in their judgments of close others? Or have they developed reappraisal-based coping strategies that allow them to regulate their increased reactivity to social threat with respect to close others? Do these involve conscious, linguistic regulatory processes, or are they unconscious strategies? While SAD can be a debilitating disorder, the majority of individuals with elevated social anxiety are able to function, so to speak, in a social world. Gaining a better understanding of the coping/regulatory strategies that patients use to enable themselves to have meaningful relationships with their social counterparts (partners, parents, children) would inform clinical approaches to therapy development.

Limited Scope of Currently Used Social Stimuli in SAD Research

Emotional face processing and social provocation paradigms have provided an excellent starting point for research into behavioral aberrations in SAD, and the neural circuits that underlie atypical behavior patterns. However, since interacting with others

in the social world involves a complex interplay of stimulus–response contingencies, a comprehensive understanding of this disorder (and, indeed, of social behavior!) can only emerge from studying more ecologically valid social interactions. Additionally, social anxiety research could benefit significantly from expanding the scope of (a) social stimulus modalities (e.g., audio and audio-visual); and (b) social behavior dimensions (e.g., social perspective taking or empathy) that are addressed in current research within the field. For instance, two recent studies examined SAD patients' responses to aversive social cues using audio stimuli (prosody of human voices). In the first study, individuals with SAD correctly identified fearful and sad voices more often, but displayed a decreased recognition of happy voices compared with healthy controls (Quadflieg, Wendt, Mohr, Miltner, & Straube, 2007). The second study found that anger prosody is associated with differential orbitofrontal activation in SAD individuals, but does not activate the amygdala or the insula any more compared with control subjects (Quadflieg et al., 2008). These data would indicate that (a) social anxiety is associated with heightened processing of negative social cues as well as decreased sensitivity to social reward embedded in human voices; and (b) although SAD patients do display biased processing of auditory cues of social threat, this bias is computed differently—and possibly in higher-order regulatory regions—than their bias for visual stimuli of social threat. These data provide tantalizing evidence that social threat detected through different modalities is processed differentially in SAD, with different social cues receiving different degrees of top-down modulation. Since real-world social interactions commonly involve both audio and visual social cues (e.g., someone engaged in face-to-face conversation), follow-up research could examine how these multimodal cues are processed in SAD, and how/to what extent modulatory influences interact to create socially anxious responses along the vigilance-avoidance orientations that characterize SAD.

Additional dimensions of social behavior remain minimally studied in SAD. For instance, previous research has suggested that elevated anxiety can reduce empathic responses to social counterparts (Deardorff, Philip, & Finch, 1977; Negd, Mallan, & Lipp, 2011). There is some data to indicate that the right premotor cortex, which was found to be thinner in SAD patients in a structural MRI analysis (Syal et al., 2012), plays a role in both the generation and the perception of emotionally expressive faces; this is consistent with *perception–action*, as well as *motor theories* of empathy (Leslie, Johnson-Frey, & Grafton, 2004). In addition to right lateralized thinning in motor and sensorimotor cortices, Syal et al. also noted significant thinning in the supramarginal region of the inferior parietal lobule, and a trend toward significant thinning in the right pars opercularis. Both of these areas have been posited to be part of the mirror neuron system involved in imitative and empathic social behavior (Carr, Iacoboni, Dubeau, Mazziotta, & Lenzi, 2003), and have been found to be thinner in individuals with other disorders of social affect, such as autism spectrum disorders (Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2006). At this time, it is difficult to predict what form, if any, atypical empathic or mentalizing responses might take in SAD. On the one hand, greater self-directed attention and rumination are proposed perpetuators of SAD (Clark & Wells, 1995), which could potentially make it difficult to disengage from the self and consider the perspective of others in mentalizing tasks. On the other hand, if SAD patients are engaged in continual evaluation of the self from

the perspective of others, they might display heightened empathic ability to assume the perspective of others. Finally, it is also possible that SAD patients will display no aberrations in social perspective taking, as these processes are under higher-order control, at which level coping mechanisms may obviate any differences in behavior.

Learning About SAD from Other Disorders or Dispositions of Prosociality

SAD research can glean a fair bit from considering other disorders involving social affect in an attempt to extrapolate the research knowledge that exists in those domains to the study of social anxiety. For example, individuals with William's syndrome (WS), a neurodevelopmental condition caused by a hemizygous microdeletion on chromosome 7q11.23, provide an extremely interesting comparison group that also shows profound aberrations in social behavior and motivation. Contrary to SAD, social aberrations in WS move in a direction where hyperactivity of the social circuits offsets cognitive deficits in a manner that provides impetus to greater learning of faculties that are embedded within the social domain. For example, individuals with WS show heightened interest in face-to-face interaction, fail to recruit the amygdala in face discrimination tasks (Paul et al., 2009), and show reduced amygdala activation in response to threatening faces (Meyer-Lindenberg et al., 2005; Munoz et al., 2010)—ergo, they display a phenotypic manifestation that is almost diametrically opposite to that of SAD patients. The enhanced interest in faces in WS patients is matched by increased thickness of the FFC, an area believed to be involved in the processing of such socially relevant information (Reiss, Eckert, & Rose, 2004; Thompson et al., 2005), while a recent study of gray matter integrity in SAD noted decreased thickness of the FFC in SAD patients compared with control participants (Syal et al., 2012). Another recent study showed that WS is associated with abnormal anatomy of white matter fibers projecting through the fusiform gyrus (Haas et al., 2012). Although it is possible that genetic abnormalities that lead to impaired cytoskeletal neuron dynamics might have led to these deficits in WS (Marenco et al., 2007), exploring white matter integrity of fibers through the fusiform, as well as ones connecting between the fusiform and the amygdala, might be a worthwhile endeavor in the study of SAD.

On a related note, increased connectivity between the OFC and the striatum has been associated with increased responsiveness to socially defined reward and increased disposition to social relationships and attachments in healthy individuals (Cohen, Schoene-Bake, Elger, & Weber, 2009; Lebreton, Barnes, & Miettunen, 2009). Would decreased responsiveness to social reward predict decreased connectivity between OFC and striatum in SAD patients?

Conclusion

In spite of high point prevalence estimates ranging from 4% to 6%, with a lifetime risk of 7% to 13% (Kessler et al., 2005; Wittchen & Fehm, 2001), SAD remains one of the most under-treated anxiety disorders (Cuthbert, 2002). Although SAD is highly

distressing in itself, it also constitutes a risk factor for the development of additional psychiatric problems, including depression, substance abuse, and suicidal behaviors (Weiller, Bisserbe, Boyer, & Lepine, 1996).

Given current evidence, SAD appears to involve a pathological manifestation of the dysregulation of social behavior. This dysfunction spans increased negative self-evaluations, inability to cognitively reappraise these negative self-beliefs, exaggerated fear sensitivity to real or imagined social threats, and possibly the inability to source reward from social interactions. SAD is associated with aberrant activation of a network of brain regions that is typically involved in social behavior, including the amygdala, the striatum, the FFC, the insular cortex, the cingulate, and the PFC. Aberrant activity has also been noted in the OFC, the visual cortices, and the parahippocampal gyrii. As some of these regions are also involved in the processing of nonsocial threat, it is important to note that SAD patients do not appear to show an increased amygdala (Goldin, Hakimi, et al., 2009) or physiological (McTeague et al., 2009) response to physical threat (i.e., exaggerated threat bias noted in SAD patients is specific to social stimuli), in keeping with the notion that SAD is a disorder of *social* behavior.

Both human and animal research has indicated that molecular mechanisms underlying increased social anxiety include perturbations of the central dopamine and serotonin systems. Studies on social subordination in nonhuman primates reveal that subordinates spend more time alone, are fearful of their social environment, and have impaired serotonergic and dopaminergic neurotransmission (Shively, 1998). Similarly, SAD patients demonstrate reduced serotonin-1A receptor binding (Lanzenberger & Mitterhauser, 2007) and decreased striatal dopamine function (Schneier & Liebowitz, 2000). In healthy volunteers, increased serotonin-1A receptor binding predicts decreased levels of general anxiety (Tauscher et al., 2001); and a specific polymorphic variant of the serotonin transporter gene predicts greater amygdala response to fearful faces (Hariri et al., 2002) as well as greater amygdala–PFC coupling (Heinz et al., 2004). In SAD patients, presence of the same serotonin gene variant (5-HTT short allele) is associated with symptom severity as well as amygdala excitability, and SSRI treatments show significant improvement in levels of social anxiety (Bouwer & Stein, 1998), as well as brain responses (Carey et al., 2004). These data raise the intriguing possibility that serotonin dysfunction could contribute to anxiety symptoms, whereas dopamine dysfunction could lead to reduced reward from social interaction in SAD. Additionally, since many of the brain regions that display increased activation in SAD contain glutamate receptors, glutamatergic transmission is an interesting area for future research of this disorder. Similarly, opioid neurotransmission remains central to social bonding behavior and thus also warrants further research in SAD.

Although much has therefore been learned about the cognitive-affective disruptions in SAD, their neural circuitry, and their molecular underpinnings, a great deal of work remains to be done. In this chapter, we have underscored the value of a social neuroscience perspective for future work on areas that remain relatively unexplored in SAD. In particular, we emphasize the value of considering the multidimensionality of human social behavior when studying its aberrations. Genetic, immune, endocrine, and nervous systems act in concert to orchestrate human social living. It is only through holding all these perspectives in sight that a holistic understanding of the neurobiology of SAD can evolve.

References

Adam, E. K., Hawkley, L. C., Kudielka, B. M., & Cacioppo, J. T. (2006). Day-to-day dynamics of experience-cortisol associations in a population-based sample of older adults. *Proceedings of the National Academy of Sciences of the United States of America, 103*(45), 17058–17063.

Amir, N., Klumpp, H., Elias, J., Bedwell, J. S., Yanasak, N., & Miller, L. S. (2005). Increased activation of the anterior cingulate cortex during processing of disgust faces in individuals with social phobia. *Biological Psychiatry, 57*(9), 975–981.

Anderson, A. K., & Phelps, E. A. (2002). Is the human amygdala critical for the subjective experience of emotion? Evidence of intact dispositional affect in patients with amygdala lesions. *Journal of Cognitive Neuroscience, 14*(5), 709–720.

Bachner-Melman, R., Zohar, A. H., Bacon-Shnoor, N., Elizur, Y., Nemanov, L., Gritsenko, I., & Ebstein, R. P. (2005). Link between vasopressin receptor AVPR1A promoter region microsatellites and measures of social behavior in humans. *Journal of Individual Differences, 26*(1), 2–10.

Beaton, E. A., Schmidt, L. A., Schulkin, J., Antony, M. M., Swinson, R. P., & Hall, G. B. (2008). Different neural responses to stranger and personally familiar faces in shy and bold adults. *Behavioral Neuroscience, 122*(3), 704–709.

Beaton, E. A., Schmidt, L. A., Schulkin, J., Antony, M. M., Swinson, R. P., & Hall, G. B. (2009). Different fusiform activity to stranger and personally familiar faces in shy and social adults. *Social Neuroscience, 4*(4), 308–316.

Bell, C., Malizia, A., & Nutt, D. (1999). The neurobiology of social phobia. *European Archives of Psychiatry and Clinical Neuroscience, 249*(Suppl. 1), S11–S18.

Berkman, L. (1995). The role of social relations in health promotion. *Psychosomatic Medicine, 57*(3), 245–254.

Bickart, K. C., Wright, C. I., Dautoff, R. J., Dickerson, B. C., & Barrett, R. F. (2011). Amygdala volume and social network size in humans. *Nature Neuroscience, 14*, 163–164.

Bierhaus, A., Wolf, J., Andrassy, M., Rohleder, N., Humpert, P. M., Petrov, D., . . .Nawroth, P. P. (2003). A mechanism converting psychosocial stress into mononuclear cell activation. *Proceedings of the National Academy of Sciences of the United States of America, 100*(4), 1920–1925.

Birbaumer, N., Grodd, W., Diedrich, O., Klose, U., Erb, M., Lotze, M., . . .Flor, H. (1998). fMRI reveals amygdala activation to human faces in social phobics. *Neuroreport, 9*(6), 1223.

Blair, K., Geraci, M., Devido, J., McCaffrey, D., Chen, G., Vythilingam, M., . . .Pine, D. S. (2008). Neural response to self- and other referential praise and criticism in generalized social phobia. *Archives of General Psychiatry, 65*(10), 1176–1184.

Blair, K. S., Geraci, M., Hollon, N., Otero, M., Devido, J., Majestic, C., . . .Pine, D. S. (2010). Social norm processing in adult social phobia: Atypically increased ventromedial frontal cortex responsiveness to unintentional (embarrassing) transgressions. *American Journal of Psychiatry, 167*(12), 1526–1532.

Blair, K. S., Geraci, M., Otero, M., Majestic, C., Odenheimer, S., Jacobs, M., . . .Pine, D. S. (2011). Atypical modulation of medial prefrontal cortex to self-referential comments in generalized social phobia. *Psychiatry Research: Neuroimaging, 193*(1), 38–45.

Blair, K., Shaywitz, J., Smith, B. W., Rhodes, R., Geraci, M., Jones, M., . . .Pnie, D. S. (2008). Response to emotional expressions in generalized social phobia and generalized anxiety disorder: Evidence for separate disorders. *American Journal of Psychiatry, 165*(9), 1193–1202.

Bögels, S. M., & Mansell, W. (2004). Attention processes in the maintenance and treatment of social phobia: Hypervigilance, avoidance and self-focused attention. *Clinical Psychology Review, 24*(7), 827–856.

Bos, P., Panksepp, J., & Bluthe, R. (2012). Acute effects of steroid hormones and neuropeptides on human social-emotional behavior: A review of single administration studies. *Frontiers in Neuroendocrinology, 33*, 17–35.

Bouwer, C., & Stein, D. J. (1998). Use of the selective serotonin reuptake inhibitor citalopram in the treatment of generalized social phobia. *Journal of Affective Disorders, 49*(1), 79–82.

Brumariu, L. E., & Kerns, K. A. (2008). Mother–child attachment and social anxiety symptoms in middle childhood. *Journal of Applied Developmental Psychology, 29*(5), 393–402.

Cacioppo, J. T., & Hawkley, L. C. (2009). Perceived social isolation and cognition. *Trends in Cognitive Sciences, 13*(10), 447–454.

Cacioppo, J. T., Hawkley, L. C., & Berntson, G. G. (2003). The anatomy of loneliness. *Current Directions in Psychological Science, 12*(3), 71–74.

Cacioppo, J. T., Hawkley, L. C., Norman, G. J., & Berntson, G. G. (2011). Social isolation. *Annals of the New York Academy of Sciences, 1231*, 17–22.

Cacioppo, J. T., Norris, C. J., Decety, J., Monteleone, G., & Nusbaum, H. (2009). In the eye of the beholder: Individual differences in perceived social isolation predict regional brain activation to social stimuli. *Journal of Cognitive Neuroscience, 21*(1), 83–92.

Carey, P. D., Warwick, J., Niehaus, D. J. H., van der Linden, G., van Heerden, B. B., Harvey, B. H., . . . Stein, D. J. (2004). Single photon emission computed tomography (SPECT) of anxiety disorders before and after treatment with citalopram. *BioMed Central Psychiatry, 4*, 30.

Carr, L., Iacoboni, M., Dubeau, M.-C., Mazziotta, J. C., & Lenzi, G. L. (2003). Neural mechanisms of empathy in humans: A relay from neural systems for imitation to limbic areas. *Proceedings of the National Academy of Sciences of the United States of America, 100*(9), 5497–5502.

Clark, D., & Wells, A. (1995). A cognitive model of social phobia. In R. G. Heimberg, M. R. Liebowitz, D. Hope, & F. R. Schneier (Eds.), *Social phobia – Diagnosis, assessment, and treatment* (pp. 69–93). New York, NY: Guilford Press.

Cohen, M. X., Schoene-Bake, J.-C., Elger, C. E., & Weber, B. (2009). Connectivity-based segregation of the human striatum predicts personality characteristics. *Nature Neuroscience, 12*(1), 32–34.

Cohen, S., Doyle, W. J., Skoner, D. P., Rabin, B. S., & Gwaltney, J. M. (1997). Social ties and susceptibility to the common cold. *Journal of the American Medical Association, 277*(24), 1940–1944.

Cole, S. W., Hawkley, L. C., Arevalo, J. M. G., & Cacioppo, J. T. (2011). Transcript origin analysis identifies antigen-presenting cells as primary targets of socially regulated gene expression in leukocytes. *Proceedings of the National Academy of Sciences of the United States of America, 108*(7), 3080–3085.

Critchley, H. D. (2000). The functional neuroanatomy of social behaviour: Changes in cerebral blood flow when people with autistic disorder process facial expressions. *Brain, 123*(11), 2203–2212.

Cuthbert, B. N. (2002). Social anxiety disorder: Trends and translational research. *Biological Psychiatry, 51*(1), 4–10.

Darcy, K., Davila, J., & Beck, J. G. (2005). Is social anxiety associated with both interpersonal avoidance and interpersonal dependence? *Cognitive Therapy and Research, 29*(2), 171–186.

Davila, J., & Beck, J. (2002). Is social anxiety associated with impairment in close relationships? A preliminary investigation. *Behavior Therapy, 33*(3), 427–446.

Deardorff, P., Philip, C., & Finch, J. A. (1977). Empathy, locus of control and anxiety in college students. *Psychological Reports, 40*(3 Pt. 2), 1236–1238.

Deeley, Q., Daly, E. M., Surguladze, S., Page, L., Toal, F., Robertson, D., . . .Murphy, D. G. (2007). An event related functional magnetic resonance imaging study of facial emotion processing in Asperger syndrome. *Biological Psychiatry, 62*(3), 207–217.

Denkova, E., Wong, G., Dolcos, S., Sung, K., Wang, L., Coupland, N., & Dolcos, F. (2010). The impact of anxiety-inducing distraction on cognitive performance: A combined brain imaging and personality investigation. *PloS One, 5*(11), e14150.

Dunbar, R. (1998). The social brain hypothesis. *Evolutionary Anthropology, 6*(5), 178–190.

Eisenberger, N. I., Lieberman, M. D., & Williams, K. D. (2003). Does rejection hurt? An FMRI study of social exclusion. *Science, 302*(5643), 290–292.

Ershler, W. B., & Keller, E. T. (2000). Age-associated increased interleukin-6 gene expression, late-life diseases, and frailty. *Annual Review of Medicine, 51*, 245–270.

Etkin, A., & Wager, T. (2007). Functional neuroimaging of anxiety: A meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. *American Journal of Psychiatry, 164*(10), 1476–1488.

Evans, K., Wright, C., & Wedig, M. (2008). A functional MRI study of amygdala responses to angry schematic faces in social anxiety disorder. *Depression and Anxiety, 25*, 496–505.

Freitas-Ferrari, M. C., Hallak, J. E. C., Trzesniak, C., Filho, A. S., Machado-de-Sousa, J. P., Chagas, M. H. N., . . .Crippa, J. A. (2010). Neuroimaging in social anxiety disorder: A systematic review of the literature. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 34*(4), 565–580.

Furmark, T. (2009). Neurobiological aspects of social anxiety disorder. *Israel Journal of Psychiatry and Related Sciences, 46*(1), 5–12.

Furmark, T., Appel, L., Henningsson, S., Ahs, F., Faria, V., Linnman, C., . . .Fredrikson, M. (2008). A link between serotonin-related gene polymorphisms, amygdala activity, and placebo-induced relief from social anxiety. *Journal of Neuroscience, 28*(49), 13066–13074.

Furmark, T. T., Appel, L. L., Michelgård, A. A., Wahlstedt, K. K., Ahs, F. F., Zancan, S. S., . . .Fredrikson, M. (2005). Cerebral blood flow changes after treatment of social phobia with the neurokinin-1 antagonist GR205171, citalopram, or placebo. *Biological Psychiatry, 58*(2), 132–142.

Furmark, T., Tillfors, M., Marteinsdottir, I., Fischer, H., Pissiota, A., Långström, B., & Fredrikson, M. (2002). Common changes in cerebral blood flow in patients with social phobia treated with citalopram or cognitive-behavioral therapy. *Archives of General Psychiatry, 59*(5), 425–433.

Gentili, C., Gobbini, M. I., Ricciardi, E., Vanello, N., Pietrini, P., Haxby, J. V., & Guazzelli, M. (2008). Differential modulation of neural activity throughout the distributed neural system for face perception in patients with social phobia and healthy subjects. *Brain Research Bulletin, 77*(5), 286–292.

Gilbert, P. (2001). Evolution and social anxiety. *Psychiatric Clinics of North America, 24*(4), 723–751.

Goldin, P. R., Hakimi, S., & Gross, J. J. (2009). Neural bases of social anxiety disorder: Emotional reactivity and cognitive regulation during social and physical threat. *Archives of General Psychiatry, 66*(2), 170–180.

Goldin, P. R., Manber-Ball, T., Werner, K., Heimberg, R., & Gross, J. J. (2009). Neural mechanisms of cognitive reappraisal of negative self-beliefs in social anxiety disorder. *Biological Psychiatry, 66*(12), 1091–1099.

Grant, D., Beck, J., & Farrow, S. (2007). Do interpersonal features of social anxiety influence the development of depressive symptoms? *Cognition & Emotion, 21*(3), 646–663.

Grant, K. A., Shively, C. A., Nader, M. A., Ehrenkaufer, R. L., Line, S. W., Morton, T. E., . . .Mach, R. H. (1998). Effect of social status on striatal dopamine D2 receptor binding characteristics in cynomolgus monkeys assessed with positron emission tomography. *Synapse, 29*(1), 80–83.

Guyer, A. E., Lau, J. Y. F., McClure-Tone, E. B., Parrish, J., Shiffrin, N. D., Reynolds, R. C., . . .Nelson, E. E. (2008). Amygdala and ventrolateral prefrontal cortex function during anticipated peer evaluation in pediatric social anxiety. *Archives of General Psychiatry, 65*(11), 1303–1312.

Haas, B. W., Hoeft, F., Barnea-Goraly, N., Golarai, G., Bellugi, U., & Reiss, A. L. (2012). Preliminary evidence of abnormal white matter related to the fusiform gyrus in Williams syndrome: A diffusion tensor imaging tractography study. *Genes, Brain, and Behavior, 11*(1), 62–68.

Hadjikhani, N., Joseph, R. M., Snyder, J., & Tager-Flusberg, H. (2006). Anatomical differences in the mirror neuron system and social cognition network in autism. *Cerebral Cortex, 16*(9), 1276–1282.

Hahn, A., Stein, P., Windischberger, C., Weissenbacher, A., Spindelegger, C., Moser, E., . . .Lanzenberger, R. (2011). Reduced resting-state functional connectivity between amygdala and orbitofrontal cortex in social anxiety disorder. *NeuroImage, 56*(3), 881–889.

Hammock, E. A. D., & Young, L. J. (2005). Microsatellite instability generates diversity in brain and sociobehavioral traits. *Science, 308*(5728), 1630–1634.

Hawkley, L. C., Hughes, M. E., Waite, L. J., Masi, C. M., Thisted, R. A., & Cacioppo, J. T. (2008). From social structural factors to perceptions of relationship quality and loneliness: The Chicago health, aging, and social relations study. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 63*(6), S375–S384.

Heinrichs, M., Baumgartner, T., Kirschbaum, C., & Ehlert, U. (2003). Social support and oxytocin interact to suppress cortisol and subjective responses to psychosocial stress. *Biological Psychiatry, 54*(12), 1389–1398.

Heinz, A., Braus, D. F., Smolka, M. N., Wrase, J., Puls, I., Hermann, D., . . .Buchel, C. (2004). Amygdala-prefrontal coupling depends on a genetic variation of the serotonin transporter. *Nature Neuroscience, 8*(1), 20–21.

House, J., Landis, K., & Umberson, D. (1988). Social relationships and health. *Science, 241*(4865), 540–545.

Israel, S., Lerer, E., Shalev, I., Uzefovsky, F., Riebold, M., Laiba, E., . . .Ebstein, R. P. (2009). The oxytocin receptor (OXTR) contributes to prosocial fund allocations in the dictator game and the social value orientations task. *PloS One, 4*(5), e5535.

Jacob, S., Brune, C. W., Carter, C. S., Leventhal, B. L., Lord, C., & Cook, E. H. (2007). Association of the oxytocin receptor gene (OXTR) in Caucasian children and adolescents with autism. *Neuroscience Letters, 417*(1), 6–9.

James, W. (1894). Discussion: The physical basis of emotion. *Psychological Review, 1*, 516–529.

Kendler, K., Karkowski, L., & Prescott, C. (1999). Fears and phobias: Reliability and heritability. *Psychological Medicine, 29*, 539–553.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 62*(6), 593–602.

Kiecolt-Glaser, J. K., Gouin, J.-P., & Hantsoo, L. (2010). Close relationships, inflammation, and health. *Neuroscience and Biobehavioral Reviews, 35*(1), 33–38.

Kim, S.-J., Young, L. J., Gonen, D., Veenstra-VanderWeele, J., Courchesne, R., Courchesne, E., . . .Insel, T. R. (2002). Transmission disequilibrium testing of arginine vasopressin receptor 1A (AVPR1A) polymorphisms in autism. *Molecular Psychiatry, 7*(5), 503–507.

Klumpp, H., Angstadt, M., & Phan, K.L (2012). Insula reactivity and connectivity to anterior cingulate cortex when processing threat in generalized social anxiety disorder. *Biological Psychology, 89*(1), 273–276.

Kuo, J. R., Goldin, P. R., Werner, K., Heimberg, R. G., & Gross, J. J. (2011). Childhood trauma and current psychological functioning in adults with social anxiety disorder. *Journal of Anxiety Disorders, 25*(4), 467–473.

Lange, K. (1885). *The emotions* (I. A. Haupt, Trans.). Williams and Wilkins.

Lanzenberger, R., & Mitterhauser, M. (2007). Reduced serotonin-1A receptor binding in social anxiety disorder. *Biological Psychiatry, 61*(9), 1081–1089.

Lebreton, M., Barnes, A., & Miettunen, J. (2009). The brain structural disposition to social interaction. *European Journal of Neuroscience, 29*, 2247–2252.

Lerer, E., Levi, S., Salomon, S., Darvasi, A., Yirmiya, N., & Ebstein, R. P. (2007). Association between the oxytocin receptor (OXTR) gene and autism: Relationship to Vineland Adaptive Behavior Scales and cognition. *Molecular Psychiatry, 13*(10), 980–988.

Leslie, K. R., Johnson-Frey, S. H., & Grafton, S. T. (2004). Functional imaging of face and hand imitation: Towards a motor theory of empathy. *NeuroImage, 21*(2), 601–607.

Liao, W., Qiu, C., Gentili, C., Walter, M., Pan, Z., Ding, J., …Chen, H. (2010). Altered effective connectivity network of the amygdala in social anxiety disorder: A resting-state FMRI study. *PloS One, 5*(12), e15238.

Liu, X., Kawamura, Y., Shimada, T., Otowa, T., Koishi, S., Sugiyama, T., …Sasaki, T. (2010). Association of the oxytocin receptor (OXTR) gene polymorphisms with autism spectrum disorder (ASD) in the Japanese population. *Journal of Human Genetics, 55*(3), 137–141.

Lochner, C., Hemmings, S., Seedat, S., Kinnear, C., Schoeman, R., Annerbrink, K., …Stein, D. J. (2007). Genetics and personality traits in patients with social anxiety disorder: A case-control study in South Africa. *European Neuropsychopharmacology, 17*(5), 321–327.

Lorberbaum, J. P., Kose, S., Johnson, M. R., Arana, G. W., Sullivan, L. K., Hamner, M. B., …George, M. S. (2004). Neural correlates of speech anticipatory anxiety in generalized social phobia. *Neuroreport, 15*(18), 2701.

Lundh, L., & Ost, L. (1996). Recognition bias for critical faces in social phobics. *Behaviour Research and Therapy, 34*(10), 787–794.

Marenco, S., Siuta, M. A., Kippenhan, J. S., Grodofsky, S., Chang, W.-L., Kohn, P., …Berman, K. F. (2007). Genetic contributions to white matter architecture revealed by diffusion tensor imaging in Williams syndrome. *Proceedings of the National Academy of Sciences of the United States of America, 104*(38), 15117–15122.

McTeague, L. M., Lang, P. J., Laplante, M.-C., Cuthbert, B. N., Strauss, C. C., & Bradley, M. M. (2009). Fearful imagery in social phobia: Generalization, comorbidity, and physiological reactivity. *Biological Psychiatry, 65*(5), 374–382.

Meyer-Lindenberg, A., Hariri, A. R., Munoz, K. E., Mervis, C. B., Mattay, V. S., Morris, C. A., & Berman, K. F. (2005). Neural correlates of genetically abnormal social cognition in Williams syndrome. *Nature Neuroscience, 8*(8), 991–993.

Meyer-Lindenberg, A., Kolachana, B., Gold, B., Olsh, A., Nicodemus, K. K., Mattay, V., … Weinberger, D. R. (2009). Genetic variants in AVPR1A linked to autism predict amygdala activation and personality traits in healthy humans. *Molecular Psychiatry, 14*(10), 968–975.

Miller, G. E., Rohleder, N., & Cole, S. W. (2009). Chronic interpersonal stress predicts activation of pro- and anti-inflammatory signaling pathways 6 months later. *Psychosomatic Medicine, 71*(1), 57–62.

Miskovic, V., & Schmidt, L. A. (2012). Social fearfulness in the human brain. *Neuroscience and Biobehavioral Reviews, 36*(1), 459–478.

Mitchell, J. P., Banaji, M. R., & Macrae, C. N. (2005). The link between social cognition and self-referential thought in the medial prefrontal cortex. *Journal of Cognitive Neuroscience, 17*(8), 1306–1315.

Mitchell, J. P., Macrae, C. N., & Banaji, M. R. (2006). Dissociable medial prefrontal contributions to judgments of similar and dissimilar others. *Neuron, 50*(4), 655–663.

Mogg, K., & Bradley, B. (1998). A cognitive-motivational analysis of anxiety. *Behaviour Research and Therapy, 36*(9), 809–848.

Moll, H., & Tomasello, M. (2007). Cooperation and human cognition: The Vygotskian intelligence hypothesis. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences, 362*(1480), 639–648.

Moser, J. S., Huppert, J. D., Duval, E., & Simons, R. F. (2008). Face processing biases in social anxiety: An electrophysiological study. *Biological Psychology, 78*(1), 93–103.

Mueller, E. M., Hofmann, S. G., Santesso, D. L., Meuret, A. E., Bitran, S., & Pizzagalli, D. A. (2008). Electrophysiological evidence of attentional biases in social anxiety disorder. *Psychological Medicine, 39*(7), 1141.

Munoz, K. E., Meyer-Lindenberg, A., Hariri, A. R., Mervis, C. B., Mattay, V. S., Morris, C. A., & Berman, K. F. (2010). Abnormalities in neural processing of emotional stimuli in Williams syndrome vary according to social vs. non-social content. *NeuroImage, 50*(1), 340–346.

Negd, M., Mallan, K. M., & Lipp, O. V. (2011). The role of anxiety and perspective-taking strategy on affective empathic responses. *Behaviour Research and Therapy, 49*(12), 852–857.

Nesse, R. M. (2001). The smoke detector principle. Natural selection and the regulation of defensive responses. *Annals of the New York Academy of Sciences, 935*, 75–85.

Ochsner, K. N., Beer, J. S., Robertson, E. R., Cooper, J. C., Gabrieli, J. D. E., Kihsltrom, J. F., & D'Esposito, M. (2005). The neural correlates of direct and reflected self-knowledge. *NeuroImage, 28*(4), 797—814.

Ochsner, K., & Gross, J. (2005). The cognitive control of emotion. *Trends in Cognitive Sciences, 9*(5), 242–249.

Ochsner, K. N., & Gross, J. J. (2007). The neural architecture of emotion regulation. In J. J. Gross & R. Buck (Eds.), *The handbook of emotion regulation* (pp. 87–109). New York, NY: Guilford Press.

Park, J., Willmott, M., Vetuz, G., Toye, C., Kirley, A., Hawi, Z., . . . Kent, L. (2010). Evidence that genetic variation in the oxytocin receptor (OXTR) gene influences social cognition in ADHD. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 34*(4), 697–702.

Paul, B. M., Snyder, A. Z., Haist, F., Raichle, M. E., Bellugi, U., & Stiles, J. (2009). Amygdala response to faces parallels social behavior in Williams syndrome. *Social Cognitive and Affective Neuroscience, 4*(3), 278–285.

Paulus, M. P., & Stein, M. B. (2006). An insular view of anxiety. *Biological Psychiatry, 60*(4), 383–387.

Phan, K., Fitzgerald, D., & Nathan, P. (2006). Association between amygdala hyperactivity to harsh faces and severity of social anxiety in generalized social phobia. *Biological Psychiatry, 59*, 424–429.

Phan, K. L., Orlichenko, A., Boyd, E., Angstadt, M., Coccaro, E. F., Liberzon, I., & Arfanakis, K. (2009). Preliminary evidence of white matter abnormality in the uncinate fasciculus in generalized social anxiety disorder. *Biological Psychiatry, 66*(7), 691–694.

Prehn-Kristensen, A., Wiesner, C., Bergmann, T. O., Wolff, S., Jansen, O., Mehdorn, H. M., . . . Pause, B. M. (2009). Induction of empathy by the smell of anxiety. *PloS One, 4*(6), e5987.

Pujol, J., Harrison, B. J., Ortiz, H., Deus, J., Soriano-Mas, C., López-Solà, M., . . . Cardoner, N. (2009). Influence of the fusiform gyrus on amygdala response to emotional faces in the non-clinical range of social anxiety. *Psychological Medicine, 39*(7), 1177–1187.

Quadflieg, S., Mohr, A., Mentzel, H.-J., Miltner, W. H. R., & Straube, T. (2008). Modulation of the neural network involved in the processing of anger prosody: The role of task-relevance and social phobia. *Biological Psychology, 78*(2), 129–137.

Quadflieg, S., Wendt, B., Mohr, A., Miltner, W. H. R., & Straube, T. (2007). Recognition and evaluation of emotional prosody in individuals with generalized social phobia: A pilot study. *Behaviour Research and Therapy, 45*(12), 3096–3103.

Rapee, R. M., & Heimberg, R. G. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy, 35*(8), 741–756.

Reiss, A., Eckert, M., & Rose, F. (2004). An experiment of nature: Brain anatomy parallels cognition and behavior in Williams syndrome. *The Journal of Neuroscience, 24*(21), 5009–5015.

Rodrigues, S., & Saslow, L. (2009). Oxytocin receptor genetic variation relates to empathy and stress reactivity in humans. *Proceedings of the National Academy of Sciences of the United States of America, 106*(50), 21437–21441.

Ruan, H., & Wu, C.-F. (2008). Social interaction-mediated lifespan extension of Drosophila Cu/Zn superoxide dismutase mutants. *Proceedings of the National Academy of Sciences of the United States of America, 105*(21), 7506–7510.

Ruby, P., & Decety, J. (2004). How would you feel versus how do you think she would feel? A neuroimaging study of perspective-taking with social emotions. *Journal of Cognitive Neuroscience, 16*(6), 988–999.

Sallet, J., Mars, R. B., Noonan, M. P., Andersson, J. L., O'Reilly, J. X., Jbabdi, S., . . . Rushworht, M. F. (2011). Social network size affects neural circuits in macaques. *Science, 334*(6056), 697–700.

Sareen, J., Campbell, D. W., Leslie, W. D., Malisza, K. L., Stein, M. B., Paulus, M. P., . . . Reiss, J. P. (2007). Striatal function in generalized social phobia: A functional magnetic resonance imaging study. *Biological Psychiatry, 61*(3), 396–404.

Schmidt, S., Mohr, A., Miltner, W. H. R., & Straube, T. (2010). Task-dependent neural correlates of the processing of verbal threat-related stimuli in social phobia. *Biological Psychology, 84*(2), 304–312.

Schneier, F., & Liebowitz, M. (2000). Low dopamine D2 receptor binding potential in social phobia. *American Journal of Psychiatry, 157*(3), 457–459.

Schultz, R. T., Grelotti, D. J., Klin, A., Kleinman, J., Van der Gaag, C., Marois, R., & Skudlarski, P. (2003). The role of the fusiform face area in social cognition: Implications for the pathobiology of autism. *Philosophical Transactions of the Royal Society B: Biological Sciences, 358*(1430), 415–427.

Schwartz, C. E., Wright, C. I., Shin, L. M., Kagan, J., & Rauch, S. L. (2003). Inhibited and uninhibited infants "grown up": Adult amygdalar response to novelty. *Science, 300*(5627), 1952–1953.

Shively, C. A. (1998). Social subordination stress, behavior, and central monoaminergic function in female Cynomolgus monkeys. *Biological Psychiatry, 44*(9), 882–891.

Silk, J. B. (2007). The adaptive value of sociality in mammalian groups. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences, 362*(1480), 539–559.

Sotres-Bayon, F., & Quirk, G. J. (2010). Prefrontal control of fear: More than just extinction. *Current Opinion in Neurobiology, 20*(2), 231–235. doi:10.1016/j.conb.2010.02.005

Sripada, C. S., Angstadt, M., Banks, S., Nathan, P. J., Liberzon, I., & Phan, K. L. (2009). Functional neuroimaging of mentalizing during the trust game in social anxiety disorder. *Neuroreport, 20*(11), 984–989.

Staugaard, S. R. (2010). Threatening faces and social anxiety: A literature review. *Clinical Psychology Review, 30*(6), 669–690.

Stein, M. B., Goldin, P. R., Sareen, J., Zorrilla, L. T. E., & Brown, G. G. (2002). Increased amygdala activation to angry and contemptuous faces in generalized social phobia. *Archives of General Psychiatry, 59*(11), 1027–1034.

Stein, M. B., & Stein, D. J. (2008). Social anxiety disorder. *The Lancet, 371*(9618), 1115–1125.

Sterck, E. H. M., Watts, D. P., & van Schaik, C. P. (1997). The evolution of female social relationships in nonhuman primates. *Behavioral Ecology and Sociobiology, 41*(5), 291–309.

Straube, T., Kolassa, I.-T., Glauer, M., Mentzel, H.-J., & Miltner, W. H. R. (2004). Effect of task conditions on brain responses to threatening faces in social phobics: An event-related functional magnetic resonance imaging study. *Biological Psychiatry, 56*(12), 921–930.

Syal, S., Hattingh, C. J., Fouché, J.-P., Spottiswoode, B., Carey, P. D., Lochner, C., & Stein, D. J. (2012). Grey matter abnormalities in social anxiety disorder: A pilot study. *Metabolic Brain Disease, 27*(3), 299–309.

Tauscher, J., Bagby, R., Javanmard, M., Christensen,B., Kasper, S., & Kapur, S. (2001). Inverse relationship between serotonin 5-HT1A receptor binding and anxiety: A [11C] WAY-100635 PET investigation in healthy volunteers. *American Journal of Psychiatry, 158*(8), 1326–1328.

Thomas, P. A. (2012). Trajectories of social engagement and mortality in late life. *Journal of Aging and Health, 24*(4), 547–568.

Thompson, P. M., Lee, A. D., Dutton, R. A., Geaga, J. A., Hayashi, K. M., Eckert, M. A., … Reiss, A. L. (2005). Abnormal cortical complexity and thickness profiles mapped in Williams syndrome. *Journal of Neuroscience, 25*(16), 4146–4158.

Thompson, R., Gupta, S., Miller, K., Mills, S., & Orr, S. (2004). The effects of vasopressin on human facial responses related to social communication. *Psychoneuroendocrinology, 29*(1), 35–48.

Thompson, R. J., Parker, K. J., Hallmayer, J. F., Waugh, C. E., & Gotlib, I. H. (2011). Oxytocin receptor gene polymorphism (rs2254298) interacts with familial risk for psychopathology to predict symptoms of depression and anxiety in adolescent girls. *Psychoneuroendocrinology, 36*(1), 144–147.

Tiihonen, J., Kuikka, J., Bergström, K., Lepola, U., Koponen, H., & Leinonen, E. (1997). Dopamine reuptake site densities in patients with social phobia. *American Journal of Psychiatry, 154*(2), 239–242.

Tillfors, M. (2001). Cerebral blood flow in subjects with social phobia during stressful speaking tasks: A PET study. *American Journal of Psychiatry, 158*(8), 1220–1226.

Tost, H., Kolachana, B., Hakimi, S., Lemaitre, H., Verchinski, B. A., Mattay, V. S., … Meyer-Lindenberg, A (2010). A common allele in the oxytocin receptor gene (OXTR) impacts prosocial temperament and human hypothalamic-limbic structure and function. *Proceedings of the National Academy of Sciences of the United States of America, 107*(31), 13936–13941.

Tottenham, N., & Sheridan, M. A. (2009). A review of adversity, the amygdala and the hippocampus: A consideration of developmental timing. *Frontiers in Human Neuroscience, 3*, 68.

Tricomi, E. M., Delgado, M. R., & Fiez, J. A. (2004). Modulation of caudate activity by action contingency. *Neuron, 41*(2), 281–292.

Turk, C., Heimberg, R., & Luterek, J. (2005). Emotion dysregulation in generalized anxiety disorder: A comparison with social anxiety disorder. *Cognitive Therapy and Research, 29*(1), 89–106.

Van Ameringen, M., Mancini, C., Szechtman, H., Nahmias, C., Oakman, J. M., Hall, G. B. C., … Farvolden, P. (2004). A PET provocation study of generalized social phobia. *Psychiatry Research, 132*(1), 13–18.

van Schaik, C. P., & Burkart, J. M. (2011). Social learning and evolution: The cultural intelligence hypothesis. *Philosophical Transactions of the Royal Society B: Biological Sciences, 366*(1567), 1008–1016.

Veit, R., Flor, H., Erb, M., Hermann, C., Lotze, M., Groddc, W., & Birbaumer N. (2002). Brain circuits involved in emotional learning in antisocial behavior and social phobia in humans. *Neuroscience Letters, 328*, 233–236.

Warwick, J., Carey, P., & Van der Linden, G. (2006). A comparison of the effects of citalopram and moclobemide on resting brain perfusion in social anxiety disorder. *Metabolic Brain Disease, 21*(2–3), 230–241.

Wassink, T. H., Piven, J., Vieland, V. J., Pietila, J., Goedken, R. J., Folstein, S. E., & Sheffield, V. C. (2004). Examination of AVPR1a as an autism susceptibility gene. *Molecular Psychiatry, 9*(10), 968–972.

Weiller, E., Bisserbe, J., Boyer, P., & Lepine, J. (1996). Social phobia in general health care: An unrecognised undertreated disabling disorder. *The British Journal of Psychiatry, 168*(2), 169–174.

Weissman, D., Gopalakrishnan, A., & Hazlett, C. (2005). Dorsal anterior cingulate cortex resolves conflict from distracting stimuli by boosting attention toward relevant events. *Cerebral Cortex, 15*, 229–237.

Wilms, M., Schilbach, L., Pfeiffer, U., Bente, G., Fink, G. R., & Vogeley, K. (2010). It's in your eyes – using gaze-contingent stimuli to create truly interactive paradigms for social cognitive and affective neuroscience. *Social Cognitive and Affective Neuroscience, 5*(1), 98–107.

Winton, E. C., Clark, D. M., & Edelmann, R. J. (1995). Social anxiety, fear of negative evaluation and the detection of negative emotion in others. *Behaviour Research and Therapy, 33*(2), 193–196.

Wittchen, H. U., & Fehm, L. (2001). Epidemiology, patterns of comorbidity, and associated disabilities of social phobia. *The Psychiatric Clinics of North America, 24*(4), 617–641.

Wu, S., Jia, M., Ruan, Y., Liu, J., Guo, Y., Shuang, M., … Zhang, D. (2005). Positive association of the oxytocin receptor gene (OXTR) with autism in the Chinese Han population. *Biological Psychiatry, 58*(1), 74–77.

Yirmiya, N., Rosenberg, C., Levi, S., Salomon, S., Shulman, C., Nemanov, L., …Ebstein, R. P. (2006). Association between the arginine vasopressin 1a receptor (AVPR1a) gene and autism in a family-based study: Mediation by socialization skills. *Molecular Psychiatry, 11*(5), 488–494.

Yrigollen, C. M., Han, S. S., Kochetkova, A., Babitz, T., Chang, J. T., Volkmar, F. R., …Grigorenko, E. L. (2008). Genes controlling affiliative behavior as candidate genes for autism. *Biological Psychiatry, 63*(10), 911–916.

5

The Pathophysiology of Social Anxiety

Wieke de Vente, Mirjana Majdandžić, and Susan Bögels

University of Amsterdam, Amsterdam

Introduction

The pathophysiology of social anxiety refers to physiological factors related to the predisposition or presence, the etiology, and/or the maintenance of social anxiety. This area of research is highly relevant for the study of social anxiety or social anxiety disorder (SAD) because physiological information may help clarify the nature of any social anxiety predisposition that may, for example, lie in higher anxiety-related physiological activation, or in a specific social anxiety-related physiological response, such as blushing. Further, studying the interplay between physiological responses and cognitive characteristics such as interoceptive awareness may also enhance the understanding of social anxiety etiology and maintaining mechanisms. Finally, physiological profiles may add to the distinction of subtypes of SAD, which may be relevant for treatment purposes, and cultural differences in prevalence rates of SAD.

With regard to predispositions to social anxiety, physiological correlates of a fearful temperament, in particular *behavioral inhibition* (e.g., Hirschfeld-Becker et al., 2008; Kagan & Snidman, 1999; see also **Chapter 7**), are reviewed. Concerning presence of social anxiety, physiological correlates of social anxiety or SAD (i.e., social phobia, DSM-IV-TR; American Psychiatric Association [APA], 2000) are reported. With respect to etiology and maintenance, studies assessing etiological and/or maintaining mechanisms of social anxiety or SAD, including physiological parameters, are discussed. Before providing an overview about the pathophysiology of social anxiety, the following paragraph provides a concise overview of the biological anxiety system and physiological anxiety indices.

Physiological Correlates of Anxiety

The pathophysiological indices of interest discussed in this chapter are those that reflect activation of peripheral systems or organs that are affected by brain structures

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

involved in emotion processing. These central structures include the amygdala that receive sensory information from cortical sensory areas and project to structures involved in emotional reactions, such as the hypothalamus and the frontal cortex (Bradley & Lang, 2007; see also **Chapter 4**). The amygdala and lateral hypothalamus also regulate the sympathetic branch of the autonomic nervous system in emotions (Bradley & Lang, 2007). In addition, the lateral extended amygdala and areas in the brainstem regulate the parasympathetic nervous system, in particular the nervus vagus (Xth cranial nerve; Bradley & Lang, 2007). Nearly all peripheral organs are innervated by sympathetic and parasympathetic nerves, and may thus be affected by central emotion processing (Bradley & Lang, 2007). The emotion of fear emerges in response to threat. On the physiological level, encountering a threatening situation results in the so-called *fight–flight* response (Cannon, 1914), consisting of sympathetic stimulation of the adrenal medulla. This stimulation results in a release of catecholamines in the blood, such as adrenaline and noradrenaline, which prepares the organism to defend itself or help itself to reach safety. The connection between the hypothalamus and the adrenal medulla via sympathetic nerves is known as the sympathetic–adrenal medullary (SAM) axis. A physiological fear response further consists of direct sympathetic stimulation of muscles and organs (Bradley & Lang, 2007). The parasympathetic system, which stimulates digestion and other body restoring processes, also modulates the sympathetic system (Bradley & Lang, 2007; Brownley, Hurwitz, & Schneiderman, 2000) in order to maintain the body's homeostasis (Cannon, 1929). Porges (1991) has argued that, in mildly threatening situations, autonomic arousal is predominantly determined by activity of the nervus vagus, affecting the heart and other organs; including, for example, the bronchi, facial muscles, and the larynx. In others words, Porges argues that, under mild threat, sympathetic dominance increases due to the reduction of vagal activity.

In the case of anxiety, peripheral indices generally reflect a state of enhanced arousal, due to enhanced sympathetic activation, reduced parasympathetic activation, or both. Enhanced arousal becomes apparent in, for example, increased heart rate, increased stroke volume, increased blood pressure, reduced high-frequency heart rate variability (or, comparably, reduced beat-to-beat heart rate variability or respiratory sinus arrhythmia), increased sweating resulting in improved skin conductance, enhanced muscle tension, trembling, and an enhanced startle response (Bradley & Lang, 2007). Heart rate, stroke volume, blood pressure, and the startle response are sympathetically and parasympathetically affected and reflect the balance of these systems (Bradley & Lang, 2007). High-frequency heart rate variability, and similar measures such as beat-to-beat heart rate variability and respiratory sinus arrhythmia, reflect variations in rate that are due to parasympathetic influences (Penttilä et al., 2001; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). Since the sweat glands in the skin are only innervated by sympathetic nerves, skin conductance reflects relatively pure sympathetic activation (Dawson, Schell, & Filion, 2007). Trembling also seems to be due to sympathetic activation specifically (Nickell & Uhde, 1995).

One peripheral response that seems typical for social anxiety is blushing (Drummond & Su, 2012; Hofmann, Moscovitch, & Kim, 2006). According to Leary, Britt, Cutlip, and Templeton (1992), blushing is a response to undesired social

attention, which is closely related to the core symptom of social anxiety: fear of (negative) evaluation (DSM-IV-TR; APA, 2000). The physiological mechanism underlying the blushing response has not been fully clarified yet (e.g., Hofmann et al., 2006; Leary et al., 1992), but it has been suggested to reflect a specific beta-adrenergic response resulting in vasodilatation of the vessels in the face and neck (Mellander, Andersson, Afzelius, & Hellstrand, 1982). The specificity is explained by the fact that vessels in these areas contain beta-adrenergic receptors, which are absent in vessels in the rest of the body. The function of the blushing response is thought to be primarily social (i.e., appeasement; Keltner & Anderson, 2000). Of note, indices associated with the hypothalamic–pituitary–adrenocortical (HPA) axis, a neuroendocrine system, are excluded from this chapter as they are discussed in **Chapter 21**.

Physiological Correlates of a Fearful Temperament

In observational studies, a fearful temperament is often operationalized as behavioral inhibition (BI). BI refers to a tendency to demonstrate reticence to interact with novel people and situations (Garcia-Koll, Kagan, & Reznick, 1984; see also **Chapter 7**). BI is a predictor of anxiety disorders, including SAD (e.g., Hirschfeld-Becker et al., 2007; Kagan & Snidman, 1999; Rosenbaum et al., 1993). An anxious temperament before the age of 1 year is characterized by enhanced reactivity to novel sensory stimuli, for example, by increased motor activity or crying (Kagan & Snidman, 1991a; Moehler et al., 2008). A fearful temperament is expected to be characterized by autonomic *activity* and *reactivity* patterns characteristic of anxiety. From the fight–flight response to threat, it is predicted to be characterized by enhanced sympathetic activation and reduced parasympathetic activation when exposed to threat. In addition, Kagan and Snidman (1991b) hypothesized that BI would be characterized by a lower response threshold of the amygdala, which would result in stronger physiological arousal during resting states as well.

Increasing evidence illustrates that a fearful temperament is indeed associated with autonomic activity and reactivity patterns that are known to be related to anxiety. The review of Fox, Henderson, Marshall, Nichols, and Ghera (2005) provides an overview of associations between BI and autonomic fear responses. They report support for the hypothesis that BI is associated with a lower response threshold of the amygdala, as evidenced by increased heart rate during rest and stronger heart rate reactivity to unfamiliarity in children of different ages (1–7 years old). In addition, Fox (1989) found that 14-month-old infants who demonstrated higher heart rate variability, indicative of stronger vagal (parasympathetic) activity, were more sociable and characterized by a stronger approach tendency. A positive association between muscle tension and BI has also been reported, as the variability of the muscles in the larynx and vocal cords was smaller (indicating higher muscle tension) in preschoolers high on BI than low on BI during single-word utterances under stress but also in non-stress conditions (Kagan, Reznick, & Snidman, 1987). Studies using skin conductance measures have also found support for stronger sympathetic activation in children with a fearful temperament. First, Scarpa, Raine, Venables, and Mednick (1997) reported that behavioral inhibition was positively associated with skin conductance level during

a task in which 3-year-old preschoolers were exposed to loud tones (75 dB). Similarly, Fowles, Kochanska, and Murray (2000) showed that a fearful temperament (measured by behavioral fear responses to mildly frightening stimuli) was positively associated with skin conductance level during fear-inducing tasks among 4-year-olds. Finally, Turner, Beidel, and Roberson-Nay (2005) found that 7–12-year-old offspring of parents with an anxiety disorder (and thus at risk for having a fearful temperament) demonstrated a higher frequency of spontaneous skin conductance fluctuations during rest, indicative of higher basal arousal, than did the offspring of parents without an anxiety disorder.

In sum, a fearful temperament appears to be associated with relatively higher levels of sympathetic activation and sympathetic reactivity to unfamiliar stimuli. It has to be noted though that evidence of enhanced sympathetic tone or reactivity is not consistently obtained (see, e.g., Talge, Donzella, & Gunnar, 2008; Van Brakel, Muris, & Derks, 2006). Fox et al. (2005) stress the fact that effects are relatively small and may not always be found in samples that do not include children with extreme levels of BI.

Less studied than basal physiological activation and reactivity is physiological *adaptation* to unfamiliar stimuli in the context of BI. Initial support has been obtained for slower adaptation to unfamiliar stimuli in children with high BI. Moehler, Kagan, Parzer, et al. (2006) found that slower habituation to acoustic stimuli (i.e., loud, 75 dB noises) at the age of 2 weeks, as measured by heart rate responses, was associated with higher BI at 14 months. In addition, at the age of 7–12 years, habituation to pictures of snakes or loud acoustic tones measured with galvanic skin responses was less likely among offspring of parents with an anxiety disorder, and thus at risk for having a fearful temperament, than offspring of parents without an anxiety disorder (Turner et al., 2005). Taken together, a fearful temperament seems to be characterized by slower or reduced physiological adaptation to new stimuli. However, since the number of studies addressing this hypothesis is very small, replication of these studies is needed.

Physiological Correlates of Social Anxiety and SAD

Research on physiological correlates of social anxiety has focused on sympathetic activity, parasympathetic activity, and on blushing when exposed to social stressors. Similar to a fearful temperament, social anxiety is expected to be characterized by enhanced sympathetic activation and reduced parasympathetic activation, in resting states as well as in social fear states. A state of increased sympathetic activity, which may result from more sympathetic activation, less parasympathetic activation, or both, is also referred to as a state of *hyperarousal*. Some studies attempt to disentangle sympathetic and parasympathetic activity. Studying parasympathetic activity in particular is of interest, since Porges (1991) has argued that it is an indicator of emotion regulation. It is hypothesized that highly anxious individuals have lower parasympathetic activation and reduced parasympathetic modulation (Porges, 1991). Concerning blushing, socially anxious individuals are expected to blush more intensely and more frequently.

Hyperarousal, Sympathetic, and Parasympathetic Activity

Support for *basal* hyperarousal as a characteristic of social anxiety in childhood comes from a study of Krämer et al. (2012). During the *Trier social stress test*, consisting of a mental arithmetic task, a speech preparation task, and a speech task (Kirschbaum, Pirke, & Hellhammer, 1993), 41 children (aged 8–12 years) diagnosed with SAD showed elevated heart rate during the entire task procedure, including baseline and stressors, compared with a healthy reference group ($n = 40$). Similarly, Schmitz, Krämer, Tuschen-Caffier, Heinrichs, and Blechert (2011) also found that children aged 8–12 years with SAD demonstrated elevated baseline heart rate and electrodermal activity, lower levels of parasympathetic activation, and reduced parasympathetic reactivity to the Trier social stress test relative to a healthy reference group. Moreover, they found that children with SAD demonstrated slower recovery of heart rate than healthy children.

In adults, comparable results are obtained. Social anxiety is characterized by basal hyperarousal; individuals with higher levels of social anxiety during a baseline phase prior to a social performance task demonstrated elevated startle responses (Cornwell, Johnsson, Berardi, & Grillon, 2006). Support is also obtained for enhanced autonomic *reactivity* to social threat, as high socially anxious women reacted with stronger heart rate acceleration to animated movie stimuli that reacted with a direct gaze (as if toward the participant) as opposed to averted gaze, in comparison with medium and low socially anxious women (Wieser, Pauli, Alpers, & Mühlberger, 2009). A similar increase in heart rate was demonstrated in high versus low socially anxious women during speech preparation and speech tasks (Gramer, Schild, & Lurz, 2012). Furthermore, larger startle responses were found in socially anxious versus nonsocially anxious individuals during speech anticipation in a virtual reality environment and a counting-backward task relative to baseline (Cornwell et al., 2006) and to *emotional* relative to *neutral* cues (Garner, Clarke, Graystone, & Baldwin, 2011). Interestingly, in the latter study, increased startle responses were found for positive *and* negative facial expressions, suggesting lower threshold of amygdala activation for emotional stimuli in general, rather than for negative stimuli alone. Additional support for fear of *positive* evaluation as a cognitive component of social anxiety is provided in **Chapter 20**.

In patients diagnosed with SAD, higher heart rate and muscle tension in the corrugator (a muscle involved in frowning which is associated with negative affect) has been found during a baseline period prior to a stressor (McTeague et al., 2009) than in controls, suggesting *basal* hyperarousal. Support has also been found for enhanced *reactivity* to social threat. First, individuals diagnosed with SAD showed greater heart rate responses than healthy controls when delivering a speech (Boone et al., 1999; Heimberg, Hope, Dodge, & Becker, 1990; Hofmann, Newman, Ehlers, & Roth, 1995; Levin et al., 1993), during singing and reviewing back their performance on video (Gerlach, Wilhelm, & Roth, 2003), or listening to narrative scripts about social threat, survival threat, and an idiographic personal fear (McTeague et al., 2009). Second, patients with SAD demonstrated larger startle responses than controls when listening to narrative scripts about various threat situations (see above; McTeague et al., 2009) or during speech preparation in a virtual-reality environment (Cornwell,

Heller, Biggs, Pine, & Grillon, 2011). Third, patients with SAD demonstrated larger skin conductance reactivity when listening to narrative scripts about various threat situations than controls (McTeague et al., 2009).

The majority of the results suggest that physiological differences between socially anxious and nonsocially anxious groups concerning basal activation and reactivity may involve both withdrawal of parasympathetic activity and increase of sympathetic activation. In some studies, though, just one of these effects (i.e., withdrawal of parasympathetic activity or increase of sympathetic activation) seems to dominate. Regarding basal autonomic activity, McTeague et al. (2009) found elevated baseline heart rate in SAD patients compared with healthy controls, while no differences from healthy controls were found on skin conductance, suggesting a reduced parasympathetic tone was the source of the higher heart rate.

With regard to reactivity, the initial heart acceleration demonstrated by high socially anxious women to animated movie stimuli reacting with a direct gaze (Wieser et al., 2009) was not accompanied by differences in skin conductance, suggesting that, similar to the findings for basal activity, the acceleration was a result of parasympathetic withdrawal rather than sympathetic activation. In line with this, no differences in skin conductance response were found between high and low socially anxious individuals during baseline and a conversation with an unknown person, reflecting no differences in sympathetic activation, whereas high socially anxious individuals demonstrated more facial coloration, suggesting induction of physiological change (Bögels, Rijsemus, & De Jong, 2002). Support for reduced parasympathetic *modulation* in social anxiety, indicating reduced emotion regulation (Porges, 1991), has also been reported. Movius and Allen (2005) found that, in a nonclinical sample, vagal tone was less modulated during a mental arithmetic task and a relaxation exercise relative to a baseline resting phase in the subgroup reporting high social anxiety compared with the subgroup reporting low social anxiety. The reduced parasympathetic reactivity and slower recovery to the social stressor reported in children by Schmitz et al. (2011, described above) can also be seen as support for reduced parasympathetic modulation. There is also support for enhanced *sympathetic reactivity* during a social stressor in children (see Schmitz et al., 2011, described above) and during threat in the study of McTeague et al. (2009, see above), in which stronger skin conductance reactivity was found in SAD patients than in healthy controls when exposed to fear scenarios.

In sum, most studies report that social anxiety is characterized by hyperarousal, either during baseline, in reaction to a social stressor, or both. Studies distinguishing between sympathetic and parasympathetic activity suggest that hyperarousal at rest or induced by social stressors can result from attenuated parasympathetic (re)activity and/or enhanced sympathetic (re)activity. Since the sympathetic and parasympathetic systems are not always both affected by a stressor, which may be dependent on factors such as the stressor intensity or ways of coping available to deal with the stressor, it is recommended to include specific measures of both parts of the autonomic nervous system.

Null findings regarding differences in sympathetic and parasympathetic activity between socially anxious and nonsocially anxious individuals have also been reported, though. For example, in high and low socially anxious children aged 9–12 years (community sample), no differences in heart rate reactivity during public-speaking were

observed ($n = 20$; Schmitz, Blechert, Krämer, Asbrand, & Tuschen-Caffier, 2012; no report on resting states). Also, in an adolescent community sample, no enhanced physiological reactivity was found, as measured by heart rate and blood pressure, between the group diagnosed with SAD ($n = 75$) versus the group without SAD ($n = 270$) during a speech task and a conversation task (Anderson & Hope, 2009; no report on resting states). Similarly, in a community sample of young adults, no differences were found for heart rate or skin conductance during a speech task between a group scoring high ($n = 20$) versus low on social anxiety ($n = 20$; Puigcerver, Martínez-Selva, García-Sánchez, & Gómez-Amor, 1989). In a community sample of young women, no differences in basal values were found between the high socially anxious group ($n = 28$) compared with the low socially anxious group ($n = 28$); indeed, reduced heart rate reactivity to a speech task was found for the high socially anxious group (Gramer & Sprintschnik, 2008). Finally, a student sample of individuals diagnosed with SAD ($n = 24$) showed comparable blood pressure, skin conductance (levels and responses), and heart rate during rest compared with healthy controls ($n = 12$; Bergamaschi et al., 2011). In conclusion, since most of these studies had a small sample size, power problems may partially explain these null findings. Moreover, for some of these studies assessing community samples, restriction of range may have prevented detecting differences. Furthermore, with regard to null findings concerning reactivity, Schmitz et al. (2011) suggest that reduced parasympathetic modulation may result in elevated arousal during baseline combined with nondeviant reactivity to a stressor. Hence, studies that do not analyze baseline differences may overlook relevant group differences.

Blushing

Information about the blushing response during childhood and its association with social anxiety is scarce. Based on self-reports from parents (Buss, Iscoe, & Buss, 1979) and children (aged 4–9 years; Colonnesi, Engelhart, & Bögels, 2010), children are able to blush from the age of 3–4 years. Already at the age of 4, self-reported blushing in children is associated with the ability to attribute embarrassment to people who are in the center of attention (Colonnesi et al., 2010). In a study of children aged 6–17 years diagnosed with an anxiety disorder, blushing was reported by 50% of the children, which was more frequently than, for example, trembling (Ginsburg, Riddel, & Davies, 2006). Hence, blushing seems to be a prominent feature of anxiety in childhood. No studies were found on physiological measurement of blushing in children. At present, in our lab, we study the blushing response in children aged 4.5 years during a social performance task in relation with their BI, shyness (e.g., Colonnesi, Napoleone, & Bögels, under review), and their parents' social anxiety.

Support for increased blushing in social anxiety has been obtained among adults. Adults diagnosed with SAD reported more physiological symptoms, including blushing, but also trembling and sweating, than controls (Bögels & Reith, 1999). With respect to physiological blushing responses, measured by blood flow (via a plethysmograph) and temperature, high socially anxious individuals showed greater blushing during a conversation with an unknown person than low socially anxious individuals

(Bögels et al., 2002). Similar physiological blushing results were found in high versus low socially anxious individuals while reviewing video recordings of themselves singing, while unknown peers observed the video as well (Drummond & Su, 2012; Gerlach, Wilhelm, Gruber, & Roth, 2001). In line, shy people who also reported elevated social anxiety, demonstrated a stronger objectively measured blushing response than nonshy people to social performance and watching this performance together with a stranger (Hofmann et al., 2006). In another study, delayed recovery of the blushing response (blood flow) rather than enhanced blushing reactivity to social interaction tasks (conversation, speech, listening to the speech) has been observed in participants high versus low on fear of blushing (Drummond et al., 2007). In a clinical study, referred patients with SAD showed greater physiological blushing (assessed with temperature and plethysmograph; significant on temperature only) during a conversation task and a speech task than controls (Voncken & Bögels, 2009).

In sum, children may blush as young as the age of 4, which appears related to the (developing) ability to see themselves from the perspective of another person. In children, initial research shows that self-reported blushing is more characteristic for children with high levels of anxiety. Among adults, blushing during various socially stressful situations—including embarrassing situations, performance situations, and interactions with strangers—is found to be a characteristic physiological response in individuals high on social anxiety or shyness, and in patients with SAD, particularly those with fear of blushing as the predominant complaint. However, studies comparing blushing between patients with other anxiety disorders have not been performed yet; hence, it is not yet clear whether blushing is specific for social anxiety and SAD, or general to overall anxiety symptoms.

Physiological Aspects Related to the Etiology and/or Maintenance of Social Anxiety

One core feature of social anxiety that is of particular interest with respect to its pathophysiology is *anxiety sensitivity.* Anxiety sensitivity refers to the fear of anxiety experiences, due to the expected harmful consequences, such as illness or embarrassment (Reiss, 1991). Within anxiety sensitivity, three domains are distinguished: physical, social, and psychological concerns (Zinbarg, Mohlman, & Hong, 1999). Anxiety sensitivity is regarded as a causal and maintaining factor of SAD fostering avoidance behavior (Reiss, Peterson, Gursky, & McNally, 1986). One hypothesis centering the role of physiological aspects in the etiology and maintenance of anxiety in general is the "enhanced reactivity" hypothesis formulated by Reiss and McNally (1985). This hypothesis states that experience with higher autonomic reactivity in response to stress will increase the risk of developing concerns about arousal-related bodily sensations. With respect to social anxiety, higher autonomic reactivity characteristic of social anxiety in response to social situations may increase the risk of developing concerns about social consequences. Indeed, the social concerns dimension of anxiety sensitivity is typical of SAD (Naragon-Gainey & Watson, 2011). These social concerns involve the fear of drawing attention to oneself when showing bodily fear symptoms, with

blushing, sweating, and trembling as prominent examples (e.g., Capozzoli, Vonk, Bögels, & Hofmann, 2012). Of note, physical concerns, for example, *fear of hyperarousal*, are also found in SAD, but are more strongly related to and prototypical of panic disorder (Naragon-Gainey, 2010).

Stewart, Buffett-Jerrott, and Kokaram (2001) suggested that, since stronger physiological arousal and more experience with autonomic reactivity are both likely to result in interoceptive awareness, interoceptive awareness may act as a mediator in the association between physiological reactivity and anxiety sensitivity. Hence, more accurate perception of heart beat may be expected in socially anxious individuals. In support of this, Stevens et al. (2011) found that in 48 students screened with the Fear of Negative Evaluation Scale (Watson & Friend, 1969) the highly socially anxious group demonstrated better accuracy when estimating their own heart rate during baseline and during speech task preparation than the low socially anxious group. Initial support for the idea that interoceptive awareness improves when physiological arousal increases was obtained, since the increase in heart rate during the speech preparation task analyzed in the sample as a whole resulted in more accurate heart rate perception during this task than during baseline (marginally significant result; Stevens et al., 2011). Since groups did not differ in objective physiological reactivity, the authors stated that improved interoceptive awareness in the high socially anxious group may have resulted from more experience with autonomic reactivity rather than from enhanced reactivity. Another study reported similar results; students high in anxiety sensitivity demonstrated better heart rate estimation accuracy than students low in anxiety sensitivity (Stewart et al., 2001). As arousal induction did not affect accuracy, the hypothesis that higher reactivity leads to better accuracy was not confirmed, and the improved accuracy may result from more experience with autonomic reactivity. However, the authors note that the power to test this effect was low ($n = 15$ per group). By contrast, the study of Antony et al. (1995) found support for the hypothesis that higher arousal results in better heart beat perception accuracy in their study comparing panic disorder patients with SAD patients and healthy controls. While no between-group differences were found in basal physiological arousal, reactivity to physical exercise, or perception accuracy, the increased physiological arousal induced by physical exercise was associated with better perception accuracy. A study in children suggests that enhanced interoceptive awareness in social anxiety is not yet present in childhood, since no difference in heart beat perception accuracy was found between high versus low socially anxious children aged 10–12 years (Schmitz et al., 2012). This finding thus suggests that enhanced interoceptive awareness develops through experience.

In sum, initial evidence supports a mediating role of interoceptive awareness in the association between enhanced physiological arousal and anxiety sensitivity. Hence, the development of interoceptive awareness seems to reflect an etiological mechanism as well as a maintaining mechanism of social anxiety. However, it should be noted that in none of the studies reporting enhanced interoceptive awareness in social anxiety was a positive association demonstrated between physiological arousal and anxiety or anxiety sensitivity. This may result from the fact that the stressors used in these studies did not induce different levels of arousal in different diagnostic groups. For future studies assessing associations between physiological arousal, interoceptive

awareness, and social anxiety, it is recommended to use social stressors which induce considerable arousal.

Alternatively, etiological models, including the one of Clark and Wells (1995), stress the role of excessive perception of feared anxiety responses, such as blushing, trembling, or sweating, due to extensive self-focused attention. Bögels et al. (2002) points out that awareness of blushing, trembling, and sweating serves to focus attention inward, and that this heightened self-focused attention, in turn, heightens experienced social anxiety and concomitant physiological reactions. A few studies about self-focused attention report on its maintaining role in social anxiety by its effect on physiological fear responses. Results of these studies are somewhat inconsistent. In one study, negative self-focused cognitions were found to mediate the association between trait social anxiety and heart rate variability (Schulz, Alpers, & Hoffman, 2008). In another study, however, self-focused attention, as manipulated by having participants see their mirror image, did not affect the relation between social anxiety and blushing (Bögels et al., 2002). A possible explanation for this lack of support for the hypothesis that self-focused attention increases blushing, as offered by the authors, is that the mirror gave participants corrective feedback about their blushing, thereby neutralizing the heightening effect on self-focus that a mirror typically has.

In conclusion, self-focused attention is a likely maintaining mechanism of physiological responses, such as blushing, trembling, and sweating, in social anxiety. No studies have focused yet on the mediating role of self-focused attention in the association between physiological arousal and anxiety sensitivity as an etiological mechanism for social anxiety. Hence, future research may examine whether the tendencies to have higher basal physiological arousal or to react with enhanced physiological arousal in social situations may play a role in the etiology of SAD because of mutually strengthening effects between heightened arousal on the one hand and self-focused attention on the other.

Physiology Related to Possible Subtypes of Social Anxiety Disorder

Various subtypes of SAD are distinguished in the social anxiety literature, including generalized SAD versus nongeneralized, specific, or circumscribed SAD (e.g., Hook & Valentiner, 2002). In generalized SAD, an individual fears "most social situations" (APA, 2000), whereas in specific SAD, an individual fears a limited number, or even just one, social situation (Hook & Valentiner, 2002). Specific SAD is typically characterized by fear of performance situations such as public speech, while generalized SAD is tied to both performance fear and fear of social interactions, such as having a conversation with someone unfamiliar (e.g., Cox, Clara, Sareen, & Stein, 2008; Hook & Valentiner, 2002). A distinction between generalized SAD (social interaction anxiety and performance fear) and specific SAD (mainly performance fear) is indeed supported by group differences in personality dimensions, etiological characteristics, and anxiety sensitivity (see Naragon-Gainey, 2010; Naragon-Gainey & Watson, 2011,

for reviews). Social interaction anxiety, typical of generalized SAD, is characterized by low levels of positive emotionality (Hughes et al., 2006), high levels of negative affect (Norton, Cox, Hewitt, & McLeod, 1997), high levels of self-critical evaluations of inferiority and worthlessness (Hook & Valentiner, 2002; Norton, Buhr, Cox, Norton, & Walker, 2000), and increased anxiety sensitivity, in particular social (i.e., others observing one's anxiety) concerns (Naragon-Gainey, 2010). Individuals with generalized SAD also report more relatives with SAD (Mannuzza et al., 1995). By contrast, performance anxiety, typical of specific SAD, is related to increased anxiety sensitivity, in particular social *and* physical (e.g., hyperarousal) concerns (Hughes et al., 2006; Naragon-Gainey, 2010; Norton et al., 1997; Olantunji & Wolitzky-Taylor, 2009), and appears unrelated to positive emotionality (Hughes et al., 2006). Of note, performance anxiety is more strongly related to anxiety sensitivity than is generalized SAD (Naragon-Gainey, 2010). With respect to etiology, performance anxiety has a smaller genetic component than generalized SAD, is less strongly associated with BI and shyness during childhood (see Bögels et al., 2010, for a review), has a later age of onset (Mannuzza et al., 1995; McTeague et al., 2009), and is particularly responsive to beta-blockers (Davidson, 2003; Kenny, 2005). Moreover, evidence suggests that conditioning experiences rather than predisposition explain the etiology of performance anxiety (see Bögels et al., 2010).

Physiological indices support these subtypes to some extent. For example, specific SAD (mainly performance anxiety) appears to be more strongly related than generalized SAD (performance and interaction anxiety) to hyperarousal during performance situations, measured by greater heart rate responses when delivering a speech (Boone et al., 1999; Heimberg et al., 1990; Hofmann et al., 1995; Levin et al., 1993; but see Turner, Beidel, & Townsley, 1992, for conflicting results), or larger startle responses, skin conductance levels, and heart rate responses to imagining their most feared performance situation (McTeague et al., 2009; but no differences in startle responses to predefined social fear situations including performance situations). Concerning *basal* arousal, though, generalized SAD was characterized by more prominent hyperarousal, measured by higher heart rate during baseline, in comparison with specific SAD and controls (McTeague et al., 2009). Taken together, performance fear seems to be characterized by more pronounced hyperarousal during performance situations (e.g., increased heart rate, higher skin conductance, trembling), and fear that others will detect such anxiety signals which may maintain performance anxiety. Generalized SAD, by contrast, seems to be characterized by basal hyperarousal and no excessive reactivity to social situations on top of that. These distinct physiological and psychological profiles have resulted in the proposal to include the specifier *performance only* (rather than specific SAD) in the DSM-V, for those SAD patients who predominantly suffer from performance fears (Bögels et al., 2010).

Several authors have suggested distinguishing yet another subtype of social anxiety: *social anxiety due to showing anxiety symptoms*, such as blushing, sweating, or trembling (e.g., Pelissolo, Moukheiber, Lobjoie, Valla, & Lambrey, 2011; Voncken & Bögels, 2009). Voncken and Bögels (2009) compared patients with fear of blushing as the primary source of fear with other SAD patients (both groups were diagnosed with generalized SAD), during a social interaction with strangers and a social performance task. They found that SAD patients suffering from fear of blushing showed higher

physiological blushing (assessed with temperature, but no overall difference was found in blood flow) than SAD patients without this primary fear, and were identified by independent observers as blushing more often and more intensely. The only other study that specifically studied SAD patients with and without blushing fear (Gerlach et al., 2001) did not find such physiological differences between these SAD groups; however, these null results may have been due to relatively low power as group sizes were small. Two studies have found indications for hyperarousal associated with fear of blushing. First, patients with erythrophobia (i.e., fear of blushing) showed higher heart rate than healthy controls, and higher sympathetic and parasympathetic activity assessed using heart rate variability during a mental stress condition (i.e., a Stroop task; Laederach-Hofmann, Mussgay, Büchel, Widler, & Rüddel, 2002). The authors suggest that the seemingly contradicting finding of elevated parasympathetic activity may be due to different breathing patterns between groups. Taken together, the results are still indicative of hyperarousal. Moreover, SAD patients with fear of blushing demonstrated a higher heart rate response during a social task than SAD patients without such fear (Gerlach et al., 2001). Of note, though, in the study of Voncken and Bögels (2009), no difference in skin conductance response was found between generalized SAD patients with and without fear of blushing, suggesting that the sympathetic response did not differ between these groups. However, a difference in arousal may have occurred due to reduced parasympathetic activation, which was not tested in this study.

The etiology of SAD with fear of blushing is, similar to performance anxiety, associated with lower levels of BI (assessed retrospectively), later age of onset, higher self-esteem, and less comorbidity than SAD without fear of blushing (Pelissolo et al., 2011). Moreover, similar to performance anxiety, fear of blushing is characterized by more conditioning experiences than social anxiety without fear of blushing (Mulkens & Bögels, 1999). Thus, with respect to physiology and etiology, fear of blushing seems to bear more similarity to specific (i.e., performance fear) SAD than to generalized (i.e., social interaction fear either combined or not combined with performance fear) SAD. However, since performance fear is tied to specific situations (i.e., performance situations) and fear of blushing is not restricted to one type of situation, they are considered to represent different subtypes of SAD. In line with this, *fear of showing anxiety symptoms* was considered as a specifier in the Social Anxiety Disorder workgroup for the DSM-V (Bögels et al., 2010). However, it was concluded that more research is needed before its recognition as a specifier is justified.

Pathophysiology of Social Anxiety Across Cultures

In a recent review on the cultural aspects of social anxiety, Hofmann, Ansnaani, and Hinton (2010) concluded that the prevalence of SAD varies widely across cultures, with Asian, European, and some African samples having the lowest rates, US samples having higher rates, and some Russian samples having the highest rates. Despite the relatively large body of research on cross-cultural aspects of and differences in social anxiety (see **Chapter 11** for expanded discussion), studies addressing physiological aspects of social anxiety across cultures are scarce. Most studies have focused

on differences in cognitions related to cultural values regarding social interaction, and consider factors such as collectivistic versus individualistic cultural orientations, social norms, self-construals, and gender roles (Hofmann et al., 2010). The available evidence suggests that, due to different social values in collectivistic versus individualistic cultures, individuals in collectivistic cultures, which have a strong orientation toward maintaining harmony within the group, are more prone to develop social anxiety because violation of these social values is considered less acceptable. Indeed, some studies have noted that social anxiety symptoms are reported more frequently on self-report measures in East Asia, characterized by collectivistic cultures, than in the United States and Europe, which are considered individualistic cultures (Heinrichs et al., 2006; Hong & Woody, 2007; Rapee et al., 2011). The discrepancy between the higher self-report rates of social anxiety and the lower prevalence rates of SAD in East Asian cultures may stem from a tendency for countries with a collectivistic orientation to be more accepting of social anxiety symptoms, despite being less accepting of violations of social rules (Lewis-Fernández et al., 2010). This is consistent with the findings of Heinrichs et al. (2006) and Rapee et al. (2011) that collectivistic countries are more accepting toward socially reticent and withdrawn behaviors.

An example of a culturally relevant type of social anxiety is taijin kyofusho (TKS; see also **Chapter 11**). TKS is a common form of SAD in East Asian cultures, and involves the *fear of showing anxiety symptoms* (Lewis-Fernández et al., 2010). One subtype of TKS, the offensive subtype, involves the fear of displaying anxiety symptoms that offend or embarrass *others*, whereas most Western manifestations of SAD involve embarrassment of oneself (Kleinknecht, Dinnel, Kleinknecht, Hiruma, & Harada, 1997). To our knowledge, there are no studies comparing the pathophysiology of patients with SAD and TKS, but research indicating similarity in nosology and in pharmacotherapeutic effects suggest similar physiological underpinnings (Stein, 2009). Therefore, it is likely that individuals with TKS, like those with SAD, have increased self-focused attention, which in turn leads to hyperarousal in social situations. In that sense, physiology may be a maintaining mechanism of TKS, like SAD, whereas the interplay between cultural norms and self-focused attention affects its etiology.

From the above evidence, it can be inferred that the cultural context in which individuals grow up exerts a large influence on the etiology and maintenance of social anxiety (Hofmann et al., 2010; Lewis-Fernández et al., 2010), and thus, on their physiological reactivity in different social contexts. However, direct evidence of cross-cultural differences in physiological predispositions of social anxiety is limited. In general, research examining cross-cultural differences in emotions has found differences in emotional experience and expression across cultures, but not in physiology. For instance, Soto, Levenson, and Ebling (2005) found that, in response to acoustic startle, Asian Americans reported experiencing less emotion than Mexican Americans, but showed no differences in physiological responses. This suggests that observed cultural differences in emotional responding, including showing and experiencing excessive anxiety in social situations, do not involve cultural differences in physiological underpinnings.

Several studies addressed blushing in different cultures. Heinrichs et al. (2006) found that self-reported fear of blushing (including blushing frequency) was higher

in Asiatic countries than in Western countries, in line with the larger concern with violation of social rules found in collectivistic countries. Drummond and Lim (2000) addressed whether people with dark skin and fair skin differed in their experience and tendency of blushing, as blushing is difficult to detect in dark-skinned people. No support was found for a different predispositional tendency of blushing between dark-skinned and fair-skinned people; Indians and Caucasians demonstrated a similar increase in facial skin temperature and blood flow during a singing task. Blushing was, however, significantly related to self-consciousness in Caucasians but not in Indians, and Caucasians could predict their blushing with greater accuracy. This suggests that skin tone affects self-consciousness and interoceptive awareness during social situations. Hence, the better visibility of a major signal of embarrassment may place Caucasians at increased risk for developing SAD. This is in line with the larger prevalence of SAD among white Americans, relative to African Americans, Asian Americans, and Latino Americans reported by Asnaani, Richey, Dimaite, Hinton, and Hofmann (2010).

With respect to cross-cultural effects on interoceptive awareness, Ma-Kellams, Blascovich, and McCall (2012) found that Asian Americans are less sensitive to internal physiological cues than European Americans. Using tests of misattribution of arousal, including false heart rate feedback and a heartbeat detection task, they showed that Asian Americans were less viscerally perceptive, that is, less accurate in identifying their own internal physiological state. They also demonstrated that this link between culture and visceral perception was mediated by contextual dependency (i.e., the inclination to attend to and rely on contextual factors, which is characteristic for individuals in collectivistic cultures), such that Asians were more attentive to and accurate in detecting contextual cues, presumably at the expense of attention to their internal state. The findings of this study suggest that Asians may be less prone to anxiety sensitivity and thus less likely to develop SAD, in line with the lower rates of SAD reported in Asian cultures (Lewis-Fernández et al., 2010).

A line of research pointing to cross-cultural differences in physiological aspects of social anxiety addresses the relation of child BI with hair pigmentation and eye color. Caucasian children with blue eyes (e.g., Rosenberg & Kagan, 1987) and blond hair (Moehler, Kagan, Brunner, et al., 2006) have been found to score higher on BI. Moehler, Kagan, Brunner, et al. (2006) suggest a physiological explanation for this link, in which hormones related to hair and skin pigmentation are coproduced with hormones related to cortisol, which play a role in the threshold for physiological arousal. These studies suggest that cross-cultural differences in child BI may to some extent be based on actual differences at the physiological level that underlie both physical appearance and anxiety-related physiological arousal.

In sum, because the fear to be evaluated by others, the defining feature of social anxiety, is linked to social standards and role expectations which are culture-dependent (Hofmann et al., 2010), it is important to consider cultural aspects of social anxiety. Examples include the etiology and nature of TKS, the possible differential role of blushing in social anxiety in people of varying skin color, and culturally bound differences in interoceptive awareness which may relate to social anxiety. Few, if any, studies have addressed cross-cultural differences in actual physiology underlying social

anxiety, but studies addressing emotional responding suggest few differences at that level. Given the abundance of evidence of socio-cultural influences on social anxiety, there is now a strong need for research linking these cross-cultural factors to physiological underpinnings of social anxiety.

Summary and Future Directions

When taking a developmental perspective on the pathophysiology of social anxiety, which in its more extreme form results in SAD, the findings discussed in this chapter suggest that, already in infancy, a fearful temperament is characterized by a lower threshold of the amygdala to react to potentially threatening situations resulting in hyperarousal and poorer physiological adaptation to new stimuli. Extensive evidence for hyperarousal associated with higher levels of social anxiety and SAD is found during childhood as well as adulthood. The physiological reaction of blushing appears to be related to social anxiety. Although blushing is reported in early childhood, the development of blushing in association with social anxiety has not been clarified yet. For example, it is unclear whether children who blush at an early age are more susceptible for the development of SAD. Furthermore, specificity of the blushing response for social anxiety as opposed to other anxieties needs to be confirmed by empirical testing.

Concerning etiological and maintaining mechanisms of social anxiety, results provide initial support for a role of enhanced physiological arousal in the development of interoceptive awareness and anxiety sensitivity, and for a role of self-focused attention in the endurance and aggravation of physiological anxiety responses. However, studies including physiological measures are scarce; hence, more studies assessing mechanisms underlying the development and maintenance of social anxiety are required.

Together with etiological characteristics, physiological profiles add to the subtyping or refining of the phenomenology of SAD. In sum, performance fear, typical of specific SAD, seems to be characterized by more pronounced hyperarousal during performance situations, whereas generalized SAD seems to be characterized by basal hyperarousal and less so by enhanced physiological reactivity to social situations. Whether fear of showing physiological symptoms has to be considered as a specifier in the DSM needs further validation.

Concerning cultural differences, one line of research points toward a predisposed tendency to react more strongly with physiological arousal to potentially threatening situations associated with cultures characterized by blond hair and blue eyes. One study suggests that skin tone affects self-consciousness and interoceptive awareness; darker skin tones may reduce the development of anxiety sensitivity (social type) and thereby decrease the likelihood of developing SAD in relevant cultures. Another study suggests that Asians have less interoceptive awareness, which may reduce the risk of developing anxiety sensitivity and thereby decrease the likelihood of developing SAD in Asians.

Overall, it can be concluded that the number of studies including physiological anxiety measures to study social anxiety and SAD is modest. Additional studies on physiological aspects of social anxiety may help to further explain the development

and maintaining mechanisms of SAD and cross-cultural differences in prevalence rates of social anxiety and SAD.

References

Asnaani, A., Richey, J., Dimaite, R., Hinton, D., & Hofmann, S. (2010). A cross-ethnic comparison of lifetime prevalence rates of anxiety disorders. *Journal of Nervous and Mental Disease, 198*, 551–555. doi:10.1097/NMD.0b013e3181ea169f

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

Anderson, E. R., & Hope, D. A. (2009). The relationship among social phobia, objective and perceived physiological reactivity, and anxiety sensitivity in an adolescent population. *Journal of Anxiety Disorders, 23*, 18–26. doi:10.1016/j.janxdis.2008.03.011

Antony, M., Brown, T., Craske, M., Barlow, D., Mitchell, W., & Meadows, E. (1995). Accuracy of heartbeat perception in panic disorder social phobia, and non-anxious subjects. *Journal of Anxiety Disorders, 9*, 355–371. doi:10.1016/0887-6185(95)00017-I

Bergamaschi, M. M., Queiroz, R. H., Chagas, M. H., de Oliveira, D. C., De Martinis, B. S., Kapczinski, F., . . . Crippa, J. A. (2011). Cannabidiol reduces the anxiety induced by simulated public speaking in treatment-naïve social phobia patients. *Neuropsychopharmacology, 36*, 1219–1226. doi:10.1038/npp.2011.6

Bögels, S., Alden, L., Beidel, D., Clark, L., Pine, D., Stein, M. B., & Voncken, M. (2010). Social anxiety disorder: Questions and answers for the DSM-V. *Depression and Anxiety, 27*, 168–189. doi:10.1002/da.20670

Bögels, S., & Reith, W. (1999). Validity of two questionnaires to assess social fears: The Dutch social phobia and anxiety inventory and the blushing, trembling and sweating questionnaire. *Journal of Psychopathology and Behavioral Assessment, 21*, 51–66. doi:10.1023/A:1022812227606

Bögels, S., Rijsemus, W., & de Jong, P. (2002). Self-focused attention and social anxiety: The effects of experimentally heightened self-awareness on fear, blushing, cognitions and social skills. *Cognitive Therapy Research, 26*, 461–472. doi:10.1023/A:1016275700203

Boone, M., McNeil, D., Masia, C., Turk, C., Carter, L., Ries, B. J., & Lewin, M. R. (1999). Multimodal comparisons of social phobia subtypes and avoidant personality disorder. *Journal of Anxiety Disorders, 13*, 271–292. doi:10.1016/S0887-6185(99)00004-3

Bradley, M. M., & Lang, P. J. (2007). Emotion and motivation. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds.), *Handbook of psychophysiology* (3rd ed., pp. 581–607). New York, NY: Cambridge University Press.

Brownley, K., Hurwitz, B., & Schneiderman, N. (2000). Cardiovascular psychophysiology. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds.), *Handbook of psychophysiology* (2nd ed., pp. 224–264). New York, NY: Cambridge University Press.

Buss, A., Iscoe, I., & Buss, E. (1979). The development of embarrassment. *Journal of Psychology, 103*, 227–230. doi:1140-1979-103-02-000010

Cannon, W. B. (1914). The emergency function of the adrenal medulla in pain and the major emotions. *American Journal of Physiology, 33*, 356–372.

Cannon, W. B. (1929). The sympathetic division of the autonomic system in relation to homeostasis. *Archives of Neurology & Psychiatry, 22*, 282–294. doi:10.1001/archneurpsyc.1929.02220020098005

Capozzoli, M. C., Vonk, I. J., Bögels, S. M., & Hofmann, S. G. (2012). Psychological interventions for fear of blushing. In W. R. Crozier & P. J. De Jong (Eds.), *The psychological significance of the blush*. New York, NY: Cambridge University Press.

Clark, D., & Wells, A. (1995). A cognitive model of social phobia. In R. Heimberg, M. Liebowitz, D. Hope, & F. Schneier (Eds.), *Social phobia. Diagnosis, assessment, and treatment* (pp. 69–93). New York, NY: Guilford Press.

Colonnesi, C., Engelhart, I., & Bögels, S. (2010). Development in children's attribution of embarrassment and the relationship with theory of mind and shyness. *Cognition and Emotion, 24*, 514–521. doi:10.1080/02699930902847151

Colonnesi, C., Napoleone, E., & Bögels, S. Toddlers' positive and negative expression of shyness and the relation to anxiety (under review).

Cornwell, B., Heller, R., Biggs, A., Pine, D., & Grillon, C. (2011). Becoming the center of attention in social anxiety disorder: Startle reactivity to a virtual audience during speech anticipation. *The Journal of Clinical Psychiatry, 72*, 942–948. doi:10.4088/JCP.09m05731blu

Cornwell, B., Johnson, L., Berardi, L., & Grillon, C. (2006). Anticipation of public speaking in virtual reality reveals a relationship between trait social anxiety and startle reactivity. *Biological Psychiatry, 59*, 664–666. doi:10.1016/j.biopsych.2005.09.015

Cox, B., Clara, I., Sareen, J., & Stein, M. (2008). The structure of feared social situations among individuals with a lifetime diagnosis of social anxiety disorder in two independent nationally representative mental health surveys. *Behavior Research and Therapy, 46*, 477–486. doi:10.1016/j.brat.2008.01.011

Davidson, J. (2003). Pharmacotherapy of social phobia. *Acta Psychiatrica Scandinavica, 108*, 65–71. doi:10.1034/j.1600-0447.108.s417.7.x

Dawson, M., Schell, A., & Filion, D. (2007). The electrodermal system. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds.), *Handbook of psychophysiology* (3rd ed., pp. 159–181). New York, NY: Cambridge University Press.

Drummond, P., Back, K., Harrison, J., Helgadottir, F., Lange, B., Lee, C., . . . , Wheatley, L. (2007). Blushing during social interactions in people with a fear of blushing. *Behavioral Research and Therapy, 45*, 1601–1608. doi:10.1016/j.brat.2006.06.012

Drummond, P., & Lim, H. (2000). The significance of blushing for fair- and dark-skinned people. *Personality and Individual Differences, 29*, 1123–1132. doi:10.1016/S0191-8869(99)00259-7

Drummond, P., & Su, D. (2012). The relationship between blushing propensity, social anxiety and facial blood flow during embarrassment. *Cognition and Emotion, 26*, 561–567. doi:10.1080/02699931.2011.595775

Fowles, D., Kochanska, G., & Murray, K. (2000). Electrodermal activity and temperament in preschool children. *Psychophysiology, 37*, 777–787. doi:10.1111/1469-8986.3760777

Fox, N. (1989). Psychophysiological correlates of emotional reactivity during the first year of life. *Developmental Psychology, 25*, 364–372. doi:10.1037/0012-1649.25.3.364

Fox, N., Henderson, H., Marshall, P., Nichols, K., & Ghera, M. (2005). Linking biology and behavior within a developmental framework. *Annual Review of Psychology, 56*, 235–262. doi:10.1146/annurev.psych.55.090902.141532

Garcia-Koll, C., Kagan, J., & Reznick, J. S. (1984). Behavioral inhibition in young children. *Child Development, 55*, 1005–1019. doi:10.1111/1467-8624.ep12427052

Garner, M., Clarke, G., Graystone, H., & Baldwin, D. (2011). Defensive startle response to emotional social cues in social anxiety. *Psychiatry Research, 186*, 150–152. doi:10.1016/j.psychres.2010.07.055

Gerlach, A., Wilhelm, F., Gruber, K., & Roth, W. T. (2001). Blushing and physiological arousability in social phobia. *Journal of Abnormal Psychology, 110*, 247–258. doi:10.1037/0021-843X.110.2.247

Gerlach, A., Wilhelm, F., & Roth, W. (2003). Embarrassment and social phobia: The role of parasympathetic activation. *Anxiety Disorders, 17*, 197–210. doi:10.1016/S0887-6185(02)00197-4

Ginsburg, G., Riddle, M., & Davies, M. (2006). Somatic symptoms in children and adolescents with anxiety disorders. *Journal of the American Academy of Child & Adolescent Psychiatry, 45*, 1179–1187. doi:10.1097/01.chi.0000231974.43966.6e

Gramer, M., Schild, E., & Lurz, E. (2012). Objective and perceived physiological arousal in trait social anxiety and post-event processing of a prepared speaking task. *Personality and Individual Differences, 53*, 980–984. doi:10.1016/j.paid.2012.07.013

Gramer, M., & Sprintschnik, E. (2008). Social anxiety and cardiovascular responses to an evaluative speaking task: The role of stressor anticipation. *Personality and Individual Differences, 44*, 371–381. doi:10.1016/j.paid.2007.08.016

Heimberg, R., Hope, D., Dodge, C., & Becker, R. (1990). DSM-III-R subtypes of social phobia: Comparison of generalized social phobics and public speaking phobics. *Journal of Nervous and Mental Diseases, 173*, 172–179. doi:10.1097/00005053-199003000-00004

Heinrichs, N., Rapee, R., Alden, L., Bögels, S., Hofmann, S., Oh, K., & Sakao, Y. (2006). Cultural differences in perceived social norms and social anxiety. *Behaviour Research and Therapy, 44*, 1187–1197. doi:10.1016/j.brat.2005.09.006

Hirschfeld-Becker, D., Biederman, J., Henin, A., Faraone, S., Davis, S., Harrington, K., & Rosenbaum, J. F. (2007). Behavioral inhibition in pre-school children at risk is a specific predictor of middle childhood social anxiety: A five-year follow-up. *Journal of Development and Behavioral Pediatrics, 28*, 225–233. doi:10.1097/01.DBP.0000268559.34463.d0

Hirschfeld-Becker, D., Micco, J., Henin, A., Bloomfield, J., Biederman, J., & Rosenbaum, J. (2008). Behavioral inhibition. *Depression and Anxiety, 25*, 357–367. doi:10.1002/da.20490

Hofmann, S., Asnaani, A., & Hinton, D. (2010). Cultural aspects in social anxiety and social anxiety disorder. *Depression and Anxiety, 27*, 1117–1127. doi:10.1002/da.20759

Hofmann, S., Moscovitch, D., & Kim, H. (2006). Autonomic correlates of social anxiety and embarrassment in shy and non-shy individuals. *International Journal of Psychophysiology, 61*, 134–142. doi:10.1016/j.ijpsycho.2005.09.003

Hofmann, S., Newman, M., Ehlers, A., & Roth, W. (1995). Psychophysiological differences between subgroups of social phobia. *Journal of Abnormal Psychology, 104*, 224–231. doi:10.1037/0021-843X.104.1.224

Hong, J., & Woody, S. (2007). Cultural mediators of self-reported social anxiety. *Behaviour Research and Therapy, 45*, 1779–1789. doi:10.1016/j.brat.2007.01.011

Hook, J., & Valentiner, D. (2002). Are specific and generalized social phobias qualitatively distinct? *Clinical Psychology: Science and Practice, 9*, 379–395. doi:10.1093/clipsy.9.4.379

Hughes, A., Heimberg, R., Coles, M., Gibb, B., Liebowitz, M., & Schneier, F. (2006). Relations of the factors of the tripartite model of anxiety and depression to types of social anxiety. *Behaviour Research and Therapy, 44*, 1629–1641. doi:10.1016/j.brat.2005.10.015

Kagan, J., Reznick, J., & Snidman, N. (1987). The physiology and psychology of behavioral inhibition in children. *Child Development, 58*, 1459–1473. doi:10.1111/1467-8624.ep8591623

Kagan, J., & Snidman, N. (1991a). Infant predictors of inhibited and uninhibited profiles. *Psychological Science, 2*, 40–44. doi:10.1111/j.1467-9280.1991.tb00094.x

Kagan, J., & Snidman, N. (1991b). Temperamental factors in human development. *American Psychologist, 46*, 856–862. doi:10.1037/0003-066X.46.8.856

Kagan, J., & Snidman, N. (1999). Early childhood predictors of adult anxiety disorders. *Society of Biological Psychiatry, 46*, 1536–1541. doi:10.1016/S0006-3223(99)00137-7

Keltner, D., & Anderson, C. (2000). Saving face for Darwin: The functions and uses of embarrassment. *Current Directions in Psychological Science, 9*, 187–192. doi:10.1111/1467-8721.00091

Kenny, D. T. (2005). A systematic review of treatments for music performance anxiety. *Anxiety, Stress and Coping, 18*, 183–208. doi:10.1080/10615800500167258

Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. (1993). The "Trier Social Stress Test" – A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology, 28*, 76–81.

Kleinknecht, R., Dinnel, D., Kleinknecht, E., Hiruma, N., & Harada, N. (1997). Cultural factors in social anxiety: A comparison of social phobia symptoms and Taijin Kyofusho. *Journal of Anxiety Disorders, 11*, 157–177. doi:10.1016/S0887-6185(97)00004-2

Krämer, M., Seefeldt, W. L., Heinrichs, N., Tusschen-Caffier, B., Schmitz, J., Wolf, O. T., & Blechert, J. (2012). Subjective, autonomic, and endocrine reactivity during social stress in children with social phobia. *Journal of Abnormal Child Psychology, 40*, 95–104. doi:10.1007/s10802-011-9548-9

Laederach-Hofmann, K., Mussgay, L., Büchel, B., Widler, P., & Rüddel, H. (2002). Patients with erythrophobia (fear of blushing) show abnormal autonomic regulation in mental stress conditions. *Psychosomatic Medicine, 64*, 358–365. doi:10.1097/01.psy.0000228010.96408.ed

Leary, M., Britt, T., Cutlip, W., & Templeton, J. (1992). Social blushing. *Psychological Bulletin, 112*, 446–460. doi:10.1037/0033-2909.112.3.446

Levin, A. P., Saoud, J. B., Strauman, T., Gorman, J. M., Fyer, A. J., Crawford, R., & Liebowitz, M. R. (1993). Responses of "generalized" and "discrete" social phobics during public speaking. *Journal of Anxiety Disorders, 7*, 207–221. doi:10.1016/0887-6185(93)90003-4

Lewis-Fernández, R., Hinton, D., Amaro, J., Patterson, E., Hofmann, S., Craske, M. G., . . . , Liao, B. (2010). Culture and the anxiety disorders: Recommendations for DSM-V. *Depression and Anxiety, 27*, 212–229. doi:10.1002/da.20647

Ma-Kellams, C., Blascovich, J., & McCall, C. (2012). Culture and the body: East–west differences in visceral perception. *Journal of Personality and Social Psychology, 102*, 718–728. doi:10.1037/a0027010

Mannuzza, S., Schneier, F. R., Chapman, T. F., Liebowitz, M. R., Klein, D. F., & Fyer, A. J. (1995). Generalized social phobia: Reliability and validity. *Archives of General Psychiatry, 52*, 230–237. doi:10.1001/archpsyc.1995.03950150062011

McTeague, L., Lang, P., Laplante, M., Cuthbert, B., Strauss, C., & Bradley, M. (2009). Fearful imagery in social phobia: Generalization, comorbidity, and physiological reactivity. *Biological Psychiatry, 65*, 374–382. doi:10.1016/j.biopsych.2008.09.023

Mellander, S., Andersson, P., Afzelius, L., & Hellstrand, P. (1982). Neural beta-aderenergic dilation of the facial vein in man—Possible mechanism in emotional blushing. *Acta Physiologica Scandinavica, 114*, 393–434. doi:10.1111/j.1748-1716.1982.tb07000.x

Moehler, E., Kagan, J., Brunner, R., Wiebel, A., Kaufmann, C., & Resch, F. (2006). Association of behavioral inhibition with hair pigmentation in a European sample. *Biological Psychology, 72*, 344–346. doi:10.1016/j.biopsycho.2005.12.001

Moehler, E., Kagan, J., Oelkers-Ax, R., Brunner, R., Poustka, K., Haffner, J., & Resch, F. (2008). Infant predictors of behavioral inhibition. *British Journal of Developmental Psychology, 26*, 145–150. doi:10.1348/026151007×206767

Moehler, E., Kagan, J., Parzer, P., Wiebel, A., Brunner, R., & Resch, F. (2006). Relation of behavioral inhibition to neonatal and infant cardiac activity, reactivity and habituation. *Personality and Individual Differences, 41*, 1349–1358. doi:10.1016/j.paid.2006.05.008

Movius, H. L., & Allen, J. J. (2005). Cardiac vagal tone, defensiveness, and motivational style. *Biological Psychology, 68*, 147–162. doi:10.1016/j.biopsycho.2004.03.019

Mulkens, S., & Bögels, S. (1999). Learning history in fear of blushing. *Behaviour Research and Therapy, 37*, 1159–1168. doi:10.1016/S0005-7967(99)00022-4

Naragon-Gainey, K. (2010). Meta-analysis of the relations of anxiety sensitivity to the depressive and anxiety disorders. *Psychological Bulletin, 136*, 128–150. doi:10.1037/a0018055

Naragon-Gainey, K., & Watson, D. (2011). Clarifying the dispositional basis of social anxiety: A hierarchical perspective. *Personality and Individual Differences, 50*, 926–934. doi:10.1016/j.paid.2010.07.012

Nickell, P. V., & Uhde, T. W. (1995). Neurobiology of social phobia. In R. G. Heimberg, M. R. Liebowitz, D. A. Hope, & F. R. Schneier (Eds.), *Social phobia. Diagnosis, assessment and treatment* (pp. 113–133). New York, NY: Guilford Press.

Norton, G., Buhr, K., Cox, B., Norton, P., & Walker, J. (2000). The role of depressive versus anxiety-related cognitive factors in social anxiety. *Personality and Individual Differences, 28*, 309–314. doi:10.1016/S0191-8869(99)00099-9

Norton, G., Cox, B. J., Hewitt, P. L., & McLeod, L. (1997). Personality factors associated with generalized and non-generalized social anxiety. *Personality and Individual Differences, 22*, 655–660. doi:10.1016/S0191-8869(96)00243-7

Olantunji, B. O., & Wolitzky-Taylor, K. B. (2009). Anxiety sensitivity and the anxiety disorders: A meta-analytic review and synthesis. *Psychological Bulletin, 135*, 974–999. doi:10.1037/a0017428

Pelissolo, A., Moukheiber, A., Lobjoie, C., Valla, J., & Lambrey, S. (2011). Is there a place for fear of blushing in social anxiety spectrum? *Depression and Anxiety, 29*, 62–70. doi:10.1002/da.20851

Penttilä, J., Helminen, A., Jartti, T., Kuusula, T., Huikuri, H. V., Tulppo, M. P.,..., Scheinin, H. (2001). Time domain, geometrical and frequency domain analysis of cardiac vagal outflow: Effects of various respiratory patterns. *Clinical Physiology, 21*, 365–376. doi:10.1046/j.1365-2281.2001.00337.x

Porges, S. W. (1991). Vagal tone: An autonomic mediator of affect. In J. Garber, & K. A. Dodge (Eds.), *The development of emotion regulation and dysregulation* (pp. 111–128). New York, NY: Cambridge University Press.

Puigcerver, A., Martínez-Selva, J., García-Sánchez, F., & Gómez-Amor, J. (1989). Individual differences in psychophysiological and subjective correlates of speech anxiety. *Journal of Psychophysiology, 3*, 75–81.

Rapee, R., Kim, J., Wang, J., Liu, X., Hofmann, S. G., Chen, J.,..., Alden, L. E. (2011). Perceived impact of socially anxious behaviors on individuals' lives in Western and East Asian countries. *Behavior Therapy, 42*, 485–492. doi:10.1016/j.beth.2010.11.004

Reiss, S. (1991). Expectancy model of fear, anxiety, and panic. *Clinical Psychology Review, 11*, 141–153. doi:10.1016/0272-7358(91)90092-9

Reiss, S., & McNally, R. (1985). Expectancy model of fear. In S. Reiss & R. R. Bootzin (Eds.), *Theoretical issues in behavior therapy* (pp. 107–121). New York, NY: Academic Press.

Reiss, S., Peterson, R., Gursky, D., & McNally, R. (1986). Anxiety sensitivity, anxiety frequency and the prediction of fearfulness. *Behavioral Research and Therapy, 24*, 1–8. doi:10.1016/0005-7967(86)90143-9

Rosenbaum, J. F., Biederman, J., Bolduc-Murphy, E. A., Faraone, S. V., Chaloff, J., Hirshfeld, D. R., & Kagan, J. (1993). Behavioral inhibition in childhood: A risk factor for anxiety disorders. *Harvard Review of Psychiatry, 1*, 2–16. doi:10.3109/10673229309017052

Rosenberg, A., & Kagan, J. (1987). Iris pigmentation and behavioral inhibition. *Developmental Psychobiology, 20*, 377–392. doi:10.1002/dev.420200403

Scarpa, A., Raine, A., Venables, P., & Mednick, S. (1997). Heart rate and skin conductance in behaviorally inhibited Mauritian children. *Journal of Abnormal Psychology, 106*, 182–190. doi:10.1037/0021-843X.106.2.182

Schmitz, J., Blechert, J., Krämer, M., Asbrand, J., & Tuschen-Caffier, B. (2012). Biased perception and interpretation of bodily anxiety symptoms in childhood social anxiety. *Journal of Clinical Child & Adolescent Psychology, 41*, 92–102. doi:10.1080/15374416.2012.632349

Schmitz, J., Krämer, M., Tuschen-Caffier, B., Heinrichs, N., & Blechert, J. (2011). Restricted autonomic flexibility in children with social phobia. *Journal of Child Psychology and Psychiatry, 52*, 1203–1211. doi:10.1111/j.1469-7610.2011.02417.x

Schulz, S., Alpers, G., & Hofmann, S. (2008). Negative self-focused cognitions mediate the effect of trait social anxiety on state anxiety. *Behaviour Research and Therapy, 46*, 438–449. doi:10.1016/j.brat.2008.01.00

Soto, J. A., Levenson, R. W., & Ebling, R. (2005). Cultures of moderation and expression: Emotional experience, behavior, and physiology in Chinese Americans and Mexican Americans. *Emotion, 5*, 154–165. doi:10.1037/1528-3542.5.2.154

Stein, D. J. (2009). Social anxiety disorder in the West and in the East. *Annals of Clinical Psychiatry, 21*, 109–117.

Stevens, S., Gerlach, A., Cludius, B., Silkens, A., Craske, M., & Hermann, C. (2011). Heartbeat perception in social anxiety before and during speech anticipation. *Behavior Research and Therapy, 49*, 138–143. doi:10.1016/j.brat.2010.11.009

Stewart, S., Buffett-Jerrott, S., & Kokaram, R. (2001). Heartbeat awareness and heart rate reactivity in anxiety sensitivity: A further investigation. *Anxiety Disorders, 15*, 535–553. doi:10.1016/S0887-6185(01)00080-9

Talge, N., Donzella, B., & Gunnar, M. (2008). Fearful temperament and stress reactivity among preschool-aged children. *Infant and Child Development, 17*, 427–445. doi:10.1002/icd.585

Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. (1996). Heart rate variability: Standards of measurement, physiological interpretation and clinical use. *Circulation, 93*, 1043–1065. doi:10.1161/01.CIR.93.5.1043

Turner, S., Beidel, D., & Roberson-Nay, R. (2005). Offspring of anxious parents: Reactivity, habituation, and anxiety proneness. *Behaviour Research and Therapy, 43*, 1263–1279. doi:10.1016/j.brat.2004.09.005

Turner, S., Beidel, D., & Townsley, R. (1992). Social phobia: A comparison of specific and generalized subtypes and avoidant personality disorder. *Journal of Abnormal Psychology, 101*, 326–331. doi:10.1037/0021-843X.101.2.326

Van Brakel, A., Muris, P., & Derks, W. (2006). Eye blink startle responses in behaviorally inhibited and uninhibited children. *International Journal of Behavioral Development, 30*, 460–465. doi:10.1177/0165025406071903

Voncken, M. J., & Bögels, S. M. (2009). Physiological blushing in social anxiety disorder patients with and without blushing complaints: Two subtypes? *Biological Psychology, 81*, 86–94. doi:10.1016/j.biopsycho.2009.02.004

Watson, D., & Friend, R. (1969). Measurement of social-evaluative anxiety. *Journal of Consulting and Clinical Psychology, 33*, 448–457. doi:10.1037/h0027806

Wieser, M., Pauli, P., Alpers, G., & Mühlberger, A. (2009). Is eye to eye contact really threatening and avoided in social anxiety? – An eye-tracking and physiological study. *Journal of Anxiety Disorders, 23*, 93–103. doi:10.1016/j.janxdis.2008.04.004

Zinbarg, R., Mohlman, J., & Hong, N. (1999). Dimensions of anxiety sensitivity. In S. Taylor (Ed.), *Anxiety sensitivity, theory, research, and treatment of the fear of anxiety* (pp. 83–114). Mahwah, NJ: Lawrence Erlbaum.

6

Personality

Understanding the Socially Anxious Temperament

Cheri A. Levinson, Simona C. Kaplan, and Thomas L. Rodebaugh

Washington University in St. Louis, USA

The importance of personality to social anxiety disorder (SAD) is rapidly becoming clear. Traditionally, division of Axis I and II disorders might imply that disordered social anxiety and personality are separate constructs. Yet, social and personality psychology reserachers have routinely treated social anxiety as a personality construct (e.g., Leary, Kowalski, & Campbell, 1988). Research on the personality disorders also does not support the notion that Axis I disorders are distinct from personality (e.g., Krueger & Tackett, 2003), indicating that social anxiety and personality may be more closely related than might be assumed given a naïve read of diagnostic manuals. Indeed, the majority of findings from research specifically on SAD and avoidant personality disorder (AVPD) has suggested that any meaningful distinction is a product of severity of social anxiety and impairment (e.g., Chambless, Fydrich, & Rodebaugh, 2008; Heimberg, 1996; Heimberg, Hope, Dodge, & Becker, 1990; although see Hummelen, Wilberg, Pedersen, & Karterud, 2007; Huppert, Strunk, Ledley, Davidson, & Foa, 2008, for some evidence of a distinction between AVPD and SAD other than severity). It is worth noting, in passing, that because this line of research suggests that AVPD and SAD differ in severity but not in kind, we will include findings regarding AVPD in our review unless there is a specific reason to believe that such findings are distinct from those related to social anxiety.

Research also suggests that personality may play a role in the development and maintenance of social anxiety. For example, an inhibited temperament in children, which we view as an early indication of a high tendency toward avoidance behavior coupled with a low tendency toward approach behavior, has been found to increase risk for SAD in adolescence (e.g., Essex, Klein, Slattery, Goldsmith, & Kalin, 2010; Hirshfeld-Becker et al., 2007; see also **Chapter 7** for a more extensive review of behavioral inhibition and SAD). Further, Bienvenu, Hettema, Neale, Prescott, and Kendler (2007) found evidence that extraversion and neuroticism may be direct expressions of

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

genetic risk for SAD. Thus, personality's influence on SAD may be crucial for understanding how social anxiety develops and is maintained. Before proceeding, we will first define what we mean by the term *personality*.

Personality

Personality has been defined as "characteristics of an individual that describe and account for consistent patterns of feeling, cognition, and behavior" (Weinstein, Capitanio, & Gosling, 2008, p. 330). These patterns are often thought of as stable traits (Ewen, 2003), which might suggest that someone high in extraversion will be outgoing and talkative in every situation. However, evidence suggests that personality is better conceptualized as *patterns of behavior* than as stable traits. For example, Fleeson (2001) asked participants to carry an ecological momentary assessment (EMA) device for 1 week and report on their personality and mood several times throughout the day. He found that there was more variation in extraversion within individuals throughout the day than between individuals: even the least talkative individuals typically show considerable variation in how talkative they are across a given day. Further, Fleeson found that the degree of variation within a given individual was highly predictable and stable. Rather than having a certain set, stable point of extraversion, people may display relatively stable *distributions* of extraverted behavior during their daily lives.

Mischel, Shoda, and Mendoza-Denton (2002) argue that personality should be conceptualized as meaningful "patterns of situation–behavior relations" (p. 730). For instance, children display varied social behaviors, but these varying behaviors are nevertheless stable *within situations* (Shoda, Mischel, & Wright, 1994). Mischel et al. (2002) propose that these behavioral patterns can be conceptualized as "if . . . then" behavioral signatures (e.g., if in situation "A," then the individual does "X"). Mischel's argument implies that we must account for the context of behavior, whereas most research we will review is based on self-report measures of global personality traits. If Mischel's conceptualization is fundamentally correct (and we believe it is), then it is important to recognize that most research available has a fundamental weakness of focusing on personality as if it can be understood separately from situations. The research we review is thus just the beginning of the story of SAD and personality, because it focuses only on the relationship between the *average* of an individual's distribution of behavior and their problematic social anxiety.

A Broad Theory of Personality and Social Anxiety

We present a broad theory of personality and social anxiety, depicted in Figure 6.1, as an aid to conceptualizing the rest of the material reviewed. To date, the aspects of this theory which have been empirically tested have received support, though much of it is nevertheless speculative and awaits full testing. The first aspect of our theory is that there is no fundamental discontinuity in experience or behavior between what is typically regarded as personality and any particular level of social anxiety *per se*. That is, we expect that social anxiety and SAD symptoms operate much like a personality trait.

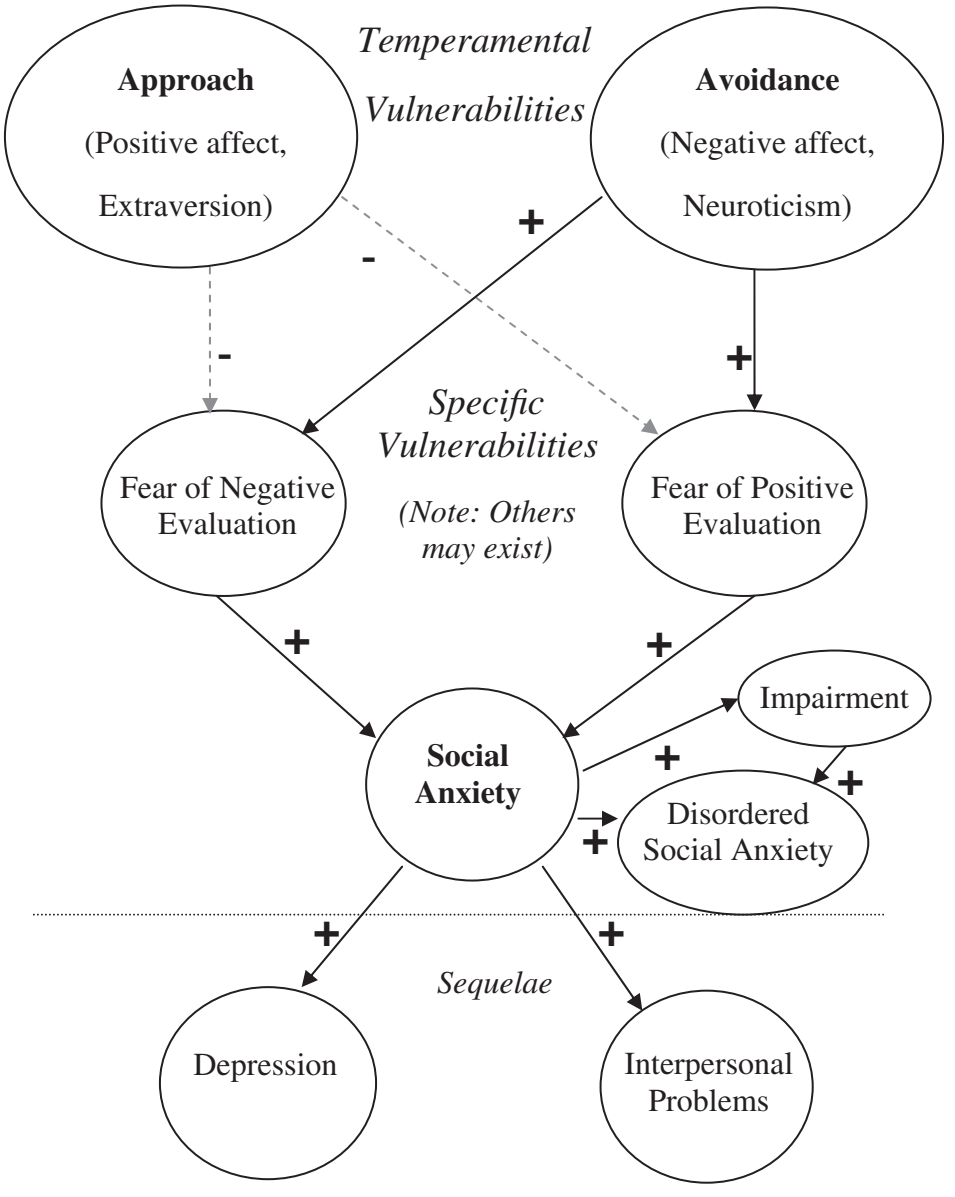

Figure 6.1 A broad model of personality's influence on social anxiety. Of note, we do not discuss specific vulnerabilities because they are less clearly defined in the literature. However, for more information on one of these vulnerabilities please see **Chapter 20** reviewing fear of positive evaluation. Minus signs/dashed lines represent a negative relationship between variables; plus signs/solid lines represent a positive relationship between variables.

On the one hand, this means that in the absence of interventions, we expect social anxiety to remain relatively stable as a profile of behavior in given contexts. On the other hand, however, the assumption that social anxiety is essentially a personality trait does not imply that social anxiety is always global, generally without trajectory over time, or not amenable to change (i.e., with treatment). As reviewed above, personality psychology researchers have, overall, accepted the proposition that personality traits are often more accurately expressed in terms of patterns of behavior, and social anxiety is no different in this respect. It has also been found that even the most stable global traits can have gradual trajectories over time (e.g., neuroticism levels tending to go down over time; Mroczek & Spiro, 2003). The propositions that social anxiety is not necessarily global (e.g., some people only fear a limited number of situations), can change over time without treatment, and can change with treatment, are fully compatible with modern conceptions of personality.

Does our initial argument suggest that SAD is a personality disorder or that there is no distinction between disordered social anxiety and normal social anxiety? For example, perhaps we ascribe to the belief, often echoed in the popular press (Cain, 2011), that SAD is an attempt to pathologize the normal personality trait of shyness (where *shyness* is a similar construct to SAD characterized by less avoidance and impairment; e.g., Chavira, Stein, & Malcarne, 2002). We reject such conclusions. Instead, we propose that the sole reliable difference between disordered social anxiety and normal social anxiety is in terms of level of impairment (e.g., inability to accomplish valued goals), which is correlated with, but not identical to, social anxiety severity. Measurement of any marker of severity of social anxiety will never allow a researcher or clinician to say with complete certainty which individuals have SAD and which do not: there will always be some individuals who report being satisfied with their lives despite what might seem to be debilitating levels of social anxiety.

Available studies regarding the dimensionality of social anxiety might seem to imply that even the distinction between problematic and nonproblematic social anxiety is only a matter of degree and not a matter of kind, but we believe that a careful reading of that literature suggests that there may additionally be a qualitative distinction. In these studies, *taxometric* analyses (e.g., Meehl, 1999) have been used to test whether social anxiety is primarily dimensional or taxonic, where the latter suggests underlying, qualitatively different groups. Kollman, Brown, Liverant, and Hofmann (2006) examined this question in a large sample of individuals diagnosed with SAD using three different types of taxometric analyses. In all three analyses, they found no evidence of underlying groups. Rather, these data supported the idea that there is a dimensional latent structure underlying social anxiety among people who have SAD. Ruscio (2010), similarly, used taxometric procedures and found that a dimensional solution for SAD was the best way to characterize social anxiety symptoms for people who had reported at least some problems with social anxiety.

In contrast, Weeks, Carleton, Asmundson, McCabe, and Antony (2010) examined the structure of social anxiety in both clinical (diagnosed with generalized SAD [GSAD]) and community/student participants (who may or may not have had SAD). These authors found evidence that social anxiety demonstrated a taxonic structure, with two groups underlying the apparently dimensional construct. The authors further found that one taxon, or group, included all participants with GSAD in addition

to undiagnosed participants, whereas the other taxon did not include any individuals with GSAD. Our interpretation of this literature is that severity of social anxiety among *people who have problems with social anxiety* is almost certainly dimensional, as it probably is among people who report no problems with social anxiety, but there may be a categorical difference between people who have trouble with social anxiety and those who do not. In this way, there may be both dimensional, as well as categorical, aspects to the construct of social anxiety in the context of SAD.

The most immediately clinically relevant of the components of our model can therefore be summarized as a continuous dimension of social anxiety that is associated with, but not subsumed by, groups defined by whether social anxiety has been classified by the individual as a personal difficulty. We further propose that the development and maintenance of the dimensional aspect of social anxiety is influenced by (a) broad temperamental factors and (b) specific vulnerability factors. In that regard, we largely echo the suggestions of other researchers who propose that there are generalized biological, generalized psychological, and specific psychological vulnerabilities underlying anxiety (e.g., Barlow, 2000). In a notable example of previous research, Kotov, Watson, Robles, and Schmidt (2007) found evidence that trait negative emotionality may be a generalized psychological vulnerability factor underlying anxiety, whereas specific vulnerabilities underlying social anxiety may be trait negative evaluation sensitivity and low positive emotionality.

Our approach is similar to that of Kotov et al. (2007), but is further informed by more recent evidence. We conceptualize the broad temperamental factors as those related to approach and avoidance. This conceptualization is similar to that of Elliot and Thrash (2002), who suggest that an approach factor consists of extraversion, positive emotionality (e.g., happiness, excitement, pride), and behavioral activation (e.g., sensitivity to rewards), whereas an avoidance factor includes neuroticism, negative emotionality (e.g., anxiety, depression), and behavioral inhibition (e.g., sensitivity to punishment). In contrast to a focus on positive and negative affect (e.g., Watson, Gamez, & Simms, 2005), a focus on approach and avoidance offers the ability to integrate the strongly compatible findings for positive and negative affect on the one hand, and extraversion and neuroticism on the other (i.e., by combining negative affect and neuroticism, as well as extraversion and positive affect). We do not examine specific vulnerability factors in as much depth in this chapter because these factors are (a) less clearly defined in the literature (i.e., many have been proposed but few fully tested), and (b) are not generally described as personality factors *per se*. However, more information on fear of positive evaluation (one of our proposed specific factors) can be found in **Chapter 20**.

In addition to broad temperamental factors and specific vulnerability factors, we propose that other constructs' relationships with social anxiety can be conceptualized primarily as *consequences* of social anxiety. For example, interpersonal problems involving personality may not be factors that produce social anxiety, but rather primarily an outcome of social anxiety; such consequences might also be referred to as *sequelae*. This interpretation of cross-sectional data seems compelling to us, but it must be noted that longitudinal work will ultimately be necessary to determine whether such factors are primarily a consequence or cause of social anxiety, or potentially are involved in a complicated reciprocal relationship with social anxiety across time.

A second type of consequence of social anxiety is not stressed here: additional symptoms of psychological disorders, such as depression. However, we include this factor in the model to demonstrate our overall impression of such factors. That is, we expect that depression arises from vulnerabilities that are shared with social anxiety as well as some that are distinct. In addition, we expect that social anxiety itself becomes a vulnerability for depression, whereas depression might not be an additional vulnerability for social anxiety (e.g., Stein et al., 2001). Although the status of depression as a clinical problem is not particularly relevant to the current chapter, it is a useful example for fully explicating the implications of our broad model. For further information on the topic of comorbidity between depression and SAD, please see **Chapter 10**. We believe that the broad model described above provides a useful conceptual framework for understanding the available research regarding personality, social anxiety, and SAD.

Personality Theories and Social Anxiety

The Five-Factor Theory

The five-factor theory (FFT) is arguably the single most influential trait theory of personality and is an attempt to summarize empirical findings on the co-variation of personality traits (McCrae & Costa, 2008). It thus offers a particularly clear way to understand personality's relationship with social anxiety. The FFT organizes personality traits into a hierarchical structure, with five personality traits representing the highest-order, broad, dimensional domains of personality (extraversion, neuroticism, openness to experience, agreeableness, and conscientiousness). Falling under each of the five factors are six lower-order *facets* (e.g., trust as a lower-order facet stemming from the higher-order domain of agreeableness) (Costa & McCrae, 1992). These traits and facets of personality are theorized to be relatively stable patterns of feelings, thoughts, and behaviors that are consistent between situations and can be quantitatively measured. However, it must be noted that measures of the five factor model typically fail to show good model fit in confirmatory factor analysis (CFA) (e.g., Vassend & Skrondal, 1997). More recent evidence suggests that the problem may be that typical CFA methods force a strict interpretation of the structure of factors and facets (Marsh et al., 2010). In other words, although available instruments probably have a structure in which items cluster under facets, which cluster under factors, the clustering is unlikely to be complete or absolute: an item measuring a facet of neuroticism is unlikely to have no relationship at all to any of the other facets or factors. We will put the issue of structure aside for the remainder of our review.

Studies are remarkably consistent regarding the relationships between social anxiety, as well as related disorders, with the big five traits of neuroticism and extraversion. Neuroticism consists of tendencies toward negative affect, particularly anxiety and depression, as well as difficulty controlling emotions and behavior. Extraversion, in contrast, refers to tendencies toward positive affect, sociability, and activity more generally. It is not difficult to speculate how social anxiety might relate to these broad constructs. More specifically, higher social anxiety (and SAD) is related to higher

levels of neuroticism and lower levels of extraversion across a wide variety of samples (Bagby, Costa, Widiger, Ryder, & Marshall, 2005; Bienvenu et al., 2004, 2007; Kotov et al., 2007; Samuel & Widiger, 2008; Trull & Sher, 1994; Watson et al., 2005). It has also been found that genetic risk for SAD is mediated by genetic risk for extraversion and neuroticism, suggesting that the genetic risk for SAD is identical to the genetic risk for (low) extraversion and (high) neuroticism (Bienvenu et al., 2007). Associations with the other FFT factors have been less consistent, but are mentioned below.

In regard to lower-order facets of personality, there is evidence that social anxiety correlates inversely with trust (a facet of *agreeableness*, which refers to likeability, pleasantness, and being harmonious in relation with others), as well as competence, self-discipline, and achievement striving (facets of *conscientiousness*, which refers to orderliness, responsibility, and attention to detail). Further, both AVPD and SAD are associated with lower scores, in comparison to participants without these conditions, on gregariousness, assertiveness, excitement seeking (facets of extraversion), and higher scores on self-consciousness, anxiousness, and vulnerability (facets of neuroticism) (Bagby et al., 2005; Bienvenu et al., 2001, 2007).

Findings regarding facets raise an issue that has generally remained unexplored: some lower-order facets relate to social anxiety in the absence of a clear relationship between their corresponding higher-order trait and social anxiety, whereas some lower-order facets do not relate to social anxiety, though their higher-order trait clearly does. For example, there is clearly a significant relationship between social anxiety and trust (lower-order facet of agreeableness), but not consistently between social anxiety and the trait of agreeableness (e.g., Bienvenu et al., 2004, did not find this relationship, although Saulsman & Page, 2004, did). Further, although SAD has a strong relationship with neuroticism, no relationship between some lower-order facets of neuroticism (e.g., immoderation) and social anxiety has been found to the best of our knowledge. These findings suggest that measurement of lower-order facets is necessary to fully understand the specific relationships between social anxiety and personality as defined by the FFT. Further, it may be that if there are sporadic findings between personality and social anxiety (i.e., occasional findings of correlations with agreeableness or conscientiousness), these may be driven by lower-order facets (i.e., trust or competence). We find it plausible that relationships with the lower-order facets associated with agreeableness and conscientiousness may be consequences (i.e., low trust), rather than causes, of higher social anxiety (whereas we expect that neuroticism and extraversion are causes, and perhaps additionally consequences). However, the longitudinal work needed to establish temporal precedents for these facets simply has not been done.

In fact, because of a general lack of longitudinal data in this area, temporal precedence is a generally unresolved issue. The research that has been conducted (Brown, 2007) found that a combination of (higher) neuroticism/behavioral inhibition (BI) and (lower) positive affect/behavioral activation (BA) prospectively predicted less improvement in SAD symptoms over time in patients with SAD. Interestingly, Brown also found a relationship in the other direction (social anxiety to neuroticism/BI) suggesting a reciprocal relationship between neuroticism and social anxiety. Research by Bienvenu et al. (2007) implies that neuroticism and extraversion lead to social anxiety,

because it is difficult to understand how genetic risk for neuroticism and extraversion could mediate genetic risk unless these two personality traits exist in some form earlier than SAD. That is, neuroticism and extraversion must either precede SAD or be highly correlated with another factor that precedes SAD.

A second unresolved issue is the potential for interactions between personality factors or facets. In our review of the literature, we have found no research directly examining any interactive relationships between personality factors or facets and social anxiety. However, Degnan and Fox (2007) postulated that there are important resilience (or moderating) characteristics that interact with temperamental reactivity (i.e., negative affect) and lead to anxiety pathology. In support of this notion, Lonigan and Vasey (2009) found that high levels of negative affect and low levels of self-control predicted the highest levels of attention to threatening stimuli. It therefore seems plausible that there may be interactions between traits and facets in the five-factor model that may impact levels of social anxiety. Indeed, in our laboratory we have preliminarily found support for an interaction between openness and trust predicting social anxiety, such that individuals low in both trust and openness are at the greatest risk for high levels of social anxiety (Kaplan, Levinson, Rodebaugh, Menatti, & Weeks, 2012).

Trait-Affect Theory

Although trait-affect theories are not always described in terms of personality, these theories imply that people have a trait-like tendency to experience certain classes of affect more or less frequently. Most of this work has been dominated by positive and negative affect, which are strikingly similar to extraversion and neuroticism. This line of research has focused on the tripartite model of anxiety and depression (Clark & Watson, 1991) that consists of three factors: Negative affect, physiological hyperarousal, and positive affect. Tests of this theory in individuals diagnosed with a wide range of anxiety and depressive disorders find that negative affect underlies all of the anxiety disorders, whereas, in SAD and depression, there is an additional unique component of low positive affect (e.g., Brown, Chorpita, & Barlow, 1998). These findings are similar to research reviewed above demonstrating that (higher) social anxiety has a relationship with (lower) extraversion, in addition to its strong relationship with neuroticism. The relationships between social anxiety and both negative and positive affect are now firmly established in the literature (e.g., Hughes et al., 2006; Kashdan, 2007; Naragon-Gainey, Watson, & Markon, 2009).

Notably, positive and negative affect are often only measured based on the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988), which, in its standard form, measures *activated* affect only. Activated affect refers to high levels of arousal in addition to how pleasant or unpleasant the emotion may be, whereas deactivated affect refers to low-arousal emotions that may also be pleasant or unpleasant (Feldman-Barrett & Russell, 1998). Examples of activated affect states are *thrilled* (activated pleasant) and *tense* (activated unpleasant), whereas examples of deactivated states are *calm* (deactivated pleasant) and *lethargic* (deactivated unpleasant). The distinction between activated and deactivated affect is based on the theory that there are two systems that motivate emotion: hedonic value (valence) and arousal (level of

motivational activation) (Bradley, Codispoti, Cuthbert, & Lang, 2001). In this model, emotion is motivated by both pleasure and arousal, and can be viewed as having four basic quadrants. The PANAS accounts for two of these quadrants (positive and negative activated affect), whereas other measures, such as the Self-Assessment Manikin (Bradley & Lang, 1994), could be used to measure all four quadrants (including positive and negative deactivated affect). Future research should consider utilizing measures of both activated and deactivated affect, lest the field be blinded by a tendency to use only a single measure, regardless of how good a measure it is.

As with the FFT, there is limited research on interactions between types of affect in predicting social anxiety. However, in contrast to research on the FFT, there is a burgeoning literature testing temporal relationships using methodologies such as EMA. These studies find a prospective relationship between social anxiety at baseline and daily positive and negative affect (Brown, Silvia, Myin-Germeys, & Kwapil, 2007; Kashdan & Steger, 2006; however, see somewhat conflicting findings from Tan et al., 2012).

The available EMA studies have begun to assess the temporal relationships of affect and social anxiety. However, with the exception of Brown (2007), who (appropriately, we believe) combined FFT and affect constructs, we have not located other research testing anxiety and affect in particular over an extended period of time (i.e., over months or years instead of days or weeks). Research assessing similar constructs has found that across 2–4 years, children's negative emotionality predicted lower social functioning (Eisenberg et al., 1997) and that low levels of positive emotionality and high levels of negative emotionality predicted shyness over time (Eggum et al., 2011). Eggum et al. also found relationships in the opposite direction: high shyness at Time 1 predicted high anger and sadness, and low positive emotionality at Time 3. Generally, the research noted above, in conjunction with broadly similar investigations (De Bolle, De Clerq, Decuyper, & De Fruyt, 2011; Lonigan, Phillips, & Hooe, 2003), provides evidence that there are temporal relationships between positive and negative affect and social anxiety. However, in many cases, there seem to be reciprocal relationships between affect and social anxiety over time.

Interpersonal Circumplex Theory

Another theory that may be important for understanding SAD in terms of personality is the interpersonal circumplex theory. The interpersonal circumplex theory conceptualizes personality as persistent interpersonal tendencies that exhibit characteristic patterns (Alden, Wiggins, & Pincus, 1990; Wiggins, 1995). Wiggins and Trobst (1997) describe interpersonal situations as social exchanges balancing negotiations between the resources of status and love. A successful negotiation would maintain both security (status) and nurturance (love) needs. For example, a person with SAD may approach social situations with concerns about status (e.g., perceived inferiority or lack of dominance: for a review of the relationship between dominance and social anxiety, please see Johnson, Leedom, & Muhtadie, 2012; see also **Chapter 2**) as well as love (e.g., "can I be accepted by people given how inferior I am?"). The circumplex model provides a way to understand how different people attempt to

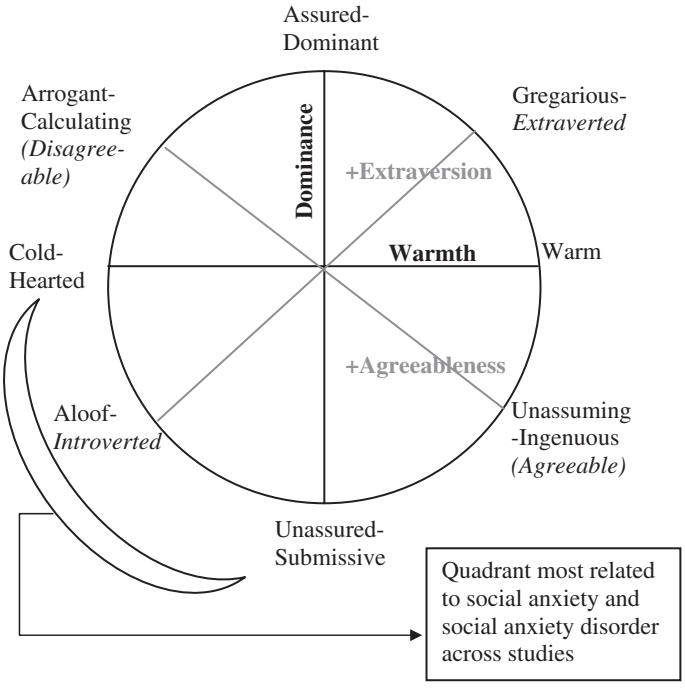

Figure 6.2 The interpersonal circumplex model. Lines for extraversion and agreeableness represent where these factors would be represented on the circumplex based on correlations between the interpersonal circumplex constructs and big five factors. Adapted from Markey and Markey (2009).

manage both their status and closeness to others. The resulting levels of needs and associated behavior can be illustrated as a circumplex; that is, status and love needs are arranged in a circle (illustrated in Figure 6.2) defined by two orthogonal dimensions: dominance (vs. submissiveness) and nurturance (vs. coldness). Psychometric work on measures of the interpersonal circumplex has found support for these two dimensions that then form eight octants. Starting with assured–dominant at the top, the remaining octants, in a clockwise direction, are gregarious–extraverted, warm–agreeable, unassuming–ingenuous, unassured–submissive, aloof–introverted, cold-hearted, and arrogant–calculating (Markey & Markey, 2009; Wiggins, Trapnell, & Phillips, 1988). Each of these octants represents a mixture between its two adjacent poles. These combinations of dominance and nurturance behavior can lead to perceptions of traits on the part of individuals (as in the five-factor model), but the circumplex theory focuses on the interpersonal aspects of these apparent traits. For example, an individual who is high in dominance and warmth might be considered a likeable, outgoing person (e.g., extraverted and moderately agreeable), whereas an individual who is dominant and low in warmth might be considered arrogant and hostile (e.g., disagreeable yet moderately extraverted). The circumplex structure has also been used as a framework for understanding interpersonal problems; assessments of such interpersonal problems have also constituted the main application of the circumplex theory to social anxiety

thus far (Alden et al., 1990; see also **Chapter 8** for extended review of interpersonal circumplex theory and social anxiety/SAD).

Overall, findings suggest several conclusions regarding clinically significant social anxiety and interpersonal problems. Foremost, individuals with clinically significant social anxiety report more problems of all types. For example, Kachin, Newman, and Pincus (2001) describe two groups of individuals with SAD: one reporting interpersonal problems with unassertiveness, exploitability, and over-nurturance, and another group reporting more problems with hostile–dominant behaviors (anger, hostility, and mistrustfulness). Despite the fact that one of these groups reported fewer problems with dominance, *both* groups reported more problems in general (including problems with dominance) than a normal control group. However, at least some of these reports appear likely to be exaggerated: it is difficult to understand how people with SAD could, as a group, be more inappropriately dominant than people without the disorder.

A second important conclusion from this literature is that, within groups of people with clinically significant social anxiety, the most elevated interpersonal problems are those related to unassertiveness/low dominance and coldness or detachment (Alden & Phillips, 1990). Multiple studies have suggested potential subgroups of people with SAD related primarily to variations in warmth; for example, Cain, Pincus, and Holtforth (2010) found two subgroups within individuals diagnosed with SAD: a friendly–submissive and a cold–submissive subtype. Those in the friendly–submissive group reported higher well-being and satisfaction, and lower social anxiety after psychotherapy than did socially anxious individuals who were cold–unassertive.

Another study conservatively combined information across self and peers and concluded that associations of social avoidance with interpersonal problems involving unassertiveness and coldness were apparent in the interpersonal environment (Rodebaugh, Gianoli, Turkheimer, & Oltmanns, 2010). In other words, people who displayed more social avoidance, according to a combination of self- and peer report, also displayed more problems due to unassertiveness and coldness in the interpersonal environment. Although self-report indicated positive associations between social avoidance and most interpersonal problems, including problems with dominance, these associations were not as strong when only combined data were considered, suggesting that individuals with SAD may tend to over-report these problems. Further, the association between social avoidance and problems being overly dominant became significantly *negative* when peer report was integrated. The implication may be that people with SAD are likely to feel as if they are displaying overly dominant behavior, but they are actually somewhat less likely to display such behavior than people without SAD.

Self-Regulatory Theory

One final theory that has not received as much attention in the area of social anxiety is self-regulatory theory and its relation to social anxiety. We highlight self-regulatory theory in this chapter because (a) several studies have addressed it, and (b) it offers the potential to place social anxiety more fully in the context of modern personality theories (i.e., beyond trait theories). Self-regulatory theory has many variants, but the

theories most frequently applied to social anxiety have been self-discrepancy theory and regulatory focus theory.

Self-discrepancy theory focuses on specific self-guides (Higgins, 1987), which represent hypothetical selves that a person may move toward. The *ideal self* involves aspirational goals, whereas the *ought self* involves attributes that the individual feels should be possessed. Increasing amounts of discrepancy between the person's current (actual) self and self-guides has been found to produce negative emotions, with discrepancies between actual and ideal producing sadness and discrepancies between actual and ought producing anxiety (Higgins, 1997). Regulatory focus theory, which offers a refinement on self-discrepancy theory, proposes that there are two regulatory systems (prevention and promotion focus) concerned with meeting basic needs through the pursuit of different goal types (Higgins, 1997). For example, security needs (and greater reliance on the *ought* self-guide) relate to prevention focus, whereas accomplishment needs (and greater reliance on the *ideal* self-guide) relate to promotion focus. In other words, prevention focus centers on avoidance, whereas promotion focus centers on approach. Previous research has linked prevention focus with anxiety and behavioral avoidance, and promotion focus with the emotions of dejection (if the goal is not reached) and approach strategies (Shah, Brazy, & Higgins, 2004). Thus, we would expect that ought self-discrepancy and prevention focus may have particular relevance for social anxiety.

Strauman (1989) compared clinically depressed individuals to individuals with SAD and found that individuals with SAD exhibited higher levels of ought self-discrepancy, whereas depressed individuals exhibited higher levels of ideal self-discrepancy. Additionally, Strauman (1989) found that depressed individuals became more dejected than individuals with SAD when primed with their ideal self-discrepancies, whereas individuals with SAD became more anxious than depressed individuals when primed with ought self-discrepancies. Weilage and Hope (1999) built upon Strauman's (1989) work and compared levels of self-discrepancies in individuals with nongeneralized SAD, GSAD, dysthymia, comorbid GSAD and dysthymia, and demographically matched normal controls. As expected, they found that participants with GSAD and comorbid GSAD and dysthymia had higher ought self-discrepancies than did matched controls. Other authors have had difficulty replicating aspects of self-discrepancy theory's predictions, although it appears that this difficulty may have at least as much to do with measurement as with the theory itself (e.g., Levinson & Rodebaugh, 2012; Rodebaugh & Donahue, 2007; Tangney, Niedenthal, Covert, & Barlow, 1998).

Scheier and Carver (1988) propose a more general theory of self-regulation, in which behavior is motivated by goals, and affect is generated based on progress toward goals. They further propose two monitoring systems: an avoidance system that gives feedback ranging from anxiety to calm, and an approach system that gives feedback ranging from happiness to sadness. Using a self-regulation framework, Kocovski and Endler (2000) found that, in undergraduates, high levels of social anxiety were associated with lower expectations that one would accomplish goals, as well as lower frequency of rewarding oneself for accomplishing goals. Research on the characteristics of avoidance and approach goals has revealed that, as would be expected, factors related to avoidance goals are related to negative affect and social anxiety during a speaking task (Rodebaugh, 2007; Rodebaugh & Shumaker, 2012). However, these

same studies have found unexpected relationships between theoretically approach-related constructs (i.e., approach goals, positive affect) and theoretically avoidance-related constructs. For example, Rodebaugh and Shumaker found that confidence and importance of avoidance goals combined to predict *positive* affect related to a public-speaking task, despite theory suggesting they should predict only negative affect. A similar relationship was also found in recent models tested by Trew and Alden (2012).

A final related area of research that should be noted here is a self-control theory proposed by Kashdan, Weeks, and Savostyanova (2011). These authors suggest that, given that self-control is a resource that can be depleted (Muraven & Baumeister, 2000), individuals who are high in social anxiety may experience chronic depletion of self-control due to excessive efforts to manage negative affect and make positive social impressions. Kashdan et al. further suggest that when self-control is depleted, people with higher social anxiety may act in an impulsive or self-destructive manner (i.e., binge drinking). As of yet, direct evidence of this model is limited. Further, it should be noted that some research indicates that the self-control depletion hypothesis itself may be limited in application primarily to individuals who *believe* that self-control is a resource that can be depleted (e.g., Job, Dweck, & Walton, 2010). That is, although everyone gets physically tired eventually, it may only be that people who believe their self-control is a limited resource are likely to act in an impulsive manner after encountering emotional stressors.

Common Themes Among Theories

There are several common themes across the theories reviewed here. As implied by our broad model, we believe that much of the influence of personality on social anxiety can be integrated in terms of approach and avoidance tendencies. Approach tendencies are motivated by a positive/desirable event or potential outcome, whereas avoidance tendencies are motivated by a negative/undesirable event or potential outcome (Elliot, 1999). Elliot and Covington (2001) have argued that approach and avoidance tendencies are a central theme for all living creatures. They argue, based on the work of Schneirla (1959), that approach and avoidance tendencies exist even in single-celled amoebas. Elliot and Covington (2001) suggest that humans have a behavioral predisposition to approach or avoid certain stimuli depending on the content. For example, consider a circumstance in which participants must respond to positively valenced stimuli, and are either told to respond by pushing a lever away or toward themselves. Just such a study suggests that people respond more quickly when the instructions match theoretically preexisting tendencies: participants more quickly pulled a lever toward themselves in response to positive stimuli in comparison to participants tasked with pushing the lever away in response to the same stimuli (Chen & Bargh, 1999).

All of the theories reviewed above can be mapped (at least in part) onto a generalized theory of temperamental tendencies toward approach and avoidance. In the FFT, extraversion can be viewed as a tendency to be motivated by rewards, and neuroticism as a tendency to have higher sensitivity toward negative outcomes (e.g., punishment). As a result, individuals who are extraverted may seek out social situations because of the reward humans typically derive from these situations. Individuals higher in neuroticism

may experience more negative affect and avoid stimuli that others do not see as negative because of their higher sensitivity to negative cues. The same conceptualization can be applied to theories of positive and negative affect. Elliot and Thrash (2002) found support in six studies for the conceptualization of extraversion and positive affect as parts of an approach temperament and negative affect and neuroticism as parts of an avoidance temperament.

The interpersonal circumplex theory presents a slightly more complex picture, but remains interpretable within a slightly broadened approach and avoidance framework. Our interpretations of the relationship between the circumplex and approach and avoidance are guided by correlations found between circumplex measures of dominance and warmth and FFT measures (e.g., Markey & Markey, 2009). Specifically, both the high warmth and high dominance ends of the two poles represent high approach, but the approaches differ in tone. Thus, extraversion can be seen as crossing the dimensions of dominance and warmth, such that high extraversion is related to a combination of high warmth and high dominance, whereas low extraversion (or introversion) is related to both low dominance and low warmth. Avoidance tendencies tend to decrease both warmth and dominance, albeit indirectly, because the general tendency toward both warmth and dominance appear more clearly related to approach than avoidance. One might then ask what determines whether a more extraverted person will appear primarily dominant or primarily warm. In terms of the FFT, a primary determinant of warmth versus dominance appears to be agreeableness, which would cross warmth and dominance in a manner similar to extraversion. However, in this case high warmth and low dominance would be related to high agreeableness, whereas high dominance and low warmth would be related to low agreeableness. We have depicted this relationship in our Figure 6.2 to clarify how extraversion and agreeableness appear to cross warmth and dominance. We have also depicted the quadrant most associated with social anxiety: low warmth and low dominance.

Future Directions

We would like to highlight some particularly pressing research needs in the area of SAD and personality. Few of the relationships reported in this chapter have been tested across gender, ethnicity, culture, or age, with some notable exceptions (e.g., Austin & Chorpita, 2004; Chorpita, 2002). Lack of tests across important individual differences is a major shortcoming of the literature: all researchers in this area should be vigilant for opportunities to conduct such tests.

As indicated in our discussion of interpersonal problems, there is evidence that social anxiety may be related to systematic tendencies to endorse problems in general. This feature is an obvious problem for self-report, but we do not want to emphasize that issue here (please see **Chapter 14**). Rather, it seems striking to us that social anxiety, and particularly SAD, is strongly related to a tendency to view the self as insufficient, faulty, or unacceptable (i.e., negative self-portrayal or core extrusion schema; Moscovitch, Orr, Rowa, Reimer, & Antony, 2009; Rodebaugh, 2009). It seems plausible that this tendency may simply be a result of elevated neuroticism, but we are aware of no demonstration that neuroticism alone can account for this type of

finding. Over and above neuroticism generally, a specific tendency to view the self as less worthy than other selves may be trait-like as well as have an association with social anxiety. We believe that viewing oneself to be less worthy or inferior may represent a particularly important personality feature in relation to SAD, possibly reflecting a specific vulnerability to SAD. Future research is needed to test the specificity of this relationship.

Clinical Implications

One major clinical implication that becomes apparent from the relationship between SAD and personality is the malleability of personality within treatment. Treatment approaches that tackle aspects of personality, such as the high level of avoidance temperament that is associated with multiple anxiety disorders, show that personality can change with treatment. For example, in the unified treatment for anxiety, the goal of therapy is to treat the *negative affect syndrome* underlying all anxiety disorders (Barlow, Allen, & Choate, 2004), and evidence suggests that these treatments may be similar in efficacy to treatments that focus on single disorders (Moses & Barlow, 2006; Wilamowska et al., 2010). Further support for the malleability of personality underlying social anxiety comes from the proposition that AVPD is a more severe form of SAD (Chambless et al., 2008; Heimberg, 1996; Heimberg et al., 1990). This proposition would suggest that a personality disorder can be treated using similar methods to anxiety disorders. Indeed, research on the treatment of AVPD shows that it typically improves or remits during the course of successful therapy for SAD and does not routinely interfere with dropout or treatment outcomes in comparison to individuals who do not meet criteria for AVPD (e.g., Brown, Heimberg, & Juster, 1995; Huppert et al., 2008; Reich, 2000; although see Feske, Perry, Chambless, Renneberg, & Goldstein, 1996, for evidence that AVPD may interfere with treatment).

There are several additional clinical implications of our model of personality that await further testing. If longitudinal research continues to support a model of social anxiety in which avoidance and approach tendencies are precursors of SAD, it seems plausible that prevention efforts could target early identification and treatment of at-risk individuals who are high in avoidance tendencies and low in approach tendencies. If these constructs prove to be malleable, it seems worth investigating novel treatments that focus on such tendencies, even prior to the development of disorders, rather than disorder symptoms specifically. Notably, multiple studies suggest that such tendencies routinely change during treatment for anxiety disorders (Gi, Egger, Kaarsemaker, & Kreutzkamp, 2010; Glinski & Page, 2010). This line of research could potentially lead to the development of alternate areas of treatment, which could aid in the prevention of the development of SAD and help individuals who do not benefit from standard treatments.

Final Remarks

We have reviewed four major theories that unite the study of personality and social anxiety: the five-factor theory, trait-affect theory, interpersonal circumplex theory,

and self-regulatory theory. These theories are typically examined seperately in SAD-related studies, if they are examined at all. As the field moves forward, we hope that researchers will consider a unified theory of personality and social anxiety, in which these personality constructs are incorporated together. Perhaps this unification will increase our insight into SAD and lead to improved clinical treatments and alleviation of the suffering stemming from this disorder.

References

Alden, L. E., & Phillips, N. (1990). An interpersonal analysis of social anxiety and depression. *Cognitive Therapy and Research, 14*, 499–513. doi:10.1007/BF01172970

Alden, L. E., Wiggins, J. S., & Pincus, A. L. (1990). Construction of circumplex scales for the inventory of interpersonal problems. *Journal of Personality Assessment, 55*, 521–536. doi:10.1207/s15327752jpa5503&4_10

Austin, A. A., & Chorpita, B. F. (2004). Temperament, anxiety, and depression: Comparisons across five ethnic groups of children. *Journal of Clinical Child and Adolescent Psychology, 33*, 216–226. doi:10.1207/s15374424jccp3302_2

Bagby, R. M., Costa, P. T., Jr., Widiger, T. A., Ryder, A. G., & Marshall, M. (2005). DSM-IV personality disorders and the five-factor model of personality: A multi-method examination of domain- and facet-level predictions. *European Journal of Personality, 19*, 307–324. doi:10.1002/per.563

Barlow, D. H. (2000). Unraveling the mysteries of anxiety and its disorders from the perspective of emotion theory. *American Psychologist, 55*, 1247–1263. doi:10.1037/0003-066X.55.11.1247

Barlow, D. H., Allen, L. B., & Choate, M. L. (2004). Toward a unified treatment for emotional disorders. *Behavior Therapy, 35*, 205–230. doi:10.1016/S0005-7894(04)80036-4

Bienvenu, O. J., Hettema, J. M., Neale, M. C., Prescott, C. A., & Kendler, K. S. (2007). Low extraversion and high neuroticism as indices of genetic and environmental risk for social phobia, agoraphobia, and animal phobia. *American Journal of Psychiatry, 164*, 1714–1721. doi:10.1176/appi.ajp.2007.06101667

Bienvenu, O. J., Nestadt, G., Samuels, J. F., Costa, P. T., Howard, W. T., & Eaton, W. W. (2001). Phobic, panic, and major depressive disorders and the five-factor model of personality. *The Journal of Nervous and Mental Disease, 189*, 154–161. doi:10.1097/00005053-200103000-00003

Bienvenu, O. J., Samuels, J. F., Costa, P. T., Reti, I. M., Eaton, W. W., & Nestadt, G. (2004). Anxiety and depressive disorders and the five-factor model of personality: A higher-and-lower-order personality trait investigation in a community sample. *Depression and Anxiety, 20*, 92–97. doi:10.1002/da.20026

Bradley, M. M., Codispoti, M., Cuthbert, B. N., & Lang, P. J. (2001). Emotion and motivation I: Defensive and appetitive reactions in picture processing. *Emotion, 1*, 276–298. doi:10.1037/1528-3542.1.3.276

Bradley, M. M., & Lang, P. J. (1994). Measuring emotion: The self-assessment manikin and the semantic differential. *Journal of Behavior Therapy and Experimental Psychiatry, 25*, 49–59. doi:10.1016/0005-7916(94)90063-9

Brown, E. J., Heimberg, R. G., & Juster, H. R. (1995). Social phobia subtype and avoidant personality disorder: Effect on severity of social phobia, impairment, and outcome of cognitive behavioral treatment. *Behavior Therapy, 26*, 467–486. doi:10.1016/S0005-7894(05)80095-4

Brown, L. H., Silvia, P. J., Myin-Germeys, I., & Kwapil, T. R. (2007). When the need to belong goes wrong: The expression of social anhedonia and social anxiety in daily life. *Psychological Science, 18*, 778–782. doi:10.1111/j.1467-9280.2007.01978.x

Brown, T. A. (2007). Temporal course and structural relationships among dimensions of temperament and *DSM-IV* anxiety and mood disorder constructs. *Journal and Abnormal Psychology, 116*, 313–328. doi:10.1037/0021-843X.116.2.313

Brown, T. A., Chorpita, B. F., & Barlow, D. H. (1998). Structural relationship among dimensions of the DSM-IV anxiety and mood disorders and dimensions of negative affect, positive affect, and autonomic arousal. *Journal of Abnormal Psychology, 107*, 179–192. doi:10.1037/0021-843X.107.2.179

Cain, N. M., Pincus, A. L., & Holtforth, M. G. (2010). Interpersonal subtypes in social phobia: Diagnostic and treatment implications. *Journal of Personality Assessment, 92*, 514–527. doi:10.1080/00223891.2010.513704

Cain, S. (2011, June 25). Shyness: Evolutionary tactic? *The New York Times*. Retrieved from http://www.nytimes.com/2011/06/26/opinion/sunday/26shyness.html?page wanted=all (accessed February 24, 2012).

Chambless, D. L., Fydrich, T., & Rodebaugh, T. L. (2008). Generalized social phobia and avoidant personality disorder: Meaningful distinction or useless duplication? *Depression and Anxiety, 25*, 8–19. doi:10.1002/da.20266

Chavira, D. A., Stein, M. B., & Malcarne, V. L. (2002). Scrutinizing the relationship between shyness and social phobia. *Journal of Anxiety Disorders, 16*, 585–598. doi:10.1016/S0887-6185(02)00124-X

Chen, M., & Bargh, J. A. (1999). Consequences of automatic evaluation: Immediate behavioral predispositions to approach or avoid the stimulus. *Personality Social Psychology Bulletin, 25*, 215–223. doi:10.1177/0146167299025002007

Chorpita, B. F. (2002). The tripartite model and dimensions of anxiety and depression: An examination of structure in a large school sample. *Journal of Abnormal Child Psychology, 30*, 177–190. doi:10.1023/A:1014709417132

Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology, 100*, 316–336. doi:10.1037/0021-843X.100.3.316

Costa, P. T., Jr., & McCrae, R. R. (1992). *Revised NEO personality inventory (NEO-PI-R) and NEO five-factor model of personality (NEO-FFI)* [Professional Manual]. Odessa, FL: Psychological Assessment Resources.

De Bolle, M., De Clercq, B., Decuyper, M., & De Fruyt, F. (2011). Affective determinants of anxiety and depression development in children and adolescents: An individual growth curve analysis. *Child Psychiatry & Human Development, 42*, 694–711. doi:10.1007/s10578-011-0241-6

Degnan, K. A., & Fox, N. A. (2007). Behavioral inhibition and anxiety disorders: Multiple levels of a resilience process. *Development and Psychopathology, 19*, 729–746. doi:10.1017/S0954579407000363

Eggum, N. D., Eisenberg, N., Reiser, M., Spinrad, T. L., Valiente, C., Sallquist, J., . . ., Liew, J. (2011). Relations over time among children's shyness, emotionality, and internalizing problems. *Social Development, 21*, 109–129. doi:10.1111/j.1467-9507.2011.00618.x

Eisenberg, N., Fabes, R. A., Shepard, S. A., Murphy, B. C., Guthrie, I. K., Jones, S., . . ., Maszk, P. (1997). Contemporaneous and longitudinal prediction of children's social functioning from regulation and emotionality. *Child Development, 68*, 642–664. doi:10.1111/1467-8624.ep9710021681

Elliot, A. J. (1999). Approach and avoidance motivation and achievement goals. *Educational Psychologist, 34*, 169–189. doi:10.1207/s15326985ep3403_3

Elliot, A. J., & Covington, M. V. (2001). Approach and avoidance motivation. *Educational Psychological Review, 13*, 73–92. doi:10.1023/A:1009009018235

Elliot, A. J., & Thrash, T. M. (2002). Approach-avoidance motivation in personality: Approach and avoidance temperaments and goals. *Journal of Personality and Social Psychology, 82*, 804–818. doi:10.1037//0022-3514.82.5.804

Essex, M. J., Klein, M. H., Slattery, M. J., Goldsmith, H. H., & Kalin, N. H. (2010). Early risk factors and developmental pathways to chronic high inhibition and social anxiety disorder in adolescence. *American Journal of Psychiatry, 167*, 40–46. doi:10.1176/appi.ajp.2009.07010051

Ewen, R. B. (2003). *An introduction to theories on personality*. Mahwah, NJ: Lawrence Erlbaum Associates.

Feldman Barrett, L., & Russell, J. A. (1998). Independence and bipolarity in the structure of current affect. *Journal of Personality and Social Psychology, 74*, 967–984. doi:10.1037/0022-3514.74.4.967

Feske, U., Perry, K. J., Chambless, D. L., Renneberg, B., & Goldstein, A. J. (1996). Avoidant personality disorder as a predictor for treatment outcome among generalized social phobics. *Journal of Personality Disorders, 10*, 174–184. doi:10.1521/pedi.1996.10.2.174

Fleeson, W. (2001). Toward a structure- and process-integrated view of personality: Traits as density distributions of states. *Journal of Personality and Social Psychology, 80*, 1011–1027. doi:10.1037//0022-3514.80.6.1011

Gi, S. T. P., Egger, J., Kaarsemaker, M., & Kreutzkamp, R. (2010). Does symptom reduction after cognitive behavioural therapy of anxiety disordered patients predict personality change? *Personality and Mental Health, 4*, 237–245. doi:10.1002/pmh.142

Glinski, K., & Page, A. C. (2010). Modifiability of neuroticism, extraversion, and agreeableness by group cognitive behaviour therapy for social anxiety disorder. *Behaviour Change, 27*, 42–52. doi:10.1375/bech.27.1.42

Heimberg, R. G. (1996). Social phobia, avoidant personality disorder, and the multiaxial conceptualization of interpersonal anxiety. In P. M. Salkovskis (Ed.), *Trends in cognitive and behavioural therapies* (pp. 43–62). Sussex, UK: John Wiley & Sons.

Heimberg, R. G., Hope, D. A., Dodge, C. S., & Becker, R. E. (1990). DSM-III—R subtypes of social phobia: Comparison of generalized social phobics and public speaking phobics. *Journal of Nervous and Mental Disease, 178*, 172–179. doi:10.1097/00005053-199003000-00004

Higgins, T. E. (1987). Self-discrepancy: A theory relating self and affect. *Psychological Review, 94*, 319–340. doi:10.1037/0033-295X.94.3.319

Higgins, T. E. (1997). Beyond pleasure and pain. *American Psychologist, 52*, 1280–1300. doi:10.1037/0003-066X.52.12.1280

Hirshfeld-Becker, D. R., Biederman, J., Henin, A., Faraone, S. J., Davis, S., Harrington, K., & Rosenbaum, J. F. (2007). Behavioral inhibition in preschool children at risk is a specific predictor of middle childhood social anxiety: A five-year follow-up. *Journal of Developmental and Behavioral Pediatrics, 28*, 225–233. doi:10.1097/01.DBP.0000268559.34463.d0

Hughes, A. A., Heimberg, R. G., Coles, M. E., Gibb, B. E., Liebowitz, M. R., & Schneier, F. R. (2006). Relations of the factors of the tripartite model of anxiety and depression to types of social anxiety. *Behaviour Research and Therapy, 44*, 1629–1641. doi:10.1016/j.brat.2005.10.015

Hummelen, B., Wilberg, T., Pederson, G., & Karterud, S. (2007). The relationship between avoidant personality disorder and social phobia. *Comprehensive Psychiatry, 48*, 348–356. doi:10.1016/j.comppsych.2007.03.004

Huppert, J. D., Strunk, D. R., Ledley, D. R., Davidson, J. R. T., & Foa, E. B. (2008). Generalized social anxiety disorder and avoidant personality disorder: Structural analysis and treatment outcome. *Depression and Anxiety, 25*, 441–448. doi:10.1002/da.20349

Job, V., Dweck, C. S., & Walton, G. M. (2010). Ego depletion-is it all in your head?: Implicit theories about willpower affect self-regulation. *Psychological Science, 21*, 1686–1693. doi:10.1177/0956797610384745

Johnson, S. L., Leedom, L. J., & Muhtadie, L. (2012). The dominance behavioral system and psychopathology: Evidence from self-report, observational, and biological studies. *Psychological Bulletin, 138*, 692–743. doi:10.1037/a0027503

Kachin, K. E., Newman, M. G., & Pincus, A. L. (2001). An interpersonal problem approach to the division of social phobia subtypes. *Behavior Therapy, 32*, 479–501. doi:10.1016/S0005-7894(01)80032-0

Kaplan, S. C., Levinson, C. A., Rodebaugh, T. L., Menatti, A., & Weeks, J. W. (2012). *Social anxiety and the big five personality traits: The interactive relationship of trust and openness.* Poster presented at the Association for Behavioral and Cognitive Therapies, National Harbor, MD.

Kashdan, T. B. (2007). Social anxiety spectrum and diminished positive experiences: Theoretical synthesis and meta-analysis. *Clinical Psychology Review, 27*, 348–365. doi:10.1016/j.cpr.2006.12.003

Kashdan, T. B., & Steger, M. F. (2006). Expanding the topography of social anxiety: An experience-sampling assessment of positive emotions, positive events, and emotion suppression. *Psychological Science, 17*, 120–128. doi:10.1111/j.1467-9280.2006.01674.x

Kashdan, T. B., Weeks, J. W., & Savostyanova, A. A. (2011). Whether, how, and when social anxiety shapes positive experiences and events: A self-regulatory framework and treatment implications. *Clinical Psychology Review, 31*, 786–799. doi:10.1016/j.cpr.2011.03.012

Kocovski, N. L., & Endler, N. S. (2000). Self-regulation: Social anxiety and depression. *Journal of Applied Biobehavioral Research, 5*, 80–91. doi:10.1111/j.1751-9861.2000.tb00065.x

Kollman, D. M., Brown, T. A., Liverant, G. I., & Hofmann, S. G. (2006). A taxometric investigation of the latent structure of social anxiety disorder in outpatients with anxiety and mood disorders. *Depression and Anxiety, 23*, 190–199. doi:10.1002/da.20158

Kotov, R., Watson, D., Robles, J. P., & Schmidt, N. B. (2007). Personality traits and anxiety symptoms: The multilevel trait predictor model. *Behavior Research and Therapy, 45*, 1485–1503. doi:10.1016/j.brat.2006.11.011

Krueger, R. F., & Tackett, J. L. (2003). Personality and psychopathology: Working toward the bigger picture. *Journal of Personality Disorders, 17*, 109–128. doi:10.1521/pedi.17.2.109.23986

Leary, M. R., Kowalski, R. M., & Campbell, C. D. (1988). Self-presentational concerns and social anxiety: The role of generalized impression expectancies. *Journal of Research in Personality, 22*, 308–321. doi:10.1016/0092-6566(88)90032-3

Levinson, C. A., & Rodebaugh, T. L. (2012). Anxiety, self-discrepancy, and regulatory focus theory: Accultuation matters. *Anxiety, Stress and Coping: An International Journal, 26*, 171–186. doi:10.1080/10615806.2012.659728

Lonigan, C. J., Phillips, B. M., & Hooe, E. S. (2003). Relations of positive and negative affectivity to anxiety and depression in children: Evidence from a latent longitudinal study. *Journal of Consulting and Clinical Psychology, 71*, 465–481. doi:10.1037/0022-006X.71.3.465

Lonigan, C. J., & Vasey, M. W. (2009). Negative affectivity, effortful control, and attention to threat-relevant stimuli. *Journal of Abnormal Child Psychology, 37*, 387–399. doi:10.1007/s10802-008-9284-y

Markey, P. M., & Markey, C. N. (2009). A brief assessment of the interpersonal circumplex: The IPIP-IPC. *Assessment, 16*, 352–361. doi:10.1177/1073191109340382

Marsh, H. W., Lüdtke, O., Muthén, B., Asparouhov, T., Morin, A. J. S., Trautwein, U., & Nagengast, B. (2010). A new look at the big five factor structure through exploratory structural equation modeling. *Psychological Assessment, 22*, 471–491. doi:10.1037/a0019227

McCrae, R. R., & Costa, P. T., Jr. (2008). The five-factor theory of personality. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality psychology: Theory and research* (pp. 159–181). New York, NY: Guildford Press.

Meehl, P. E. (1999). Clarifications about taxometric method. *Applied & Preventive Psychology, 8*, 165–174. doi:10.1016/S0962-1849(05)80075-7

Mischel, W., Shoda, Y., & Mendoza-Denton, R. (2002). Situation-behavior profiles as a locus of consistency in personality. *Current Directions in Psychological Science, 11*, 50–54. doi:10.1111/1467-8721.00166

Moscovitch, D. A., Orr, E., Rowa, K., Reimer, S. G., & Antony, M. M. (2009). In the absence of rose-colored glasses: Ratings of self-attributes and their differential certainty and importance across multiple dimensions in social phobia. *Behaviour Research and Therapy, 47*, 66–70. doi:10.1016/j.brat.2008.10.007

Moses, E. B., & Barlow, D. H. (2006). A new unified treatment approach for emotional disorders based on emotion science. *Current Directions in Psychological Science, 15*, 146–150. doi:10.1111/j.0963-7214.2006.00425.x

Mroczek, D. K., & Spiro, A., III. (2003). Modeling intraindividual change in personality traits: Findings from the normative aging study. *Journal of Gerontology: Psychological Sciences, 58B*, 153–165. doi:10.1093/geronb/58.3.P153

Muraven, M., & Baumeister, R. F. (2000). Self-regulation and depletion of limited resources: Does self-control resemble a muscle? *Psychological Bulletin, 126*, 247–259. doi:10.1037/0033-2909.126.2.247

Naragon-Gainey, K., Watson, D., & Markon, K. E. (2009). Differential relations of depression and social anxiety symptoms to the facets of extraversion/positive emotionality. *Journal of Abnormal Psychology, 118*, 299–310. doi:10.1037/a0015637

Reich, J. (2000). The relationship of social phobia to avoidant personality disorder: A proposal to reclassify avoidant personality disorder based on clinical empirical findings. *European Psychiatry, 15*, 15–19. doi:10.1016/S0924-9338(00)00240-6

Rodebaugh, T. L. (2007). The effect of different types of goal pursuit on experience and performance during a stressful social task. *Behaviour Research and Therapy, 45*, 951–963. doi:10.1016/j.brat.2006.08.003

Rodebaugh, T. L. (2009). Hiding the self and social anxiety: The core extrusion schema measure. *Cognitive Therapy and Research, 33*, 90–109. doi:10.1007/s10608-007-9143-0

Rodebaugh, T. L., & Donahue, K. L. (2007). Could you be more specific, please: Self-discrepancies, affect, and variation in specificity and relevance. *Journal of Clinical Psychology, 63*, 1193–1207. doi:10.1002/jclp.20425

Rodebaugh, T. L., Gianoli, M. O., Turkheimer, E., & Oltmanns, T. F. (2010). The interpersonal problems of the socially avoidant: Self and peer shared variance. *Journal of Abnormal Psychology, 119*, 331–340. doi:10.1037/a0019031

Rodebaugh, T. L., & Shumaker, E. A. (2012). Avoidance goals for a specific social situation influence activated negative and positive affect. *Cognitive Therapy and Research, 36*, 36–46. doi:10.1007/s10608-010-9313-3

Ruscio, A. M. (2010). The latent structure of social anxiety disorder: Consequences of shifting to a dimensional diagnosis. *Journal of Abnormal Psychology, 119*, 662–671. doi:10.1037/a0019341

Samuel, D. B., & Widiger, T. A. (2008). A meta-analytic review of the relationships between the five-factor model and DSM-IV-TR personality disorders: A facet level analysis. *Clinical Psychology Review, 28*, 1326–1342. doi:10.1016/j.cpr.2008.07.002

Saulsman, L. M., & Page, A. C. (2004). The five-factor model and personality disorder empirical literature: A meta-analytic review. *Clinical Psychology Review, 23*, 1055–1085. doi:10.1016/j.cpr.2002.09.001

Scheier, M. F., & Carver, C. S. (1988). A model of behavioral self-regulation: Translating intention into action. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (pp. 303–346). New York, NY: Academic Press.

Schneirla, T. C. (1959). An evolutionary and developmental theory of biphasic processes underlying approach and withdrawal. In M. R. Jones (Ed.), *Nebraska symposium on motivation* (pp. 1–42). Oxford, UK: University of Nebraska Press.

Shah, J. Y., Brazy, P. C., & Higgins, T. E. (2004). Promoting us or preventing them: Regulatory focus and manifestations of intergroup bias. *Personality and Social Psychology Bulletin, 30*, 433–446. doi:10.1177/0146167203261888

Shoda, Y., Mischel, W., & Wright, J. C. (1994). Intraindividual stability in the organization and patterning of behavior: Incorporating psychological situations into the idiographic analysis of personality. *Journal of Personality and Social Psychology, 65*, 1023–1035. doi:10.1037/0022-3514.67.4.674

Stein, M. B., Fuetsch, M., Müller, N., Höfler, M., Lieb, R., & Wittchen, H. (2001). Social anxiety disorder and the risk of depression: A prospective community study of adolescents and young adults. *Archives of General Psychiatry, 58*, 251–256. doi:10.1001/archpsyc.58.3.251

Strauman, T. J. (1989). Self-discrepancies in clinical depression and social phobia: Cognitive structures that underlie emotional disorders? *Journal of Abnormal Psychology, 98*, 14–22. doi:10.1037/0021-843X.98.1.14

Tan, P. Z., Forbes, E. E., Dahl, R. E., Ryan, N. D., Siegle, G. J., Ladouceur, C. D., & Silk, J. S. (2012). Emotional reactivity and regulation in anxious and nonanxious youth: A cellphone ecological momentary assessment study. *Journal of Child Psychology and Psychiatry, 53*, 197–206. doi:10.1111/j.1469-7610.2011.02469.x

Tangney, J. P., Niedenthal, P. M., Covert, M. V., & Barlow, D. H. (1998). Are shame and guilt related to distinct self-discrepancies? A test of Higgins's (1987) hypotheses. *Journal of Personality and Social Psychology, 75*, 256–268. doi:10.1037/0022-3514.75.1.256

Trew, J. L., & Alden, L. E. (2012). Positive affect predicts avoidance goals in social interaction anxiety: Testing a hierarchical model of social goals. *Cognitive Behaviour Therapy, 41*, 174–183. doi:10.1080/16506073.2012.663402

Trull, T. J., & Sher, K. J. (1994). Relationship between the five-factor model of personality and Axis I disorders in a nonclinical sample. *Journal of Abnormal Psychology, 103*, 350–360. doi:10.1037/0021-843X.103.2.350

Vassend, O., & Skrondal, A. (1997). Validation of the NEO personality inventory and the five-factor model. Can findings from exploratory and confirmatory factor analysis be reconciled? *European Journal of Personality, 11*, 147–166. doi:10.1002/(SICI)1099-0984(199706)11:2<147:AID-PER278>3.0.CO;2-E

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*, 1063–1070. doi:10.1037/0022-3514.54.6.1063

Watson, D., Gamez, W., & Simms, L. J. (2005). Basic dimensions of temperament and their relation to anxiety and depression: A symptom-based perspective. *Journal of Research in Personality, 39*, 46–66. doi:10.1016/j.jrp.2004.09.006

Weeks, J. W., Carleton, R. N., Asmundson, G. J. G., McCabe, R. E., & Antony, M. M. (2010). Social anxiety disorder carved at its joints: Evidence for the taxonicity of social anxiety disorder. *Journal of Anxiety Disorders, 24*, 734–742. doi:10.1016/j.janxdis.2010.05.006

Weilage, M., & Hope, D. A. (1999). Self-discrepancy in social phobia and dysthymia. *Cognitive Therapy and Research, 23*, 637–650. doi:10.1023/A:1018788925223

Weinstein, T. A. R., Capitanio, J. P., & Gosling, S. D. (2008). Personality in animals. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality: Theory and research* (pp. 328–350). New York, NY: Guilford Press.

Wiggins, J. S. (1995). *Interpersonal Adjective Scales: Professional manual.* Odessa, FL: Psychological Assessment Resources.

Wiggins, J. S., & Trobst, K. K. (1997). When is a circumplex an "Interpersonal Circumplex?" The case of supportive actions. In R. Plutchik & H. R. Conte (Eds.), *Circumplex models of personality and emotions* (pp. 57–80). Washington, DC: American Psychological Association.

Wiggins, J. S., Trapnell, P., & Phillips, N. (1988). Psychometric and geometric characteristics of the Revised Interpersonal Adjectives Scale (IAS-R). *Multivariate Behavioral Research, 23*, 517–530. doi:10.1207/s15327906mbr2304_8

Wilamowska, Z. A., Thompson-Hollands, J., Fairholme, C. P., Ellard, K. K., Farchione, T. J., & Barlow, D. H. (2010). Conceptual background, development, and preliminary data from the unified protocol for transdiagnostic treatment of emotional disorders. *Depression and Anxiety, 27*, 882–890. doi:10.1002/da.20735

7

Behavioral Inhibition

A Discrete Precursor to Social Anxiety Disorder?

Dina R. Hirshfeld-Becker, Jamie A. Micco, Christine H. Wang, and Aude Henin

Massachusetts General Hospital, USA

Introduction

Across multiple species, including humans, a small subset of individuals are born with a consistent tendency to be "neophobic," that is, fearful and hesitant to approach novel objects, individuals, or environments. In children, we refer to this temperamental bias as "behavioral inhibition to the unfamiliar" (BI). Studies have begun to unpack BI's underlying neurobiology and genetics, explore its specific association with social anxiety disorder (SAD; i.e., social phobia), and examine whether early intervention with children with BI can prevent future anxiety disorders. In this chapter, we evaluate what is known about BI and its outcomes and what still remains to be researched.

Defining the Construct

BI is the temperamental tendency to display restraint, fearfulness, or withdrawal when faced with unfamiliar people, situations, and objects (Kagan, Reznick, & Snidman, 1988). It occurs in approximately 10–15% of children, with moderate stability from toddlerhood through middle childhood, especially for children most extreme in BI (Hirshfeld-Becker, Biederman, & Rosenbaum, 2004).

Characteristics of BI differ by developmental stage. At 3–4 months, "high reactive" infants who respond to novel sensory input with distress and motor reactivity are more likely than their non-reactive peers to display BI as toddlers (Kagan, Snidman, Zetner, & Peterson, 1999). Toddlers with BI show distress; fear and avoidance of unfamiliar settings, objects, and peers; decreased vocalization; reduced smiling; and a tendency

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

to remain proximate to caretakers (Garcia-Coll, Kagan, & Reznick, 1984). At ages 3–5 years, they show quiet restraint and are hesitant to smile, approach, and speak with strangers (Kagan, Reznick, & Snidman, 1987) and are reticent with peers (Rubin, Burgess, & Hastings, 2002). By early elementary school, BI most frequently manifests in unfamiliar group settings, and may negatively influence social competence (Bohlin, Hagekull, & Andersson, 2005). As adults, BI individuals remain cautious and ill at ease with strangers; have smaller social networks; delay leaving home; and avoid risky activities, leadership roles, and demanding projects (Caspi & Silva, 1995; Caspi et al., 2003; Gest, 1997).

Behaviors similar to BI, including the tendency to withdraw and cease ongoing activity when faced with novelty, have been observed in fish (Martins et al., 2011), birds, rodents (Trullas & Skolnick, 1993), and primates (Suomi, 1997). This neophobia confers adaptive advantages, such as avoiding predation or other dangers, but may be detrimental to mate selection or exploration of new food sources. In animals, these behaviors are genetically mediated and associated with activity in the amygdala, striatum, or hypothalamic–pituitary–adrenal axis (Kalin, Shelton, Fox, Oakes, & Davidson, 2005; Suomi, 1997). Thus, BI may be an evolutionarily conserved adaptation to novelty.

Neurobiological Underpinnings

Physiologic Reactivity

BI has been associated with a lower threshold to sympathetic nervous system activation (Kagan et al., 1988), reflected in a higher heart rate and reduced heart period variability under stress (Schmidt, Fox, Schulkin, & Gold, 1999), with reduced cardiac habituation to novelty observable even in BI newborns (Moehler et al., 2006). Children with BI also display pupillary dilation, laryngeal muscle tension, and, in some cases, elevated salivary cortisol (Kagan et al., 1987, 1988; Schmidt et al., 1997). Additionally, BI and high reactivity have been linked to a pattern of right frontal EEG asymmetry, suggesting heightened withdrawal responses to aversive stimuli (Fox, Henderson, Rubin, Calkins, & Schmidt, 2001). Infants or children who were originally high reactive or inhibited also show increased electrophysiologic responses to novel or unusual stimuli (Fox, Henderson, Marshall, Nichols, & Ghera, 2005; Marshall, Reeb, & Fox, 2009) and increased startle responses (Schmidt & Fox, 1998).

Neuroimaging Studies

Several studies have confirmed heightened amygdala reactivity in adult individuals with BI observed in early childhood (Perez-Edgar et al., 2007; Schwartz, Wright, Shin, Kagan, & Rauch, 2003) or retrospectively self-reported (Blackford, Avery, Shelton, & Zald, 2009; Blackford, Avery, Cowan, Shelton, & Zald, 2011; Clauss, Cowan, & Blackford, 2011). Adolescents with BI in early childhood also show greater striatal activation to monetary incentives (Guyer et al., 2006), which appears related to *motivated agency* not reward sensitivity (Bar-Haim et al., 2009). BI subjects showed

higher response in the caudate to feedback about losing but not gaining money, suggesting higher striatal sensitivity to aversive information (Helfinstein, Benson, et al., 2011). Cortical differences have been noted as well, with BI associated with weak inhibitory dorsal anterior cingulate cortex control (Clauss et al., 2011), and infant high reactivity associated in adolescence with greater thickness in the right ventromedial prefrontal cortex (Schwartz et al., 2010). Conflicting structural associations with the orbitofrontal cortex, an area associated with behavioral control, have also been reported.

It is possible that individuals with BI and disinhibition show different striatal response to rewards and punishments, with BI subjects more easily conditioned to avoid aversive stimuli (Helfinstein, Fox, & Pine, 2011). Myers et al. (2012) reported that adults with BI showed more rapid acquisition of an experimental conditioned response. Alternatively, results may reflect heightened arousal around choices with potential negative consequences, or increased performance anxiety. Also, since many of the comparison non-BI children in these studies were selected for high positive reactivity and were disinhibited as toddlers, and since disinhibited toddlers are at higher risk for attention deficit hyperactivity disorder (ADHD) (Hirshfeld-Becker et al., 2002; Hirshfeld-Becker, Biederman, Henin, Faraone, Micco, et al., 2007), comparison subjects may have shown striatal hyposensitivity to the expectation of reward, associated with ADHD (Scheres, Milham, Knutson, & Castellanos, 2007). Because most of these neuroimaging studies used comparison subjects extreme in disinhibition, to truly understand whether the functional and structural features identified are associated with BI, BI individuals should be contrasted with non-BI, non-disinhibited controls.

Genetic Underpinnings

BI shows moderate heritability, with estimates in toddlerhood ranging from 0.41 to 0.64 (DiLalla, Kagan, & Reznick, 1994; Emde et al., 1992; Matheny, 1989; Robinson, Kagan, Reznick, & Corley, 1992), with heritability highest among children extreme in BI (DiLalla et al., 1994). Several candidate genes have been found to be associated with BI, including the glutamic acid decarboxylase gene (65 kDa isoform), which encodes an enzyme involved in GABA synthesis, for which a mouse knockout is associated with increased inhibition (Smoller et al., 2001). Another is the gene for corticotropin-releasing hormone (CRH), a mediator of stress responses that affects the hypothalamic–pituitary–adrenal axis and limbic system (Smoller et al., 2003, 2005). Transgenic mice that over-express this hormone display BI-like behaviors. A third is the gene encoding regulator-of-G-protein-signaling-2 (RGS2), which regulates mouse anxiety (Smoller et al., 2008); it is associated with laboratory-observed BI in children, self-rated introversion in adults, and increased limbic amygdala and insular cortex activation during emotion processing in adults.

In addition, interesting gene–environment interactions have been identified for 5-HTTLPR. Children who had both the short allele (homo- or heterozygous) and low social support displayed increased rates of BI in middle childhood (Fox, Nichols et al., 2005). In 8- to 12-year-olds (Burkhouse, Gibb, Coles, Knopik, & McGeary, 2011), child-rated maternal overprotection was positively associated with social fears

in children with the short–short allele, but not among others, after controlling for mother and child psychopathology.

Associations with Anxiety Disorders

Family Studies

If BI is a heritable risk factor for anxiety disorders, it should be more prevalent among offspring of parents with anxiety disorders. Indeed, four family studies found elevated rates of laboratory-observed BI among preschool-age offspring of parents with panic disorder (Battaglia et al., 1997; Manassis, Bradley, Goldberg, Hood, & Swinson, 1995; Rosenbaum et al., 1988, 2000). However, no associations between BI in infants and toddlers and maternal panic disorder were observed (Warren et al., 2003), perhaps because of the younger age of the children or other methodological differences. Rosenbaum and colleagues (1991) found that parents of community children with BI had higher rates of SAD, childhood avoidant disorder, and persistent anxiety disorders than parents of uninhibited children. Similarly, mothers of 4-year-olds rated as "shy" had nearly eight times the rate of lifetime SAD in controls (Cooper & Eke, 1999). Mood and anxiety disorders were also elevated in mothers of shy children, but were not specifically related to child shyness.

It is likely that these associations are in part genetically mediated. Associations between BI and the CRH locus are particularly pronounced among children at risk for panic disorder (Smoller et al., 2003), suggesting that BI may have stronger genetic influences in offspring of anxious parents. A well-powered study also found that, although BI at preschool age was associated with parental panic disorder, BI showed no association with environmental factors such as family adversity or exposure to parental disorders (Hirshfeld-Becker, Biederman, Faraone et al., 2004).

Prospective Studies

The most methodologically rigorous way to examine the link between BI and anxiety disorders is through prospective longitudinal studies that assess BI early in childhood and then assess anxiety disorders later in development. As seen in Table 7.1, studies have converged to suggest that BI represents a specific risk factor for SAD.

Our group's initial pilot studies suggested that BI conferred risk for anxiety disorders in general or for overanxious or phobic disorders (Biederman et al., 1990, 1993; Hirshfeld-Becker et al., 1992). However, these studies were limited in that they did not have the power to covary parental anxiety disorders (which are significantly associated with both BI and childhood anxiety disorders; Micco et al., 2009), and only used psychiatric controls. Also, early diagnostic instruments did not distinguish childhood social phobia from other phobic disorders. Indeed, the most common phobias associated with BI in these studies were social in nature (i.e., speaking in class, strangers, and crowds).

In the past 15 years, larger and better-controlled studies have suggested that BI is specifically associated with risk for social anxiety in childhood and early adolescence (Biederman et al., 2001; Chronis-Tuscano et al., 2009; Hayward et al., 1998;

Table 7.1 Prospective Longitudinal Studies Examining Associations Between Behavioral Inhibition and Anxiety Disorders

Study	*Methods*	*Results*
Pilot studies (in chronological order)		
Biederman et al. (1990)	Children were rated as BI or uninhibited (bold) via lab observations between ages 2 and 7 years ($N = 71$); disorders were assessed at age 7 years with the DICA-P; children were contrasted with 20 healthy controls	Compared with healthy controls, BI children had increased multiple (≥ 2) anxiety disorders (accounted for by phobic or overanxious disorder)
Hirshfeld-Becker et al. (1992)	Children were assessed for BI in the lab at 21 months, 4, 5, and 7 years ($N = 41$) and classified as "stable inhibited" if they had BI at all four time points; disorders were assessed using the DICA-P	The "stable inhibited" group had higher rates of any anxiety disorders, multiple (≥ 2) anxiety disorders, and phobic disorders
Biederman et al. (1993)	Children from Biederman et al. (1990) were followed up 3 years later using the DICA-P and anxiety modules of the K-SADS-E	Children with baseline BI had higher rates of multiple (≥ 2) anxiety disorders, agoraphobia, avoidant disorder, separation anxiety disorder, and were more likely to have new anxiety disorders
Larger longitudinal studies (in chronological order)		
Hayward, Killen, Kraemer, and Taylor (1998)	Adolescents were retrospectively assessed in 9th grade for childhood BI using the RSRI; from 9th to 12th grade, they were assessed yearly for SAD and depression using the SPAI and KSADS ($N = 2242$)	Among those without SAD at baseline, students in the top 15th percentile on both social avoidance and fearfulness (factors on the RSRI) had a greater than fivefold chance of developing SAD
Schwartz, Snidman, and Kagan (1999)	Children from two Kagan studies who were characterized in the lab as either inhibited or uninhibited (bold) at 21 and 31 months were assessed for anxiety at age 13 years ($N = 79$) via adapted DISC	Current SAD during adolescence was higher in those who had been inhibited as toddlers (61% vs. 27%), with the effect stronger for girls (44% vs. 6%)
Biederman et al. (2001)	Children assessed for BI in the lab once at age 2–6 years were evaluated for disorders via parental K-SADS-E at mean age 6 years ($N = 216$; 25% concurrently assessed)	Children with BI had increased rates of childhood avoidant disorder (9% vs. 1%) and SAD (17% vs. 5%) compared with non-BI children

(*continued*)

Table 7.1 (*Continued*)

Study	*Methods*	*Results*
Hirshfeld-Becker, Biederman, Henin, Faraone, Davis, et al. (2007)	Children from Biederman et al. (2001) were re-assessed via parental KSADS-E at mean age 9.6 years ($N = 215$)	Children with BI were more likely than non-BI children to have higher rates of SAD (28% vs. 14%) and to have new-onset SAD (22.2% vs. 8%)
Chronis-Tuscano et al. (2009)	Children assessed for BI at 14 months, 24 months, 4 years, and 7 years via lab observation and mother report were evaluated for anxiety at mean age 15 years using the K-SADS and SCARED-R (child and parent report)	Lab-observed BI did not predict SAD; stable mother-reported BI from age 14 months through 7 years predicted a 3.79-fold increase in lifetime SAD and higher SCARED-R Social Phobia scores
Essex, Klein, Slattery, Goldsmith, and Kalin (2010)	BI was assessed from birth to grade 9 ($N = 238$) via multiple informants; child psychopathology was assessed in ninth grade using the K-SADS-PL ($N = 60$)	About 50% of children rated chronically high in BI met criteria for SAD, versus 27% in the middle–high group, and none in the low–middle and chronic low groups; BI beginning in third grade predicted lifetime SAD
Hudson, Dodd, and Bovopoulos (2011)	At age 4 years, children were characterized as BI ($N = 102$) or uninhibited (extremely low BI) ($N = 100$); at age 6 years, child anxiety was assessed using the ADIS-P-IV	Lab-observed BI specifically predicted SAD (48% vs. 2%) and GAD (18% vs. 1%); parent-reported BI predicted any anxiety disorder and number of anxiety disorders
Muris et al. (2011)	Children of ages 5–8 years were characterized as behaviorally inhibited ($N = 124$) or as controls ($N = 137$) using the BII; child anxiety symptoms were assessed using the Dominic-R and SCARED	BI predicted later social anxiety symptoms
Hudson and Dodd (2012)	This was a follow-up at age 9 years of Hudson, Dodd, and Bovopoulos (2011), using the ADIS-P-IV to assess child anxiety disorders	Parent-reported BI predicted anxiety disorders, as well as SAD (37% vs. 3%), GAD (21% vs. 9%), and separation anxiety disorder (10% vs. 1%)

Abbreviations: ADIS-P-IV, Anxiety Disorders Interview Schedule for DSM-IV, Parent Version; BI, behavioral inhibition; BII, Behavioral Inhibition Instrument (includes the Behavioral Inhibition Scale and one additional item); DICA-P, Diagnostic Interview Schedule for Children and Adolescents, Parent Version; DISC, Diagnostic Interview Schedule for Children; GAD, generalized anxiety disorder; K-SADS, Kiddie-Schedule for Affective Disorders and Schizophrenia; KSADS-E, Kiddie-Schedule for Affective Disorders and Schizophrenia—Epidemiological Version; K-SADS-PL, Kiddie-Schedule for Affective Disorders and Schizophrenia—Present and Lifetime Version; RSRI, Retrospective Self-Report of Inhibition; SAD, social anxiety disorder; SCARED, Screen for Child Anxiety-Related Emotional Disorders; SCARED-R, Screen for Child Anxiety-Related Emotional Disorders—Revised; SPAI, Social Phobia and Anxiety Inventory.

Hirshfeld-Becker, Biederman, Henin, Faraone, Davis, et al., 2007; Schwartz et al., 1999). In a prospective 10-year study of 284 preschoolers (Biederman et al., 2001; Hirshfeld-Becker, Biederman, Henin, Faraone, Davis, et al., 2007; Rosenbaum et al., 2000), we used DSM-IV based instruments, which included comparison children of parents without anxiety or mood disorders, classified children as BI if they were more extreme in inhibition than the top 20% of comparison children, and covaried parental diagnoses in analyses of child outcomes. We found that BI assessed at mean age of 4 years specifically predicted SAD at mean ages of 6 and 10 years. Data suggested that BI may be most predictive of anxiety disorders in offspring of parents with anxiety disorders (Biederman et al., 2001; Rosenbaum et al., 1992).

The association between BI and SAD has also been observed in six community samples, which were either unselected (Essex et al., 2010; Hayward et al., 1998; Muris, van Brakel, Arntz, & Schouten, 2011), or selected on the basis of extremes in temperament during early childhood (Chronis-Tuscano et al., 2009; Hudson, Dodd, Lyneham, & Bovopoulous, 2011; Schwartz et al., 1999) (see Table 7.1).

Prospective Studies Assessing Symptoms Other Than Social Anxiety

Four additional studies looked at associations between BI and symptoms of other disorders. These studies found that BI was inversely associated with posttraumatic stress disorder (PTSD) symptoms after viewing events of 9/11 on television, accounted for by lower television viewing in the BI children (Otto et al., 2007); that observed BI was associated with internalizing symptoms persisting from age 4 through age 15 (Williams et al., 2009); that BI at age 3 predicted symptoms of separation and overanxious disorders at age 7 (Volbrecht & Goldsmith, 2010); and that self-rated BI, anxiety sensitivity, and negative affect together predicted severity of panic attacks and post-panic symptoms in adolescents (Wilson & Hayward, 2005).

Cross-Sectional Studies and Retrospective Studies

Some cross-sectional studies have found associations between BI and anxiety in general (Muris, Meesters, & Spinder, 2003; Muris, Merckelbach, Schmidt, Gadet, & Bogie, 2001; Muris, Merckelbach, Wessel, & van de Ven, 1999), whereas others found larger or more specific associations with SAD (Brumariu & Kerns, 2010; Hudson, Dodd, & Bovopoulos, 2011; Muris et al., 1999). Others did not distinguish social anxiety but looked at associations with anxiety in general, finding that BI correlated with anxiety independently of parenting factors in 11–15-year-olds (van Brakel, Muris, Bögels, & Thomassen, 2006); predicted self-reported anxiety symptoms independently of extraversion and neuroticism in 9–12-year-olds (Muris et al., 2009); and predicted anxiety but not depression or sleep problems in 8–13-year-olds (Broeren, Muris, Bouwmeester, van der Heijden, & Abee, 2011).

Retrospective studies are limited by possible biased recall among individuals already affected with anxiety disorders, and the potential for concurrent BI to be confounded by inhibition secondary to symptom onset. Some retrospective studies have documented associations between BI and anxiety in general (Fincham, Smit, Carey, Stein,

& Seedat, 2008; van Ameringen, Mancini, & Oakman, 1998), or anxiety disorders such as obsessive-compulsive disorder (OCD; Coles, Schofield, & Pietrefesa, 2006). In contrast, others have found larger or more specific associations with SAD (Dalrymple, Herbert, & Gaudiano, 2007; Gladstone & Parker, 2006; Gladstone, Parker, Mitchell, Wilhelm, & Malhi, 2005; Knappe et al., 2011; Mick & Telch, 1998; Schofield, Coles, & Gibb, 2009; Wittchen, Stein, & Kessler, 1999).

BI and Its Association with Depression

There is mixed evidence of a potential link between BI and depression. Two family studies observed elevated rates of BI among young offspring of parents with depression compared with offspring of parents without depression (Kochanska, 1991; Rosenbaum et al., 2000), while two others found no association (Kochanska & Radke-Yarrow, 1992) or lower BI in offspring of depressed parents (Jones, Field, & Almeida, 2009). Similarly, in two community studies that assessed both BI and parental depression, one found no association (Durbin, Klein, Hayden, Buckley, & Moerk, 2005), whereas another found that BI in toddlerhood was associated with prior and concurrent symptoms of maternal depression (Moehler et al., 2007). It is possible that other factors moderate the association; for example, in 3-year-olds with high or moderate positive emotionality only, BI was related to parental depression (Olino, Klein, Dyson, Rose, & Durbin, 2010).

Prospective studies have also yielded mixed results, with some observing a relationship between BI in childhood and major depression in adulthood (Beesdo et al., 2007; Caspi, Moffitt, Newman, & Silva, 1996) and others failing to find an association with major depression in childhood (Brozina & Abela, 2006). Cross-sectional (Muris et al., 2001, 2003) and retrospective (Gladstone & Parker, 2006; Reznick, Hegeman, Kaufman, Woods, & Jacobs, 1992) questionnaire report studies with adolescents and adults have also noted associations between BI and depression (Rotge et al., 2011), but in two of these studies, the link was mediated by social anxiety or arousal (Gladstone & Parker, 2005; Schofield et al., 2009). Further follow-up of prospective samples assessed for BI in childhood is needed to understand whether BI confers later risk for depression, and whether this risk is independent of social anxiety.

Cultural Considerations

The studies reviewed above were conducted in Western cultures, which tend to value sociability and assertiveness. BI may function more adaptively in cultures that emphasize collectivism and value reserve, restraint, and obedience (Rubin et al., 2006). For example, two studies have reported higher rates of BI in 2-year-olds from countries such as China or Korea than in toddlers from Canada or Australia (Chen et al., 1998; Rubin et al., 2006). In contrast, Kagan et al. (1994) observed that rates of "high reactivity" at age 4 months were highest in the United States, intermediate in Ireland, and lowest in China. Maternal attitudes to 2-year-olds with BI also vary between cultures: in Canada, BI was associated positively with punishment orientation and negatively with acceptance and encouragement, whereas in China, BI was associated positively

with warm and accepting attitudes and negatively with rejection and punishment orientation (Chen et al., 1998). Interestingly, in a Chinese sample (Chen, Chen, Li, & Wang, 2009), BI at age 2 years predicted greater cooperative behavior, peer affection, social integration, positive school attitudes, school competence, lower scores on antagonistic behaviors and learning problems, and overall better adjustment at school age. It may be that cultural influences impact the stability and outcomes of BI. It is also possible, especially if high reactivity is less prevalent in Chinese infants, that some inhibited toddlers in Eastern samples had *behavioral* phenocopies of BI (i.e., BI learned through cultural factors rather than genetically based). This possibility could be further examined by studying variations in heredity of BI between cultures.

Is BI in Western Cultures a Discrete Risk Factor for Social Anxiety Disorder?

This question encompasses two important issues: (1) is BI a specific risk factor for SAD, and (2) is it discrete from SAD, or simply the early manifestation of SAD itself? As described above, most, though not all, prospective studies that followed children through middle childhood or adolescence found BI to be a specific risk factor for SAD, with some suggesting that the predictions are stronger for BI measured at age 4 years or later, for early-onset SAD, and for generalized SAD (rather than fear of specific performance situations).

There are several differences between studies that find a specific association between BI and SAD and those that find broader associations. First, studies differ in the contrast groups used. In studies that compare extremely inhibited against extremely uninhibited children, one would expect greater difference in rates of social or other anxiety than in studies that use unselected samples, where comparison children are moderate on approach or avoidance of novelty. Second, in most studies of community samples, parental psychopathology is not fully assessed or covaried. Since BI is more common among offspring of parents with anxiety disorders, individuals endorsing BI in community or clinical samples have a greater probability of being offspring of anxious parents. Since such offspring are known to have elevated rates of anxiety disorders in general, this may confound associations between BI and anxiety. Covarying parental anxiety in analyses of BI as a predictor of childhood anxiety disorders (e.g., Biederman et al., 2001; Hirshfeld-Becker, Biederman, Henin, Faraone, Davis, et al., 2007) is essential to more accurately examine this issue. Third, most prospective studies extend only to middle adolescence at the latest, which may limit ability to examine associations with later-onset disorders (e.g., panic disorder, depression).

Most importantly, studies differ in which aspects of BI they assess, including both social and non-social stimuli. These different aspects of BI are usually found to be at best modestly related (Dyson, Klein, Olino, Dougherty, & Durbin, 2011; Geng, Hu, Wang, & Chen, 2011; Kochanska, 1991; Rubin, Hastings, Stewart, Henderson, & Chen, 1997), particularly in samples less extreme in BI, and to be associated with different symptoms. For example, Dyson and colleagues (2011) found that non-social BI in preschoolers was related to concurrent parent-reported fears, whereas social BI

was related to shyness. DSM-IV social phobia was related only to social BI, and specific phobia was associated more strongly with non-social BI. It is possible that studies finding links with SAD emphasize social over non-social BI in their assessments. This is borne out in retrospective studies finding associations between non-social BI and OCD or depression; unsurprisingly, since childhood anxiety is a known risk factor for adult depression. This may also explain why, in Chronis-Tuscano et al. (2009), parent ratings of BI on shyness/social fearfulness scales predicted adolescent social phobia better than observational measures that also included reactions to non-social stimuli (e.g., unfamiliar toys, robots, and tunnels). Similarly, BI at later ages may better predict SAD because laboratory observations at these later ages typically include fewer non-social stimuli. Further studies are needed to deconstruct BI measures and determine which aspects of BI predict social versus other anxiety disorders. Regarding the possibility that early BI is simply a measure of infant-onset SAD, several pieces of evidence suggest that this is not the case. First, BI in early childhood does not reach the level of psychiatric disorder because it is not itself associated with impairment; in fact, in some cultures, it appears adaptive. SAD, even in younger children, cannot be diagnosed in the absence of impaired function. Second, only a minority of BI individuals develop SAD during their lifetime; the rest become at most, cautious, risk-aversive adults. Third, not all individuals with SAD initially have BI; in fact, a subset of adult SAD patients is temperamentally disinhibited and risk-taking, yet has generalized fears of social interaction with earlier-than-average onset (Kashdan, McKnight, Richey, & Hofmann, 2009). Fourth, the heritability of BI is higher than the heritability of SAD (Smoller & Tsuang, 1998). Finally, neophobic tendencies occur across species, and BI has many behavioral and physiologic commonalities with these tendencies as observed in other animals. All this suggests that BI is a temperamental trait rather than a disorder.

Clearly, one could still regard extreme BI as a subtype of very early-onset subclinical generalized SAD, recognizing that not all BI children need develop SAD and not all SAD patients need to have had BI for this subtype to exist. However, theoretical considerations aside, we would still be left with a construct measurable in preschool or earlier that predicts increased risk for a potentially debilitating disorder later in life (Hayward et al., 1998; Hirshfeld-Becker, Biederman, Henin, Faraone, Davis, et al., 2007); this points to the need for preventive intervention.

Toward Preventive Intervention

Several studies have explored factors that explain why some BI children but not others develop SAD or other anxiety disorders. For example, parental overprotectiveness is associated both with stability of BI (Belsky, Hsieh, & Crnic, 1998; Degnan, Henderson, Fox, & Rubin, 2008; Hudson, Dodd, Lyneham, et al., 2011) and with onset of anxiety disorders (Hudson & Dodd, 2012). Other factors associated with stability of BI are parental criticism (Hudson, Dodd, & Bovopoulos, 2011; Rubin et al., 2002), modeling of avoidant responses (Murray et al., 2008), family conflict (Pauli-Pott & Beckmann, 2007), and possibly insecure attachment (Shamir-Essakow, Ungerer, & Rapee, 2005). Factors internal to the child that may also increase the stability of BI

or onset of anxiety include lower approach motivation (Henderson, Fox, & Rubin, 2001), higher conditionability (Myers et al., 2012); or heightened vigilance and monitoring (McDermott et al., 2009), neural responses to novel stimuli (Reeb-Sutherland, Vanderwert, et al., 2009), and attention to threat (Pérez-Edgar et al., 2011), startle response (Reeb-Sutherland, Helfinstein, et al., 2009), and inhibitory control of prepotent responses (White, McDermott, Degnan, Henderson, & Fox, 2011). In contrast, children who show greater social competence (Asendorpf, 1994), the ability to flexibly shift attention away from distressing stimuli, or to broaden attention to avoid fixating on distressing stimuli, tend to reduce their shyness or inhibition (Eisenberg, Shepard, Fabes, Murphy, & Guthrie, 1998; White et al., 2011).

We may hypothesize from these studies that teaching parents to model and reward approach behaviors, reduce overprotection and criticism, and promote autonomy, and that teaching children to flexibly direct their attention, undergo exposure and habituation to novel or anxiety-provoking stimuli, and improve social skills may be helpful in reducing BI and thus subsequent anxiety disorders.

BI and Preventive Intervention

Screening Measures: To effectively apply preventive interventions, it is important to have easy-to-administer, cost-effective screening measures to identify children with BI. While the "state-of-the-art" method to assess BI in research studies involves laboratory observation of children's interactions with unfamiliar adults and peers, such observations are time-intensive to administer and code, making them costly to administer widely. Research is also limited by a lack, with few exceptions, of standardized laboratory assessments, with different protocols assessing different aspects of BI.

Fortunately, several screening tools have been developed, and although correlations are modest with observational measures, they have the advantage of being much easier and less costly to administer. In addition, several longitudinal studies have shown that onset of SAD can be predicted from BI solely on the basis of parent-report measures (Chronis-Tuscano et al., 2009) or even retrospective child-report measures (Hayward et al., 1998). Adult self-reports of inhibition or similar constructs (e.g., introversion) are also associated with some of the same brain functions (Blackford et al., 2009, 2011) and genes (Smoller et al., 2008) that have been linked to laboratory-observed BI. Table 7.2 summarizes details of BI screening measures.

Is BI a Useful Target for Preventive Intervention?

If BI is a causal risk factor for the onset of SAD, then interventions that reduce BI should also reduce subsequent SAD. Studies have suggested that BI can be targeted using cognitive behavioral therapy (CBT) approaches identical to those used to treat anxiety.

Several recent studies have reported that CBT interventions aimed at young children with BI are efficacious in reducing anxiety disorders in these youth, compared with

Table 7.2 Questionnaire Measures for Assessing Behavioral Inhibition (BI)

	Measure	*References*	*Ages/number of items*	*Factors/scales*	*Correlation with observational measures*	*Comments*
Concurrent report by multiple reporters	Behavioral Inhibition Questionnaire (BIQ)	Bishop, Spence, and McDonald (2003), Broeren and Muris (2010), Kim et al. (2011)	3–15 years/30 items	Six factors within three domains (social novelty, situational novelty, and physical activities)	.25–.48 for the whole sample and .35–.60 for children extremely high or low in BI	*Pros*: Distinguishes social and non-social BI; items reflect multiple contexts *Cons:* Lower convergent validity for fathers
Concurrent report by parents and teachers	Infant Behaviour Questionnaire (IBQ)	Rothbart (1981, 1986), Lamb, Frodi, Hwang, and Frodi (1983), Goldsmith, Rieser-Danner, and Briggs (1991), Marysko, Finke, Wiebel, Resch, and Moehler (2010)	Infants (3–12 months old)/78 items	One total scale and five subscales measuring smiling, distress to limitations, distress to novelty, motor activity, and soothability	.37 between IBQ and temperament at 9 months; .30 for "distress to novelty" at 4 months and fear at 14 months	*Pros:* Distress to novelty scale captures social and non-social BI, (without distinguishing them) and predicts later BI *Cons:* Psychometrics are weaker for infants <6 months
	Behavioral Inhibition Scale for Children Aged 3–6 (BIS 3–6)	Ballespi, Jane, and Riba (2012a, 2012b)	3–6 years/37 items	One broadband scale	.26 (parent report) and .49 (teacher report) for whole sample; 54 (parent report) and .64 (teacher report) children extremely high or low in BI	*Pros:* Captures social and non-social BI (without distinguishing them) *Cons:* Poor inter-rater reliability for items measuring fear or negative affect

Concurrent report by children/adolescents and parents	Behavioral Inhibition Scale (BIS)	Muris et al. (1999, 2003), Muris, Rassin, Franken, and Leemreis (2007), Gest (1997), van Brakel, Muris, and Bogels (2004), van Brakel et al. (2006)	All ages (self-reported ages 11+ years)/4–8 items	One scale assessing BI in response to novel social interactions	With observational measures: parent report = .44 with social and .13 with non-social behaviors; no data available for self-report version	*Pros:* Very brief, can be used in longitudinal research *Cons:* Items are limited to concern about talking to unfamiliar people (social BI)
Retrospective reports by adolescents or adults	Retrospective Self-Report of Inhibition (RSRI)	Reznick et al. (1992), Hayward et al. (1998), Neal, Edelmann, and Glachan (2002), Rohrbacher et al. (2008)	Adult and adolescent report/30 items	Two factors for adults: social/school inhibition and illness/fear behaviors; three factors for adolescents: fearfulness, social avoidance, and illness behavior	None available	*Pros*: Widely used in adolescent and adult samples to ask about elementary school behavior; social inhibition factor captures social BI *Cons:* Fear/illness items overlap with a broad range of anxiety symptoms; no data on association with observed BI in early childhood; possibility of recall bias

(*continued*)

Table 7.2 (*Continued*)

	Measure	*References*	*Ages/number of items*	*Factors/scales*	*Correlation with observational measures*	*Comments*
	Retrospective Measure of Behavioral Inhibition (RMBI)	Gladstone and Parker (2005), Gladstone et al. (2005)	Adult report (18+ years)/18 items	Four factors assessing BI during elementary school: non-approach, fearful inhibition, risk avoidance, and shyness and sensitivity	None available	*Pros:* Gets reports of the kind of childhood BI behaviors measured in laboratory protocols (e.g., withdrawal, reticence, crying to novelty), recalled from elementary school; distinguishes social and non-social BI *Cons:* No data on association with observed BI in early childhood; possibility of recall bias

monitoring-only conditions (Hirshfeld-Becker et al., 2008, 2010; Kennedy, Rapee, & Edwards, 2009; Rapee, Kennedy, Ingram, Edwards, & Sweeney, 2005), with treatment gains maintained or intensified over time (e.g., 2–3-year follow-up; Rapee, Kennedy, Ingram, Edwards, & Sweeney, 2010). Interestingly, child CBT interventions have demonstrated efficacy whether delivered to parents and children (e.g., Hirshfeld-Becker et al., 2010) or through parents alone (e.g., Rapee et al., 2005).

In contrast to their utility in reducing anxiety disorders, the efficacy of these interventions in reducing BI is less clear. Some studies have not observed an impact of treatment on BI (Rapee et al., 2005, 2010), whereas others report significant post-treatment decreases in BI (Kennedy et al., 2009) or its associated features (Hirshfeld-Becker et al., 2010). Pahl and Barreet (2010), in a universal prevention intervention for preschoolers, reported that on teacher reports (but not parent reports), the intervention group had greater decreases in BI than a control group, with the largest decrease in girls in this group (notably, the intervention group had higher BI at baseline). At 12-month follow-up, children from the intervention group showed decreases in anxiety from baseline and, for girls only, decreased BI and increased social–emotional competence. Conversely, in our controlled trial of preschoolers at high risk for anxiety, BI was a negative predictor of treatment response (94% improvement rate in non-BI children vs. 63% for those with BI).

Overall, these studies offer promise that BI can be targeted in interventions that ultimately reduce anxiety disorders, but point to the idea that it may be difficult to carry out primary prevention, given that many preschool-aged children with extreme BI may already meet criteria for anxiety disorders. It should also be noted that despite the efficacy of treatment, a significant percentage of children persist with anxiety disorders. Finally, these studies point to the complex interplay of sample characteristics, including gender, severity of BI, and intervention characteristics on treatment outcomes. Although these initial findings suggest that CBT may be promising for young children with or at-risk for anxiety, further studies are needed to compare which of the available approaches are most efficacious and cost-effective.

Summary and Future Directions

To summarize, BI in early childhood has been shown in multiple prospective studies to be an early risk factor predicting subsequent SAD. This risk is particularly pronounced in children who remain stably inhibited across early childhood, whose parents are overprotective or who model avoidant coping, and possibly in children with poorer attention modulation, poor social skills, low approach motivation, or physiologic correlates of sensitivity to novelty. BI may also be associated with later generalized or separation anxiety disorder (Hudson & Dodd, 2012; Hudson, Dodd, and Bovopoulos 2011) and with depression, especially in individuals affected with SAD. Associations with panic disorder and OCD are less clear. CBT for young children, implemented through parents alone or also to children directly, appears promising in reducing anxiety disorders in these children, and screening measures have been developed to assist in identifying BI children with whom to intervene.

As we have noted, several questions remain. First, it is unclear whether all children with BI or only those with parental diathesis for anxiety disorders are at increased risk for social anxiety. To answer this question, researchers looking at non-high-risk samples would need to examine and covary lifetime history of parental anxiety disorders. Second, because many studies of BI children contrast them with children extreme in disinhibition, we cannot determine whether correlates truly relate to BI rather than (inversely) to disinhibition. Studies that compare BI children with non-BI–non-disinhibited children are needed. Third, although studies have raised promising hypotheses, prospective longitudinal studies are needed to better characterize factors that account for the transition from BI to SAD. Additional questions that require clarification include whether inhibited children are more easily conditionable to aversive stimuli or less able to learn to extinguish fears; whether BI is indeed a negative predictor of response to CBT; and whether targeted preventive interventions ought to be offered to all young children with BI, to those with additional risk factors (such as parental anxiety disorder), or only to those who have already developed anxiety disorders.

References

Asendorpf, J. (1994). The malleability of behavioral inhibition: A study of individual developmental functions. *Developmental Psychology, 30*(6), 912–919. doi:10.1037/0012-1649.30.6.912

Ballespi, S., Jane, M. C., & Riba, M. D. (2012a). Parent and teacher ratings of temperamental disposition to social anxiety: The BIS 3–6. *Journal of Personality Assessment, 94*, 164–174. doi:10.1080/00223891.2011.645929

Ballespi, S., Jane, M. C., & Riba, M. D. (2012b). Who should report abnormal behavior at preschool age? The case of behavioral inhibition. *Child Psychiatry and Human Development, 43*(1), 48–69. doi:10.1007/s10578-011-0250-5

Bar-Haim, Y., Fox, N. A., Benson, B., Guyer, A. E., Williams, A., Nelson, E. E., . . . , Ernst, M. (2009). Neural correlates of reward processing in adolescents with a history of inhibited temperament. *Psychological Science, 20*(8), 1009–1018. doi:10.1111/j.1467-9280.2009.02401.x

Battaglia, M., Bajo, S., Strambi, L. F., Brambilla, F., Castronovo, C., Vanni, G., & Bellodi, L. (1997). Physiological and behavioral responses to minor stressors in offspring of patients with panic disorder. *Journal of Psychiatric Research, 31*(3), 365–376. doi:10.1016/S0022-3956(97)00003-4

Beesdo, K., Bittner, A., Pine, D. S., Stein, M. B., Hofler, M., Lieb, R., & Wittchen, H. U. (2007). Incidence of social anxiety disorder and the consistent risk for secondary depression in the first three decades of life. *Archives of General Psychiatry, 64*(8), 903–912. doi:10.1001/archpsyc.64.8.903

Belsky, J., Hsieh, K.-H., & Crnic, K. (1998). Mothering, fathering, and infant negativity as antecedents of boys' externalizing problems and inhibition at age 3 years: Differential susceptibility to rearing experience? *Development and Psychopathology, 10*, 301–319. doi:10.1017/S095457949800162X

Biederman, J., Hirshfeld-Becker, D. R., Rosenbaum, J. F., Herot, C., Friedman, D., Snidman, N., . . . , Faraone, S. V. (2001). Further evidence of association between behavioral inhibition and social anxiety in children. *American Journal of Psychiatry, 158*(10), 1673–1679. doi:10.1176/appi.ajp.158.10.1673

Biederman, J., Rosenbaum, J. F., Bolduc-Murphy, E. A., Faraone, S. V., Chaloff, J., Hirshfeld, D. R., & Kagan, J. (1993). A 3-year follow-up of children with and without behavioral inhibition. *Journal of the American Academy of Child and Adolescent Psychiatry, 32*(4), 814–821. doi:10.1097/00004583-199307000-00016

Biederman, J., Rosenbaum, J., Hirshfeld, D., Faraone, S., Bolduc, E., Gersten, M.,..., Reznick, J. S. (1990). Psychiatric correlates of behavioral inhibition in young children of parents with and without psychiatric disorders. *Archives of General Psychiatry, 47*, 21–26.

Bishop, G., Spence, S. H., & McDonald, C. (2003). Can parents and teachers provide a reliable and valid report of behavioral inhibition? *Child Development, 74*(6), 1899–1917. doi:10.1046/j.1467-8624.2003.00645.x

Blackford, J. U., Avery, S. N., Cowan, R. L., Shelton, R. C., & Zald, D. H. (2011). Sustained amygdala response to both novel and newly familiar faces characterizes inhibited temperament. *Social Cognitive & Affective Neuroscience, 6*(5), 621–629. doi: 10.1093/scan/nsq073

Blackford, J. U., Avery, S. N., Shelton, R. C., & Zald, D. H. (2009). Amygdala temporal dynamics: Temperamental differences in the timing of amygdala response to familiar and novel faces. *BMC Neuroscience, 10*, 145. doi:10.1186/1471-2202-10-145

Bohlin, G., Hagekull, B., & Andersson, K. (2005). Behavioral inhibition as a precursor of peer social competence in early school age: The interplay with attachment and nonparental care. *Merrill-Palmer Quarterly, 51*(1), 1–19. doi:10.1353/mpq.2005.0001

Broeren, S., & Muris, P. (2010). A psychometric evaluation of the behavioral inhibition questionnaire in a non-clinical sample of Dutch children and adolescents. *Child Psychiatry and Human Development, 41*(2), 214–229. doi:10.1007/s10578-009-0162-9

Broeren, S., Muris, P., Bouwmeester, S., van der Heijden, K. B., & Abee, A. (2011). The role of repetitive negative thoughts in the vulnerability for emotional problems in non-clinical children. *Journal of Child and Family Studies, 20*(2), 135–148. doi:10.1007/s10826-010-9380-9

Brozina, K., & Abela, J. R. (2006). Behavioural inhibition, anxious symptoms, and depressive symptoms: A short-term prospective examination of a diathesis-stress model. *Behaviour Research and Therapy, 44*(9), 1337–1346. doi:10.1016/j.brat.2005.09.010

Brumariu, L. E., & Kerns, K. A. (2010). Mother-child attachment patterns and different types of anxiety symptoms: Is there specificity of relations? *Child Psychiatry and Human Development, 41*(6), 663–674. doi:10.1007/s10578-010-0195-0

Burkhouse, K. L., Gibb, B. E., Coles, M. E., Knopik, V. S., & McGeary, J. E. (2011). Serotonin transporter genotype moderates the link between children's reports of overprotective parenting and their behavioral inhibition. *Journal of Abnormal Child Psychology, 39*(6), 783–790. doi:10.1007/s10802-011-9526-2

Caspi, A., Harrington, H., Milne, B., Amell, H. W., Theodore, R. F., & Moffit, T. E. (2003). Children's behavioral styles at age 3 are linked to their adult personality traits at age 26. *Journal of Personality, 71*(4), 495–513. doi:10.1111/1467-6494.7104001

Caspi, A., Moffitt, T. E., Newman, D. L., & Silva, P. A. (1996). Behavioral observations at age 3 years predict adult psychiatric disorders. *Archives of General Psychiatry, 53*(11), 1033–1039

Caspi, A., & Silva, P. A. (1995). Temperamental qualities at age 3 predict personality traits in young adulthood: Longitudinal evidence from a birth cohort. *Child Development, 66*, 486–498

Chen, X., Chen, H., Li, D., & Wang, L. (2009). Early childhood behavioral inhibition and social and school adjustment in Chinese children: A 5-year longitudinal study. *Child Development, 80*(6), 1692–1704. doi:10.1111/j.1467-8624.2009.01362.x

Chen, X., Hastings, P. D., Rubin, K. H., Chen, H., Cen, G., & Stewart, S. (1998). Child-rearing attitudes and behavioral inhibition in Chinese and Canadian toddlers: A cross-cultural study. *Developmental Psychology, 34*(4), 677–686. doi:10.1037/0012-1649.34.4.677

Chronis-Tuscano, A., Degnan, K. A., Pine, D. S., Perez-Edgar, K., Henderson, H. A., Diaz, Y., . . . , Fox, N. A. (2009). Stable early maternal report of behavioral inhibition predicts lifetime social anxiety disorder in adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry, 48*(9), 928–935. doi:10.1097/CHI.0b013e3181ae09df

Clauss, J. A., Cowan, R. L., & Blackford, J. U. (2011). Expectation and temperament moderate amygdala and dorsal anterior cingulate cortex responses to fear faces. *Cognitive, Affective, and Behavioral Neuroscience, 11*(1), 13–21. doi:10.3758/s13415-010-0007-9

Coles, M. E., Schofield, C. A., & Pietrefesa, A. S. (2006). Behavioral inhibition and obsessive-compulsive disorder. *Journal of Anxiety Disorders, 20*(8), 1118–1132. doi:10.1016/j.janxdis.2006.03.003

Cooper, P. J., & Eke, M. (1999). Childhood shyness and maternal social phobia: A community study. *The British Journal of Psychiatry, 174*, 439–443. doi:10.1192/bjp.174.5.439

Dalrymple, K. L., Herbert, J. D., & Gaudiano, B. A. (2007). Onset of illness and developmental factors in social anxiety disorder: Preliminary findings from a retrospective interview. *Journal of Psychopathology and Behavioral Assessment, 29*(2), 101–110. doi:10.1007/s10862-006-9033-x

Degnan, K. A., Henderson, H. A., Fox, N. A., & Rubin, K. H. (2008). Predicting social wariness in middle childhood: The moderating roles of childcare history, maternal personality and maternal behavior. *Social Development, 17*(3), 471–487. doi:10.1111/j.1467-9507.2007.00437.x

DiLalla, L. F., Kagan, J., & Reznick, J. S. (1994). Genetic etiology of behavioral inhibition among 2-year-old children. *Infant Behavior and Development, 17*, 405–412. doi:10.1016/0163-6383(94)90032-9

Durbin, C. E., Klein, D. N., Hayden, E. P., Buckley, M. E., & Moerk, K. C. (2005). Temperamental emotionality in preschoolers and parental mood disorders. *Journal of Abnormal Psychology, 114*(1), 28–37. doi:10.1037/0021-843X.114.1.28

Dyson, M. W., Klein, D. N., Olino, T. M., Dougherty, L. R., & Durbin, C. E. (2011). Social and non-social behavioral inhibition in preschool-age children: Differential associations with parent-reports of temperament and anxiety. *Child Psychiatry and Human Development, 42*(4), 390–405. doi:10.1007/s10578-011-0225-6

Eisenberg, N., Shepard, S. A., Fabes, R. A., Murphy, B. C., & Guthrie, I. K. (1998). Shyness and children's emotionality, regulation, and coping: Contemperaneous, longitudinal, and across-contect relations. *Child Development, 69*(3), 767–790.

Emde, R. N., Plomin, R., Robinson, J., Corley, R., DeFries, J., Fulker, D. W., . . . , Zahn-Waxler, C. (1992). Temperament, emotion, and cognition at fourteen months: The MacArthur Longitudinal Twin Study. *Child Development, 63*, 1437–1455. doi:10.2307/1131567

Essex, M. J., Klein, M. H., Slattery, M. J., Goldsmith, H. H., & Kalin, N. H. (2010). Early risk factors and developmental pathways to chronic high inhibition and social anxiety disorder in adolescence. *American Journal of Psychiatry, 167*(1), 40–46. doi:10.1176/appi.ajp.2009.07010051

Fincham, D., Smit, J., Carey, P., Stein, D. J., & Seedat, S. (2008). The relationship between behavioural inhibition, anxiety disorders, depression and CD4 counts in HIV-positive adults: A cross-sectional controlled study. *AIDS Care, 20*(10), 1279–1283. doi:10.1080/09540120801927025

Fox, N. A., Henderson, H. A., Marshall, P. J., Nichols, K. E., & Ghera, M. M. (2005). Behavioral inhibition: Linking biology and behavior within a developmental framework. *Annual Review of Psychology, 56*, 235–262. doi:10.1146/annurev.psych.55.090902.141532

Fox, N. A., Henderson, H. A., Rubin, K. H., Calkins, S. D., & Schmidt, L. A. (2001). Continuity and discontinuity of behavioral inhibition and exuberance: Psychophysiological and behavioral influences across the first four years of life. *Child Development, 72*(1), 1–21. doi:10.1111/1467-8624.00262

Fox, N. A., Nichols, K. E., Henderson, H. A., Rubin, K., Schmidt, L., Hamer, D., . . . , Pine, D. S. (2005). Evidence for a gene-environment interaction in predicting behavioral inhibition in middle childhood. *Psychological Science, 16*(12), 921–926. doi:10.1111/j.1467-9280.2005.01637.x

Garcia-Coll, C., Kagan, J., & Reznick, J. S. (1984). Behavioral inhibition in young children. *Child Development, 55*, 1005–1019. doi:10.2307/1130152

Geng, F., Hu, Y., Wang, Y., & Chen, F. (2011). Two types of behavioral inhibition: Relations to effortful control and attention in school children. *Journal of Research in Personality, 45*(6), 662–669.

Gest, S. D. (1997). Behavioral inhibition: Stability and associations with adaptation from childhood to early adulthood. *Journal of Personality and Social Psychology, 72*(2), 467–475. doi:10.1037/0022-3514.72.2.467

Gladstone, G., & Parker, G. (2005). Measuring a behaviorally inhibited temperament style: Development and initial validation of new self-report measures. *Psychiatry Research, 135*(2), 133–143. doi:10.1016/j.psychres.2005.03.005

Gladstone, G. L., & Parker, G. B. (2006). Is behavioral inhibition a risk factor for depression? *Journal of Affective Disorders, 95*(1–3), 85–94. doi:10.1016/j.jad.2006.04.015

Gladstone, G. L., Parker, G. B., Mitchell, P. B., Wilhelm, K. A., & Malhi, G. S. (2005). Relationship between self-reported childhood behavioral inhibition and lifetime anxiety disorders in a clinical sample. *Depression and Anxiety, 22*(3), 103–113. doi:10.1002/da.20082

Goldsmith, H. H., Rieser-Danner, L. A., & Briggs, S. (1991). Evaluating convergent and discriminant validity of temperament questionnaires for preschoolers, toddlers, and infants. *Developmental Psychology, 27*(4), 566–579. doi:10.1037/0012-1649.27.4.566

Guyer, A., Nelson, E. E., Perez-Edgar, K., Hardin, M. G., Robserson-Nay, R., Monk, C. S., . . . , Ernst, M. (2006). Striatal functional alteration in adolescents characterized by early childhood behavioral inhibition. *Journal of Neuroscience, 26*(24), 6399–6405. doi:10.1523/JNEUROSCI.0666-06.2006

Hayward, C., Killen, J., Kraemer, K., & Taylor, C. (1998). Linking self-reported childhood behavioral inhibition to adolescent social phobia. *Journal of the American Academy of Child and Adolescent Psychiatry, 37*(12), 1308–1316. doi:10.1097/00004583-199812000-00015

Helfinstein, S. M., Benson, B., Perez-Edgar, K., Bar-Haim, Y., Detloff, A., Pine, D., . . . , Ernst, M. (2011). Striatal responses to negative monetary outcomes differ between temperamentally inhibited and non-inhibited adolescents. *Neuropsychologia, 49*, 479–485. doi:10.1016/j.neuropsychologia.2010.12.015

Helfinstein, S. M., Fox, N. A., & Pine, D. S. (2011). Approach-withdrawal and the role of the striatum in the temperament of behavioral inhibition. *Developmental Psychology, 48*(3), 815–826. doi:10.1037/a0026402

Henderson, H. A., Fox, N. A., & Rubin, K. H. (2001). Temperamental contributions to social behavior: The moderating roles of frontal EEG asymmetry and gender. *Journal of the American Academy of Child and Adolescent Psychiatry, 40*(1), 68–74.

Hirshfeld-Becker, D. R., Biederman, J., Faraone, S., Segool, N., Buchwald, J., & Rosenbaum, J. F. (2004). Lack of association between behavioral inhibition and psychosocial adversity factors in children at risk. *American Journal of Psychiatry, 161*, 547–555. doi:10.1176/appi.ajp.161.3.547

Hirshfeld-Becker, D. R., Biederman, J., Faraone, S. V., Violette, H., Wrightsman, J., & Rosenbaum, J. F. (2002). Temperamental correlates of disruptive behavior disorders in young children: Preliminary findings. *Biological Psychiatry, 51*, 563–574.

Hirshfeld-Becker, D. R., Biederman, J., Henin, A., Faraone, S. V., Davis, S., Harrington, K., & Rosenbaum, J. F. (2007). Behavioral inhibition in preschool children at risk is a specific predictor of middle childhood social anxiety: A five-year follow-up. *Journal of Developmental and Behavioral Pediatrics, 28*(3), 225–233. doi:10.1097/01.DBP.0000268559.34463.d0

Hirshfeld-Becker, D. R., Biederman, J., Henin, A., Faraone, S. V., Micco, J. A., van Grondelle, A., . . . , Rosenbaum, J. F. (2007). Clinical outcomes of laboratory-observed preschool behavioral disinhibition at five-year follow-up. *Biological Psychiatry, 62*, 565–572. doi:10.1016/j.biopsych.2006.10.021

Hirshfeld-Becker, D. R., Biederman, J., & Rosenbaum, J. F. (2004). Behavioral inhibition. In T. L. Morris & J. S. March (Eds.), *Anxiety disorders in children and adolescents* (2nd ed., pp. 27–58). New York, NY: Guilford Press.

Hirshfeld-Becker, D. R., Masek, B., Henin, A., Blakely, L. R., Pollock-Wurman, R. A., McQuade, J., . . . , Biederman, J. (2010). Cognitive behavioral therapy for 4- to 7-year-old children with anxiety disorders: A randomized clinical trial. *Journal of Consulting and Clinical Psychology, 78*(4), 498–510.

Hirshfeld-Becker, D. R., Masek, B., Henin, A., Blakely, L. R., Rettew, D. C., Dufton, L., . . . , Biederman, J. (2008). Cognitive-behavioral intervention with young anxious children. *Harvard Review of Psychiatry, 16*(2), 113–125.

Hirshfeld-Becker, D. R., Rosenbaum, J. F., Biederman, J., Bolduc, E. A., Faraone, S. V., Snidman, N., . . . , Kagan, J. (1992). Stable behavioral inhibition and its association with anxiety disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 31*(1), 103–111. doi:10.1097/00004583-199201000-00016

Hudson, J. L., & Dodd, H. F. (2012). Informing early intervention: Preschool predictors of anxiety disorders in middle childhood. *PLoS One, 7*(8), e42359. doi:10.1371/journal.pone.0042359

Hudson, J. L., Dodd, H. F., & Bovopoulos, N. (2011). Temperament, family environment and anxiety in preschool children. *Journal of Abnormal Child Psychology, 39*(7), 939–951. doi:10.1007/s10802-011-9502-x

Hudson, J. L., Dodd, H. F., Lyneham, H. J., & Bovopoulous, N. (2011). Temperament and family environment in the development of anxiety disorder: Two-year follow-up. *Journal of the American Academy of Child and Adolescent Psychiatry, 50*(12), 1255–1264. doi:10.1016/j.jaac.2011.09.009

Jones, N. A., Field, T., & Almeida, A. (2009). Right frontal EEG asymmetry and behavioral inhibition in infants of depressed mothers. *Infant Behavior and Development, 32*(3), 298–304. doi:10.1016/j.infbeh.2009.04.004

Kagan, J., Arcus, D., Snidman, N., Feng, W. Y., Hendler, J., & Greene, S. (1994). Reactivity in infants: A cross-national comparison. *Developmental Psychology, 30*, 342–345.

Kagan, J., Reznick, J. S., & Snidman, N. (1987). The physiology and psychology of behavioral inhibition in children. *Child Development, 58*, 1459–1473.

Kagan, J., Reznick, J. S., & Snidman, N. (1988). Biological bases of childhood shyness. *Science, 240*, 167–171. doi:10.1126/science.3353713

Kagan, J., Snidman, N., Zetner, M., & Peterson, E. (1999). Infant temperament and anxious symptoms in school age children. *Development and Psychopathology, 11*, 209–224. doi:10.1017/S0954579499002023

Kalin, N. H., Shelton, S. E., Fox, A. S., Oakes, T. R., & Davidson, R. J. (2005). Brain regions associated with the expression and contextual regulation of anxiety in primates. *Biological Psychiatry, 58*, 796–804. doi:10.1016/j.biopsych.2005.05.021

Kashdan, T. B., McKnight, P. E., Richey, J. A., & Hofmann, S. G. (2009). When social anxiety disorder co-exists with risk-prone, approach behavior: Investigating a neglected, meaningful subset of people in the National Comorbidity Survey-Replication. *Behavior Research and Therapy, 47*(7), 559–568.

Kennedy, S. J., Rapee, R. M., & Edwards, S. L. (2009). A selective intervention program for inhibited preschool-aged children of parents with an anxiety disorer: Effects on current anxiety disorders and temperament. *Journal of the American Academy of Child and Adolescent Psychiatry, 48*(6), 602–609.

Kim, J., Klein, D., Olino, T., Dyson, M., Dougherty, L., & Durbin, C. (2011). Psychometric properties of the behavioral inhibition questionnaire in preschool children. *Journal of Personality Assessment, 93*(6), 545–555. doi:10.1080/00223891.2011.608756

Knappe, S., Beesdo-Baum, K., Fehm, L., Stein, M. B., Lieb, R., & Wittchen, H.-U. (2011). Social fear and social phobia types among community youth: Differential clinical features and vulnerability factors. *Journal of Psychiatric Research, 45*(1), 111–120. doi:10.1016/j.jpsychires.2010.05.002

Kochanska, G. (1991). Patterns of inhibition to the unfamiliar in children of normal and affectively ill mothers. *Child Development, 62*, 250–263. doi:10.2307/1131001

Kochanska, G., & Radke-Yarrow, M. (1992). Inhibition in toddlerhood and the dynamics of the child's interaction with an unfamiliar peer at age five. *Child Development, 63*, 325–335. doi:10.2307/1131482

Lamb, M. E., Frodi, A. M., Hwang, C.-P., & Frodi, M. E. (1983). Interobserver and test-retest reliability of Rothbart's infant behavior questionnaire. *Scandinavian Journal of Psychology, 24*, 153–156. doi:10.1111/j.1467-9450.1983.tb00487.x

Manassis, K., Bradley, S., Goldberg, S., Hood, J., & Swinson, R. (1995). Behavioural inhibition, attachment and anxiety in children of mothers with anxiety disorders. *Canadian Journal of Psychiatry, 40*, 87–92.

Marshall, P. J., Reeb, B. C., & Fox, N. A. (2009). Electrophysiological responses to auditory novelty in temperamentally different 9-month-old infants. *Developmental Science, 12*, 568–582. doi:10.1111/j.1467-7687.2008.00808.x

Martins, C. I. M., Silva, P. I. M., Conceicao, L. E. C., Costas, B., Hoglund, E., Overli, O., & Schrama, J. W. (2011). Linking fearfulness and coping styles in Fish. *PLoS One, 6*(11), e28084. doi:10.1371/journal.pone.0028284

Marysko, M., Finke, P., Wiebel, A., Resch, F., & Moehler, E. (2010). Can mothers predict childhood behavioural inhibition in early infancy? *Child and Adolescent Mental Health, 15*(2), 91–96. doi:10.1111/j.1475-3588.2009.00539.x

Matheny, A. P. (1989). Children's behavioral inhibition over age and across situations: Genetic similarity for a trait during change. *Journal of Personality, 57*(2), 215–235. doi:10.1111/j.1467-6494.1989.tb00481.x

McDermott, J. M., Perez-Edgar, K., Henderson, H. A., Chronis-Tuscano, A., Pine, D. S., & Fox, N. A. (2009). A history of childhood behavioral inhibition and enhanced response monitoring in adolescence are linked to clinical anxiety. *Biological Psychiatry, 65*(5), 445–448. doi:10.1016/j.biopsych.2008.10.043

Micco, J. A., Henin, A., Mick, E., Kim, S., Hopkins, C. A., Biederman, J., & Hirshfeld-Becker, D. R. (2009). Anxiety and depressive disorders in offspring at high risk for anxiety: A meta-analysis. *Journal of Anxiety Disorders, 23*(8), 1158–1164. doi:10.1016/j.janxdis.2009.07.021

Mick, M., & Telch, M. (1998). Social anxiety and history of behavioral inhibition in young adults. *Journal of Anxiety Disorders, 12*(1), 1–20. doi:10.1016/S0887-6185(97)00046-7

Moehler, E., Kagan, J., Parzer, P., Brunner, R., Reck, C., Wiebel, A., . . . , Resch, F. (2007). Childhood behavioral inhibition and maternal symptoms of depression. *Psychopathology, 40*(6), 446–452. doi:10.1159/000107429

Moehler, E., Kagan, J., Parzer, P., Wiebel, A., Brunner, R., & Resch, F. (2006). Relation of behavioral inhibition to neonatal and infant cardiac activity, reactivity and habituation. *Personality and Individual Differences, 41*(7), 1349–1358. doi:10.1016/j.paid.2006.05.008

Muris, P., Bos, A. E. R., Mayer, B., Verkade, R., Thewissen, V., & Dell'Avvento, V. (2009). Relations among behavioral inhibition, big five personality factors, and anxiety disorder symptoms in non-clinical children. *Personality and Individual Differences, 46*(4), 525–529. doi:10.1016/j.paid.2008.12.003

Muris, P., Meesters, C., & Spinder, M. (2003). Relationships between child- and parent-reported behavioral inhibition and symptoms of anxiety and depression in normal adolescents. *Personality and Individual Differences, 34*, 759–771. doi:10.1016/S0191-8869(02)00069-7

Muris, P., Merckelbach, H., Schmidt, H., Gadet, B., & Bogie, N. (2001). Anxiety and depression as correlates of self-reported behavioural inhibition in normal adolescents. *Behaviour Research and Therapy, 39*, 1051–1061. doi:10.1016/S0005-7967(00)00081-4

Muris, P., Merckelbach, H., Wessel, I., & van de Ven, M. (1999). Psychopathological correlates of self-reported behavioural inhibition in normal children. *Behaviour Research and Therapy, 37*(6), 575–584. doi:10.1016/S0005-7967(98)00155-7

Muris, P., Rassin, E., Franken, I., & Leemreis, W. (2007). Psychometric properties of the Behavioral Inhibition Scale in young adults. *Journal of Individual Differences, 28*(4), 219–226.

Muris, P., van Brakel, A. M. L., Arntz, A., & Schouten, E. (2011). Behavioral inhibition as a risk factor for the development of childhood anxiety disorders: A longitudinal study. *Journal of Child and Family Studies, 20*(2), 157–170. doi:10.1007/s10826-010-9365-8

Murray, L., de Rosnay, M., Pearson, J., Bergeron, C., Schofield, E., Royal-Lawson, M., & Cooper, P. J. (2008). Intergenerational transmission of social anxiety: The role of social referencing processes in infancy. *Child Development, 79*(4), 1049–1064. doi:10.1111/j.1467-8624.2008.01175.x

Myers, C. E., VanMeenen, K. M., McAuley, J. D., Beck, K. D., Pang, K. C. H., & Servatius, R. J. (2012). Behaviorally inhibited temperament is associated with severity of post-traumatic stress disorder symptoms and faster eyeblink conditioning in veterans. *Stress: The International Journal on the Biology of Stress, 15*(1), 31–44. doi:10.3109/10253890.2011.578184

Neal, J. A., Edelmann, R. J., & Glachan, M. (2002). Behavioural inhibition and symptoms of anxiety and depression: Is there a specific relationship with social phobia? *British Journal of Clinical Psychology, 41*(Pt. 4), 361–374. doi:10.1348/014466502760387489

Olino, T. M., Klein, D. N., Dyson, M. W., Rose, S. A., & Durbin, C. E. (2010). Temperamental emotionality in preschool-aged children and depressive disorders in parents: Associations in a large community sample. *Journal of Abnormal Psychology, 119*(3), 468–478. doi:10.1037/a0020112

Otto, M. W., Henin, A., Hirshfeld-Becker, D. R., Pollack, M. H., Biederman, J., & Rosenbaum, J. F. (2007). Posttraumatic stress disorder symptoms following media exposure to

tragic events: Impact of 9/11 on children at risk for anxiety disorders. *Journal of Anxiety Disorders, 21*(7), 888–902. doi:10.1016/j.janxdis.2006.10.008

Pahl, K. M., & Barrett, P. M. (2010). Preventing anxiety and promoting social and emotional strength in preschool children: A universal evaluation of the Fun FRIENDS program. *Advances in School Mental Health Promotion, 3*(3), 14–25.

Pauli-Pott, U., & Beckmann, D. (2007). On the association of interparental conflict with developing behavioral inhibition and behavior problems in early childhood. *Journal of Family Psychology, 21*, 529–532. doi:10.1037/0893-3200.21.3.529

Pérez-Edgar, K., Reeb-Sutherland, B. C., McDermott, J. M., White, L. K., Henderson, H. A., Degnan, K. A., . . . , Fox, N. A. (2011). Attention biases to threat link behavioral inhibition to social withdrawal over time in very young children. *Journal of Abnormal Child Psychology, 39*, 885–895. doi:10.1007/s10802-011-9495-5

Pérez-Edgar, K., Roberson-Nay, R., Hardin, M. G., Poeth, K., Guyer, A. E., Nelson, E. E., . . . , Ernst, M. (2007). Attention alters neural responses to evocative faces in behaviorally inhibited adolescents. *Neuroimage, 35*, 1538–1546. doi:10.1016/j.neuroimage.2007.02.006

Rapee, R. M., Kennedy, S., Ingram, M., Edwards, S., & Sweeney, L. (2005). Prevention and early intervention of anxiety disorders in inhibited preschool children. *Journal of Consulting and Clinical Psychology, 73*(3), 488–497.

Rapee, R. M., Kennedy, S. J., Ingram, M., Edwards, S. L., & Sweeney, L. (2010). Altering the trajectory of anxiety in at-risk young children. *American Journal of Psychiatry, 167*(12), 1518–1525.

Reeb-Sutherland, B. C., Helfinstein, S. M., Degnan, K. A., Pérez-Edgar, K., Henderson, H. A., Lissek, S., . . . , Fox, N. A. (2009). Startle response in behaviorally inhibited adolescents with a lifetime occurrence of anxiety disorders. *Journal of the American Academy of Child and Adolescent Psychiatry, 48*(6), 610–617. doi:10.1097/CHI.0b013e31819f70fb

Reeb-Sutherland, B. C., Vanderwert, R. E., Degnan, K. A., Marshall, P. J., Pérez-Edgar, K., Chronis-Tuscano, A., . . . Fox, N. A. (2009). Attention to novelty in behaviorally inhibited adolescents moderates risk for anxiety. *Journal of Child Psychology and Psychiatry, 50*(11), 1365–1372. doi:10.1111/j.1469-7610.2009.02170.x

Reznick, J. S., Hegeman, I. M., Kaufman, E., Woods, S. W., & Jacobs, M. (1992). Retrospective and concurrent self-report of behavioral inhibition and their relation to adult mental health. *Development and Psychopathology, 4*, 301–321. doi: 10.1017/S095457940000016X

Robinson, J. L., Kagan, J., Reznick, J. S., & Corley, R. (1992). The heritability of inhibited and uninhibited behavior: A twin study. *Developmental Psychology, 28*(6), 1030–1037. doi: 10.1037/0012-1649.28.6.1030

Rohrbacher, H., Hoyer, J., Beesdo, K., Höfler, M., Bittner, A., Lieb, R., & Wittchen, H. U. (2008). Psychometric properties of the Retrospective Self Report of Inhibition (RSRI) in a representative German sample. *International Journal of Methods in Psychiatric Research, 17*(2), 80–88. doi: 10.1002/mpr.233

Rosenbaum, J. F., Biederman, J., Bolduc, E. A., Hirshfeld, D. R., Faraone, S. V., & Kagan, J. (1992). Comorbidity of parental anxiety disorders as risk for child-onset anxiety in inhibited children. *American Journal of Psychiatry, 149*, 475–478.

Rosenbaum, J. F., Biederman, J., Gersten, M., Hirshfeld, D. R., Meminger, S. R., Herman, J. B., . . . , Snidman, N. (1988). Behavioral inhibition in children of parents with panic disorder and agoraphobia: A controlled study. *Archives of General Psychiatry, 45*, 463–470.

Rosenbaum, J. F., Biederman, J., Hirshfeld, D. R., Faraone, S. V., Bolduc, E. A., Kagan, J., . . . , Reznick, J. S. (1991). Further evidence of an association between behavioral inhibition

and anxiety disorders: Results from a family study of children from a non-clinical sample. *Journal of Psychiatric Research, 25*(25), 49–65. doi: 10.1016/0022-3956(91)90015-3

Rosenbaum, J. F., Biederman, J., Hirshfeld-Becker, D. R., Kagan, J., Snidman, N., Friedman, D.,. . . , Faraone, S. V. (2000). A controlled study of behavioral inhibition in children of parents with panic disorder and depression. *American Journal of Psychiatry, 157*(12), 2002–2010. doi:10.1176/appi.ajp.157.12.2002

Rotge, J. Y., Grabot, D., Aouizerate, B., Pélissolo, A., Lépine, J. P., & Tignol, J. (2011). Childhood history of behavioral inhibition and comorbidity status in 256 adults with social phobia. *Journal of Affective Disorders, 129*(1–3), 338–341. doi:10.1016/j.jad.2010.07.031

Rothbart, M. (1981). Measurement of temperament in infancy. *Child Development, 52*, 569–578.

Rothbart, M. (1986). Longitudinal observation of infant temperament. *Developmental Psychology, 22*, 356–365. doi:10.1037/0012-1649.22.3.356

Rubin, K., Burgess, K. B., & Hastings, P. D. (2002). Stability and social-behavioral consequences of toddlers' inhibited temperament and parenting behaviors. *Child Development, 73*(2), 483–495. doi:10.1111/1467-8624.00419

Rubin, K., Hastings, P., Stewart, S., Henderson, H., & Chen, X. (1997). The consistency and concomitants of inhibition: Some of the children, all of the time. *Child Development, 68*(3), 467–483. doi:10.2307/1131672

Rubin, K. H., Hemphill, S. A., Chen, X., Hastings, P., Sanson, A., Coco, A. L.,. . . , Liyin, C. (2006). A cross-cultural study of behavioral inhibition in toddlers: East-West-North-South. *International Journal of Behavioral Development, 30*(3), 219–226. doi:10.1177/0165025406066723

Scheres, A., Milham, M. P., Knutson, B., & Castellanos, F. X. (2007). Ventral striatal hyporesponsiveness during reward anticipation in attention-deficit hyperactivity disorder. *Biological Psychiatry, 61*(5), 720–724. doi:10.1016/j.biopsych.2006.04.042

Schmidt, L. A., & Fox, N. A. (1998). Fear-potentiated startle responses in temperamentally different human infants. *Developmental Psychobiology, 32*, 113–120. doi:10.1002/(SICI)1098-2302(199803)32:2<113::AID-DEV4>3.0.CO;2-S

Schmidt, L. A., Fox, N. A., Rubin, K. H., Sternberg, E. M., Gold, P. W., Smith, C. C., & Schulkin, J. (1997). Behavioral and neuroendocrine responses in shy children. *Developmental Psychobiology, 30*, 127–140. doi:10.1002/(SICI)1098-2302(199703)30:2<127::AID-DEV4>3.0.CO;2-S

Schmidt, L. A., Fox, N. A., Schulkin, J., & Gold, P. W. (1999). Behavioral and psychophysiological correlates of self-presentation in temperamentally shy children. *Developmental Psychobiology, 35*, 119–135. doi:10.1002/(SICI)1098-2302(199909)35:2<119::AID-DEV5>3.0.CO;2-G

Schofield, C. A., Coles, M. E., & Gibb, B. E. (2009). Retrospective reports of behavioral inhibition and young adults' current symptoms of social anxiety, depression, and anxious arousal. *Journal of Anxiety Disorders, 23*(7), 884–890. doi:10.1016/j.janxdis.2009.05.003

Schwartz, C. E., Kunwar, P. S., Greve, D. N., Moran, L. R., Viner, J. C., Covino, J. M.,. . . , Wallace, S. R. (2010). Structural differences in adult orbital and ventromedial prefrontal cortex predicted by infant temperament at 4 months of age. *Archives of General Psychiatry, 67*(1), 78–84. doi:10.1001/archgenpsychiatry.2009.171

Schwartz, C.E., Snidman, N., & Kagan, J. (1999). Adolescent social anxiety as an outcome of inhibited temperament in childhood. *Journal of the American Academy of Child and Adolescent Psychiatry, 38*(8), 1008–1015. doi:10.1097/00004583-199908000-00017

Schwartz, C. E., Wright, C. I., Shin, L. M., Kagan, J., & Rauch, S. L. (2003). Inhibited and uninhibited infants "grown up": Adult amygdalar response to novelty. *Science, 300*, 1952–1953. doi:10.1126/science.1083703

Shamir-Essakow, G., Ungerer, J. A., & Rapee, R. M. (2005). Attachment, behavioral inhibition, and anxiety in preschool children. *Journal of Abnormal Child Psychology, 33*(2), 131–143. doi:10.1007/s10802-005-1822-2

Smoller, J. W., Acierno, J. S., Jr., Rosenbaum, J. F., Biederman, J., Pollack, M. H., Memimger, S., . . . , Slaughenhaupt, S. A. (2001). Targeted genome screen of panic disorder and anxiety disorder proneness using homology to murine QTL region. *American Journal of Medical Genetics (Neuropsychiatric Genetics), 105*(2), 195–206.

Smoller, J. W., Paulus, M. P., Fagerness, J. A., Purcell, S., Yamaki, L. H., Hirshfeld-Becker, D. R., . . . , Stein, M. B. (2008). Influence of RGS2 on anxiety-related temperament, personality, and brain function. *Archives of General Psychiatry, 65*(3), 298–308. doi:10.1001/archgenpsychiatry.2007.48

Smoller, J. W., Rosenbaum, J. F., Biederman, J., Kennedy, J., Dai, D., Racette, S., . . . , Slaughenhaupt, S. A. (2003). Association of a genetic marker at the corticotropin releasing hormone locus with behavioral inhibition. *Biological Psychiatry, 54*, 1376–1381. doi:10.1016/S0006-3223(03)00598-5

Smoller, J. W., & Tsuang, M. T. (1998). Panic and phobic anxiety: Defining phenotypes for genetic studies. *American Journal of Psychiatry, 155*(9), 1152–1162.

Smoller, J.W., Yamaki, L., Fagerness, J., Biederman, J., Racette, S., Laird, N., . . . , Sklar, P. B. (2005). The corticotropin releasing hormone gene and behavioral inhibition in children at risk for panic disorder. *Biological Psychiatry, 57*(12), 1485–1492. doi:10.1016/j.biopsych.2005.02.018

Suomi, S. J. (1997). Early determinants of behaviour: Evidence from primate studies. *British Medical Bulletin, 53*(1), 170–184.

Trullas, R., & Skolnick, P. (1993). Differences in fear motivated behaviors among inbred mouse strains. *Psychopharmacology, 111*, 323–331. doi:10.1007/BF02244948

van Ameringen, M., Mancini, C., & Oakman, J. (1998). The relationship of behavioral inhibition and shyness to anxiety disorder. *Journal of Nervous and Mental Disease, 186*, 425–431. doi:10.1097/00005053-199807000-00007

van Brakel, A. M. L., Muris, P., & Bogels, S. M. (2004). Relations between parent- and teacher-reported behavioral inhibition and behavioral observations of this temperamental trait. *Journal of Clinical Child and Adolescent Psychology, 33*(3), 579–589. doi:10.1207/s15374424jccp3303_15

van Brakel, A. M. L., Muris, P., Bögels, S. M., & Thomassen, C. (2006). A multifactorial model for the etiology of anxiety in non-clinical adolescents: Main and interactive effects of behavioral inhibition, attachment and parental rearing. *Journal of Child and Family Studies, 15*(5), 569–579. doi:10.1007/s10826-006-9061-x

Volbrecht, M. M., & Goldsmith, H. H. (2010). Early temperamental and family predictors of shyness and anxiety. *Developmental Psychology, 46*(5), 1192–1205. doi:10.1037/a0020616

Warren, S. L., Gunnar, M. R., Kagan, J., Anders, T. F., Simmens, S. J., Rones, M., . . . , Sroufe, L. A. (2003). Maternal panic disorder: Infant temperament, neurophysiology, and parenting behaviors. *Journal of the American Academy of Child and Adolescent Psychiatry, 42*(7), 814–825. doi:10.1097/01.CHI.0000046872.56865.02

White, L. K., McDermott, J. M., Degnan, K. A., Henderson, H. A., & Fox, N. A. (2011). Behavioral inhibition and anxiety: The moderating roles of inhibitory control and attention shifting. *Journal of Abnormal Child Psychology, 39*(5), 735–747. doi:10.1007/s10802-011-9490-x

Williams, L. R., Degnan, K. A., Pérez-Edgar, K. E., Henderson, H. A., Rubin, K. H., Pine, D. S., . . . , Fox, N. A. (2009). Impact of behavioral inhibition and parenting style on internalizing and externalizing problems from early childhood through adolescence. *Journal of Abnormal Child Psychology, 37*(8), 1063–1075. doi:10.1007/s10802-009-9331-3

Wilson, K. A., & Hayward, C. (2005). A prospective evaluation of agoraphobia and depression symptoms following panic attacks in a community sample of adolescents. *Journal of Anxiety Disorders, 19*(1), 87–103.

Wittchen, H. U., Stein, M. B., & Kessler, R. C. (1999). Social fears and social phobia in a community sample of adolescents and young adults: Prevalence, risk factors and comorbidity. *Psychological Medicine, 29*(2), 309–323. doi:10.1017/S0033291798008174

8

Relational Processes in Social Anxiety Disorder

Lynn E. Alden, Marci J. Regambal, and Leili Plasencia

University of British Columbia, Canada

Social Acceptance: A Fundamental Motivation

Humans are inherently social animals. The drive for social inclusion is believed to be an evolved mechanism that was as important to survival as physical self-preservation (Baumeister & Leary, 1995; DeWall & Bushman, 2011; see also **Chapter 2**). For our forebears, group membership would have provided access to shared resources (food, shelter) and defense against physical threats. In support of the evolutionary significance of social inclusion, research shows that ostracism and other negative social events result in what has been called *social pain*. Social pain activates the same neural circuits and produces many of the same emotional and behavioral effects as physical pain and other threats to survival (e.g., Eisenberger & Cole, 2012; Eisenberger, Inagaki, Muscatell, Haltom, & Leary, 2011; Eisenberger, Lieberman, & Williams, 2003; Kross, Berman, Mischel, Smith, & Wager, 2011; Riva, Wirth, & Williams, 2011). Indeed, the need for social inclusion is so deeply embedded that even ostracism via a computer threatens self-esteem and life meaning (Zadro, Williams, & Richardson, 2004).

We social anxiety researchers have tended to focus on self-related processes, for example, negative self-beliefs and negatively biased self-judgments. Relational theorists argue that what appear to be self-related cognitive processes, as well as the *self-conscious emotions* (e.g., pride, shame, guilt), actually arise in the service of promoting social relatedness. The most widely recognized example is self-esteem, one's overall evaluation of one's self-worth. Relational theorists propose that self-esteem evolved to function as a *sociometer* that monitors cues of potential rejection and motivates us to adopt strategies to avoid exclusion (e.g., Baumeister & Leary, 1995; Leary, 2007; see also **Chapter 2**). Consistent with this hypothesis, social inclusion is associated with higher self-esteem (Denissen, Penke, Schmitt, & van Aken, 2008; Leary, Haupt, Strausser, & Chokel, 1998). Conversely, negative social events (e.g., bullying) are associated with lower self-esteem and subsequent difficulties maintaining friendships (e.g., Schafer et al., 2004). Moreover, reductions in friendships and increased

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

interpersonal conflict have been found to lead to decreases in self-esteem over time (Stinson et al., 2008). Although self-esteem fluctuates in response to social events, our chronic (trait) background level of self-esteem appears to act as an interpersonal guidance system that influences our signature social motivations, perceptions, and behaviors (e.g., seeking belonging or avoiding rejection). Among other things, low trait self-esteem has been shown to impede our ability to detect acceptance cues and to fuel self-defeating social strategies (Cameron, Stinson, Gaetz, & Balchen, 2010). Interestingly, following social exclusion, individuals with lower self-esteem display stronger activation of the dorsal anterior cingulate cortex (dACC), one of the brain structures that mediates social pain, which suggests that individuals with chronically low self-esteem are biologically more sensitive to social rejection (Onoda et al., 2010). This finding again underscores the link between self-processes and social inclusion.

Social Bonds and Human Well-Being

Social relationships have profound implications for health (Uchino, Cacioppo, & Kiecolt-Glaser, 1996). Prospective studies consistently reveal that low social integration predicts increased mortality even when baseline biomedical and lifestyle factors are controlled (e.g., House, Robbins, & Metzner, 1982; Schoenbach, Kaplan, Fredman, & Kleinbaum, 1986). Specifically, individuals with few social contacts and relationships have roughly double the risk of dying over the subsequent decade relative to their socially connected counterparts. Indeed, low social integration is a major risk factor for mortality, comparable to obesity and possibly even smoking (see House, Landis, & Umberson, 1988). The link between social integration and mortality is attributed in part to the close link between personal relationships and immune system regulation (Kiecolt-Glaser, Gouin, & Hantsoo, 2010). Inflammation is a reliable positive predictor of mortality in older adults and is specifically linked to a variety of health problems, including cardiovascular disease, diabetes, arthritis, osteoporosis, and Alzheimer's disease. Evidence from a variety of research paradigms all reveal that low social support and greater interpersonal conflict are associated with higher proinflammatory cytokines and immunodysregulation. Even brief situational threats to one's social esteem, acceptance, or status increase cortisol and proinflammatory cytokine activity (e.g., Dickerson, Gable, Irwin, Aziz, & Kemeny, 2009).

Relational researchers distinguish objectively defined *social isolation*, the number or frequency of social contacts, from *loneliness*, the perception that one's social needs are not being met by the quantity or especially the quality of one's social relationships. The hypothesized need to belong implicated in evolutionary theories has generally emphasized the psychological impact of social interactions and relationships rather than their presence or absence (e.g., Hawkley & Cacioppo, 2010). Cacioppo and his colleagues have argued that loneliness has a more powerful effect on health than social isolation *per se*. Research indicates that loneliness, not living alone, is associated with non-normative cardiovascular activation and sleep impairment, which are believed to contribute to negative health outcomes (Hawkley, Preacher, & Cacioppo, 2011). Loneliness is also associated with dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis and impaired glucocorticoid-receptor-mediated signal transduction, which

are believed to induce proinflammatory gene expression and alter immunoregulation, thereby promoting the wide range of diseases as described above (Cole et al., 2007). Prospective studies reveal that loneliness predicts more rapid cognitive decline and, more ominously, higher incidence of Alzheimer's disease; indeed, those in the top decile of loneliness were more than twice as likely to develop Alzheimer's disease than those with a stronger sense of social connectedness (Wilson et al., 2007). Furthermore, interventions that increase social contacts result in improved health in older adults (Pitkala, Routasalo, Kautiainen, & Tilvis, 2009; Routasalo, Tilvis, Kautiainen, & Pitkala, 2009).

Social relationships also contribute to *emotional* well-being. As noted above, social inclusion is associated with higher self-esteem (Leary et al., 1998). Social relationships have other benefits as well. Social affirmation facilitates self-growth (DiDonato & Krueger, 2010). The number of social relationships correlates positively with happiness and subjective well-being (Diener & Ryan, 2009; Diener & Seligman, 2002; although see Lucas, Dyrenforth, & Diener, 2008, for discussion of effect size). *Close* relationships, such as intimate friendships and romantic partnerships, seem particularly crucial to our happiness. A cross-cultural study of 42 countries revealed that married individuals were consistently highest in happiness and well-being relative to single and particularly to divorced or widowed individuals (Diener, Gohm, Suh, & Oishi, 2000; see also Haring-Hidore, Stock, Okun, & Witter, 1985). Conversely, social exclusion leads to decline in life meaning (Stillman et al., 2009). Finally, low social support and greater loneliness are consistently linked to depression (e.g., Russell & Cutrona, 1991).

Social Anxiety Disorder and Relationships

Keeping in mind the association between social relationships and physical and emotional well-being, let us examine the SAD literature. First and foremost, it is abundantly clear that individuals with SAD have significant relationship deficiencies. Clinical studies reveal that individuals with SAD have smaller social networks (Davidson, Hughes, George, & Blazer, 1994; Schneier et al., 1994). The same pattern is found in broader community samples, where more severe SAD symptoms were associated with greater rates of living alone, fewer friends, and lower involvement in club or association activities (Falk Dahl & Dahl, 2010).

In addition to objectively smaller social networks, SAD is associated with fewer close (intimate) relationships. Compared with other disorders, individuals with SAD are more likely to report having no close friends (Whisman, Sheldon, & Goering, 2000). The link between SAD and frequency of contact with and number of friends remains even when demographics, comorbid disorders, and absence of other types of relationships (e.g., with family) are controlled (Chou, Liang, & Sareen, 2011; Rodebaugh, 2009). Not only do individuals with SAD have fewer friends, they are less satisfied with the frequency and quality of contact with the friends they do have (Bech & Angst, 1996; Cramer, Polit, Torgersen, & Kringlen, 2005). SAD symptoms are also associated with lower rates of marriage or marriage-like (cohabitation) relationships (Falk Dahl & Dahl, 2010). Even if they are married, people with SAD are less satisfied with their partners (Bech & Angst, 1996) and experience significant

marital distress (albeit not as much as found in individuals with bipolar disorder, alcohol abuse, or generalized anxiety disorder [GAD]; Whisman, 2007).

What are the broader implications of these relational deficiencies? Given the dearth of close relationships, it is not surprising that individuals with SAD perceive an absence of social support, both instrumental and emotional (Cramer et al., 2005; Furmark et al., 1999), and reduced quality of life (e.g., Eng, Coles, Heimberg, & Safren, 2005; Stein & Kean, 2000). Harkening back to the relationship literature above, individuals with SAD also report greater loneliness (Falk Dahl & Dahl, 2010). In light of the established relationship between social isolation and health, surprisingly little research has examined health status in individuals with SAD. However, one study revealed that individuals with SAD were twice as likely to visit their family physicians (Stein et al., 1999), and another found that SAD was associated with poorer overall health, more consultations with doctors, and poorer health-related quality of life (Acarturk, de Graaf, van Straten, ten Have, & Cuijpers, 2008). In addition, SAD has significant comorbidity rates with alcohol and drug-related disorders (e.g., Morris, Stewart, & Ham, 2005), which bodes poorly for health outcomes. Thus, what little empirical data exist suggest that the emotional and health effects of low social integration may extend to individuals with SAD. Before we can more fully understand these relationship deficiencies, we first need to understand adaptive relational functioning. To do so, we turn to relational models.

Relational Theories

The relational literature is extensive, ranging from attachment research to dynamic interpersonal theories to various social psychological perspectives. We will not attempt to cover the entirety of this rich domain but instead will use three contemporary relational models to illustrate key constructs and processes involved in adaptive relational functioning.

Circumplex Models

Circumplex models take their name from the well-established observation that interpersonal behavior can be viewed in terms of two orthogonal dimensions, dominance and affiliation, which can be combined to create a circular space (see Fournier, Moskowitz, & Zuroff, 2011, for more detailed description). The domain of interpersonal problems can also be viewed within this framework, with problematic social behavior reflecting the underlying dimensions of domineering (vs. nonassertive) and overly nurturant (vs. cold/distant) problem behaviors (Alden, Wiggins, & Pincus, 1990). Circumplex researchers have made many contributions to our understanding of interpersonal behavior, and in particular have outlined certain basic principles of interpersonal interactions. We will highlight several that are relevant to our current discussion (see also **Chapter 6**).

The first is the concept of *complementarity*, which refers to a balance or harmony between the behavioral patterns of interaction partners. Research reveals that interactions are more satisfying when interaction partners display *correspondence* on affiliation

(e.g., when friendly actions on one's part are met with friendliness from one's partner) and *reciprocity* on dominance (i.e., when one's attempt to dominate is met with submission from one's partner and vice versa) (Tracey, 2004). Complementarity has been shown to increase positive feelings about one's interaction partners (e.g., Tiedens & Fragale, 2003). It also affects psychophysiological responding, in that noncomplementary interactions produce increases in blood pressure and heart rate (Smith & Ruiz, 2007). Another relevant circumplex concept is that of *mutual influence*. Interaction partners tend to adjust their behaviors to move toward more complementary patterns (e.g., Sadler, Ethier, Gunn, Duong, & Woody, 2009, see also Markey & Kurtz, 2006; Sadler & Woody, 2003). Moving the other way, dyads with decreasing levels of act-by-act complementarity experience greater levels of interpersonal distress, resulting in likely dissolution (Tracey, 2005). The take-home message here is that our interpersonal behavior is sensitive to and shaped by the behavior of those around us, both over the course of a single interaction and over repeated interactions. In addition, social interactions fare better when there is mutual accommodation and balance.

A third circumplex concept is the notion of the *impact message*. Any social behavior (even doing nothing) sends an interpersonal message that "invites" (pulls for) a response (Kiesler, 1996). Following the above, my behavior exerts an interpersonal pull for a complementary response from you. A related point is that my preconceptions, or expectations, about you influence my initial behavior toward you, and can evoke a response that confirms the preconceptions in a self-fulfilling prophecy, a process that Kiesler (and others) describe as an *interpersonal transaction cycle*. Interestingly, people generally expect others to behave in ways that are complementary to their own style even when they have not yet met the other person (Tiedens, Unzueta, & Young, 2007). The apparent confirmation of expectancies reinforces the original behavioral patterns even when those patterns lead to unsatisfying outcomes. Thus, if I expect you to be cold and distant, you are more likely to respond by emotionally distancing yourself from me.

In addition to conceptual contributions, circumplex researchers have developed research procedures and assessment tools to study fine-grained changes in interactions over time. One such example is Moskowitz's (1994) event-contingent recording, a procedure in which individuals rate their daily social interactions on an interpersonal grid. The grid is used to rate the behavior of those with whom they interact in the circumplex space (i.e., on dimensions of dominance and affiliation), and also to rate their own return behavior and emotional responses (see Moskowitz, 1994; Moskowitz, Ho, & Turcotte-Tremblay, 2007). These and other circumplex methods allow us to move out of the lab to study interaction patterns as they unfold in real life.

Friendship Development Models

In light of the deficiencies in close relationships associated with SAD, research that addresses the general development of intimacy is particularly relevant to understanding what goes wrong for those with SAD. Reis and Shaver's (1988) intimacy model specifically deals with the processes involved in moving relationships from emotionally superficial to intimate. Intimacy is defined here as "feeling understood, validated, cared

for, and closely connected with another person" (p. 385). The model implicates two elements as essential to intimacy. The first is self-disclosure, defined as any revelation of information unknown to the other person, including thoughts or feelings. Reis and his colleagues drew on Altman and Taylor's (1973) seminal social penetration theory, which proposed that relationships develop through a process of mutual self-disclosure in which each person gradually reveals more personal opinions, feelings, and experiences while gauging his or her partner's response. At any point in this process, either partner can choose to respond with lesser openness to stop emotional deepening and thereby regulate the level of intimacy of the relationship. Indeed, when asked to define a "close relationship," the predominant characteristic listed by most people was a relationship in which one can be open with the other person (Parks & Floyd, 1996). One of the most robust findings in the relationship literature is that self-disclosure on the part of an individual leads to greater feelings of similarity and liking by others (e.g., Collins & Miller, 1994; Laurenceau, Barrett, & Pietromonaco, 1998). Several qualifications apply; self-disclosure is most effective when it is *reciprocal* (i.e., matches the other person's openness) or is modestly more intimate. Revealing information that greatly exceeds the other's openness, or responding with less openness than the other person, counteracts the similarity/liking effects. Valence is also a consideration. Persistent sharing of negative feelings and experiences (i.e., complaining) places an emotional burden on others that causes them to disengage from emotional contact.

The second element of the intimacy model is partner responsiveness, which is hypothesized to lead to the experience of feeling understood, or *felt understanding* (Reis, 2007). Responsiveness has been shown to contribute to social intimacy and perceived social relatedness beyond self-disclosure alone (Laurenceau et al., 1998; Reis, Sheldon, Gable, Roscoe, & Richard, 2000). People who perceive their partners as responsive become more responsive themselves, which leads to even more positive partner perceptions and so on, in an emerging process that leads to higher-quality relationships for both the partner and the self (Laurenceau et al., 1998). To take one example, individuals who shared positive events to an enthusiastic, positive listener (compared with a neutral one) experienced greater enjoyment, had more positive partner perceptions, and were more willing to self-disclose, both immediately after the interaction and even more openly 1 week later (Reis et al., 2010).

Together, self-disclosure and partner responsiveness were found to facilitate rapport in interactions between unacquainted strangers (Butler et al., 2003) and to contribute to feelings of intimacy in dating and married couples (e.g., Lippert & Prager, 2001; Manne et al., 2004). Felt understanding not only deepens relationships (Reis, 2007), it increases the individual's subjective well-being and positive affect (Lun, Kesebir, & Oishi, 2008; Oishi, Koo, & Akimoto, 2008). Research indicates that felt understanding is greater when one's interaction partners respond to and validate *central* aspects of the self (Reis & Patrick, 1996). There are individual and cultural influences on this validation process. For example, individuals from independent cultures experience greater felt understanding and satisfaction in interactions in which their partners attended to and validated the personal self, whereas individuals from collectivist cultures experienced greater felt understanding when interaction partners attended to and validated features of their collective self (Oishi et al., 2008; Oishi, Krochik, & Akimoto, 2010). To summarize, emotional intimacy develops when both parties are

open with and responsive to the other person and deficiencies in either behavior prevents the development of closeness.

Risk-Regulation Models

As described above, people with SAD are less likely to marry, and when they do, their marriages are characterized by distress. Therefore, the final model we will consider addresses the factors essential to maintain marriage and marriage-like partnerships. Here, Holmes, Murray, and their colleagues proposed a risk-regulation model (e.g., Holmes & Murray, 2011). This model underscores two preconditions for commitment in close relationships. The first is based on the somewhat cynical idea that people engage in ongoing consideration of alternative partners (i.e., weigh the characteristics of their current partner against those of other possible partners). People are said to be more motivated to commit to and remain in relationships with partners who are perceived as having more desirable features and strengths than the viable options. The second and more positive factor is trust. In successful intimate relationships, a person comes to trust that their partner loves and values them and thereby develops a sense of *felt security* (Holmes & Murray, 2007). Perceptions of partner love are partly contingent on the belief that one's partner sees positive qualities in one's self. According to these writers, we search for behavioral evidence of our partner's positive regard and commitment. But behavioral evidence alone is not enough.

In more positive romantic relationships, individuals go beyond the behavioral cues and suspend disbelief to project positivity onto their partner (i.e., believe that their partner values and loves them even at times when the behavioral cues are less clear). Holmes and Murray (2007) describe this as *audacious trust.* Interestingly, people who are more responsive to their partners first project their own responsiveness onto partners and perceive them as more responsive than they actually are, which fuels positive behaviors on the part of the first individual and eventually facilitates deepening of the relationship (e.g., Lemay & Clark, 2008; Lemay, Clark, & Feeney, 2007; Murray, Holmes, & Griffin, 1996). Harkening back to the social pain literature described above, trust is associated with lower self-reported social pain and with greater activation of brain structures responsible for regulating social pain in social rejection situations (Yanagisawa et al., 2011). Thus, individuals with audacious trust may experience less negative emotional reactions and engage in fewer distancing behaviors to minor slights by their partners.

Self-doubts weaken the cognitive and behavioral processes associated with trust in close relationships (Murray, Holmes, & Collins, 2006). Self-doubting individuals assume that others see them in the same way that they see themselves and therefore underestimate the strengths their partners see in them (Murray, Holmes, Griffin, Bellavia, & Rose, 2001). As a result, they experience lower felt security and display *cautious trust.* Murray et al. (2001) studied married and dating couples and found that individuals with negative self-models were significantly more likely to feel that they were loved less than their partner's actual sentiment. In fact, people with negative self-beliefs were loved just as much as individuals with more positive self-regard. Furthermore, individuals who (incorrectly) felt less loved perceived their partners

less positively. Other work suggests that people react to perceptions of a partner's disaffection by finding fault with their partners and distancing themselves from their relationship (Lemay & Clark, 2008). This strategy has been shown to erode felt security, increase anticipated rejection, and result in behaviors that actually elicit less accepting behaviors from their romantic partners (Downey, Freitas, Michaelis, & Khouri, 1998). In short, people with negative self-beliefs view their relationships in ways that mirror their self-doubts, and therefore have difficulty fulfilling their need for security and belonging even in relationships with loving, committed partners.

Summary

The above theories, as well as other interpersonal models, and accompanying research share certain methodological and conceptual features. First, social behavior is studied as an interactive process with methods that encourage measuring both parties in interactions/relationships. Second, there is an emphasis on studying real-life relationships (i.e., with friends, roommates, and romantic partners). Third, relational models attempt to define and measure *prosocial* concepts like intimacy, complementarity, trust, felt security, and responsiveness, and to identify the specific actions that facilitate those states. These prosocial concepts are not well represented in the SAD literature despite growing evidence that others' liking for us, and hence willingness to be in relationships with us, is almost entirely determined by prosocial behaviors (e.g., Reis et al., 2010; Stinson, Cameron, Wood, Gaucher, & Holmes, 2009). It is also important to note that simply engaging in such actions also enhances self-esteem and reduces self-protective social concerns for the individual (Alden & Trew, 2012; Grant & Gino, 2010; Mongrain, Chin, & Shapira, 2011).

Having considered relational models, we next move to empirical studies of relational functioning in individuals with social anxiety and with SAD to assess whether these models describe their relational deficiencies and might be useful in suggesting future directions for research.

SAD and Interpersonal Interactions

We will consider three levels of relational functioning. We begin with a micro-analysis of the specific behaviors associated with SAD, and then broaden our focus to consider relational patterns involved in friendship development and finally in intimate relationships.

Dysfunctional Behavior as Relational Strategies

People with SAD fear negative responses from others. Ironically, they actually can elicit such responses (Clark & Wells, 1995; Rapee & Heimberg, 1997). Others perceive socially anxious individuals as less attractive and friendly, less effective as leaders, and having weaker strength of character (Jones & Russell, 1982; Purdon, Antony,

Monteiro, & Swinson, 2001). Creed and Funder (1998) found that conversational partners of socially anxious individuals expressed less interest and liking for them, engaged in less eye contact, talked at their partner instead of with them, dominated and controlled the interaction, and behaved in a more irritated manner. They also found that college peers described socially anxious individuals as vulnerable to threat, sensitive to demands, lacking personal meaning in life, and moody, compared with individuals low in social anxiety. Heerey and Kring (2007) found that conversational partners of socially anxious students failed to experience the increase in positive affect found in partners of nonsocially anxious students. Even after brief conversations, socially anxious participants tend to be liked less by their conversational partners than are nonanxious people (e.g., Alden & Bieling, 1998; Meleshko & Alden, 1993). Why is this?

Researchers agree that individuals with SAD can behave in a manner that evokes negative social reactions. Cognitive-behavioral theorists view these dysfunctional social behaviors as *safety behaviors* (Clark & Wells, 1995; Rapee & Heimberg, 1997; see also **Chapter 16**). Safety behaviors are defined as overt or covert acts intended to manage or avert a perceived threat and increase the person's sense of safety (Salkovskis, 1991). Using safety behaviors may reduce anxiety in the short term; however, it interferes with processing evidence that the situation is not really dangerous, thus impeding threat disconfirmation and maintaining fear in the long term (e.g., Kim, 2005; Taylor & Alden, 2010; Wells et al., 1995). Research suggests that clinical populations with SAD are able to identify many of their habitual safety behaviors and to drop them in subsequent social interactions with corresponding reductions in threat perceptions and anxiety (Kim, 2005; Taylor & Alden, 2010; Wells et al., 1995). These findings support the idea that the dysfunctional social behaviors of individuals with SAD are often deliberate and strategic.

Plasencia, Alden, and Taylor (2011) asked patients with SAD to identify the safety behaviors they used during a laboratory interaction. The two most common strategies were *avoidance* and *impression management*. Avoidance included such actions as limiting speech, avoiding eye contact, and low self-disclosure, essentially attempting to hide the self. Impression management included attempts to tightly monitor and control one's behavior, over-preparation (e.g., rehearsing what to say before and during social interactions), and feigned friendliness. The latter behaviors are strikingly similar to the adaptive social behaviors used by most people to present themselves favorably and facilitate social interactions. In the case of SAD, however, the actions are adopted because the individual believes they are necessary to avoid rejection, rather than because the person is genuinely engaged in the interaction. These behaviors thus function to present an "artificial self" that the person believes will be less likely to evoke rejection. Both self-concealment and impression-management can be viewed as *relational strategies* that are adopted to manage perceived social threats.

Relational models predict that these strategies would have social consequences. Consistent with this idea, Plasencia et al. (2011) found that both avoidance and impression management were associated with negative outcomes. Avoidance was associated with lower partner interest in future interactions. Interestingly, after controlling for avoidance, impression management was not associated with partner response but rather with higher predicted cost of feared outcomes in subsequent interactions. Thus,

those who used this strategy felt they had more to lose if they were unable to maintain the facade the next time they talked with their partner. Notably, both strategies were associated with a subjective sense of inauthenticity (i.e., the person recognized that their behavior was fake). Harkening back to the relational models above, the failure to present one's genuine self would likely impede satisfying interactions.

To directly examine the effects of safety behaviors on social outcomes, Taylor and Alden (2011) asked one group of patients with SAD to eliminate their safety behaviors prior to a second interaction with a conversational partner. Importantly, their partners' perceptions of them became more positive and the partners became more interested in further interaction, whereas no changes in social outcomes were observed for controls (Taylor & Alden, 2011). Thus, safety behaviors appeared to link the negative self-beliefs of individuals with SAD to poor relational outcomes.

How could relational theories expand on these observations? Applying the circumplex model, Russell et al. (2011) examined the behavior of people with SAD, measuring both inhibited, submissive behaviors as well as prosocial, affiliative behaviors. These researchers used event-contingent monitoring that allowed examination of daily patterns of social interaction. They found that people with SAD showed increased submissive behavior in response to anxiety, but that in situations where they experienced emotional security, they responded with complementary affiliative behavior. These findings demonstrate that socially anxious people recognize when they can connect, and adjust their behavior depending on their impressions of the social environment.

Where do we go from here? A similar study to the one by Russell et al. (2011) could be conducted but instead or additionally monitor the use of safety behaviors so as to identify links between others' behavior, reliance on dysfunctional relational strategies, and social outcomes in daily social interactions. Potential research questions include: In what contexts do people with SAD feel emotionally secure? When they feel secure, do they spontaneously drop their safety strategies? And when socially anxious people drop their safety strategies, do they see others more positively, thereby establishing positive transaction cycles?

SAD and Friendship Development

The intimacy model above indicates that reciprocal self-disclosure is a key factor in the development of friendships. A number of studies have examined how individuals with SAD respond to self-disclosure by others. Work in our lab used the classic self-disclosure paradigm in which socially anxious individuals interact with a confederate who engages in either intimate or nonintimate self-disclosure. Findings revealed that socially anxious individuals were less likely to reciprocate the level of intimacy displayed by their conversational partners and, in turn, their partners were less interested in future contact with them (Meleshko & Alden, 1993). Vonken and her colleagues used structural equation modeling (SEM) to outline the sequence of events linking the behavior of individuals with SAD to social rejection. The results indicated that these individuals evoked negative emotional reactions in both their conversational partners and objective observers, which led their partners to perceive them as dissimilar

to themselves. Partner's negative emotions and perceptions of dissimilarity together predicted their rejection of people with SAD (Vonken, Alden, Bögels, & Roelofs, 2008). The picture that emerges from this work is that socially anxious individuals establish negative interpersonal transaction cycles between themselves and others, in which their attempts to avoid disapproval actually reduce their partner's interest in interacting with them in the future, thereby shutting off the development of closer relationships.

It is important to note that this pattern emerges only, or at least more strongly, when socially anxious people are threatened with negative evaluation. Alden and Bieling (1998) used the self-disclosure paradigm above but manipulated threat expectancy. When faced with threat of negative evaluation, socially anxious individuals displayed the pattern described above and, as before, their partners rated them as less likeable. In contrast, in the positive expectancy condition, the socially anxious participants were as intimate in their disclosures as were the nonanxious controls, and their partners rated them as equally likeable. These findings indicate that socially anxious individuals are able to be open with others but only when they are not concerned about the possibility of negative reactions. This pattern is consistent with circumplex research indicating that social behavior is shaped by the social environment.

Research on SAD has yet to examine other aspects of the intimacy model, such as partner responsiveness and felt understanding. For example, do individuals with SAD ever experience felt understanding or do they feel perpetually misunderstood? Does partner validation of key aspects of self-identity lead to greater openness on the part of individuals with SAD as it does with nonanxious people? Harkening back to the concept of mutual influence, it would also be interesting to see if people with SAD do eventually accommodate to the level of a partner's disclosure over time—perhaps they simply take longer to warm up. Another factor to consider is that most extant findings on self-disclosure are based on nonclinical samples and laboratory interactions. The lab environment may impact socially anxious people differently than controls. It would be valuable to draw on circumplex event-recording methods to examine self-disclosure in real life to determine whether these individuals are able to engage in self-disclosure in their lives and if so, under what conditions.

SAD and Intimate Relationships

Examining intimate relationships in individuals with SAD is important because, unlike interactions with strangers that are primarily motivated by a need to avoid social rejection, interactions within closer relationships may be partially motivated by a need for closeness, which may present particular challenges for people with SAD. The three relational models above indicate that the development and maintenance of intimate relationships require a complex set of behaviors (e.g., self-disclosure), processes (e.g., complementarity), and cognitions (e.g., felt understanding and trust). Although relatively little research has been conducted, the extant literature indicates that socially anxious individuals may experience difficulty in some of these areas.

Grant, Beck, Farrow, and Davila (2007) found that SAD symptoms were associated with the avoidance of expressing emotion, lack of assertion, and interpersonal

dependency in interactions with family and friends. Furthermore, avoidance of expressing emotions predicted depressive symptoms 1 year later. Davila and Beck (2002) identified similar interpersonal styles with the addition of a tendency to avoid conflict. The avoidant and nonassertive style found in these studies is consistent with SAD research on less intimate relationships but also suggests that, in close relationships, SAD is concurrently associated with an over-reliance on others (i.e., dependency).

The tendency to avoid self-disclosure and emotional expression also extends to clinical samples. Sparrevohn and Rapee (2009) found that individuals diagnosed with SAD endorsed lower self-disclosure and expression of emotions within their romantic relationships, compared with individuals without SAD. Importantly, this association remained after controlling for dysphoria (Sparrevohn & Rapee, 2009). Later work suggested this pattern may be stronger in women than in men and for negative than for positive emotions (Cuming & Rapee, 2010). Given the role of self-disclosure in creating intimacy, it is not surprising that the lack of self-disclosure was associated with reduction in perceived social support (Sparrevohn & Rapee, 2009).

Valence also influences the effects of self-disclosure. In any relationship, it is possible that expressing negative emotions will elicit a negative response from a partner. Kashdan, Volkmann, Breen, and Han (2007) demonstrated that for socially anxious individuals, the perceived closeness of their romantic relationship decreased with expression of negative emotions. The opposite was found for nonsocially anxious individuals. These results suggest that the way in which socially anxious individuals share their negative emotions may place a burden on their partners.

Observational studies of social behavior add to this picture. Wenzel, Graff-Dolezal, Macho, and Brendle (2005) examined the communication skills of individuals high and low in SAD symptoms as they and their romantic partners discussed positive, neutral, and negative topics. During all types of conversations, socially anxious individuals displayed fewer "positive" behaviors (e.g., using feeling statements, giving compliments, providing empathy, and using positive nonverbal behaviors), and when discussing a problem topic, displayed more "very negative behaviors" (e.g., put-downs, blaming, and "yes, buts"). There were no differences in behavior of the partners of the socially anxious and nonanxious participants. Beck, Davila, Farrow, and Grant (2006) had couples work their way through a stressful situation. Relationship satisfaction moderated the behavior of socially anxious individuals. Specifically, at high levels of satisfaction, socially anxious individuals demonstrated more negative behaviors (e.g., criticism, rejection, and blame) than nonanxious individuals, whereas at lower levels of satisfaction, the two groups did not differ. Additionally, socially anxious individuals reported more negative affect when their partners displayed positive behaviors. The authors concluded that individuals with SAD might feel safe to display negative behaviors and affect under conditions of high satisfaction. Harkening back to the risk regulation model, it is possible that relationships characterized by trust may offset some of the fears that otherwise interfere with the genuine expression of emotion and self-disclosure. Nonetheless, the observed deficiency in appreciating their partner's positive behavior may not bode well for ongoing relationship satisfaction.

The application of relational models offers promising ways to expand this research. For example, the principles of complementarity and correspondence suggest that individuals with SAD might be more likely to have partners with a dominant and cold

style. It would also be interesting to determine if the negative self-beliefs held by individuals with SAD interfere with the development of trust and felt security within intimate relationships. Interestingly, neither of the two studies that included romantic partners found any difference in the behavior of the partners of individuals with or without SAD (Beck et al., 2006; Wenzel et al., 2005). Including close/romantic partners in future research will be essential in studying the processes central to relational models, to determine the extent to which dysfunctional intimate relationships are a function of both partners, or whether they arise primarily from the anxious individual's perceptions of self and others.

Underexplored Areas

Perhaps the most important direction for future research is to determine whether existing treatments significantly increase prosocial behaviors and social relationships in patients with SAD. Given that quality of life measures repeatedly point to lack of satisfaction with social functioning, surprisingly little research has included relational measures. One exception is work by Heimberg and his colleagues, who found significant improvements in satisfaction with social functioning in SAD patients following Group Cognitive Behavioral Therapy (GCBT), although social satisfaction remained lower than that in the general population (Eng et al., 2005). We have recently experimented with a regimen that integrates principles from the circumplex and intimacy models with standard CBT techniques. A wait list controlled study indicated that the regimen produced significant increases in prosocial behaviors (i.e., social initiation behaviors) and satisfaction with relationships (Alden & Taylor, 2011). Preliminary results from a larger treatment comparison study indicate that addressing relational issues as part of CBT produces greater change in relational functioning than a comparison behavior therapy regimen. These results highlight an area worth pursuing further.

Future comparative studies are necessary to determine whether the interpersonal features of SAD are specific or shared with other anxiety disorders, many of which are also associated with impaired social functioning (see Alden & Regambal, 2011; Beck, 2010). An excellent example is provided by Rodebaugh (2009), who examined epidemiological data and found that among the anxiety disorders, only SAD was uniquely linked with perceived friendship impairment, which suggests that friendship development may be of particular importance to these individuals and needs to be addressed in treatment. Another interesting comparison is between individuals with SAD and those with GAD, as these disorders are often comorbid, raising diagnostic dilemmas. Whereas individuals with SAD have difficulty entering marriage-like relationships (Falk Dahl & Dahl, 2010), individuals with GAD are not only more likely to seek out these relationships but are significantly more likely to experience relationship distress and disruption (Whisman, 2007).

A third under-explored research frontier pertains to cultural differences and other sources of diversity in the social functioning of individuals with SAD. Among other things, culture has been shown to influence felt understanding (Lun, Oishi, Coan, Akimoto, & Miao, 2010; Oishi et al., 2010) and the relationship between social

support and stress (e.g., Taylor, Welch, Kim, & Sherman, 2007). Given our increasingly multi-cultural world, identifying such differences might contribute to our ability to understand and treat SAD in individuals from non-Western cultures (see also **Chapter 11**). There is also a dearth of research examining the effects of SAD on long-term relationships and relationships over the lifespan.

Summary

Our goals in this chapter were to outline the relational deficiencies of individuals with SAD and to suggest how relational models might be useful in understanding those deficiencies. Given the importance of social connections to human health and well-being, further research on SAD and social relationships is badly needed.

References

Acarturk, D., de Graaf, R., van Straten, A., ten Have, M., & Cuijpers, P. (2008). Social phobia and number of social fears, and their association with comorbidity, health-related quality of life and help-seeking. A population-based study. *Social Psychiatry and Psychiatric Epidemiology, 43*, 273–279. doi:10.1007/s00127-008-0309-1

Alden, L. E., & Bieling, P. M. (1998). The interpersonal consequences of the pursuit of safety. *Behaviour Research and Therapy, 36*, 1–9.

Alden, L. E., & Regambal, M. J. (2011). Interpersonal processes in the anxiety disorders. In L. M. Horowitz & S. Strack (Eds.), *The handbook of interpersonal psychology* (pp. 449–470). New York, NY: John Wiley & Sons.

Alden, L. E., & Taylor, C. T. (2011). Relational treatment strategies increase social approach behaviors in patients with generalized social anxiety disorder. *Journal of Anxiety Disorders, 25*, 309–318. doi:10.1016/j.janxdis.2010.10.003

Alden, L. E., & Trew, J. (2012). If it makes you happy: Engaging in kind acts increases positive affect in socially anxious individuals. *Emotion, 13*, 64–75. doi:10.1037/a0027761

Alden, L. E., Wiggins, J. S., & Pincus, A. L. (1990). Construction of circumplex scales for the inventory of interpersonal problems. *Journal of Personality Assessment, 55*, 521–536.

Altman, I., & Taylor, D. A. (1973). *Social penetration: The development of interpersonal relationships.* New York, NY: Rinehart & Winston.

Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychological Bulletin, 11*, 497–529.

Bech, P., & Angst, J. (1996). Quality of life in anxiety and social phobia. *International Clinical Psychopharmacology, 11*, 97–100.

Beck, J. G. (Ed.). (2010). *Interpersonal processes in the anxiety disorders.* Washington, DC: American Psychological Association Press.

Beck, J. G., Davila, J., Farrow, S., & Grant, D. (2006). When the heat is on: Romantic partner responses influence distress in socially anxious women. *Behaviour Research and Therapy, 44*, 737–748. doi:10.1016/j.brat.2005.05.004

Butler, E. A., Egloff, B., Wilhelm, F. H., Smith, N. C., Erickson, E. A., & Gross, J. J. (2003). The social consequences of expressive suppression. *Emotion, 3*, 48–67.

Cameron, J. J., Stinson, D. A., Gaetz, R., & Balchen, S. (2010). Acceptance is in the eye of the beholder: Self-esteem and motivation perceptions of acceptance from the opposite sex. *Journal of Personality and Social Psychology, 99*, 513–529. doi:10.1037/a0018558

Chou, K.-L., Liang, K., & Sareen, J. (2011). The association between social isolation and the DSM-IV mood, anxiety, and substance use disorders: Wave 2 of the National Epidemiologic Survey on Alcohol and Related Conditions. *Journal of Clinical Psychiatry, 72*, 1468–1476.

Clark, D. M., & Wells, A. (1995). A cognitive model of social phobia. In R. G. Heimberg, M. R. Liebowitz, D. A. Hope, & F. R. Schneier (Eds.), *Social phobia: Diagnosis, assessment, and treatment* (pp. 69–93). New York, NY: Guilford Press.

Cole, S. W., Hawkley, L. C., Arevalo, J. M., Sung, C. Y., Rose, R. M., & Cacioppo, J. T. (2007). Social regulation of gene expression in humans: Glucocorticoid resistance in the leukocyte transcriptome. *Genome Biology, 8*, R189.1–R189.13. doi:10.1186/gb-2007-8-9-r189

Collins, N. L., & Miller, L. C. (1994). Self-disclosure and liking: A meta-analytic review. *Psychological Bulletin, 116*, 457–475.

Cramer, V., Polit, C., Torgersen, T., & Kringlen, E. (2005). Quality of life and anxiety disorders: A population study. *Journal of Nervous and Mental Disease, 193*, 196–202.

Creed, A. T., & Funder, D. C. (1998). Social anxiety: From the inside and outside. *Personality and Individual Differences, 25*(1), 19–33. doi:10.1016/S0191-8869(98)00037-3

Cuming, S., & Rapee, R. M. (2010). Social anxiety and self-protective communication style in close relationships. *Behaviour Research and Therapy, 48*, 87–96.

Davidson, J. R. T., Hughes, D. C., George, L. K., & Blazer, D. G. (1994). The boundary of social phobia. Exploring the threshold. *Archives of General Psychiatry, 5*, 975–983.

Davila, J., & Beck, J. G. (2002). Is social anxiety associated with impairment in close relationships? A preliminary investigation. *Behavior Therapy, 33*, 427–444.

Denissen, J. J., Penke, L., Schmitt, D. P., & van Aken, M. A. (2008). Self-esteem reactions to social interactions: Evidence for sociometer mechanisms across days, people, and nations. *Journal of Personality and Social Psychology, 95*, 181–196.

DeWall, C. N., & Bushman, B. J. (2011). Social acceptance and rejection: The sweet and the bitter. *Current Directions in Psychological Science, 20*, 256–260. doi:0.1177/0963721411417545

Dickerson, S. S., Gable, S. L., Irwin, M. R., Aziz, N., & Kemeny, M. E. (2009). Social-evaluative threat and proinflammatory cytokine regulation: An experimental laboratory investigation. *Psychological Science, 20*, 1237–1244.

DiDonato, T. E., & Krueger, J. L. (2010). Interpersonal affirmation and self-authenticity: A test of Rogers' self-growth hypothesis. *Self and Identity, 9*, 322–336. doi:10.1080/15298860903135008

Diener, E. R., Gohm, C. L., Suh, E., & Oishi, S. (2000). Similarity of the relations between marital status and subjective well-being across culture. *Journal of Cross-Cultural Psychology, 31*, 419–436.

Diener, E. R., & Ryan, K. (2009). Subjective well-being: A general overview. *South African Journal of Psychology, 39*, 391–406.

Diener, E. R., & Seligman, M. (2002). Very happy people. *Psychological Science, 13*, 81–84.

Downey, G., Freitas, A. L., Michaelis, B., & Khouri, H. (1998). The self-fulfilling prophecy in close relationships: Rejection sensitivity and rejection by romantic partners. *Journal of Personality and Social Psychology, 75*, 545–560.

Eisenberger, N. I., & Cole, S. W. (2012) Social neuroscience and health: Neurophysiological mechanisms linking social ties with physical health. *Nature Neuroscience, 15*, 669–674. doi:10.1038/nn.3086

Eisenberger, N. I., Inagaki, T. K., Muscatell, K. A., Haltom, K. E. B., & Leary, M. R. (2011). The neural sociometer: Brain mechanisms underlying state self-esteem. *Journal of Cognitive Neuroscience, 23*, 3448–3455. doi:10.1162/jocn_a_00027

Eisenberger, N. I., Lieberman, M. D., & Williams, K. D. (2003). Does rejection hurt? An fMRI study of social exclusion. *Science, 302*, 290–292. doi:10.1126/science.1089134

Eng, W., Coles, M. E., Heimberg, R. G., & Safren, S. A. (2005). Domains of life satisfaction in social anxiety disorder: Relation to symptoms and response to cognitive-behavioral therapy. *Journal of Anxiety Disorders, 19*, 143–156. doi:10.1016/j.janxdis.2004.01.007

Falk Dahl, C. A., & Dahl, A. A. (2010). Lifestyle and social network in individuals with high levels of social phobia/anxiety symptoms: A community-based study. *Social Psychiatry and Psychiatric Epidemiology, 45*, 309–317. doi:10.1007/s00127-009-0069-6

Fournier, M. A., Moskowitz, D. S., & Zuroff, D. C. (2011). Origins and applications of the interpersonal circumplex. In L. M. Horowitz & S. Strack (Eds.), *The handbook of interpersonal psychology* (pp. 57–73). New York, NY: John Wiley & Sons.

Furmark, T., Tillfors, M., Everz, P.-O., Marteinsdottir, I., Gefvert, O., & Fredrikson, M. (1999). Social phobia in the general population: Prevalence and sociodemographic profile. *Social Psychiatry and Psychiatric Epidemiology, 34*, 416–424.

Grant, A. M., & Gino, F. (2010). A little thanks goes a long way: Explaining why gratitude expressions motivate prosocial behaviour. *Journal of Personality and Social Psychology, 6*, 946–955.

Grant, D. M., Beck, J. G., Farrow, S. M., & Davila, J. (2007). Do interpersonal features of social anxiety influence the development of depressive symptoms. *Cognition and Emotion, 21*, 646–663. doi:10.1080/02699930600713036

Haring-Hidore, M., Stock, W. A., Okun, M. A., & Witter, R. A. (1985). Marital status and subjective well-being: A research synthesis. *Journal of Marriage and the Family, 47*, 947–953.

Hawkley, L. C., & Cacioppo, J. T. (2010). Loneliness matters: A theoretical and empirical review of consequences and mechanisms. *Annals of Behavioral Medicine, 40*, 218–227. doi:10.1007/s12160-010-9210-8

Hawkley, L. C., Preacher, K., & Cacioppo, J. (2011). As we said, loneliness (not living alone) explains individual differences in sleep quality: Reply. *Health Psychology, 30*, 136. doi:10.1037/a0022366

Heerey, E. A., & Kring, A. M. (2007). Interpersonal consequences of social anxiety. *Journal of Abnormal Psychology, 116*(1), 125–134. doi:10.1037/0021-843X.116.1.125

Holmes, J. G., & Murray, S. L. (2007). Felt security as a normative resource: Evidence for an elemental risk regulation system. *Psychological Inquiry, 18*, 163–167.

Holmes, J. G., & Murray, S. L. (2011). *Interdependent minds: The dynamics of close relationships.* New York, NY: Guilford Press.

House, J. S., Landis, K. R., & Umberson, D. (1988). Social relationships and health. *Science, 241*, 540–545.

House, J. S., Robbins, C., & Metzner, H. M. (1982). The association of social relationships and activities with mortality: Prospective evidence from the Tecumseh Community Health Study. *American Journal of Epidemiology, 116*, 123–140.

Jones, W. H., & Russell, D. (1982). The social reticence scale: An objective instrument to measure shyness. *Journal of Personality Assessment, 46*(6), 629–631. doi:10.1207/s15327752jpa4606_12

Kashdan, T. B., Volkmann, J., Breen, W. E., & Han, S. (2007). Social anxiety and emotion regulation in romantic relationships: The costs and benefits of negative emotion expression are context-dependent. *Journal of Anxiety Disorders, 21*, 475–492. doi:10.1016/j.janxdis.2006.08.007

Kiecolt-Glaser, J. K., Gouin, J.-P., & Hantsoo, L. (2010). Close relationships, inflammation, and health. *Neuroscience and Biobehavioral Reviews, 35*, 33–38. doi:10.1016/j.neubiorev.2009.09.003

Kiesler, D. J. (1996). *Contemporary interpersonal theory and research: Personality, psychopathology, and psychotherapy.* New York, NY: John Wiley & Sons.

Kim, E.-J. (2005). The effect of the decreased safety behaviors on anxiety and negative thoughts in social phobics. *Journal of Anxiety Disorders, 19*, 69–86.

Kross, E., Berman, M. G., Mischel, W., Smith, E. E., & Wager, T. D. (2011). Social rejection shares somatosensory representations with physical pain. *Proceedings of the National Academy of Science of the United States of America, 108*, 6270–6275.

Laurenceau, J.-P., Barrett, L. F., & Pietromonaco, P. R. (1998). Intimacy as an interpersonal process: The importance of self-disclosure, partner disclosure, and perceived partner responsiveness in interpersonal exchanges. *Journal of Personality and Social Psychology, 74*, 1238–1251.

Leary, M. R. (2007). Motivational and emotional aspects of the self. *Annual Review of Psychology, 58*, 317–344. doi:10.1146/annurev.psych.58.110405.085658

Leary, M. R., Haupt, A., Strausser, K., & Chokel, J. (1998). Calibrating the sociometer: The relationship between interpersonal appraisals and state self-esteem. *Journal of Personality and Social Psychology, 74*, 1290–1299.

Lemay, E. P., & Clark, M. S. (2008). How the head liberates the heart: Projection of communal responsiveness guides relationship promotion. *Journal of Personality and Social Psychology, 94*, 647–671.

Lemay, E. P., Clark, M. S., & Feeney, B. C. (2007). Projection of responsiveness to needs and construction of satisfying communal relationships. *Journal of Personality and Social Psychology, 92*, 834–853. doi:10.1037/0022-3514.92.5.834

Lippert, T., & Prager, K. (2001). Daily experiences of intimacy: A study of couples. *Personal Relationships, 8*, 283–298.

Lucas, R. E., Dyrenforth, P. S., & Diener, E. (2008). Four myths about subjective well-being. *Social and Personality Psychology Compass, 25*, 2001–2015. doi:10.1111/j.1751-9004.2008.00140.x

Lun, J., Kesebir, S., & Oishi, S. (2008). On feeling understood and feeling well: The role of interdependence. *Journal of Research in Personality, 42*, 1623–1628. doi:10.1016/j.jrp.2008.06.009

Lun, J., Oishi, S., Coan, J. A., Akimoto, S., & Miao, F. F. (2010). Cultural variations in motivational responses to felt misunderstanding. *Personality and Social Psychology Bulletin, 36*, 986–996. doi:10.1177/0146167210362979

Manne, S., Ostroff, J., Rini, C., Fox, K., Goldstein, L., & Grana, G. (2004). The interpersonal process model of intimacy: The role of self-disclosure, partner disclosure, and partner responsiveness in interactions between breast cancer patients and their partners. *Journal of Family Psychology, 18*, 589–599. doi:10.1037/0893-3200.18.4.589

Markey, P. M., & Kurtz, J. E. (2006). Increasing acquaintanceship and complementarity of college roommates. *Personality and Social Psychology Bulletin, 32*, 907–916.

Meleshko, K. A., & Alden, L. E. (1993). Anxiety and self-disclosure: Toward a motivational model. *Journal of Personality and Social Psychology, 64*, 1000–1009.

Mongrain, M., Chin, J. M., & Shapira, L. B. (2011). Practicing compassion increases happiness and self-esteem. *Journal of Happiness Studies, 12*, 963–981. doi:10.1007/s10902-010-9239-1

Morris, E. P., Stewart, S. H., & Ham, L. S. (2005). The relationship between social anxiety disorder and alcohol use disorders: A critical review. *Clinical Psychology Review, 25*, 734–760. doi:10.1016/j.cpr.2005.05.004

Moskowitz, D. S. (1994). Cross-situational generality and the interpersonal circumplex. *Journal of Personality and Social Psychology, 66*, 921–933.

Moskowitz, D. S., Ho, M.-H. R., & Turcotte-Tremblay, A.-M. (2007). Contextual influences on interpersonal complementarity. *Personality and Social Psychology Bulletin, 33*, 1051–1063. doi:10.1177/0146167207303024

Murray, S. L., Holmes, J. G., & Collins, N. L. (2006). Optimizing assurance: The risk regulation system in relationships. *Psychological Bulletin, 132*, 641–666. doi:10.1037/0033-2909.132.5.641

Murray, S. L., Holmes, J. G., & Griffin, D. W. (1996). The benefits of positive illusions: Idealization and the construction of satisfaction in close relationships. *Journal of Personality and Social Psychology, 70*, 79–98. doi:10.1177/0146167201274004

Murray, S. L., Holmes, J. G., Griffin, D. W., Bellavia, G., & Rose, P. (2001). The mismeasure of love: How self-doubt contaminates relationship beliefs. *Personality and Social Psychology Bulletin, 27*, 423–436. doi:10.1177/0146167201274004

Oishi, S., Koo, M., & Akimoto, S. (2008). Culture, interpersonal perceptions and happiness in social interactions. *Personality and Social Psychology Bulletin, 34*, 307–320. doi:10.1177/0146167207311198

Oishi, S., Krochik, M., & Akimoto, S. (2010). Felt understanding as a bridge between close relationships and subjective well-being: Antecedents and consequences across individuals and culture. *Social and Personality Psychology Compass, 10*, 403–416. doi:10.1111/j.1751-9004.2010.00264.x

Onoda, K., Okamoto, Y., Nakashima, K., Nittono, H., Yoshimura, S., Yamawaki, S., . . . , Ura, M. (2010). Does low self-esteem enhance social pain? The relationship between trait self-esteem and anterior cingulate cortex activation induced by ostracism. *Social Cognitive and Affective Neuroscience, 5*, 385–391. doi:10.1093/scan/nsq002

Parks, M. R., & Floyd, K. (1996). Meaning for closeness and intimacy in friendship. *Journal of Social and Personal Relationships, 13*, 85–107.

Pitkala, K. H., Routasalo, P., Kautiainen, H., & Tilvis, R. S. (2009). Effects of psychosocial group rehabilitation on health, use of health care services and mortality of older persons suffering from loneliness: A randomized controlled trial. *Journal of Gerontology, 64*, 792–800. doi:10.1093/gerona/glp011

Plasencia, M. L., Alden, L. E., & Taylor, C. T. (2011). Functional implications of safety behaviour subtypes in social anxiety disorder. *Behaviour Research and Therapy, 49*, 665–675. doi:10.1016/j.brat.2011.07.005

Purdon, C., Antony, M., Monteiro, S., & Swinson, R. P. (2001). Social anxiety in college students. *Journal of Anxiety Disorders, 15*, 203–215. doi:10.1016/S0887-6185(01)00059-7

Rapee, R. M., & Heimberg, R. G. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy, 35*(8), 741–756. doi:10.1016/S0005-7967(97)00022-3

Reis, H. T. (2007). Steps toward the ripening of relationship science. *Personal Relationships, 14*, 1–23.

Reis, H. T., & Patrick, B. C. (1996). Attachment and intimacy: Component processes. In A. Kruglanski & E. T. Higgins (Eds.), *Social psychology: Handbook of basic principles* (pp. 523–563). New York, NY: Guilford Press.

Reis, H. T., & Shaver, P. (1988). Intimacy as an interpersonal process. In S. Duck, D. F. Hay, S. E. Hobfoll, W. Ickes, & B. M. Montgomery (Eds.), *Handbook of personal relationships: Theory, research and interventions* (pp. 367–389). Oxford, UK: John Wiley & Sons.

Reis, H. T., Sheldon, K. M., Gable, S. L., Roscoe, J. R., & Richard, M. (2000). Daily well-being: The role of autonomy, competence, and relatedness. *Personality and Social Psychology Bulletin, 26*, 419–435.

Reis, H. T., Smith, S. M., Carmichael, C. L., Caprariello, P. A., Tsai, F., Rodrigues, A., & Maniaci, M. R. (2010). Are you happy for me? How sharing positive events with others provides personal and interpersonal benefits. *Journal of Personality and Social Psychology, 99*, 311–329. doi:10.1037/a0018344

Riva, P., Wirth, J. H., & Williams, I. D. (2011). The consequences of pain: The social and physical pain overlap on psychological responses. *European Journal of Social Psychology, 41*, 681–687.

Rodebaugh, T. L. (2009). Social phobia and perceived friendship quality. *Journal of Anxiety Disorders, 23*, 872–878. doi:10.1016/j.janxdis.2009.05.001

Routasalo, P. E., Tilvis, R. S., Kautiainen, H., & Pitkala, K. H. (2009). Effects of psychosocial group rehabilitation on social functioning, loneliness and well-being of lonely older people: Randomized controlled trial. *Journal of Advanced Nursing, 65*, 297–305. doi:10.1111/j.1365-2648.2008.04837.x

Russell, D. W., & Cutrona, C. T. (1991). Social support, stress, and depressive symptoms among the elderly: Test of a process model. *Psychology and Aging, 6*, 190–201.

Russell, J. J., Moskowitz, D. S., Zuroff, D. C., Bleau, P. P., Pinard, G. G., & Young, S. N. (2011). Anxiety, emotional security and the interpersonal behavior of individuals with social anxiety disorder. *Psychological Medicine, 41*, 545–554. doi:10.1017/S0033291710000863

Sadler, P., Ethier, N., Gunn, G. R., Duong, D., & Woody, E. (2009). Are we on the same wavelength? Interpersonal complementarity as shared cyclical patterns during interactions. *Journal of Personality and Social Psychology, 97*, 1005–1020. doi:10.1037/a0016232

Sadler, P., & Woody, E. (2003). Is who you are who you're talking to? Interpersonal style and complementarity in mixed-sex interactions. *Journal of Personality and Social Psychology, 84*, 80–96. doi:10.1037/0022-3514.84.1.80

Salkovskis, P. M. (1991). The importance of behavior in the maintenance anxiety and panic: A cognitive account. *Behavioural Psychotherapy, 19*, 6–19.

Schafer, M., Korn, S., Smith, P. E., Hunger, S. C., Mora-Merchan, J. A., Singer, M. M., & van der Meulen, K. (2004). Lonely in the crowd: Recollections of bullying. *British Journal of Developmental Psychology, 22*, 379–394.

Schneier, R. F. R., Heckelman, L. R., Garfinkel, R., Campeas, R., Fallon, B. X., Gitow, A., . . . , Liebowitz, M. R. (1994). Functional impairment in social phobia. *Journal of Clinical Psychiatry, 55*, 322–331.

Schoenbach, V. J., Kaplan, B. H., Fredman, L., & Kleinbaum, D. G. (1986). Social ties and mortality in Evans County, Georgia. *American Journal of Epidemiology, 123*, 577–591.

Smith, J. L., & Ruiz, J. M. (2007). Interpersonal orientation in context: Correlates and effect of interpersonal complementarity on subjective and cardiovascular experience. *Journal of Personality, 75*, 670–708. doi:10.1111/j.1467-6494.2007.00453.x

Sparrevohn, R. M., & Rapee, R. M. (2009). Self-disclosure, emotional expression and intimacy within romantic relationships of people with social phobia. *Behaviour Research and Therapy, 47*, 1074–1078. doi:10.1016/j.brat.2009.07.016

Stein, M. B., & Kean, Y. M. (2000). Disability and quality of life in social phobia: Epidemiologic findings. *American Journal of Psychiatry, 157*, 1606–1613.

Stein, M. B., McQuaid, J. R., Laffaye, C., & McCahill, M. E. (1999). Social phobia in the primary care medical setting. *Journal of Family Practice, 48*, 514–519.

Stillman, T. F., Baumeister, R. F., Lambert, A., Crescion, A. W., DeWall, C. N., & Fincham, F. D. (2009). Alone and without purpose: Life loses meaning following social exclusion. *Journal of Experimental Social Psychology, 45*, 686–694. doi:10.1016/j.jesp.2009.03.007

Stinson, D. A., Cameron, J. J., Wood, J. V., Gaucher, D., & Holmes, J. G. (2009). Deconstructing the "reign of error": Interpersonal warmth explains the self-fulfilling prophecy of anticipated acceptance. *Personality and Social Psychology Bulletin, 35*, 1165–1178. doi:10.1177/0146167209338629

Stinson, D. A., Logel, C., Zanna, M. P., Holmes, J. G., Cameron, J. J., Wood, J. V., & Spencer, S. J. (2008). The cost of lower self-esteem: Testing a self- and social-bonds model

of health. *Journal of Personality and Social Psychology, 94*, 412–428. doi:10.1037/0022-3514.94.3.412

Taylor, C. T., & Alden, L. E. (2010). Safety behaviors and judgment biases in generalized social anxiety disorder. *Behavior Research and Therapy, 48*, 226–237. doi:10.1016/j.brat.2009.11.005

Taylor, C. T., & Alden, L. E. (2011). To see ourselves as others see us: An experimental integration of the intra and interpersonal consequences of self-protection in social anxiety disorder. *Journal of Abnormal Psychology, 120*, 129–141. doi:10.1037/a0022127

Taylor, S. E., Welch, W. T., Kim, H. S., & Sherman, D. K. (2007). Cultural differences in the impact of social support on psychology and biological stress responses. *Psychological Science, 18*, 831–837.

Tiedens, L. Z., & Fragale, A. R. (2003). Power moves: Complementarity in dominant and submissive nonverbal behavior. *Journal of Personality and Social Psychology, 84*, 558–568. doi:10.1037/0022-3514.84.3.558

Tiedens, L. Z., Unzueta, M. M., & Young, M. J. (2007). An unconscious desire for hierarchy? The motivated perception of dominance complementarity in task partners. *Journal of Personality and Social Psychology, 93*, 402–414. doi:10.1037/0022-3514.93.3.402

Tracey, T. J. G. (2004). Levels of interpersonal complementarity: A simplex representation. *Personality and Social Psychology Bulletin, 30*, 1211–1225. doi:10.1177/0146167204264075

Tracey, T. J. G. (2005). Interpersonal rigidity and complementarity. *Journal of Research in Personality, 39*, 592–614. doi:10.1016/j.jrp.2004.12.001

Uchino, B. N., Cacioppo, J. T., & Kiecolt-Glaser, J. (1996). The relationship between social support and physiological processes: A review with emphasis on underlying mechanism and implications for health. *Psychological Bulletin, 119*, 488–531.

Vonken, M. J., Alden, L. E., Bögels, S. M., & Roelofs, J. (2008). Social rejection in social anxiety disorder: The role of performance deficits. *British Journal of Clinical Psychology, 47*, 439–450. doi:10.1348/014466508×334745

Wells, A., Clark, D. M., Salkovskis, P. M. S., Ludgate, J., Hackmann, A., & Gelder, M. (1995). Social phobia: The role of in-situation safety behaviors in maintaining anxiety and negative beliefs. *Behavior Therapy, 26*, 153–161.

Wenzel, A., Graff-Dolezal, J., Macho, M., & Brendle, J. R. (2005). Communication and social skills in socially anxious and nonanxious individuals in the context of romantic relationships. *Behaviour Research and Therapy, 43*, 505–519. doi:10.1016/j.brat.2004.03.010

Whisman, M. A. (2007). Marital distress and DSM-IV psychiatric disorders in a population-based national survey. *Journal of Abnormal Psychology, 116*, 638–643. doi:10.1037/0021-843X.116.3.638

Whisman, M. A., Sheldon, C., & Goering, P. (2000). Psychiatric disorders and dissatisfaction with social relationships. Does type of relationship matter? *Journal of Abnormal Psychology, 109*, 803–808.

Wilson, R. S., Krueger, K. R., Arnold, S. E., Schneider, J. A., Kelly, J. F., Barnes, L., . . . , Tang, Y., & Bennett, D. A. (2007). Loneliness and risk of Alzheimer disease. *Archives of General Psychiatry, 64*, 234–240.

Yanagisawa, K., Masui, K., Furutani, K., Nomura, M., Ura, M., & Yoshida, H. (2011). Does higher general trust serve as a psychosocial buffer against social pain? An NIRS study of social exclusion. *Social Neuroscience, 6*, 190–197.

Zadro, L., Williams, K. D., & Richardson, R. (2004). How low can you go? Ostracism by a computer is sufficient to lower self-reported levels of belonging, control, self-esteem, and meaningful existence. *Journal of Experimental Social Psychology, 40*, 560–567. doi:10.1016/j.jesp.2005.10.007

II

Variability Within Social Anxiety Disorder

9

Social Anxiety Disorder in Children and Adolescents

Thomas H. Ollendick[1], Kristy E. Benoit[1], and Amie E. Grills-Taquechel[2]

[1] *Virginia Tech, USA*
[2] *Boston University, USA*

Age-related increases in social evaluative anxiety are part of normal development. As young children emerge into childhood and then into adolescence, they place increasing importance on how peers, friends, and adults perceive them and how they "come across" in their social worlds. Transient episodes of social anxiety are common and part of normal development. However, for a small but significant number of youth, normal developmental processes go awry and social anxiety disorder (SAD) develops (Ollendick & Benoit, 2012).

In this chapter, we explore developmental factors associated with SAD. Our primary objective is to present a coherent theoretical framework in which knowledge of the antecedents, processes, and consequences of change that occur with development can be incorporated into the understanding of SAD and its sequellae. The chapter concludes with a discussion of the implications of these observations for theory, research, and practice.

Social Anxiety Disorder

Social anxiety disorder (SAD) consists of a marked and persistent fear of social or performance situations in which embarrassment or humiliation might occur (American Psychiatric Association [APA], 1994). Frequently, when exposed to possible scrutiny by others, youth with SAD fear they might do something wrong or act in a way that will make them feel ashamed or embarrassed. Exposure to social or performance situations frequently provokes an immediate anxiety response that is excessive or unreasonable. In young children, this anxiety response may take the form of crying, screaming, or clinging to familiar persons or objects. In contrast, older children and adolescents may experience panic-like symptoms when confronted by anxiety-provoking social situations. Other behavioral manifestations of SAD include gaze aversion, stooped shoulders, nail biting, and a trembling voice. Although behavioral avoidance of social

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

or performance situations is common among socially anxious adolescents and adults, socially anxious children rarely have the opportunity to avoid these social situations. As a result, they may evidence disinterest in age-appropriate social, academic, and athletic activities. Youth who attempt to avoid anxiety-provoking situations are frequently perceived by adults as oppositional, negativistic, or noncompliant since they refuse to do as they are asked (Beidel & Turner, 1998; Hofmann & DiBartolo, 2001; Ollendick, Costa, & Benoit, 2010; Ollendick & Hirshfeld-Becker, 2002).

Older children and adolescents with SAD also report anxious thoughts concerning embarrassment, inadequacy, and self-criticism. Alternatively, some youth report that when confronted by anxiety-provoking social situations, they are overcome by maladaptive thoughts and are unable to think clearly, and become inept or awkward in their social interchanges. Although adolescents and adults typically recognize their social fears as irrational, children may not. They report feeling justified in their "distorted" and "dysfunctional" beliefs. Thus, one of the most important contextual factors associated with the expression of SAD is age.

The average age of onset for SAD is mid-adolescence (APA, 1994), which coincides with the normal vulnerability to social embarrassment seen in adolescents. However, a number of researchers report diagnoses in prepubertal children as young as 7 years of age (Albano & Hayward, 2004; Beidel & Turner, 1998; Ollendick & Hirshfeld-Becker, 2002). SAD is the most common anxiety disorder seen in late adolescence and adulthood and ranks third among all psychiatric disorders, following major depression and alcohol dependence. Results of the National Comorbidity Survey indicate that the adult lifetime prevalence rate of SAD is 12.1% (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). Research also indicates SAD lifetime prevalence rates of between 10% and 15% in adolescents in the United States (Heimberg, Stine, Hiripi, & Kessler, 2000; Merikangas et al., 2011) and Europe (Essau, Conradt, & Petermann, 1999; Wittchen, Stone, & Kessler, 1999). Although prevalence rates for children are not well established, most adults with SAD are frequently unable to recall a period in their lives when they were *not* socially anxious. Of course, these retrospective reports must be viewed with appropriate caution. Findings also suggest that SAD, at least in adolescence, follows a chronic, unremitting course in the absence of effective treatment (Albano & Hayward, 2004). Thus, SAD represents a disorder of considerable magnitude and significance in childhood and adolescence. What are the antecedents to this disorder and what processes characterize this development gone awry? Although the answer to this question is complex, developmental psychology and developmental psychopathology provide us with important clues.

Basic Premises of Developmental Psychology and Developmental Psychopathology

Developmental Theory

Within the field of developmental psychology, theorists have long debated which theoretical model best helps researchers and clinicians understand the many changes that occur in individuals throughout their development and across their life spans,

and the processes responsible for them. Early debates focused attention on issues of autonomy and organization and were tied to two major world views in what came to be known as the "mechanistic" and "organismic" models of development. Supporters of the mechanistic model (Baer, 1982; Skinner, 1938) viewed organisms similar to machines; they were acted upon largely by forces from the outside world. Specifically, with regard to development, these theorists viewed organisms primarily as passive recipients of information and relatively passive respondents to increasingly complex and varied stimulus input (i.e., a *tabula rasa*). Furthermore, they believed that changes in behavior over time reflected gradual modifications in antecedent and consequent stimuli, with explanations for development derived largely from principles of learning theory (e.g., conditioning, reinforcement). Skinner (1938), for example, asserted that "the basic premise of behavioral psychology (was) that all organisms, human and subhuman, *young and old* (italics added), were subject to the same law of effect (principle of reinforcement) and could be studied in the same basic manner" (p. 27). From this perspective, many clinicians and researchers viewed development and developmental processes as possessing relatively little clinical or societal significance (see Ollendick & Cerny, 1981, for an extended discussion of this point).

In contrast to the passive qualities of the organism portrayed in the mechanistic point of view, proponents of the organismic model of development asserted that organisms were not passive; rather, they viewed them as agents who were actively involved in the construction of their own environments. Furthermore, organismic theorists often described development as if it passed through discrete and oftentimes invariant stages (e.g., Piaget's stages of cognitive development, Freud's psychosexual stages, Erickson's stages of identity development). These various theories maintained that basic structures and functions changed across age and they reflected emerging, qualitatively different ways of interacting with the environment. In its simplest form, this model proposed that change resulted largely from maturational processes occasioned by intrinsic organismic factors rather than extrinsic environmental ones.

Extensive debate among proponents of these opposing models, as well as increasing recognition of their limitations, led to the advent of a third model of development, namely, the *transactional* model. Also known as *developmental contextualism*, the transactional model moved beyond the mechanistic and organismic models (Lerner, Hess, & Nitz, 1991; Ollendick, Grills, & King, 2001; Ollendick & Vasey, 1999) and was highly consistent with the tenets of social learning/social cognitive theory (cf., Bandura, 1977; Ollendick & Cerny, 1981). Advocates of this model proposed that developmental changes occurred because of continuous reciprocal interactions between an active organism and its active environmental context. Organisms affected their own development by being both products and producers of their environments. Although differences in theory and philosophy remain, most developmental theorists these days agree with the transactional model and believe that development involves systematic, successive, and adaptive changes within and across life periods in the structure, function, and content of the individual's cognitive, emotional, behavioral, social, and interpersonal characteristics (Silverman & Ollendick, 1999). Because developmental changes occur in an orderly and sequential fashion (i.e., they are systematic and successive), changes observed at one point in time will likely influence subsequent events (although not necessarily in a direct, linear fashion; see below). Thus,

the diversity or variety of changes over the life span is constrained by, but not solely determined by, changes that occur at earlier points in time.

Developmental Psychopathology

Developmental psychopathology and developmental psychology are closely intertwined (Rutter & Garmezy, 1983). Sroufe and Rutter (1984) defined developmental psychopathology as "the study of the origins and course of individual patterns of behavioral maladaptation, whatever the age of onset, whatever the causes, whatever the transformations in behavioral manifestations, and however complex the course of developmental pattern may be" (p. 18). Implicit in this definition is concern with development and developmental deviations or distortions (i.e., clinical psychopathologies) that occur throughout and across the life span, and the processes associated with those perturbations. The study of psychopathology, from this perspective, organizes itself around milestones, transitions, and sequences in physical, cognitive, and social–emotional development. This theory views development as a series of qualitative reorganizations within and among systems. The character of these reorganizations is determined by factors at various levels of contextual analysis (e.g., genetic, constitutional, developmental, physiological, behavioral, environmental, and sociological) that are in dynamic transaction with one another (Cicchetti, 1989). Pathological development constitutes a lack of integration among the very systems that contribute synergistically to adaptation at particular developmental levels (i.e., toddlerhood, childhood, adolescence). Development goes awry at such times and under such conditions (Ollendick & Vasey, 1999).

Although development at one level affects later development, direct or isomorphic continuity of behavior is not expected. Rather, developmental outcomes may occur through multiple pathways. Normal *and* pathological development result from individually distinct and unique transactions between a changing organism and its ever-changing environmental context. This notion is captured in the developmental principle of equifinality—the principle that any one outcome (i.e., SAD) may result from multiple and diverse pathways. From a developmental perspective, the expectation that a singular pathway to a given disorder exists would be the exception, not the rule. In contrast to equifinality, the principle of multifinality asserts that varied outcomes can eventuate from the very same common starting points. Thus, any one risk factor for the development of SAD (e.g., *behavioral inhibition to the unfamiliar* [BI], Kagan, 1994) is likely to result in a variety of diverse outcomes, not just SAD. It is therefore important to identify and understand intra- and extra-individual characteristics (i.e., antecedents) that promote or inhibit early deviations and maintain or disrupt early adaptation and development (i.e., outcomes). Thus, the field of developmental psychopathology examines the origins and course of a given disorder, its precursors and sequellae, its variations in manifestation with development, and more broadly, its relations to nondisordered behavior patterns (Ollendick & Vasey, 1999; Toth & Cicchetti, 1999).

As may be evident, the developmental psychopathology perspective does not subscribe to nor prescribe a particular theoretical orientation or explanation for the origins

of diverse child psychopathologies (i.e., medical model, psychodynamic theory, social learning theory, family systems theory), nor does it supplant particular theories; rather, it sharpens our awareness about connections among phenomena that may otherwise seem unrelated or disconnected. In this sense, it is a macroparadigm, which serves to bridge a variety of conceptual models (i.e., microparadigms in themselves).

Antecedent Pathways to Social Anxiety Disorder as an Outcome

Several antecedent factors have been associated with the development of SAD as a developmental outcome, including genetic influences (see **Chapter 3** for a detailed review), temperament dimensions, attachment styles, emotion-regulation strategies, peer interpersonal factors, parental anxiety and parenting practices, conditioning events, and more broad ecological and developmental forces (Ollendick & Benoit, 2012; Ollendick & Hirshfeld-Becker, 2002). An examination of all of these factors is beyond the scope of the present chapter; however, five of these factors—temperamental characteristics, attachment processes, emotion-regulation strategies, parenting practices, and peer interpersonal factors—will be highlighted to illustrate the delicate interplay of child, parent, and peer influences.

Behavioral Inhibition

Research has pointed to the temperamental characteristic of BI as a precursor to SAD (see **Chapter 7** for extended review). Originally described by Kagan and colleagues, BI represents a relatively enduring, biologically based tendency, observable in 10–15% of infants and toddlers, to demonstrate distress, fear, avoidance or quiet restraint and reticence when exposed to unfamiliar situations, persons, and objects (Kagan, Reznick, & Snidman, 1987). Although BI shares features with shyness and social withdrawal, it is broader in scope in that it encompasses inhibition to nonsocial as well as social stimuli that are unfamiliar to the child. BI manifests itself differently at different age levels: inhibited toddlers react to novelty with agitation, distress, and heightened motor reactivity, as well as clinging to the caregiver; preschoolers react with hesitancy, restraint, reticence, inhibited spontaneous conversation, and limited smiling in unfamiliar situations; school children manifest inhibition through extreme reticence and constriction with unfamiliar adults, and through quiet isolation with unfamiliar peers; and adolescents frequently express behavioral inhibition through social withdrawal, social anxiety, and in some instances aggression and violence. Thus, age, a developmental marker, serves as a context for the form of this temperamental characteristic.

Kagan and colleagues (e.g., 1987) followed two independent cohorts of children identified as behaviorally inhibited as toddlers and found that BI was moderately stable through middle childhood and into early adolescence. More recent longitudinal studies, which vary widely in the ways in which behavioral inhibition has been defined, have reported similar stability of BI from toddlerhood through early childhood (Fox,

Henderson, Marshall, Nichols, & Ghera, 2005; Goldsmith & Lemery, 2000), middle childhood (Fox et al., 2005), late childhood (Degnan, Henderson, Fox, & Rubin, 2008; Scarpa, Raine, Venables, & Mednick, 1995), adolescence (Chronis-Tuscano et al., 2009; Schwartz, Snidman, & Kagan, 1999; Williams et al., 2009), and even early adulthood (Caspi & Silva, 1995).

Although BI has been shown to be relatively stable across childhood and adolescence, not all children continue to display heightened BI across development. In a follow-up of the original Kagan et al. (1987) cohort of children, Hirshfeld-Becker et al. (1992) showed that about 50% of the sample continued to display wariness and reticence as they reached childhood. Further, in another community sample, Fox et al. (2005) showed that about half of the children with BI continued to show signs of social wariness and reticence across childhood. The stably inhibited children evinced greater autonomic reactivity, elevated morning cortisol levels, heightened startle responses, and more vigilant attentional styles. They also showed heightened amygdala activation in new or threatening situations. These studies, and others (see Degnan & Fox, 2007, and **Chapter 7** for reviews) demonstrate that although the temperamental characteristic of BI is relatively stable and persistent, it is not invariant; that is, only about half of the children continue to display BI characteristics over time.

Within these stability estimates, BI has been shown to be prospectively associated with the development of social anxiety in the children of both normal and anxiety-disordered parents. Children from the Kagan et al. (1987) cohort demonstrated significantly greater SAD prevalence at age 7–8 years than controls (Biederman et al., 1990). Furthermore, stability of BI throughout early childhood (assessed at 21 months, 4 years, 5.5 years, and 7.5 years) increased risk for anxiety disorders (including SAD) in these children (Hirshfeld-Becker et al., 1992). The risk was most evident among BI children whose parents themselves had anxiety disorders (Rosenbaum et al., 1992). By age of 13 years, SAD was significantly higher in youngsters from both Kagan cohorts who had been inhibited as toddlers compared with those who had been uninhibited (34% vs. 9%).

Similar results have emerged in clinical cohorts of children who evidence BI at an early age. One clinical study, for example, compared rates of anxiety disorders among 216 inhibited and uninhibited children of parents with panic disorder, major depressive disorder, or no panic/depressive disorders (Biederman et al., 2001). This study found that inhibited youngsters (mean age 6 years) had significantly higher rates of SAD than uninhibited children (17% vs. 5%). At follow-up, when these children were approximately 10 years of age (Biederman et al., 2006; Hirshfeld-Becker et al., 2007), inhibited children were again significantly more likely than uninhibited children to manifest SAD (28% vs. 14%). In addition, BI children were significantly more likely to show new onset of SAD (22% vs. 8%) during this follow-up period. These differential patterns were not present for other anxiety disorders or the affective disorders. These findings are in accord with other longitudinal studies that have found specific links between BI and SAD (Hayward, Killen, Kraemer, & Taylor, 1998; Schwartz et al., 1999). Similar findings have been obtained by Fox and his colleagues with community samples of children and adolescents (Chronis-Tuscano et al., 2009; Degnan et al., 2008; Fox et al., 2005). For example, Chronis-Tuscano et al. (2009) used a prospective longitudinal design to determine whether stable BI predicted the presence

of psychiatric disorders in 126 adolescents. BI was assessed at 14 months, 24 months, 4 years, and 7 years of age, with approximately 13% of the overall sample of children displaying stable BI across each of these four time points. Stable BI was associated with a fourfold increase in the odds ratio of having lifetime SAD in adolescence. This association was specific to SAD and not other anxiety diagnoses.

These results suggest that BI is associated specifically with the development of SAD in childhood and adolescence. However, it is clear from these studies that not all children with BI develop SAD (in fact, across studies, only about one-fourth to one-third do so), suggesting that there must be other pathways to SAD, or a combination of risk factors that occasion its onset.

Attachment Processes

Parent–child attachment is considered to be one of the most influential and important relationships in development. The type of attachment children have is characterized by the quality of the parent–child relationship in terms of felt security and trust that children have toward their caregiver (Bowlby, 1973). More specifically, whether a child is securely or insecurely attached to their caregiver is directly related to how safe the child feels when interacting with her or his environment. Securely attached children tend to explore their environment more willingly and confidently, and feel like their caregiver is caring, loving, accessible, communicative, trustworthy, and responsive to their needs (Bowlby, 1973). A young child, for example, who is securely attached is able to separate from her caregiver and play with other children in the park or even try out new things and meet new people (with her caregiver nearby). Securely attached children are more self-confident, trusting, and competent in their attachments to other people in their life and have higher self-regulatory abilities, which can allow even fearful or inhibited children to confront perceived threat because their caregiver is available to assist them if need be (Thompson, 2001). The feelings that securely attached children have about their own abilities to handle stress or interact with their environment are not thought to be solely "in" the child, but rather embedded in the parent–child context. Given this, secure attachment is considered to be an interpersonal factor that protects the child against the development of anxiety and related disorders.

Attachment in childhood marks the beginning of children starting to use other adults and peers as a secure base to explore their environment. This is mainly due to the introduction of different school environments in which there are increasingly longer periods of separation from caregivers. Whereas physical proximity is the central theme in attachment in the early years, Bowlby (1987) proposed that *availability* of the attachment figure becomes more important during later childhood. Although children in this age group are developing more cognitive, emotional, and physical skills to begin taking responsibility for their own protection, they are still not making decisions solely on their own. Thus, knowing that a secure attachment figure is available to help if need be may be critical to the progression of normative interpersonal development during childhood, not only because of the direct effects that a secure attachment has on the parent–child relationship, but also because of the indirect effects it has on children's interactions and experiences with other people throughout life.

Bowlby (1987) suggested that early interpersonal experiences within the parent–child attachment relationship predict diverse forms of later psychopathology in the child. Building on Bowlby's seminal work, Manassis and Bradley (1994) posited that *insecure* attachments provide an environmental context that influences, promotes, and reinforces the development and maintenance of anxiety in children over time. Consistent with this model, child's insecure attachment has been found to be a risk factor for the development of childhood anxiety disorders in general and SAD in particular (Manassis, Bradley, Goldberg, Hood, & Swinson, 1994; Warren, Huston, Egeland, & Sroufe, 1997), and, notably, to be associated with BI in young children (Warren et al., 1997).

Attachment theory posits that insecure attachments convey the message to children that caregivers are unreliable, unavailable, untrustworthy, and largely uncommunicative. Children who receive these types of messages can develop a maladaptive approach to future interpersonal situations or relationships based on the expectation that their needs will not be met by significant others, causing either low interpersonal contact/avoidance behaviors or high interpersonal contact/demanding behaviors. These behaviors elicit negative reactions from others, which serve to strengthen the initial distorted beliefs. This distorted view can be expressed in maladaptive forms of coping and avoidance behaviors, creating a chronic and persistent state of anxiety within children, thus placing them at higher risk for the development of SAD and other anxiety disorders (Manassis & Bradley, 1994).

A considerable amount of research has supported insecure attachment as a risk factor in the development of anxiety. For example, Warren et al. (1997) examined 172 insecurely attached children at 12 months of age and found that they were more likely than securely attached children to have an anxiety disorder at age 17 years, including SAD, especially if they were also behaviorally inhibited (BI). Manassis et al. (1994) examined attachment patterns in clinically anxious mothers and their children and found that infants of anxious mothers not only displayed insecure attachments but also evidenced higher rates of subsequent diverse anxiety disorders. In yet another study, Manassis, Bradley, Goldberg, Hood, and Swinson (1995) found that insecurely attached children experienced higher levels of anxiety than securely attached children. In addition, Muris and Meesters (2002) demonstrated that insecure attachment independently and uniquely predicted child trait anxiety as reported by parents and children. Unfortunately, not all of these studies have specified the type of anxiety disorder that eventuates following an insecure attachment, nor have they specified the relations between insecure attachment and BI. Such interrelations remain to be established firmly, but insecure attachment, especially when combined with BI, appears to be important in the onset of SAD in at least some children and adolescents.

Emotion Regulation

Yet a third factor related to the onset of SAD in children and adolescents is poor emotion-regulation skills. Several definitions of emotion regulation have been put forth (Eisenberg, 2002). Eisenberg and Spinrad (2004), for example, define emotion regulation as "the process of initiating, avoiding, inhibiting, maintaining, or

modulating the occurrence, form, intensity, or duration of internal feeling states, emotion-related physiological, attentional processes, motivational states, and/or the behavioral concomitants of emotion in the services of accomplishing affect-related biological or social adaptation or achieving individual goals" (p. 338). Gross and Thompson (2007) suggest that we view emotion regulation as a set of processes that operate on a continuum from automatic, effortless, and unconscious to controlled, effortful, and conscious processes. These definitions illustrate the complexity of emotion regulation as a construct, as well as the diversity of processes involved in regulation of emotions. Although various definitions and nuances of emotion regulation have been put forth, most researchers agree that it encompasses physiological, cognitive, and behavioral processes that allow individuals to modulate how they experience and express positive and negative emotions.

The main mechanism through which emotion regulation is thought to occur is effortful control, or, "the ability to inhibit a dominant response to perform a subdominant response" (Rothbart & Bates, 1998, p. 137). This includes two processes, the first of which is attentional control, or, the ability to shift and focus attention as needed. Research has shown that children exhibit less distress when they are able to shift focus away from distressing stimuli and focus on nondistressing stimuli (Rothbart, Posner, & Boylan, 1990). Attentional processes can also be used to redirect attention internally by thinking positive thoughts or distracting oneself when faced with a distressing situation, such as when a child focuses on their caregiver when meeting someone for the first time or going to a new place. The second process of effortful control is inhibitory control, or, the ability to suppress inappropriate responses. This includes the ability to inhibit aggressive responses when in anger-provoking situations or inhibit avoidant responses when in anxiety-arousing situations, such as when a child answers the question asked of him by the teacher, even though he is anxious that other children in the class might laugh at him.

In addition to these inhibitory functions, Thompson (1994) emphasizes that emotion regulation also involves the ability to enhance and maintain emotion when needed, such as when children increase their anger in order to confront bullies or become courageous in anxiety-arousing situations. This also occurs with positive emotions, such as when children recall pleasant experiences to feel increased levels of positive arousal. When children successfully master the above processes, they are better able to keep their emotions in check and respond appropriately in interpersonal contexts, demonstrate flexibility in these situations, and resolve conflicts. Such interpersonal skills can then lead to more successful interpersonal relationships.

According to this view, emotions not only determine how a person feels, but also which environments or situations a person will engage in, as well as the conditions under which she or he will do so. For instance, anxious individuals may avoid situations which they view as anxiety-provoking, thereby maintaining their maladaptive beliefs about these situations and reinforcing their anxiety. In other instances, they may deploy selective attention, leading them to focus only on information confirming their anxious feelings about a given situation. Finally, other individuals may challenge their anxiety by directly engaging in the given situation, being attentive to the anxiety-provoking stimuli and, via a process of cognitive appraisal, changing their beliefs about that situation. This may lead to changes in emotional and behavioral responses in future

situations, accompanied by altered cognitive appraisals of the situations themselves. Thus, according to Gross and Thompson's (2007) model, our emotions steer the way in which we think about and engage in various situations and environments (see also Esbjørn, Bender, Reinholdt-Dunne, Munck, & Ollendick, 2012).

Most studies investigating the role of emotion regulation in anxiety disorders have focused on adults. These studies have generally found that emotion regulation is less well developed in anxious individuals (see Amstadter, 2008, for a recent review). Higher levels of anxious arousal and worry are reported to be associated with the use of suppression as an (ineffective) regulatory mechanism (Campbell-Sills, Barlow, Brown, & Hofmann, 2006), limited access to other emotion-regulation strategies, and a general nonacceptance of emotions (Kashdan, Zvolensky, & McLeish, 2008). Severity of anxiety symptoms has also been shown to be positively correlated with emotion-regulation difficulties in these adults (Salters-Pedneault, Roemer, Tull, Rucker, & Mennin, 2006; Tull, 2006).

Although research is more limited (see Hannesdottir & Ollendick, 2007, for review), several studies also illustrate the association between emotion-regulation difficulties and anxiety disorders in children and adolescents. For example, Suveg and Zeman (2004) investigated emotion management skills in children aged 8–12 years who met diagnostic criteria for an anxiety disorder (several with SAD) and compared them with a control group of children with no psychopathology. The results resembled those found in adult studies: children with an anxiety disorder reported significantly lower perceptions of self-efficacy with regard to emotion regulation and higher intensity in experience of worry and anger than nonanxious controls, as well as a less-constructive way of managing these emotions. These findings were supported by another study of children aged 8–13 years with anxiety disorders (several with SAD), that reported anxious children to be five times as likely to indicate use of maladaptive emotion-regulation strategies than adaptive ones, compared with nonanxious youth (Suveg, Sood, Hudson, & Kendall, 2008).

In addition to these studies, two recent longitudinal studies show the potential antecedent impact of emotion-regulation difficulties on the development of anxiety disorders more broadly and SAD more specifically. In one recent study, Hannesdottir, Doxie, Bell, Ollendick, and Wolfe (2010) examined the association between frontal EEG measures at $4\frac{1}{2}$ years of age, and physiological measures of emotion regulation and self- and parent-reported social anxiety symptoms at 9 years of age in 20 normally developing children. A significant association was found between right frontal asymmetry in early childhood and both decreased ability to regulate emotions and increased levels of social anxiety symptoms in middle childhood. In another longitudinal study, emotion-regulation difficulties were also related to subsequent development of anxiety disorders in children (Bosquet & Egeland, 2006; see also Sroufe, 2005). In this seminal study, 155 children were described as having "high risk" families, which were defined as having low economic status, low level of parental education, chaotic living conditions, significant life stress, and a lack of social support. Children were evaluated at nine different intervals from birth to 17.5 years of age, including assessments of emotion regulation and anxiety symptoms in childhood and preadolescence, as well as anxiety disorders in adolescence. To our knowledge, this is also the only study to assess both emotion-regulation abilities and attachment classifications in children

in relation to anxiety. Bosquet and Egeland (2006) reported that insecure attachment predicted a unique proportion of emotion-regulation difficulties experienced in the preschool years, and that these emotion-regulation difficulties, in turn, predicted childhood anxiety symptoms. They found that this pathway was specific for anxiety symptoms and, in particular, social anxiety symptoms, and that the anxiety symptoms were moderately stable during childhood and adolescence. Both of these longitudinal studies illustrate the potential impact of emotion-regulation difficulties and, in the latter study, the complex interplay between insecure attachment, emotion-regulation difficulties, and the onset of anxiety in children and adolescents.

The studies highlighted above indicate that children, adolescents, and adults who exhibit elevated levels of anxiety symptoms or suffer from anxiety disorders have emotion-regulation difficulties. Moreover, the longitudinal studies indicate that emotion-regulation difficulties often precede the development of anxiety disorders. These emotion-regulation difficulties include problems with emotional awareness and strategies for dealing with emotions. Anxious adults appear to have less access to emotion-regulation strategies in general. Whether this finding is true for children is uncertain as this has not yet been investigated empirically, although findings indicate that the strategies employed by anxious children are less effective than those utilized by children in normal control groups.

Parenting Practices

In addition to temperamental characteristics, attachment processes, and emotion-regulation difficulties, a number of investigators have examined the role of parenting practices in the development of SAD and other anxiety disorders. A vast amount of research has documented the role of parenting (e.g., parenting styles, attitudes, beliefs, and behaviors) in the persistence/desistence of BI, as well as its relations to attachment processes and emotion-regulation difficulties, and subsequent development of SAD and related anxiety disorders. As but one example, it has been shown that overly protective, controlling, and critical parenting practices are linked with continued BI and the emergence of SAD-like symptoms. For instance, Rubin, Burgess, and Hastings (2002) found that inhibition at 2 years predicted wariness with unfamiliar peers at 4 years of age, but only when mothers engaged in overly controlling and critical parenting styles. Rubin, Cheah, and Fox (2001) have shown similar findings with preschool children.

On the other hand, researchers have shown that parenting practices characterized by warmth and responsiveness are associated with less inhibited and more socially adaptive behavior in children (Hane, Fox, Henderson, & Marshall, 2008; Park, Belsky, Putnam, & Crnic, 1997). However, it should be noted that this sensitive and warm parenting may actually exacerbate inhibited behaviors in children high on BI in some instances by inadvertently reinforcing avoidant behaviors and beliefs that the world is a scary place and the child cannot cope with or master his/her fears (Degnan et al., 2008; Kagan, Arcus, & Snidman, 1993). Overall, however, parents who use supportive strategies to guide their children to engage socially and to approach and value novel situations are more likely to protect their children from persistence of inhibition over time. Thus,

whereas overly controlling, critical, and/or solicitous forms of parenting may occasion the persistence of BI, more supportive and encouraging patterns of parenting are likely to lead to its desistence over time.

Similarly, a number of investigators have shown that parenting strategies typified by overprotection and overcontrol are related to the presence and persistence of other anxiety disorders in children in addition to SAD (Ollendick & Horsch, 2007; Wood, McLeod, Sigman, Hwang, & Chu, 2003). Furthermore, Siqueland, Kendall, and Steinberg (1996) demonstrated that parents of anxiety disordered children (not just SAD) were rated by observers as granting less autonomy to their children (i.e., more controlling) than parents of nonanxious children. Whaley, Pinto, and Sigman (1999) and Hudson and Rapee (2001) demonstrated similar results. Collectively, these studies show that anxiety levels in children are related to having parents who engage in parenting practices characterized by overprotection and overcontrol.

As suggested by Wood et al. (2003) and McLeod, Wood, and Weisz (2007), we might expect specific parenting styles and strategies to qualify or moderate the effects of BI, attachment processes, or emotion-regulation difficulties on the development and expression of anxiety. More specifically, overly controlling and overly soliciting parenting might lead to SAD or other anxiety disorders, while controlling but warm parenting might protect the child with BI, insecure attachment, or emotion-regulation difficulties. Such a hypothesis for BI was examined by Williams et al. (2009) in a sample of 113 children who were assessed for BI at 14 months, 24 months, and 7 years of age. Internalizing problems (e.g., anxiety, social withdrawal, depression) were assessed at ages 4, 7, and 15 years. Although the findings were complex and varied somewhat over time, it was shown that internalizing problems were most evident in those families with children high in BI in which overly controlling and overly soliciting parenting was used. Unfortunately, this study did not examine attachment processes or emotion-regulation difficulties or report on the presence of SAD specifically, only broad internalizing problems. A study examining the interactive effects of these risk factors for SAD is yet to be undertaken.

Several studies have also demonstrated links between heightened parental anxiety (symptoms or disorders) and parenting strategies characterized by overprotection and overcontrol (see McLeod et al., 2007; and Wood et al. 2003, for reviews). Moreover, as illustrated in the early work of Hirshfeld-Becker et al. (1992) and Rosenbaum et al. (1992), the stability of BI and its relations with anxiety disorders in children are especially pronounced in those whose parents have anxiety disorders themselves. The basic idea here is that characteristics of both the parent and child impact upon the parenting practices employed. Unfortunately, however, these studies and others have not examined the complex interplay of the various factors cited and the development and expression of SAD or other childhood anxiety disorders.

Peer Relationships

In addition to parenting practices and child characteristics such as temperament factors, attachment processes, and emotion-regulation difficulties, other researchers have examined peer factors and their role in the development of SAD (see Costa, Benoit,

& Ollendick, 2010). With development, interactions with peers steadily increase and take on increasingly greater importance. Peer relationships in childhood are characterized by particular behaviors, thoughts, and emotions. For example, Eisenberg and Fabes (1998) report that positive social behaviors such as generosity, helpfulness, and cooperation increase during childhood. Children's understanding of friendships also changes. Children begin to develop a sense of continuity and reciprocity in their choice of friends. Perspective-taking abilities become salient in that children begin to appreciate the thoughts and feelings of others (Selman & Schultz, 1990).

From a developmental perspective, one of the most important tasks of childhood is to learn acceptable ways of interacting with one's peers. Although interpersonal abilities and skills accrued from a secure attachment relationship, and healthy parental socialization of emotions affect this learning, the majority of it occurs within the context of peer groups. As such, interactions with peers are thought to play an important role in children's interpersonal, social, and cognitive development. Certain social skills are necessary in order for children to form successful peer relationships (Rubin, Bukowski, & Parker, 1998). These social skills include, but are not limited to, the abilities to: (1) understand the thoughts and feelings of others, (2) begin, maintain, and end interactions in a positive way, (3) appropriately express emotions and behaviors, and (4) inhibit behaviors that might be construed as negative by others. Peer acceptance is thought to be, in part, a function of these social skills. Concerns about peer acceptance take on a significant role during childhood development. More importantly, peer acceptance is a significant predictor of short- and long-term adjustment. Hence, normative interpersonal development consists of children possessing the social skills that enable them to form peer relationships and subsequently be accepted by their peers.

The formation of peer relationships, or more precisely the inability to form such relationships, is an important interpersonal process associated with the development of anxiety disorders, particularly SAD. Two characteristics apply. The first characteristic is the outlook children have about interpersonal relationships, which is based largely on the parent–child attachment relationship. The second characteristic is the child's temperament and how it elicits or occasions certain behaviors from others. Both of these characteristics can directly and indirectly affect the formation of peer relationships (Ollendick et al., 2010) and subsequent development of SAD (Ollendick & Hirshfeld-Becker, 2002).

The optimal outcome in forming good peer relationships is for children to feel they are accepted and valued by their peers. However, when the opposite occurs and children are rejected by their peers, anxiety may develop. Rubin and colleagues (1998) have described this pathway well. Beginning with behavioral inhibition, the pathway to social wariness, withdrawal, and rejection unfolds. Parents dealing with behaviorally inhibited children may have the tendency to become insensitive and unresponsive due to the high frequency of these behaviors and because their attempts to soothe or comfort their child have failed (Rubin, Both, Zahn-Waxer, Cummings, & Wilkinson, 1991). Subsequently, the interaction of the child's behaviors and the parent's behaviors toward the child results in the solidification of an insecure parent–child attachment. It is thought that this sequence of events hinders a child's ability to form subsequent good peer relationships.

How might not being able to form peer relationships result in the development of an anxiety disorder? Insecurely attached and behaviorally inhibited children are thought to be afraid of rejection; therefore, these children withdraw from their peers to avoid rejection. Moreover, children with poor emotion-regulation difficulties are less likely to handle rejection in a constructive manner. This social withdrawal, in turn, results in children not being able to establish normal social relationships, thereby decreasing their chances of being exposed to normative social behaviors (Rubin et al., 1998). This results in children having increased anxiety when placed in settings with peers, which then results in even higher levels of withdrawal in these settings, perhaps eventuating in SAD. As children progress through childhood, their withdrawal behaviors become increasingly recognized by peer groups (Ollendick & Hirshfeld-Becker, 2002; Younger & Boyko, 1987), which then serves to increase anxiety in the already anxious and withdrawn child. In this manner, the interpersonal process of forming peer relationships, along with the other factors examined above, can have a dramatic effect on the development of SAD in childhood.

Summary Comments and Conclusions

The development of SAD is not straightforward nor is its course easily predicted. SAD may result from multiple antecedent pathways; moreover, any one pathway associated with the development of SAD can lead to outcomes other than SAD. Although genetic and temperamental factors, emotion-regulation difficulties, parental influences, peer relationships, and diverse environmental factors are all associated with SAD, they relate in a complex, transactive manner that is dependent upon contextual factors such as child age and the broader familial and community context in which the child is embedded. Any one of these antecedent factors alone is likely not sufficient, nor necessary, to occasion the onset of this (or any other) disorder. Specificity of the developmental outcome results from the combination, timing, and circumstances surrounding these influences (Ollendick & Benoit, 2012; Ollendick & Hirshfeld-Becker, 2002; Ollendick & Vasey, 1999). Put simply, antecedent risk factors serve to predispose youth to the development of an outcome such as SAD; however, they do not directly occasion it. A complex interplay of factors serves to precipitate its expression and maintenance.

Implications for Research and Practice

Five different types of studies would be helpful in advancing research and practice: (1) Longitudinal prospective studies of at-risk children (e.g., offspring of parents with an anxiety disorder and/or children with BI) which begin in infancy and assess children in terms of attachment processes, emotion-regulation capacities, peer relationships, and parenting practices. Specificity of disorder should be examined in these studies so that we will have more specific information about the onset and course of SAD in particular. (2) Twin studies and adoption studies which assess BI *and* childhood anxiety disorders (including SAD) and model the associations between temperament and disorders, along with parental psychopathology, parenting practices, and peer

relationships over time. Such studies might help to sort out genetic influences from environmental influences in the development and course of these various dimensions and resultant disorders. (3) Neurological studies that tell us more about the biological processes that underpin constructs such as BI and emotion-regulation processes. (4) Studies that take advantage of emerging fMRI technology which links brain scanners and allows multiple participants to interact with each other; such technology could be used to assess the neurological foundations of SAD and related anxiety disorders. (5) Additional prevention and intervention studies of at-risk offspring with behavioral inhibition and anxiety-disordered parents, in which antecedent factors thought to contribute to the onset of SAD are targeted and child and family processes are monitored. Although interventions cannot tell us much about etiological factors, they can inform us about maintaining factors, and importantly, about how to prevent or mitigate the course of SAD. Ultimately, such interventions could be pitted against one another in children with and without different antecedent risk factors in order to determine which factors are helpful to target and under which circumstances. The factors intervened upon might include factors in the child (e.g., emotion-regulation tendencies, cognitive factors, tendency to cope through avoidance, lower threshold to sympathetic arousal), the parents (e.g., cognitive factors, parental modeling, parental facilitation or avoidance of socialization, criticism/shaming), and parent–child (e.g., attachment processes) or peer interactions.

In short, our work is cut out for us. Although we have learned much about SAD and its antecedents, causes, and consequences in recent years, we have only just begun to scratch the surface of what we need to learn. In many respects, the study of SAD is in its own infancy. Still, we are making progress, even if only in baby steps.

References

Albano, A. M., & Hayward, C. (2004). Social anxiety disorder. In T. H. Ollendick & J. S. March (Eds.), *Phobic and anxiety disorders in children and adolescents: A clinician's guide to psychosocial and pharmacological interventions* (pp. 198–235). New York, NY: Oxford University Press.

American Psychiatric Association (1994). *Diagnostic and statistical manual for mental disorders* (4th ed.). Washington, DC: Author.

Amstadter, A. (2008). Emotion regulation and anxiety disorders. *Journal of Anxiety Disorders, 22*, 211–221. doi:10.1016/j.janxdis.2007.02.004

Baer, D. M. (1982). Behavior analysis and development. *Human Development, 25*, 357–361. doi:10.1159/000272818

Bandura, A. (1977). *Social learning theory.* Englewood Cliffs, NJ: Prentice-Hall.

Beidel, D. C., & Turner, S. M. (1998). *Shy children, phobic adults: nature and treatment of social phobia*. Washington, DC: American Psychological Association.

Biederman, J., Faraone, S. V., Hirshfeld-Becker, D. R., Friedman, D., Robin, J., & Rosenbaum, J. F. (2001). Patterns of psychopathology and dysfunction in a large sample of high-risk children of parents with panic disorder and major depression: A controlled study. *American Journal of Psychiatry, 158*, 49–57. doi:10.1176/appi.ajp.158.1.49

Biederman, J., Petty, C., Hirshfeld-Becker, D. R., Henin, A., Faraone, S. V., Dang, D., . . . , Rosenbaum, J. F. (2006). A controlled longitudinal five year follow-up study of children

at high and low risk for panic disorder and major depression. *Psychological Medicine, 36*, 1141–1152. doi:10.1017/S0033291706007781

Biederman, J., Rosenbaum, J. F., Hirshfeld, D. R., Faraone, S.V., Bolduc, E. A., Gersten, M.,. . . , Reznick, J. S. (1990). Psychiatric correlates of behavioral inhibition in young children of parents with and without psychiatric disorders. *Archives of General Psychiatry, 47*, 21–26. doi:10.1001/archpsyc.1990.01810130023004

Bosquet, M., & Egeland, B. (2006). The development and maintenance of anxiety symptoms from infancy through adolescence in a longitudinal sample. *Development and Psychopathology, 18*, 517–550. doi:10.1017/S0954579406060275

Bowlby, J. (1973). *Attachment and loss: separation anxiety and anger* (Vol. *2*). New York, NY: Basic Books.

Bowlby, J. (1987). *Attachment and the therapeutic process.* Madison, CT: International Universities Press.

Campbell-Sills, L., Barlow, D. H., Brown, T. A., & Hofmann, S. G. (2006). Acceptability and suppression of negative emotion in anxiety and mood disorders. *Emotion, 6*, 587–595. doi:10.1037/1528-3542.6.4.587

Caspi, A., & Silva, P. A. (1995). Temperamental qualities at age three predict personality traits in young adulthood: Longitudinal evidence from a birth cohort. *Child Development, 66*, 486–498. doi:10.2307/1131592

Chronis-Tuscano, A., Degnan, K. A., Pine, D. S., Perez-Edgar, K., Henderson, H. A., Diaz, Y.,. . . , Fox, N. A. (2009). Stable early maternal report of behavioral inhibition predicts lifetime social anxiety disorder in adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry, 48*, 928–935. doi:10.1097/CHI.0b013e3181ae09df

Cicchetti, D. (1989). Developmental psychopathology: Past, present, and future. In D. Cicchetti (Ed.), *The emergence of a discipline: The Rochester symposium on developmental psychopathology* (pp. 1–12). Hillsdale, NJ: Lawrence Erlbaum.

Costa, N. M., Benoit, K. E., & Ollendick, T. H. (2010). Interpersonal issues in treating children and adolescents. In L. M. Horowitz & S. Strack (Eds.), *Handbook of interpersonal psychology* (pp. 493–508). New York, NY: John Wiley & Sons. doi:10.1002/9781118001868.ch28

Degnan, K. A., & Fox, N. A. (2007). Behavioral inhibition and anxiety disorders: Multiple levels of a resilience process. *Development and Psychopathology, 19*, 729–746. doi:10.1017/S0954579400004764

Degnan, K. A., Henderson, H. A., Fox, N. A., & Rubin, K. H. (2008). Predicting social wariness in middle childhood: The moderating roles of childcare history, maternal personality, and maternal behavior. *Social Development, 17*, 471–487. doi:10.1111/j.1467-9507.2007.00437.x

Eisenberg, N. (2002). Emotion-related regulation and its relation to quality of social functioning. In W. W. Hartup & R. A. Weinberg (Eds.), *Minnesota symposia on child psychology. Child psychology in retrospect and prospect: In celebration of the 75th anniversary of the Institute of Child Development* (Vol. *32*, pp. 133–171). Mahwah, NJ: Lawrence Erlbaum.

Eisenberg, N., & Fabes, R. A. (1998). Prosocial development. In W. Damon & N. Eisenberg (Eds.), *Handbook of child psychology: Social, emotional, and personality development* (5th ed., Vol. *3*, pp. 701–778). New York, NY: John Wiley & Sons.

Eisenberg, N., & Spinrad, T. L. (2004). Emotion-related regulation: Sharpening the definition. *Child Development, 75*, 334–339. doi:10.1111/j.1467-8624.2004.00674.x

Esbjørn, B. H., Bender, P. K., Reinholdt-Dunne, M. L., Munck, L. A., & Ollendick, T. H. (2012). The development of anxiety disorders: Considering the contributions of attachment and emotion regulation. *Clinical Child and Family Psychology Review, 15*, 129–143. doi:10.1007/s10567-011-0105-4

Essau, C. A., Conradt, J., & Petermann, F. (1999). Frequency and comorbidity of social phobia and social fears in adolescents. *Behaviour Research and Therapy, 37*, 831–843. doi:10.1016/S0005-7967(98)00179-X

Fox, N. A., Henderson, H. A., Marshall, P. J., Nichols, K. E., Ghera, M. M. (2005). Behavioral inhibition: Linking biology and behavior within a developmental framework. *Annual Review of Psychology, 56*, 235–262. doi:10.1146/annurev.psych.55.090902.141532

Goldsmith, H. H., & Lemery, K. S. (2000). Linking temperamental fearfulness and anxiety symptoms: A behavior-genetic perspective. *Biological Psychiatry, 48*, 1199–1209. doi:10.1016/S0006-3223(00)01003-9

Gross, J. J., & Thompson, R. A. (2007). Emotion regulation: Conceptual foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation*. New York, NY: Guilford Press.

Hane, A. A., Fox, N. A., Henderson, H. A., & Marshall, P. J. (2008). Behavioral reactivity and approach-withdrawal bias in infancy. *Developmental Psychology, 44*, 1491–1496. doi:10.1037/a0012855

Hannesdottir, D. K., Doxie, J., Bell, M. A., Ollendick, T. H., & Wolfe, C. D. (2010). A longitudinal study of emotion regulation and anxiety in middle childhood: Associations with frontal EEG asymmetry in early childhood. *Developmental Psychobiology, 52*, 197–204. doi:10.1002/dev.20425

Hannesdottir, D. K., & Ollendick, T. H. (2007). The role of emotion regulation in the treatment of child anxiety disorders. *Clinical Child and Family Psychology Review, 10*, 275–293. doi:10.1007/s10567-007-0024-6

Hayward, C., Killen, J., Kraemer, K., & Taylor, C. (1998). Linking self-reported childhood behavioral inhibition to adolescent social phobia. *Journal of the American Academy of Child and Adolescent Psychiatry, 37*, 1308–1316. doi:10.1097/00004583-199812000-00015

Heimberg, R. G., Stine, M. B., Hiripi, E., & Kessler, R. C. (2000). Trends in the prevalence of social phobia in the United States: A synthetic cohort analysis of changes over four decades. *European Journal of Psychiatry, 15*, 29–37. doi:10.1016/S0924-9338(00)00213-3

Hirshfeld-Becker, D. R., Biederman, J., Henin, A., Faraone, S. V., Davis, S., Harrington, K., & Rosenbaum, J. F. (2007). Behavioral inhibition in preschool children at risk is a specific predictor of middle childhood social anxiety: A five-year follow-up. *Journal of Developmental and Behavioral Pediatrics, 28*, 225–233. doi:10.1097/01.DBP.0000268559.34463.d0

Hirshfeld-Becker, D. R., Rosenbaum, J. F., Biederman, J., Bolduc, E. A., Faraone, S. V., Snidman, N., . . . , Kagan, J. (1992). Stable behavioral inhibition and its association with anxiety disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 31*, 103–111. doi:10.1097/00004583-199201000-00016

Hofmann, S. G., & DiBartolo, P. M. (Eds.). (2001). *From social anxiety to social phobia: Multiple perspectives.* Boston, MA: Allyn & Bacon.

Hudson, J. L., & Rapee, R. M. (2001). Parent-child interactions and anxiety disorders: An observational study. *Behaviour Research and Therapy, 39*, 1411–1427. doi:10.1016/S0005-7967(00)00107-8

Kagan, J. (1994). *Galen's prophecy: Temperament in human nature.* New York, NY: Basic Books.

Kagan, J., Arcus, D., & Snidman, N. (1993). The idea of temperament: Where do we go from here?. In R. Plomin & G. E. McClearn (Eds.), *Nature, nurture, and psychology* (pp. 197–210). Washington, DC: American Psychological Association.

Kagan, J., Reznick, J. S., & Snidman, N. (1987). The physiology and psychology of behavioral inhibition in children. *Child Development, 58*, 1459–1473. doi:10.2307/1130685

Kashdan, T. B., Zvolensky, M. J. & McLeish, A. C. (2008). Anxiety sensitivity and affect regulatory strategies: Individual and interactive risk factors for anxiety-related symptoms. *Journal of Anxiety Disorders, 22*, 429–440. doi:10.1016/j.janxdis.2007.03.011

Kessler, R. C., Chiu, W. T., Demler, O., Merikangas, K. R., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the national comorbidity survey replication. *Archives of General Psychiatry, 62*, 617–627. doi:10.1001/archpsyc.62.6.617

Lerner, R. M., Hess, L. E., & Nitz, K. (1991). A developmental perspective on psychopathology. In M. Hersen & C. G. Last (Eds.), *Handbook of child and adult psychopathology: A longitudinal perspective* (pp. 9–32). Elmsford, NY: Pergamon Press.

Manassis, K., & Bradley, S. (1994). The development of childhood anxiety disorders: Toward an integrated model. *Journal of Applied Developmental Psychology, 15*, 345–366. doi:10.1016/0193-3973(94)90037-X

Manassis, K., Bradley, S., Goldberg, S., Hood, J., & Swinson, R. P. (1994). Attachment in mothers with anxiety disorders and their children. *Journal of the American Academy of Child and Adolescent Psychiatry, 33*, 1106–1113. doi:10.1097/00004583-199410000-00006

Manassis, K., Bradley, S., Goldberg, S., Hood, J., & Swinson, R. P. (1995). Behavioural inhibition, attachment and anxiety in children of mothers with anxiety disorders. *Canadian Journal of Psychiatry, 40*, 87–92.

McLeod, B. D., Wood, J. J., & Weisz, J. R. (2007). Examining the association between parenting and childhood anxiety: A meta-analysis. *Clinical Psychology Review, 25*, 155–172. doi:10.1016/j.cpr.2007.03.001

Merikangas, K. R., He, J.-P., Burstein, M., Swendsen, J., Avenevoli, S., Case, B., . . . , Olfson, M. (2011). Service utilization for lifetime mental disorders in U.S. adolescents: Results of the National Comorbidity Survey – Adolescent Supplement (NCS-A). *Journal of the American Academy of Child and Adolescent Psychiatry, 50*, 32–45. doi:10.1016/j.jaac.2010.10.006

Micco, J. A., Henin, A., Rosenbaum, J., Biederman, J., Simoes, N., Bloomfield, A., & Hirshfeld-Becker, D. R. (2008, March). *Temperamental pathways to anxiety disorder in children at risk: Implications for prevention and treatment*. Symposium presentation at the annual meeting of the Anxiety Disorders Association of America, Savannah, GA.

Muris, P., & Meesters, C. (2002). Attachment, behavioral inhibition, and anxiety disorders symptoms in normal adolescents. *Journal of Psychopathology and Behavioral Assessment, 24*, 97–106. doi:10.1023/A:1015388724539

Ollendick, T. H., & Benoit, K. (2012). A parent-child interactional model of social anxiety disorder in youth. *Clinical Child and Family Psychology Review, 15*, 81–91. doi:10.1007/s10567-011-0108-1

Ollendick, T. H., & Cerny, J. A. (1981). *Clinical behavior therapy with children*. New York, NY: Plenum Press.

Ollendick, T. H., Costa, N. M., & Benoit, K. E. (2010). Interpersonal processes and the anxiety disorders of childhood. In G. Beck (Ed.), *Interpersonal processes in the anxiety disorders: Implications for understanding psychopathology and treatment* (pp. 71–95). Washington, DC: APA Books. doi:10.1037/12084-003

Ollendick, T. H., Grills, A. E., & King, N. J. (2001). Applying developmental theory to the assessment and treatment of childhood disorders: Does it make a difference? *Clinical Psychology and Psychotherapy, 8*, 304–315. doi:10.1002/cpp.311

Ollendick, T. H., & Hirshfeld-Becker, D. R. (2002). The developmental psychopathology of social anxiety disorder. *Biological Psychiatry, 51*, 44–58. doi:10.1016/S0006-3223(01)01305-1

Ollendick, T. H., & Horsch, L. M. (2007). Fears in children and adolescents: Relations with child anxiety sensitivity, maternal overprotection, and maternal phobic anxiety. *Behavior Therapy, 38*, 402–411. doi:10.1016/j.beth.2006.12.001

Ollendick, T. H., & Vasey, M. W. (1999). Developmental theory and the practice of clinical child psychology. *Journal of Clinical Child Psychology, 28*, 457–466. doi:10.1207/S15374424JCCP2804_4

Park, S., Belsky, J., Putnam, S., & Crnic, K. (1997). Infant emotionality, parenting, and 3-year inhibition: Exploring stability and lawful discontinuity in a male sample. *Developmental Psychology, 33*, 218–227. doi:10.1037/0012-1649.33.2.218

Rosenbaum, J. F., Biederman, J., Bolduc, E. A., Hirshfeld, D. R., Faraone, S. V., & Kagan, J. (1992). Comorbidity of parental anxiety disorders as risk for child-onset anxiety in inhibited children. *American Journal of Psychiatry, 149*, 475–478.

Rothbart, M. K., & Bates, J. E. (1998). Temperament. In W. Damon (Series Ed.) & N. Eisenberg (Vol. Ed.), *Handbook of child psychology: Social, emotional, and personality development* (5th ed., Vol. *3*, pp. 105–176). New York, NY: John Wiley & Sons.

Rothbart, M. K., Posner, M. I., & Boylan, A. (1990). Regulatory mechanisms in infant development. In J. Enns (Ed.), *The development of attention: Research and theory*. Dordrecht, The Netherlands: Elsevier North-Holland. doi:10.1016/S0166-4115(08)60450-1

Rubin, K. H., Both, L., Zahn-Waxer, C., Cummings, M., & Wilkinson, M. (1991). The dyadic play behaviors of children of well and depressed mothers. *Development and Psychopathology, 3*, 243–251. doi:10.1017/S0954579400005289

Rubin, K. H., Bukowski, W., & Parker, J. G. (1998). Peer interactions, relationships, and groups. In N. Eisenberg (Ed.), *Handbook of child psychology: Social, emotional, and personality development* (5th ed., Vol. *3*, pp. 619–700). New York, NY: John Wiley & Sons.

Rubin, K. H., Burgess, K. B., & Hastings, P. D. (2002). Stability and social-behavioral consequences of toddlers' inhibited temperament and parenting behaviors. *Child Development, 73*, 483–495. doi:10.1111/1467-8624.00419

Rubin, K. H., Cheah, C. S., & Fox, N. A. (2001). Emotion regulation, parenting, and display of social reticence in preschoolers. *Early Education and Development, 12*, 97–115. doi:10.1207/s15566935eed1201_6

Rutter, M., & Garmezy, N. (1983). Developmental psychopathology. In E. M. Hetherington (Ed.), *Socialization, personality, and social development* (pp. 775–911). New York, NY: John Wiley & Sons.

Salters-Pedneault, K., Roemer, L., Tull, M. T., Rucker, L., & Mennin, D. S. (2006). Evidence of broad deficits in emotion regulation associated with chronic worry and generalized anxiety disorder. *Cognitive Therapy Research, 30*, 469–480. doi:10.1007/s10608-006-9055-4

Scarpa, A., Raine, A., Venables, P., & Mednick, S. (1995). The stability of inhibited/uninhibited temperament from ages 3 to 11 years in Mauritian children. *Journal of Abnormal Child Psychology, 23*, 607–618. doi:10.1007/BF01447665

Schwartz, C., Snidman, N., & Kagan, J. (1999). Adolescent social anxiety as an outcome of inhibited temperament in childhood. *Journal of the American Academy of Child and Adolescent Psychiatry, 38*, 1008–1015. doi:10.1097/00004583-199908000-00017

Selman, R., & Schultz, L. (1990). *Making a friend in youth: Developmental theory and pair therapy*. Chicago, IL: University of Chicago Press.

Silverman, W. K., & Ollendick, T. H. (Eds.) (1999). *Developmental issues in the clinical treatment of children*. Boston, MA: Allyn & Bacon.

Siqueland, L., Kendall, P. C., & Steinberg, L. (1996). Anxiety in children: Perceived family environments and observed family interaction. *Journal of Clinical Child Psychology, 25*, 225–237. doi:10.1207/s15374424jccp2502_12

Skinner, B. F. (1938). *The behavior of organisms*. New York, NY: Appleton.

Sroufe, L. A. (2005). Attachment and development: A prospective, longitudinal study from birth to adulthood. *Attachment & Human Development, 7*, 349–367. doi:10.1080/14616730500365928

Sroufe, L. A., & Rutter, M. (1984). The domain of developmental psychopathology. *Child Development, 55*, 17–29. doi:10.2307/1129832

Suveg, C., Sood, E., Hudson, J. L., & Kendall, P. C. (2008). "I'd rather not talk about it". Emotion parenting in families of children with an anxiety disorder. *Journal of Family Psychology, 22*, 875–884. doi:10.1037/a0012861

Suveg, C., & Zeman, J. (2004). Emotion regulation in children with anxiety disorders. *Journal of Clinical Child and Adolescent Psychology, 33*, 750–759. doi:10.1207/s15374424jccp3304_10

Thompson, R. A. (1994). Emotion regulation: A theme in search of definition. In N. A. Fox (Ed.), *The development of emotion regulation: Biological and behavioral considerations (Monographs of the Society for Research in Child Development)* (Vol. *59*, Serial No. 240, pp. 25–52). New York, NY: John Wiley & Sons. doi: 10.2307/1166137

Thompson, R. A. (2001). Childhood anxiety disorders from the perspective of emotion regulation and attachment. In M. Vasey & M. Dadds (Eds.), *The developmental psychopathology of anxiety* (pp. 160–182). New York, NY: Oxford University Press.

Toth, S. L., & Cicchetti, D. (1999). Developmental psychopathology and child psychotherapy. In S. W. Russ & T. H. Ollendick (Eds.), *Handbook of psychotherapies with children and families* (pp. 15–43). New York, NY: Kluwer Academic/Plenum. doi:10.1007/978-1-4615-4755-6_2

Tull, M. T. (2006). Extending an anxiety sensitivity model of uncued panic attack frequency and symptom severity: The role of emotion dysregulation. *Cognitive Therapy Research, 30*, 177–184. doi:10.1007/s10608-006-9036-7

Warren, S. L., Huston, L., Egeland, B., & Sroufe, L. A. (1997). Child and adolescent anxiety disorders and early attachment. *Journal of the American Academy of Child and Adolescent Psychiatry, 30*, 637–644. doi:10.1097/00004583-199705000-00014

Whaley, S. E., Pinto, A., & Sigman, M. (1999). Characterizing interactions between anxious mothers and their children. *Journal of Consulting and Clinical Psychology, 67*(6), 826–836. doi:10.1037/0022-006X.67.6.826

Williams, L. R., Degnan, K. A., Perez-Edgar, K. E., Henderson, H. A., Rubin, K. H., Pine, D. S., . . . , Fox, N. A. (2009). Impact of behavioral inhibition and parenting style on internalizing and externalizing problems from early childhood through adolescence. *Journal of Abnormal Child Psychology, 37*, 1063–1075. doi:10.1007/s10802-009-9331-3

Wittchen, H.-U., Stone, M. B., & Kessler, R. C. (1999). Social fears and social phobia in a community sample of adolescents and young adults: Prevalence, risk factors and comorbidity. *Psychological Medicine, 29*, 309–323. doi:10.1017/S0033291798008174

Wood, J., McLeod, B. D., Sigman, M., Hwang, W. C., & Chu, B. C. (2003). Parenting and childhood anxiety: Theory, empirical findings, and future directions. *Journal of Child Psychology and Psychiatry, 44*, 134–151. doi:10.1111/1469-7610.00106

Younger, A., & Boyko, K. (1987). Aggression and withdrawal as social schemas underlying children's peer perceptions. *Child Development, 58*, 1094–1100. doi:10.2307/1130549

10

Comorbidity

Social Anxiety Disorder and Psychiatric Comorbidity are not Shy to Co-Occur

Derek D. Szafranski, Alexander M. Talkovsky, Samantha G. Farris, and Peter J. Norton

University of Houston, USA

Social anxiety disorder (SAD; i.e., social phobia) is one of the most prevalent psychological disorders in the United States, with an initial onset frequently occurring in late childhood/adolescence (Schneier, Johnson, Hornig, Liebowtz, & Weissman, 1992). Liebowitz, Heimberg, Fresco, Travers, and Stein (2000) reported that SAD was the fourth most common psychological disorder behind major depressive disorder, alcohol abuse, and specific phobia. SAD has a 6.8% 12-month and 12.1% lifetime prevalence rate in adults, with the highest prevalence rates between the ages of 30 and 44 years (14.3%) (Kessler, Berglund, et al., 2005; Kessler, Chiu, Demler, & Walters, 2005). SAD prevalence rates are lower among children and adolescents, with 5.5% meeting criteria for SAD between the ages of 13 and 18 years (Merikangas et al., 2010). Comorbidity within SAD is highly prevalent as well. As such, a considerable amount of research has been conducted examining the effects of comorbid diagnoses within the SAD population.

Comorbid psychological disorders impair a wide variety of areas for an individual, including symptom severity, impairment level, and overall quality of life (Klein Hofmeijer-Sevink et al., 2012; Wittchen, Fuetsch, Sonntag, Müller, & Liebowitz, 2000). Comorbidity also impacts a clinician's ability to make accurate diagnoses and discover etiological causes, makes case conceptualizations more difficult, and complicates treatment plans (Marrie et al., 2009).

In a community-based survey, Acarturk, de Graaf, van Straten, ten Have, and Cuijpers (2008) reported that 66.2% of responders with SAD also met criteria for an additional psychological disorder. Recent research suggests that quality of life is substantially and negatively impacted by the addition of a comorbid diagnosis with a primary diagnosis of SAD. Furthermore, individuals with a primary diagnosis of

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

SAD and a comorbid diagnosis displayed greater decreases in quality-of-life ratings when compared with individuals with only an SAD diagnosis and a subclinical group of individuals with social fears (Wittchen et al., 2000). It is possible that comorbid diagnoses lead to additional escape and avoidant behaviors, which further impact an individual's life, thus accounting for further decreases in overall life satisfaction.

Research further suggests that individuals with a primary diagnosis of SAD and a comorbid Axis I diagnosis display more severe symptoms of SAD than individuals without a comorbid Axis I diagnosis (Erwin, Heimberg, Juster, & Mindlin, 2002). Moreover, individuals with a comorbid disorder also report more chronic symptoms of SAD and life impairment than individuals without a comorbid diagnosis (Schneier, Spitzer, Gibbon, & Fyer, 1991).

Comorbidity within SAD is associated with an increased number of social fears. In one study, 38% of individuals who reported just one feared social situation also reported a comorbid anxiety disorder diagnosis. The percentage of individuals with a comorbid anxiety disorder diagnosis substantially increased as the number of feared situations increased. For example, 92% of individuals who reported five or six feared situations also met criteria for a comorbid anxiety disorder (Acarturk et al., 2008). Similar elevations were reported with mood disorders. Twenty-seven percent of individuals who endorsed one feared social situation met criteria for a comorbid mood disorder, whereas 57% of individuals who endorsed five or six feared situations met criteria for a comorbid mood disorder. These findings suggest that the generalized subtype of SAD may be associated with higher rates of comorbidity with other anxiety disorders and mood disorders than is the nongeneralized subtype. Although Väänänen et al. (2011) have reported that SAD frequently precedes the onset of comorbid major depressive disorder, temporal or causal relationships between social fears and other comorbid diagnoses remain unclear and additional research is needed to elucidate these relationships.

Comorbid Anxiety Disorders[1]

Generalized Anxiety Disorder

SAD has a high comorbidity rate with other anxiety disorders (Chartier, Walker, & Stein, 2003). The most common comorbid anxiety disorder for individuals with a principal diagnosis of SAD is generalized anxiety disorder (GAD) (Barlow, 1986; de Ruiter, Rijken, Garssen, & Van Schaik, 1989; Sanderson, DiNardo, Rapee, & Barlow, 1990). Research suggests that between one-third (Turner, Beidel, Borden, Stanley, & Jacob, 1991) and one-quarter (Mennin, Heimberg, & Jack, 2000) of individuals with a principal diagnosis of SAD also meet criteria for GAD. Commonly, GAD is defined by high levels of cognitive and autonomic anxiety symptoms elicited by a number of environmental cues (e.g., health, finances, minor matters). However, some studies report an absence of autonomic arousal within individuals with GAD (Brown, Chorpita, & Barlow, 1998). Individuals with GAD often display high levels of impairment due to the variety of areas impacted by their anxiety

symptoms (Kessler, DuPont, Berglund, & Wittchen, 1999). For individuals with a principal diagnosis of SAD, the addition of a GAD diagnosis may further impact these areas.

SAD and comorbid GAD can be difficult to distinguish from one another. Some researchers suggest using specific physiological characteristics to differentiate between SAD and GAD. For instance, individuals with SAD display higher levels of sweating, heart palpitations, and faintness. Individuals with GAD display higher amounts of dizziness, headaches, shortness of breath, insomnia, and fear of dying (Reich, Noyes, & Yates, 1988; Versiani, Mundim, Nardi, & Liebowitz, 1988). Moreover, research suggests that SAD is more highly related to physiological arousal, whereas GAD is more cognitively based (Gross, Oei, & Evans, 1989).

Research suggests that individuals with a principal diagnosis of SAD and a comorbid diagnosis of GAD also display a larger amount of overall life impairment. In a clinical trial of individuals with a principal diagnosis of SAD with and without a comorbid diagnosis of GAD, Mennin et al. (2000) reported that individuals with comorbid GAD displayed significantly higher rates of social anxiety, avoidance, general anxiety, cognitive symptoms of anxiety, depressed mood, functional impairment, and overall psychopathology. Somatic symptoms were found to be equivalent between groups. SAD treatment is also complicated by the addition of a comorbid GAD diagnosis. Comorbid GAD decreases the likelihood of recovery from SAD when compared to individuals without a comorbid GAD diagnosis (Bruce et al., 2005).

Obsessive-Compulsive Disorder

While a substantial amount of individuals have comorbid diagnoses of SAD and GAD, a smaller subset of individuals has a principal diagnosis of SAD and comorbid obsessive-compulsive disorder (OCD). Acarturk et al. (2008) reported that 4.3% of individuals diagnosed with SAD also met criteria for OCD. These rates are higher than the national average of OCD alone, which is estimated to occur within 1–3% of the population (Karno, Golding, Sorenson, & Burnam, 1988; Ruscio, Stein, Chiu, & Kessler, 2010). OCD is a fundamentally impairing disorder characterized by intrusive nonsensical obsessive cognitions/images and compulsive ritualistic behaviors aimed at reducing anxiety symptoms. A great deal of research has highlighted the negative impact of OCD on individuals' lives, such as impairment in quality of life, job-related activities, socioeconomic status, and personal relationships (Hollander, Stein, Fineberg, Marteau, & Legault, 2010).

Similar to individuals with SAD, individuals with OCD display negative anticipatory reactions to social situations (Gilboa-Schechtman, Franklin, & Foa, 2000). Given the negative stigma associated with ritualistic hand washing and door locking and contamination fears, individuals exhibiting these behaviors often experience negative feedback from people within their environment (e.g., coworkers, family, friends) (Fennell & Liberato, 2007). A substantial portion of evidence-based treatment for

SAD is based on addressing common misinterpretations of social interactions. Therapists often attempt to challenge negative automatic thoughts related to these interactions using evidence-based thought patterns, and examine alternative explanations for events. OCD behaviors are not only problematic for individuals with the diagnosis but for those around them as well. Oftentimes, individuals with OCD require others in their lives to comply with ritualistic behaviors and rules, thus imposing upon the others' lives. Frequently, this leads to conflict and aversive social interactions (Stengler-Wenzke, Beck, Holzinger, & Angermeyer, 2004). For therapists, a diagnosis of SAD with comorbid OCD can complicate treatment. This complication is specifically noteworthy in cognitive behavioral therapy (CBT), because challenging negative automatic thoughts related to social situations may not be useful due to actual aversive social interactions taking place as a result of OCD behaviors.

Panic Disorder With and Without Agoraphobia

Research suggests that between 5.8% and 12.8% of individuals diagnosed with SAD also meet criteria for comorbid panic disorder (Chartier et al., 2003; Erwin et al., 2002). In a comparison study of individuals with and without a primary diagnosis of SAD, Chartier et al. (2003) reported that individuals diagnosed with SAD were over five times more likely to meet criteria for panic disorder than those without an SAD diagnosis. Similar trends were reported when studying agoraphobia. For individuals without an SAD diagnosis, 1.9% met criteria for agoraphobia, compared with 13.2% of individuals with SAD.

Diagnostic complications often occur when individuals display SAD symptoms and report panic attacks. Within SAD patients, anxiety symptoms will frequently increase to a level of panic while in social situations (Ball, Otto, Pollack, Uccello, & Rosenbaum, 1995). Oftentimes, these individuals remove themselves from social situations in order to reduce the panic symptoms (Clum, Clum, & Surls, 1993). However, according to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR; American Psychiatric Association [APA], 2000), the presence of panic attacks is not sufficient to diagnose panic disorder. One of the hallmark criteria of panic disorder is the presence of panic attacks "out of the blue" (Norton, Dorward, & Cox, 1986). If panic attacks are elicited strictly by social situations and do not occur outside of these situations, individuals do not meet criteria for panic disorder. The differentiation between SAD with co-occurring panic attacks and SAD with comorbid panic disorder seems easily differentiated in theory. However, in practice, it can be difficult. This may be especially true for individuals with poor insight or recollection as to triggers of panic symptoms. Furthermore, agoraphobia is defined as anxiety or worry related to fears in which it would be difficult/embarrassing to escape from while having a panic attack (Craske & Barlow, 1993). Individuals with agoraphobia frequently avoid specific situations (including social situations) in which they have a fear of high anxiety (e.g., crowds, theatres). However, these same types of avoidance behaviors are observed in individuals with SAD. Criterion C of agoraphobia in the DSM-IV-TR states that the avoidance behavior cannot be better accounted for by another mental disorder such as SAD (APA, 2000).

Posttraumatic Stress Disorder

Fewer studies have been conducted concerning comorbidity between SAD and posttraumatic stress disorder (PTSD). However, Zayfert, DeViva, and Hofmann (2005) reported that 7% of participants with a principal diagnosis of SAD had a comorbid diagnosis of PTSD. However, 43% of their participants with a principal diagnosis of PTSD had a comorbid diagnosis of SAD. Furthermore, more impairment seems to occur within individuals with principal PTSD with comorbid SAD diagnoses in comparison with those without comorbid SAD. Zayfert et al. reported that the principal PTSD with comorbid SAD participants had a higher likelihood of meeting criteria for depression and other anxiety disorders. Moreover, this subset of participants endorsed greater symptom severity and more functional impairment than patients with either PTSD or SAD alone. Similar results were reported within a combat Veteran population (Orsillo, Heimberg, Juster, & Garrett, 1996).

Comorbid Mood Disorders

Major Depressive Disorder

The comorbidity of SAD and major depression is one of the most common patterns of comorbidity. Research suggests that individuals with a diagnosis of major depressive disorder are 2.9 to 6.0 times more likely than individuals without this diagnosis to meet criteria for an additional diagnosis of SAD (Mineka, Watson, & Clark, 1998). In the DSM-IV, the *generalized subtype* specifier of SAD is used when an SAD patient's fears are related to "most" situations rather than the minimum of one situation in criterion A (APA, 2000; p. 456). Generalized SAD is more often comorbid than the non-generalized subtype, and the generalized subtype is associated with higher levels of depression. This may be due to the fact that individuals with generalized SAD have lives that are more restricted due to the social situations that they fear and avoid (Mineka et al., 1998). Hughes and colleagues (2006) found that SAD is associated with low positive affect, especially in the case of the generalized subtype of SAD. SAD that is specific to performance situations was found to be more strongly associated with the anxious arousal dimension of Clark and Watson's (1991) tripartite model of anxiety and depression than the generalized subtype. Findings from their study suggested that individuals with generalized SAD are more likely to experience low positive affect, so the investigators performed *post hoc* analyses and found that those with generalized SAD scored higher on measures of depression than did individuals with non-generalized SAD, but did not score significantly higher on measures of anxiety. Fava and colleagues (2000) reported that among individuals with major depressive disorder, 27.0% had a lifetime diagnosis of SAD, in contrast to the 13.3% lifetime prevalence rate among the general population at the time (Erwin et al., 2002), and 26.2% of the individuals met criteria for SAD at the time of assessment. Their findings suggest that comorbid SAD and major depression are associated with an earlier age of onset but not greater chronicity than major depression alone.

Both SAD and major depression are associated with differing rates across genders when analyzed alone, but this is not necessarily the case when looking at their comorbidity. Age is an important consideration in SAD with major depressive disorder. In adolescents with SAD, the risk for subsequent depression may be present among boys only. Among girls, SAD at age 15 years does not significantly predict depression at age 17 years, but depression at age 15 years increases risk of subsequent SAD at age 17 years (Väänänen et al., 2011). Ohayon and Schatzberg (2010) compared the rates of these individual disorders and their co-occurrence across genders and age groups. They found that the rate of SAD with major depression decreases with age. In both genders, rates decreased over time. The authors attributed this to the fact that comorbid individuals were more likely than those with a single diagnosis to seek treatment and were also more likely to either use an antidepressant currently or in their lifetimes. They also found that the prevalence of SAD with major depression is comparable between men and women, although each individual disorder is more common among females. Ohayon and Schatzberg (2010) continued to examine age effects beyond the interaction with gender. When examined without taking into account the presence of SAD, the prevalence of depression decreases with age. This age-related decrease was present in the comorbid group as well. SAD diagnosis typically precedes that of major depression, and this is quite consistent across ages (Väänänen et al., 2011). Earlier onset of either individual disorder was also associated with a worse prognosis for those with single or comorbid diagnoses. Childhood-onset major depression is associated with a greater lifetime prevalence of SAD (48.9%) than adult-onset major depression (25.7%) with adolescent-onset individuals falling in between (30.7%). SAD is more likely to precede major depression across ages, with this temporal relationship occurring in more than 50% of children and more than 95% of adults with both diagnoses (Alpert et al., 1999). More specifically, Chavira, Stein, Bailey, and Stein (2004) found that generalized SAD had an earlier age of onset than the nongeneralized subtype, and only the generalized subtype was associated with an increased risk for major depression; they reported that about 28% of children with SAD had a lifetime history of major depression.

Comorbid individuals have a different clinical presentation than individuals with either disorder alone. Individuals with SAD and depression reported more depressive symptoms than individuals with major depression alone. Those individuals with comorbid SAD and depression are more likely than individuals with depression alone to report moderate psychomotor agitation and hypersomnia at least three nights per week. Individuals with SAD and major depression are also more likely to present with features resembling *atypical depression* (Ohayon & Schatzberg, 2010). Alpert and colleagues (1999) found that individuals meeting full criteria for atypical depression were significantly more likely than other subjects to meet criteria for SAD than individuals with major depressive disorder (37.4% vs. 21.9%), and two-thirds of individuals meeting criteria for SAD met criteria for atypical depression, compared with one-half of the individuals without SAD meeting this criterion. In a study comparing socially anxious patients with comorbid anxiety or comorbid depression, across depressive disorders, patients with comorbid SAD and depression were more severe than those with comorbid SAD and another anxiety disorder on measures of social anxiety (and depression), and had lower global

assessments of functioning, earlier onset, and a more chronic and impairing course (Erwin et al., 2002).

Cognitive behavioral group therapy (CBGT) for SAD improved depression in individuals with: SAD only, SAD and a comorbid anxiety disorder, and SAD with comorbid depression; but, greater improvement was exhibited by the SAD and depression group. Comorbidity did not affect qualitative or quantitative improvement of SAD symptoms; all groups improved at similar rates and maintained gains at 12-month follow-up (Erwin et al., 2002). Further, Moscovitch, Hofmann, Suvak, and In-Albon (2005) found that CBGT improves both SAD and major depression. Improvements in social anxiety fully mediated (and accounted for 91% of the variance) the decreases in depression, but decreases in depression only partially mediated (and accounted for 6% of the variance) the decreases in social anxiety.

Dysthymic Disorder

SAD shows a fairly high rate of comorbidity with dysthymic disorder. Compared with individuals without dysthymic disorder, individuals that meet criteria are more likely to also meet criteria for SAD (Markowitz, Moran, Kocsis, & Francis, 1992). In a study of adolescents with dysthymia, Masi and colleagues (2003) found a total comorbidity rate with SAD of 13%, but this was 5.5% in children aged 7–11 years and 17.1% in adolescents aged 12–18 years. Wittchen and Fehm (2003) found that individuals with an SAD diagnosis were 5.03 times more likely than individuals without this diagnosis to receive a diagnosis of dysthymic disorder in their lifetime, and this odds ratio had a positive relationship with the severity of SAD. Substantiating these findings, Kessler, Stang, Wittchen, Stein, and Walters (1999) found that over one-quarter of individuals with dysthymic disorder, a greater percentage than individuals with a diagnosis of major depression, had also been diagnosed with SAD in their lifetimes, and that this effect was not significantly different across genders. Wells, Tien, Garrison, and Eaton (1994) also found that the presence of dysthymia significantly and positively predicted the onset of SAD.

The study of comorbidity between SAD and dysthymia is complicated by the different average ages of onset for these disorders. The average age of onset of SAD is in adolescence, but it is in early adulthood for dysthymic disorder and, accordingly, the rates of comorbidity increase from childhood to adolescence and even further into adulthood (Masi et al., 2003). However, the clinical presentation of those with early-onset (before age of 21 years) dysthymia differs from those with onset after age of 21 years. Barzega, Maina, Venturello, and Bogetto (2001) found that those with early onset are significantly more likely to have comorbid SAD compared with late-onset patients.

With the exception of these results, the data on changes in prevalence rates with age are mostly consistent. SAD appears to be a risk factor leading to more mood disorders, and longitudinally, the generalized subtype of SAD predisposes individuals more so than does the nongeneralized subtype (Wittchen & Fehm, 2003). Kessler, Stang, et al. (1999) found that more than three quarters (76.0%) of individuals with this particular comorbidity reported that SAD temporally preceded dysthymic disorder rather than vice versa (17.8%) or a simultaneous diagnosis (6.1%). A current diagnosis of SAD

predicted both major depression and dysthymia, but not bipolar disorder. Remitted SAD had no predictive effect (and neither did the duration of clinically significant SAD) on the onset of dysthymia; and it had only modest effects on major depression and bipolar disorder, much less robust than that of current SAD. The authors argued that successful treatment of SAD may make changes that affect the mood disorder as well. Additionally, there were no effects due to age of onset or cohort.

Bipolar Disorder

Bipolar disorder is highly comorbid with SAD. Kessler, Stang, et al. (1999) found that nearly half of those with bipolar disorder will receive a diagnosis of SAD in their lifetimes, and that a diagnosis of bipolar disorder confers a risk increase nearly double that of major depression. In contrast, Simon and colleagues (2003) found no difference in SAD rates between individuals with bipolar disorder versus those with major depression, but they found that those with bipolar disorder scored significantly higher on measures of anxiety sensitivity, a vulnerability to anxiety disorders. Krishnan (2005) reported that 47% of individuals with bipolar disorder had comorbid SAD, more than any other individual anxiety disorder. Although Kessler, Stang, et al. (1999) found no gender differences, Krishnan (2005) found that comorbid anxiety disorders, not exclusively SAD, were more common in women with bipolar disorder than men with the same diagnosis. Dilsaver and Chen (2003) found that, among inpatients with manic symptoms, only those that also had depressive symptoms experience intra-episode SAD.

Dilsaver and Chen (2003) found that SAD symptoms in bipolar patients were strongly linked to suicidality, indicating a particular need for specialized treatment of SAD in this population. Kessler, Stang, et al. (1999) found that 47.0% of individuals with this comorbidity reported that SAD had temporal precedence over bipolar disorder, 40.7% reported the opposite, and 12.3% reported a simultaneous onset. They reported that this was the only mood disorder for which less than half of individuals reported that an SAD diagnosis preceded the mood disorder diagnosis. Pharmacotherapy is the first-line treatment method for individuals with bipolar disorder, but special consideration is necessary for those with comorbid SAD. When treating these patients, use of an antidepressant before adequately stabilizing the individual's mood may exacerbate the bipolar disorder symptoms and subsequently worsen the social anxiety symptoms; therefore, it is important to begin pharmacotherapy with a mood stabilizer (Freeman, Freeman, & McElroy, 2002). Cognitive behavioral therapy may also be a good alternative to antidepressants in treating SAD in individuals with bipolar disorder (Freeman et al., 2002).

Comorbid Personality Disorders

Avoidant Personality Disorder

SAD and avoidant personality disorder (APD) show so much overlap that many believe that these disorders belong on the same spectrum, as opposed to the current

structure of the DSM-IV (APA, 2000), in which they are placed on different axes (see also **Chapter 12** for expanded discussion of this issue). Among individuals with a diagnosis of SAD, generalized subtype, 25–73% also met criteria for APD (Chambless, Fydrich, & Rodebaugh, 2008).

The SAD subtypes differ in the rates of comorbidity with APD. Tillfors, Furmark, Ekselius, and Fredrikson (2004) found that the comorbidity of APD and SAD was highest among those with the generalized subtype (68.8%) and lowest for those with the nongeneralized subtype (2.1%), with individuals who feared more than one social situation but who did not meet criteria for the generalized subtype falling in between (44.1%). Brown, Heimberg, and Juster (1995) found that the rate of comorbid SAD and APD was 44% for the generalized subtype, and 14% for the nongeneralized subtype. When comparing features across the four diagnostic groups involving those with versus without APD, and those with generalized versus non-generalized SAD, most of the differences occurred between the SAD subtypes, and not across those with or without a diagnosis of APD. Cox, Pagura, Stein, and Sareen (2009) analyzed the National Epidemiologic Survey of Alcohol and Related Conditions to determine the frequency of comorbid SAD and APD. They found that each disorder occurred at a similar rate nationally, 2.6% and 2.4%, respectively, but that the rates of each disorder were much higher in individuals with the other. A sizable proportion (39.5%) of those with APD also met criteria for SAD of the generalized subtype, and 36.4% of those with generalized SAD met criteria for APD. Schneier et al. (1991) found that 70% of socially phobic individuals in their sample met criteria for APD, but 89% of those with the generalized subtype met these criteria in contrast to 21% of those with nongeneralized SAD.

The phenomenological features of these disorders appear to occur on a continuum despite the fact that they are on separate axes. Brown et al. (1995) found that SAD patients with APD were more likely to earn lower income, be unmarried, and have an additional affective disorder diagnosis than those without comorbid APD. Those with comorbid APD were less likely to participate in social activities involving significant interpersonal contact unless certain of being liked, and were more likely to fear being embarrassed in these situations due to visible symptoms. Among individuals with generalized SAD, Tillfors et al. (2004) found that individuals with comorbid APD had greater anxiety and depression, more impairment, and were more likely to meet criteria for an additional diagnosis. Interestingly, SAD patients with and without APD could not be differentiated with respect to ratings of social distress and the number of feared situations. These data may suggest a dimensional relationship between these disorders. Tran and Chambless (1995) found that generalized SAD patients with and without APD differed significantly on functional impairment, but not fear of negative evaluation, social distress, or social avoidance; they argued that their results suggested that both SAD and APD diagnoses provide useful information, and that completely collapsing them may not be prudent. However, Herbert, Hope, and Bellack (1992) found that the presence of comorbid APD is associated with quantitative rather than qualitative differences in symptom severity.

Generalized SAD and APD have large diagnostic overlap (Van Velzen, Emmelkamp, & Scholing, 2000). Six of the seven diagnostic criteria for APD include components dealing with social interaction. Also, one of the nonsocial-specific criteria

for APD was removed in the transition from the DSM-III-R (APA, 1987) to DSM-IV, making the overlap between the two diagnoses even greater (Hofmann, Heinrichs, & Moscovitch, 2004). Huppert, Strunk, Ledley, Davidson, and Foa (2008) performed a confirmatory factor analysis on diagnostic assessments of APD and generalized SAD indicators to determine if a one- or two-factor model provided a better fit. The single-factor model had a slightly worse fit. However, Ralevski et al. (2005) found that individuals with APD but not SAD endorsed only one of seven individual APD criteria (*reluctance to take risks*) with lower frequency than those with both diagnoses.

Many who argue that APD and SAD belong on the same spectrum cite treatment outcome research in support of their arguments. The co-occurrence of SAD and APD may not result in differences in treatment outcomes. Van Velzen, Emmelkamp, and Scholing (1997) found that individuals with SAD but not APD reported less avoidance behavior, socially anxious cognitions, and depression than those with both diagnoses, but these pretreatment differences were no longer significant following at least 10 sessions of behavioral treatment. Both the comorbid and single-diagnosis groups responded to treatment and reached similar end-states. Among individuals with SAD, Brown et al. (1995) found that the presence of APD had no effect on outcomes among those with generalized SAD.

These effects remain consistent across treatment modalities as well. Huppert and colleagues (2008) found that, among individuals with generalized SAD, those with comorbid APD improved at a greater rate in the first 4 weeks of treatment across CBGT and CBGT plus fluoxetine than those not meeting criteria for APD, but that the presence of APD in individuals with generalized SAD did not predict treatment response above and beyond increased SAD symptom severity. Kose and colleagues (2009) found that CBT and pharmacotherapy were equally effective regardless of comorbidity.

Taken together, the evidence suggests that there are not major qualitative differences between individuals with generalized SAD alone and those who also meet criteria for APD. Although generalized SAD and APD are at least partially overlapping based on their criteria, Kose and colleagues (2009) suggest that they may still be considered to be two distinct disorders, with generalized SAD touching upon phobic features and APD touching upon interpersonal functioning.

Conduct Disorder and Antisocial Personality Disorder

Although it may seem counterintuitive for a disorder characterized by inhibition to be comorbid with a class of disorders characterized by disinhibition, SAD co-occurs with disruptive behavior disorders at a frequency that exceeds the likelihood based on the prevalence of the individual disorders. Community studies indicate that 34–53% of individuals with antisocial personality disorder (ASPD) receive a lifetime diagnosis of an anxiety disorder (Coid & Ullrich, 2010). The prevalence of anxiety disorders among children with conduct disorder (CD) has been estimated to be about 3.1 times greater than among children without CD (Hodgins, De Brito, Chhabra, & Cote, 2010), and the comorbidity of anxiety disorders and conduct problems in youths is three times more likely than what would be expected by chance alone (Angold, Costello, &

Erkanli, 1999). Beidel, Turner, and Morris (1999) found that 10% of socially phobic children aged 7 through 13 years in their sample met criteria for an externalizing disorder. Goodwin and Hamilton (2003) found that those with an ASPD diagnosis were 1.8 times more likely to receive a lifetime diagnosis of SAD than an individual that has never received this diagnosis, even upon controlling for demographics. After controlling for comorbid affective and substance use disorders as well, they found that only SAD and PTSD were associated with ASPD; this trend also applied to those who were diagnosed with CD but never received an ASPD diagnosis. Interestingly, Marmorstein (2006) found that, although both generalized and nongeneralized SAD patients were at greater risk for an externalizing disorder than individuals without SAD, only the nongeneralized subtype increased risk for comorbid CD between the ages of 9 and 17 years. Combined antisocial behavior and anxiety is associated with poorer quality of life and increased suicidal ideation (Brandes & Bienvenu, 2006), as well as increased likelihood of co-occurring depressive and substance use disorders (Goodwin & Hamilton, 2003), when compared with either antisocial behavior or anxiety alone.

Anxiety disorders alone are more common in females, but ASPD either with or without a comorbid anxiety disorder is more common among males (Coid & Ullrich, 2010). Moreover, Coid and Ullrich noted that males were far more likely than females (3:1) to have comorbid anxiety disorder and ASPD diagnoses. Although there is a significant association between CD and nongeneralized SAD, this is entirely accounted for by the association in males (Marmorstein, 2006). Marmorstein (2007) found that the association between SAD and CD is much stronger among males than females.

As both antisocial and socially phobic symptoms change across the lifespan, it should come as no surprise that their comorbidity changes across ages as well. Coid and Ullrich (2010) found that individuals with comorbid ASPD and anxiety diagnoses were younger in age than were individuals with an anxiety disorder alone. Angold et al. (1999) found that the rates of CD in anxious children remained relatively constant over time, but that the rate of anxiety in children with CD decreased over the same span, consistent with the findings of Loeber and Keenan (1994) that the link between CD and anxiety weakens between the ages of 11 and 18 years. Marmorstein (2007) found an association between SAD and CD in children aged 12–14 years, but not in children aged 9–11 or 15–17 years. Because the median age of onset of SAD is 13 years (Kessler et al., 2005), CD may not have occurred at a clinically significant level in the youngest group, and CD may have remitted below clinical significance in the oldest group.

The comorbidity of SAD and ASPD presents unique challenges in treatment. McMurran (2011) claimed that the antisocial behavior in this population is driven by anxiety. Compared with those with just ASPD, patients with comorbid SAD and ASPD may be more responsive to treatment, and it has been proposed that interventions should aim to improve social confidence, reduce hypervigilance, and prepare patients to better deal with threats when they present. Modalities such as cognitive therapy, mindfulness-based therapies, attentional retraining, or social problem-solving may prove effective in meeting these goals. Bubier and Drabick (2009) noted that many comorbid children improve as a result of anxiety-based treatments; thus, the processes that underlie anxiety may play a role in the onset and/or maintenance of

externalizing behaviors and vice versa. They also found it unclear whether anxiety and aggression develop concurrently or whether one precedes the other.

Comorbid Substance Use Disorders

The co-occurrence of SAD with substance use disorders is well documented in both community (Breslau, Novak, & Kessler, 2004; Conway, Compton, Stinson, & Grant, 2006; Kushner, Krueger, Frye, & Peterson, 2008; Lasser et al., 2000) and clinical (Compton, Cottler, Jacobs, Ben-Abdallah, & Spitznagel, 2003; Kushner et al., 2005) samples (see also **Chapter 25** of this book for expanded review). Moreover, longitudinal studies have examined the development, onset, and maintenance of SAD and co-occurring substance use disorders. Indeed, much of the existing work on SAD–substance use comorbidity has been thoroughly reviewed (Carrigan & Randall, 2003; Kushner, Abrams, & Borchardt, 2000; Stewart & Conrad, 2008); as such, the current chapter aims to provide only a brief glimpse of the relevant literature.

Alcohol

Alcohol use disorders are among the most frequently diagnosed disorders, with lifetime prevalence rates of alcohol abuse and dependence estimated at 13.2% and 5.4%, respectively (Kessler, Berglund, et al., 2005), and 12-month prevalence rates of 3.1% and 1.3%, respectively (Kessler, Chiu, et al., 2005). Rates of alcohol use disorders are significantly higher among individuals with a history of and current diagnosis of SAD than nonpsychiatric controls (Kessler et al., 1997). Among older adults (age 60+ years), rates of an alcohol use disorder were significantly higher among those with current SAD (35.2%) than without (20.6%; Chou, 2009). Epidemiological studies have examined the nuances between specific anxiety disorders versus a general "any anxiety" category and have documented the greater odds of alcohol dependence among persons with SAD (Kushner et al., 2008). Furthermore, the risk for comorbid SAD with an alcohol use disorder is greater among females than males (Kessler et al., 1997). SAD generally precedes the onset of substance use disorders (e.g., Buckner, Schmidt, et al., 2008; Kushner et al., 2008; Marmorstein, 2012) with approximately 75–80% of individuals with SAD reporting onset of SAD first.

In efforts to better understand the interplay between SAD and alcohol use disorders, studies have evaluated motives for alcohol use and alcohol expectancies as mediating (explanatory) processes. A recent study using data from a large nationally representative community sample (National Epidemiologic Survey on Alcohol and Related Conditions; NESARC) reported that 14.9% of adults with SAD indicated "self-medication"-oriented drinking motives (Robinson, Sareen, Cox, & Bolton, 2009). Notably, this was higher than rates reported by individuals with panic disorder with (6.7%) and without (3.0%) agoraphobia, specific phobia (4.1%), and GAD (14.1%); OCD and PTSD were not evaluated in this study. Individuals with SAD reported high rates of coping-motivated drinking before and during social situations (e.g., 50% and 80%, respectively; Thomas, Randall, Book, & Randall, 2008).

Tobacco

Approximately one in five adults in the United States smokes cigarettes, and less than half quit successfully (Lasser et al., 2000). These numbers are alarming in light of the fact that cigarette smoking is the leading cause of preventable death and disability in the United States (US Department of Health and Human Services, 2010). Moreover, rates of smoking are doubled among individuals with SAD (54.0%; Lasser et al., 2000). Among older adults, the rate of nicotine dependence is significantly higher among adults with current SAD (24.0%) than those without (12.5%; Chou, 2009). Indeed, after controlling for demographic variables and other comorbid psychiatric diagnoses (e.g., alcohol and other substance use disorders, depression), SAD was associated with a significant increased risk of heavy lifetime smoking, lifetime nicotine dependence, and at least one previous unsuccessful quit attempt (Cougle, Zvolensky, Fitch, & Sachs-Ericsson, 2010). When examining smoking behavior in the past year, an SAD diagnosis was predictive of increased risk for daily smoking, heavy smoking, and nicotine dependence; however, these results were no longer significant after controlling for additional comorbid psychiatric disorders, suggesting that it may be important to consider additional comorbidity when evaluating the SAD–smoking relationship (e.g., panic attacks; Breslau & Klein, 1999; Cougle et al., 2010; Johnson et al., 2000; Piper, Cook, Schlam, Jorenby, & Baker, 2011).

Epidemiological studies have examined the temporal onset of smoking and psychiatric disorders (e.g., Breslau et al., 2004; Johnson et al., 2000). In regards to SAD, a current (12-month) SAD diagnosis significantly increased risk for the subsequent first onset of daily smoking and development of nicotine dependence. Moreover, 81.3% of adult smokers with SAD indicated onset of their anxiety disorder prior to initiation of smoking (Cougle et al., 2010). This pattern of onset may be unique to SAD, as the reverse pattern is consistently found for other anxiety disorders (e.g., Breslau et al., 2004; Cougle et al., 2010; Johnson et al., 2000). These data suggest that the SAD–smoking relationship, in particular, may be influenced by maladaptive coping-oriented smoking motives; however, the current state of the research on anxiety–tobacco relations and motives has been dominated by research on panic-related psychopathology (e.g., Zvolensky & Bernstein, 2005). As such, this area of study warrants further attention in SAD.

Cannabis

Cannabis is the most commonly used illicit drug in the United States (Murray, Morrison, Henquet, & Di Forti, 2007), and the third most commonly used substance after alcohol and tobacco (Conway et al., 2006; Tepe, Dalrymple, & Zimmerman, 2012). Rates of use are estimated to range from 13% to 23% (Agosti, Nunes, & Levin, 2002; Conway et al., 2006; Kessler et al., 2005; Tepe et al., 2012), with rates of lifetime psychiatric comorbidities with cannabis estimated at nearly 90% (Agosti et al., 2002). When examining across illicit drug types, cannabis abuse and dependence are the most commonly occurring among individuals with SAD (17.8%; Conway et al., 2006). From a representative sample (Agosti et al., 2002), rates of lifetime cannabis dependence are estimated at 29% among individuals with a history of SAD, which

is higher than other anxiety disorders including GAD (12.1%), PTSD (18.5%), simple phobia (18.1%), agoraphobia (11.3%), and panic disorder (6.9%). This pattern of results is consistent with findings from Buckner, Schmidt, and colleagues (2008), who found that SAD, but not other anxiety disorders, was significantly predictive of increased risk for cannabis dependence. Further, the majority of individuals with SAD and cannabis use disorders (approximately 60%) report that onset of the anxiety disorder preceded substance use (Buckner, Schmidt, et al., 2008; Tepe et al., 2012). Consistent with this pattern of onset, a growing body of research documents the mediating role of tension-reduction expectancies and coping motives for marijuana use in the relation between SAD and cannabis use and problems related to use (e.g., Buckner, Bonn-Miller, Zvolensky, & Schmidt, 2007; Buckner, Heimberg, Matthews, & Silgado, 2012).

Other Illicit Substances

Among community representative samples, rates of lifetime drug use disorders range from 7.9% (abuse) to 3.0% (dependence; Kessler, Chiu, et al., 2005). In the NESARC data, 22.3% of individuals with SAD reported a lifetime substance use disorder (Conway et al., 2006). In this sample, the most common comorbid drug use disorders among individuals with SAD (aside from cannabis) were cocaine (6.3%), amphetamine (5.5%), hallucinogen (4.1%), opioid (3.6%), tranquilizer (2.9%), and sedative (2.7%) use disorders (Conway et al., 2006). SAD was associated with a significant increase in risk of substance abuse and dependence (Hofmann, Richey, Kashdan, & McKnight, 2009; Marmorstein, 2012), and in the majority of cases (>90%), SAD preceded the onset of drug use disorders (Marmorstein, 2012). Further, self-medication behavior with both alcohol and drugs among individuals with SAD is estimated to range from 4.5% in nationally representative community samples (Robinson et al., 2009) to 16.4% in representative clinical samples (Bolton, Cox, Clara, & Sareen, 2006). Notably, both of these studies evaluated self-medication of alcohol and drugs together, which limits the interpretation of these results. Unfortunately, many studies rarely examine individual drug types, and instead typically lump illicit drugs into one catch-all category term "substance use disorders." This poses difficulty for interpretation and extrapolation of results to clinical settings, and highlights a need for further attention. Although this is a more general issue for the addictions–mental health field, it is relevant to the case of SAD as well.

Clinically speaking, there are several relevant implications that warrant attention when working with the aforementioned populations. Many mental health providers often do not address substance use, frequently citing that they feel inadequate to provide such services (Zvolensky et al., 2005). Regardless, assessment of substance use, abuse, and dependence should be conducted at the outset of SAD treatment, given the documented risk for development of substance use disorders among individuals with SAD. Symptom presentation and severity should be carefully evaluated and triaged accordingly, as alcohol and other anxiolytic substances may attenuate the presentation of SAD symptoms, and severity of both anxiety and substance use appears to increase as they co-occur (Buckner, Timpano, Zvolensky, Sachs-Ericsson, & Schmidt, 2008; Thomas, Randall, & Carrigan, 2003). Further, these comorbid individuals have been

reported to have poor social support and high relational stress (Buckner, Timpano, et al., 2008), and poor physical functioning (Tepe et al., 2012), in comparison to persons with only one of these disorders. Furthermore, while patients might report substance misuse that may not meet diagnostic threshold, in the absence of adaptive coping strategies, risky substance use may continue or even increase. Notably, self-medication-oriented substance use is found to be associated with increased rates of distress, suicidal ideation, and suicide attempts among anxiety disordered, nationally representative samples (Bolton et al., 2006). Therefore, maladaptive coping-oriented substance use motives are an ideal, malleable process to target in SAD treatment.

Conclusions

Comorbidity within SAD is highly prevalent. Anxiety disorders, mood disorders, personality disorders, and substance use disorders are some of the most studied comorbid disorders within SAD research. Overall, research suggests that individuals with SAD who have a comorbid psychological disorder will likely display more severe symptoms, report more impairment, indicate lower quality of life, and display worse treatment outcomes than individuals with an SAD diagnosis alone. As such, further research and dissemination of knowledge about comorbid effects is warranted.

Note

1. Due to the lack of peer-reviewed research concerning comorbidity between SAD and specific phobias, this information will not be reviewed in this chapter.

References

Acarturk, C. C., de Graaf, R., van Straten, A. A., ten Have, M. M., & Cuijpers, P. P. (2008). Social phobia and number of social fears, and their association with comorbidity, health-related quality of life and help seeking: A population-based study. *Social Psychiatry and Psychiatric Epidemiology, 43*(4), 273–279. doi:10.1007/s00127-008-0309-1

Agosti, V., Nunes, E., & Levin, F. (2002). Rates of psychiatric comorbidity among U.S. residents with lifetime cannabis dependence. *The American Journal of Drug and Alcohol Abuse, 28*(4), 643–652. doi:10.1081/ADA-120015873

Alpert, J. E., Fava, M., Uebelacker, L. A., Nierenberg, A. A., Pava, J. A., Worthington, J. J., & Rosenbaum, J. F. (1999). Patterns of axis I comorbidity in early-onset versus late-onset major depressive disorder. *Biological Psychiatry, 46*, 202–211. doi:10.1016/S0006-3223(99)00017-7

American Psychiatric Association (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., revised). Washington, DC: Author.

American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revised). Washington, DC: Author.

Angold, A., Costello, E. J., & Erkanli, A. (1999). Comorbidity. *Journal of Child Psychology and Psychiatry, 40*, 57–87. doi:10.1111/1469-7610.00424

Ball, S. G., Otto, M. W., Pollack, M. H., Uccello, R. ., & Rosenbaum, J. F. (1995). Differentiating social phobia and panic disorder: A test of core beliefs. *Cognitive Therapy and Research, 19*(4), 473–481. doi:10.1007/BF02230413

Barlow, D. H. (1986). Co-morbidity and depression among the anxiety disorders. Issues in diagnosis and classification. *Journal of Nervous and Mental Disease, 174*, 63–72. doi:10.1097/00005053-198602000-00001

Barzega, G., Maina, G., Venturello, S., & Bogetto, F. (2001). Dysthymic disorder: Clinical characteristics in relation to age at onset. *Journal of Affective Disorders, 66*, 39–46. doi:10.1016/S0165-0327(00)00293-7

Beidel, D. C., Turner, S. M., & Morris, T. L. (1999). Psychopathology of childhood social phobia. *Journal of the American Academy of Child & Adolescent Psychiatry, 38*, 643–650. doi:10.1097/00004583-199906000-00010

Bolton, J., Cox, B., Clara, I., & Sareen, J. (2006). Use of alcohol and drugs to self-medicate anxiety disorders in a nationally representative sample. *Journal of Nervous and Mental Disease, 194*(11), 818–825. doi:10.1097/01.nmd.0000 244481.63148.98

Brandes, M., & Bienvenu, O. J. (2006). Personality and anxiety disorders. *Current Psychiatry Reports, 8*, 263–269. doi:10.1007/s11920-006-0061-8

Breslau, N., & Klein, D. F. (1999). Smoking and panic attacks: An epidemiologic investigation. *Archives of General Psychiatry, 56*, 1141–1147. doi:10.1001/archpsyc.56.12.1141

Breslau, N., Novak, S. P., & Kessler, R. C. (2004). Psychiatric disorders and stages of smoking. *Biological Psychiatry, 55*(1), 69–76. doi:10.1016/S0006-3223(03)00317-2

Brown, E. J., Heimberg, R. G., & Juster, H. R. (1995). Social phobia subtype and avoidant personality disorder: Effect on severity of social phobia, impairment, and outcome of cognitive behavioral treatment. *Behavior Therapy, 26*, 467–486. doi:10.1016/S0005-7894(05)80095-4

Brown, T. A., Chorpita, B. F., & Barlow, D. H. (1998). Structural relationships among dimensions of the DSM-IV anxiety and mood disorders and dimensions of negative affect, positive affect, and autonomic arousal. *Journal of Abnormal Psychology, 107*(2), 179–192. doi:10.1037/0021-843X.107.2.179

Bruce, S. E., Yonkers, K. A., Otto, M. W., Eisen, J. L., Weisberg, R. B., Pagano, M., . . . , Keller, M. B. (2005). Influence of psychiatric comorbidity on recovery and recurrence in generalized anxiety disorder, social phobia, and panic disorder: A 12-year prospective study. *American Journal of Psychiatry, 162*(6), 1179–1187. doi:10.1176/appi.ajp.162.6.1179

Bubier, J. L., & Drabick, D.A. (2009). Co-occurring anxiety and disruptive behavior disorders: The roles of anxious symptoms, reactive aggression, and shared risk processes. *Clinical Psychology Review, 29*, 658–669. doi:10.1016/j.cpr.2009.08.005

Buckner, J. D., Bonn-Miller, M. O., Zvolensky, M. J., & Schmidt, N. B. (2007). Marijuana use motives and social anxiety among marijuana using young adults. *Addictive Behaviors, 32*(10), 2238–2252. doi:10.1016/j.addbeh.2007.04.004

Buckner, J. D., Heimberg, R. G., Matthews, R. A., & Silgado, J. (2012). Marijuana-related problems and social anxiety: The role of marijuana behaviors in social situations. *Psychology of Addictive Behaviors, 26*(1), 151–156. doi:10.1037/a0025822

Buckner, J. D., Schmidt, N. B., Lang, A. R., Small, J. W., Schlauch, R. C., & Lewinsohn, P. M. (2008). Specificity of social anxiety disorder as a risk factor for alcohol and cannabis dependence. *Journal of Psychiatric Research, 42*(3), 230–239. doi:10.1016/j.jpsychires.2007.01.002

Buckner, J. D., Timpano, K. R., Zvolensky, M. J., Sachs-Ericsson, N., & Schmidt, N. B. (2008). Implications of comorbid alcohol dependence among individuals with social anxiety disorder. *Depression and Anxiety, 25*(12), 1028–1037. doi:10.1002/da.20442

Carrigan, M. H., & Randall, C. L. (2003). Self-medication in social phobia: A review of the alcohol literature. *Addictive Behaviors, 28*(2), 269–284. doi:10.1016/S0306-4603(01)00235-0

Chambless, D. L., Fydrich, T., & Rodebaugh, T. L. (2008). Generalized social phobia and avoidant personality disorder: Meaningful distinction or useless duplication? *Depression and Anxiety, 25*, 8–19. doi:10.1002/da.20266

Chartier, M. J., Walker, J. R., & Stein, M. B. (2003). Considering comorbidity in social phobia. *Social Psychiatry and Psychiatric Epidemiology, 38*(12), 728–734. doi:10.1007/s00127-003-0720-6

Chavira, D. A., Stein, M. B., Bailey, K., & Stein, M. T. (2004). Comorbidity of generalized social anxiety disorder and depression in a pediatric primary care sample. *Journal of Affective Disorders, 80*, 163–171. doi:10.1016/S0165-0327(03)00103-4

Chou, K. (2009). Social anxiety disorder in older adults: Evidence from the National Epidemiologic Survey on alcohol and related conditions. *Journal of Affective Disorders, 119*(1–3), 76–83. doi:10.1016/j.jad.2009.04.002

Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology, 100*(3), 316–336. doi:10.1037/0021-843X.100.3.316

Clum, G. A., Clum, G. A., & Surls, R. (1993). A meta-analysis of treatments for panic disorder. *Journal of Consulting and Clinical Psychology, 61*(2), 317–326. doi:10.1037/0022-006X.61.2.317

Coid, J., & Ullrich, S. (2010). Antisocial personality disorder and anxiety disorder: A diagnostic variant? *Journal of Anxiety Disorders, 24*, 452–460. doi:10.1016/j.janxdis.2010.03.001

Compton, W. M., Cottler, L. B., Jacobs, J. L., Ben-Abdallah, A., & Spitznagel, E. L. (2003). The role of psychiatric disorders in predicting drug dependence treatment outcomes. *American Journal of Psychiatry, 160*(5), 890–895. doi:10.1176/appi.ajp.160.5.890

Conway, K. P., Compton, W., Stinson, F. S., & Grant, B. F. (2006). Lifetime comorbidity of DSM-IV mood and anxiety disorders and specific drug use disorders: Results from the National Epidemiologic Survey on alcohol and related conditions. *Journal of Clinical Psychiatry, 67*, 247–257. doi:10.4088/JCP.v67n0211

Cougle, J. R., Zvolensky, M. J., Fitch, K. E., & Sachs-Ericsson, N. (2010). The role of comorbidity in explaining the associations between anxiety disorders and smoking. *Nicotine & Tobacco Research, 12*(4), 355–364. doi:10.1093/ntr/ntq006

Cox, B. J., Pagura, J., Stein, M. B., & Sareen, J. (2009). The relationship between generalized social phobia and avoidant personality disorder in a National Mental Health Survey. *Depression and Anxiety, 26*, 354–361. doi:10.1002/da.20475

Craske, M. G., & Barlow, D. H. (1993). Panic disorder and agoraphobia. In D. H. Barlow (Ed.), *Clinical handbook of psychological disorders: A step-by-step treatment manual* (2nd ed., pp. 1–47). New York, NY: Guilford Press.

de Ruiter, C., Rijken, H., Garssen, B., & Van Schaik, A. (1989). Comorbidity among the anxiety disorders. *Journal of Anxiety Disorders, 3*, 57–68. doi:10.1016/0887-6185(89)90001-7

Dilsaver, S. C., & Chen, Y. (2003). Social phobia, panic disorder, and suicidality in subjects with pure and depressive mania. *Journal of Affective Disorders, 77*, 173–177. doi:10.1016/S0165-0327(02)00114-3

Erwin, B. A., Heimberg, R. G., Juster, H., & Mindlin, M. (2002). Comorbid anxiety and mood disorders among persons with social anxiety disorder. *Behaviour Research and Therapy, 40*, 19–35. doi:10.1016/S0005-7967(00)00114-5

Fava, M., Rankin, M. A., Wright, E. C., Alpert, J. E., Nierenberg, A. A., Pava, J., & Rosenbaum, J. F. (2000). Anxiety disorders in major depression. *Comprehensive Psychiatry, 41*, 97–102. doi:10.1016/S0010-440X(00)90140-8

Fennell, D., & Liberato, A. Q. (2007). Learning to live with OCD: Labeling, the self, the stigma. *Deviant Behavior, 28*, 305–331. doi:10.1080/01639620701233274

Freeman, M. P., Freeman, S. A., & McElroy, S. L. (2002). The comorbidity of bipolar and anxiety disorders: Prevalence, psychobiology, and treatment issues. *Journal of Affective Disorders, 68*(1), 1–23. doi:10.1016/S0165-0327(00)00299-8

Gilboa-Schechtman, E., Franklin, M. E., & Foa, E. B. (2000). Anticipated reactions to social events: Differences among individuals with generalized social phobia, obsessive compulsive disorder, and nonanxious controls. *Cognitive Therapy and Research, 24*, 731–746. doi:10.1023/A:1005595513315

Goodwin, R. D., & Hamilton, S. P. (2003). Lifetime comorbidity of antisocial personality disorder and anxiety disorders among adults in the community. *Psychiatry Research, 117*, 159–166. doi:10.1016/S0165-1781(02)00320-7

Gross, P. R., Oei, T. P., & Evans, L. (1989). Generalized anxiety symptoms in phobic disorders and anxiety states: A test of the worry hypothesis. *Journal of Anxiety Disorders, 3*, 159–169. doi:10.1016/0887-6185(89)90010-8

Herbert, J. D., Hope, D. A., & Bellack, A. S. (1992). Validity of the distinction between generalized social phobia and avoidant personality disorder. *Journal of Abnormal Psychology, 101*(2), 332–339. doi:10.1037/0021-843X.101.2.332

Hodgins, S., De Brito, S. A., Chhabra, P., & Cote, G. (2010). Anxiety disorders among offenders with antisocial personality disorders: A distinct subtype? *Canadian Journal of Psychiatry, 55*, 784–791.

Hofmann, S. G., Heinrichs, N., & Moscovitch, D. A. (2004). The nature and expression of social phobia: Toward a new classification. *Clinical Psychology Review, 24*, 769–797. doi:10.1016/j.cpr.2004.07.004

Hofmann, S. G., Richey, J., Kashdan, T. B., & McKnight, P. E. (2009). Anxiety disorders moderate the association between externalizing problems and substance use disorders: Data from the National Comorbidity Survey—Revised. *Journal of Anxiety Disorders, 23*, 529–534. doi:10.1016/j.janxdis.2008.10.011

Hollander, E., Stein, D. J., Fineberg, N. A., Marteau, F., & Legault, M. (2010). Quality of life outcomes in patients with obsessive-compulsive disorder: Relationship to treatment response and symptom relapse. *Journal of Clinical Psychiatry, 71*, 784–792. doi:10.4088/JCP.09m05911blu

Hughes, A. A., Heimberg, R. G., Coles, M. E., Gibb, B. E., Liebowitz, M. R., & Schneier, F. R. (2006). Relations of the factors of the tripartite model of anxiety and depression to types of social anxiety. *Behaviour Research and Therapy, 44*, 1629–1641. doi:10.1016/j.brat.2005.10.015

Huppert, J. D, Strunk, D. R., Ledley, D. R., Davidson, J. R., & Foa, E. B. (2008). Generalized social anxiety disorder and avoidant personality disorder: Structural analysis and treatment outcome. *Depression and Anxiety, 25*, 441–448. doi:10.1002/da.20349

Johnson, J. G., Cohen, P., Pine, D. S., Klein, D. F., Kasen, S., & Brook, J. S. (2000). Association between cigarette smoking and anxiety disorders during adolescence and early childhood. *Journal of the American Medical Association, 284*, 2348–2351. doi:10.1001/jama.284.18.2348

Karno, M., Golding, J. M., Sorenson, S. B., & Burnam, M. (1988). The epidemiology of obsessive-compulsive disorder in five US communities. *Archives of General Psychiatry, 45*, 1094–1099. doi:10.1001/archpsyc.1988.01800360042006

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 62*, 593–602. doi:10.1001/archpsyc.62.6.593

Kessler, R. C., Chiu, W., Demler, O., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 62*, 617–627. doi:10.1001/archpsyc.62.6.617

Kessler, R. C., Crum, R., Warner, L., Nelson, C., Schulenberg, J., & Anthony, J. (1997). Lifetime co-occurrence of DSM-III-R alcohol abuse and dependence with other psychiatric disorders in the National Comorbidity Survey. *Archives of General Psychiatry, 54*, 313–321. doi:10.1001/archpsyc.1997.01830160031005

Kessler, R. C., DuPont, R. L., Berglund, P., & Wittchen, H. (1999). Impairment in pure and comorbid generalized anxiety disorder and major depression at 12 months in two national surveys. *American Journal of Psychiatry, 156*, 1915–1923.

Kessler, R. C., Stang, P., Wittchen, H. U., Stein, M., & Walters, E. E. (1999). Lifetime comorbidities between social phobia and mood disorders in the US National Comorbidity Survey. *Psychological Medicine, 29*, 555–567.

Klein Hofmeijer-Sevink, M., Batelaan, N. M., van Megen, H. M., Penninx, B. W., Cath, D. C., van den Hout, M. A., & van Balkom, A. M. (2012). Clinical relevance of comorbidity in anxiety disorders: A report from the Netherlands Study of Depression and Anxiety (NESDA). *Journal of Affective Disorders, 137*, 106–112.

Kose, S., Solmaz, M., Celikel, F. C., Citak, S., Ozturk, M., Tosun, M., . . . , Sayar, K. (2009). Comorbidity of avoidant personality disorder in generalized social phobia and its impact on psychopathology. *Bulletin of Clinical Psychopharmacology, 19*, 340–346.

Krishnan, K. R. (2005). Psychiatric and medical comorbidities of bipolar disorder. *Psychiatric Medicine, 67*, 1–8. doi:10.1097/01.psy.0000151489.36347.18

Kushner, M. G., Abrams, K., & Borchardt, C. (2000). The relationship between anxiety disorders and alcohol use disorders: A review of major perspectives and findings. *Clinical Psychology Review, 20*, 149–171. doi:10.1016/S0272-7358(99)00027-6

Kushner, M. G., Abrams, K., Thuras, P., Hanson, K. L., Brekke, M., & Sletten, S. (2005). Follow-up study of anxiety disorder and alcohol dependence in comorbid alcoholism treatment patients. *Alcoholism: Clinical and Experimental Research, 29*, 1432–1443. doi:10.1097/01.alc.0000175072.17623.f8

Kushner, M. G., Krueger, R., Frye, B., & Peterson, J. (2008). Epidemiological perspectives on co-occurring anxiety disorder and substance use disorder. In S. H. Stewart, & P. J. Conrod (Eds.), *Anxiety and substance use disorders: The vicious cycle of comorbidity* (pp. 3–17). New York, NY: Springer. doi:10.1007/978-0-387-74290-8_1

Lasser, K., Boyd, J., Woolhandler, S., Himmelstein, D. U., McCormick, D., & Bor, D. H. (2000). Smoking and mental illness: A population-based prevalence study. *Journal of the American Medical Association, 284*, 2606–2610. doi:10.1001/jama.284.20.2606

Liebowitz, M. R., Heimberg, R. G., Fresco, D. M., Travers, J., & Stein, M. B. (2000). Social phobia or social anxiety disorder: What's in a name?. *Archives of General Psychiatry, 57*, 191–192. doi:10.1001/archpsyc.57.2.191-a

Loeber, R., & Keenan, K. (1994). Interaction between conduct disorder and its comorbid conditions: Effects of age and gender. *Clinical Psychology Review, 14*(6), 497–523. doi:10.1016/0272-7358(94)90015-9

Markowitz, J. C., Moran, M. E., Kocsis, J. H., & Frances, A. J. (1992). Prevalence and comorbidity of dysthymic disorder among psychiatric outpatients. *Journal of Affective Disorders, 24*, 63–71. doi:10.1016/0165-0327(92)90020-7

Marmorstein, N. R. (2006). Generalized versus performance-focused social phobia: Patterns of comorbidity among youth. *Anxiety Disorders, 20*, 778–793. doi:10.1016/j.janxdis.2005.08.004

Marmorstein, N. R. (2007). Relationships between anxiety and externalizing disorders in youth: The influences of age and gender. *Journal of Anxiety Disorders, 21*, 420–432. doi:10.1016/j.janxdis.2006.06.004

Marmorstein, N. R. (2012). Anxiety disorders and substance use disorders: Different associations by anxiety disorder. *Journal of Anxiety Disorders, 26*, 88–94. doi:10.1016/j.janxdis.2011.09.005

Marrie, R. A., Horwitz, R. R., Cutter, G. G., Tyry, T. T., Campagnolo, D. D., & Vollmer, T. T. (2009). Comorbidity delays diagnosis and increases disability at diagnosis in MS. *Neurology, 72*, 117–124. doi:10.1212/01.wnl.0000333252.78173.5f

Masi, G., Millepiedi, S., Mucci, M., Pascale, R. R., Perugi, G., & Akiskal, H. S. (2003). Phenomenology and comorbidity of dysthymic disorder in 100 consecutively referred children and adolescents: Beyond DSM-IV. *Canadian Journal of Psychiatry, 48*, 99–105.

McMurran, M. (2011). Anxiety, alcohol intoxication, and aggression. *Legal and Criminological Psychology, 16*, 357–371. doi:10.1111/j.2044-8333.2011.02012.x

Mennin, D. S., Heimberg, R. G., & Jack, M. S. (2000). Comorbid generalized anxiety disorder in primary social phobia: Symptom severity, functional impairment, and treatment response. *Journal of Anxiety Disorders, 14*, 325–343. doi:10.1016/S0887-6185(00)00026-8

Merikangas, K., He, J., Burstein, M., Swanson, S. A., Avenevoli, S., Cui, L., . . . , Swendsen, J. (2010). Lifetime prevalence of mental disorders in U.S. adolescents: Results from the National Comorbidity Survey Replication—Adolescent Supplement (NCS-A). *Journal of the American Academy of Child & Adolescent Psychiatry, 49*, 980–989. doi:10.1016/j.jaac.2010.05.017

Mineka, S., Watson, D., & Clark, L.A. (1998). Comorbidity of anxiety and unipolar mood disorders. *Annual Review of Psychology, 49*, 377–412. doi:10.1146/annurev.psych.49.1.377

Moscovitch, D. A., Hofmann, S. G., Suvak, M. K., & In-Albon, T. (2005). Mediation of changes in anxiety and depression during treatment of social phobia. *Journal of Consulting and Clinical Psychology, 73*(5), 945–952. doi:10.1037/0022-006X.73.5.945

Murray, R. M., Morrison, P. D., Henquet, C., & Di Forti, M. (2007). Cannabis the mind and the society: The harsh realities. *Nature Reviews: Neuroscience, 11*, 885–895. doi:10.1038/nrn2253

Norton, G., Dorward, J., & Cox, B. J. (1986). Factors associated with panic attacks in nonclinical subjects. *Behavior Therapy, 17*, 239–252. doi:10.1016/S0005-7894(86)80054-5

Ohayon, M. M., & Schatzberg, A. F. (2010). Social phobia and depression: Prevalence and comorbidity. *Journal of Psychosomatic Research, 68*, 235–243. doi:10.1016/j.jpsychores.2009.07.018

Orsillo, S. M., Heimberg, R. G., Juster, H. R., & Garrett, J. (1996). Social phobia and PTSD in Vietnam veterans. *Journal of Traumatic Stress, 9*(2), 235–252. doi:10.1002/jts.2490090207

Piper, M. E., Cook, J. W., Schlam, T. R., Jorenby, D. E., & Baker, T. B. (2011). Anxiety diagnoses in smokers seeking cessation treatment: Relations with tobacco dependence, withdrawal, outcome and response to treatment. *Addiction, 106*, 418–427. doi:10.1111/j.1360-0443.2010.03173.x

Ralevski, E. E., Sanislow, C. A., Grilo, C. M., Skodol, A. E., Gunderson, J. G., Shea, M., . . . , McGlashan, T. H. (2005). Avoidant personality disorder and social phobia: Distinct enough to be separate disorders?. *Acta Psychiatrica Scandinavica, 112*, 208–214. doi:10.1111/j.1600-0447.2005.00580.x

Reich, J. H., Noyes, R., & Yates, W. (1988). Anxiety symptoms distinguishing social phobia from panic and generalized anxiety disorders. *Journal of Nervous and Mental Disease, 176*, 510–513. doi:10.1097/00005053-198808000-00011

Robinson, J., Sareen, J., Cox, B. J., & Bolton, J. (2009). Self-medication of anxiety disorders with alcohol and drugs: Results from a nationally representative sample. *Journal of Anxiety Disorders, 23*, 38–45. doi:10.1016/j.janxdis.2008. 03.013

Ruscio, A. M., Stein, D. J., Chiu, W. T., & Kessler, R. C. (2010). The epidemiology of obsessive-compulsive disorder in the National Comorbidity Survey Replication. *Molecular Psychiatry, 15*, 53–63. doi:10.1038/mp.2008.94

Sanderson, W. C., DiNardo, P. A., Rapee, R. M., & Barlow, D. H. (1990). Syndrome comorbidity in patients diagnosed with a DSM-III-R anxiety disorder. *Journal of Abnormal Psychology, 99*, 308–312. doi:10.1037/0021-843X.99.3.308

Schneier, F. R., Johnson, J., Hornig, C. D., Liebowitz, M. R., & Weissman, M. M. (1992). Social phobia: Comorbidity and morbidity in an epidemiologic sample. *Archives of General Psychiatry, 49*, 282–288.

Schneier, F. R., Spitzer, R. L., Gibbon, M., Fyer, A. J. (1991). The relationship of social phobia subtypes and avoidant personality disorder. *Comprehensive Psychiatry, 32*, 496–502. doi:10.1016/0010-440X(91)90028-B

Simon, N. M., Smoller, J. W., Fava, M., Sachs, G., Racette, S. R., Perlis, R., . . . , Rosenbaum, J. F. (2003). Comparing anxiety disorders and anxiety-related traits in bipolar disorder and unipolar depression. *Journal of Psychiatric Research, 37*, 187–192. doi:10.1016/S0022-3956(03)00021-9

Stengler-Wenzke, K., Beck, M. M., Holzinger, A. A., & Angermeyer, M. C. (2004). Stigma experiences of patients with obsessive compulsive disorders. *Fortschritte Der Neurologie, Psychiatrie, 72*, 7–13. doi:10.1055/s-2003-812450

Stewart, S. H., & Conrod, P. J. (2008). *Anxiety and substance use disorders: The vicious cycle of comorbidity.* New York, NY: Springer. doi:10.1007/978-0-387-74290-8

Tepe, E., Dalrymple, K., & Zimmerman, M. (2012). The impact of comorbid cannabis use disorders on the clinical presentation of social anxiety disorder. *Journal of Psychiatric Research, 46*, 50–56. doi:10.1016/j.jpsychires.2011.09.021

Thomas, S. E., Randall, C. L., & Carrigan, M. H. (2003). Drinking to cope in socially anxious individuals: A controlled study. *Alcoholism: Clinical and Experimental Research, 27*, 1937–1943. doi:10.1097/01.ALC.0000100942.30743.8C

Thomas, S. E., Randall, P. K., Book, S. W., & Randall, C. L. (2008). The complex relationship between co-occurring social anxiety and alcohol use disorders: What effect does treating social anxiety have on drinking?. *Alcoholism: Clinical and Experimental Research, 32*, 77–84. doi:10.1111/j.1530-0277.2007.00546.x

Tillfors, M., Furmark, T., Ekselius, L., & Fredrikson, M. (2004). Social phobia and avoidant personality disorder: One spectrum disorder? *Nordic Journal of Psychiatry, 58*, 147–152. doi:10.1080/08039480410005530

Tran, G. Q., & Chambless, D. L. (1995). Psychopathology of social phobia: Effects of subtype and of avoidant personality disorder. *Journal of Anxiety Disorders, 9*, 489–501. doi:10.1016/0887-6185(95)00027-L

Turner, S. M., Beidel, D. C., Borden, J. W., Stanley, M. A., & Jacob, R. G. (1991). Social phobia: Axis I and II correlates. *Journal of Abnormal Psychology, 100*, 102–106. doi:10.1037/0021-843X.100.1.102

US Department of Health and Human Services (2010). *How tobacco smoke causes disease: The biology and behavioral basis for smoking-attributable disease: A report of the surgeon general.* Atlanta, GA: Author.

Väänänen, J., Fröjd, S., Ranta, K., Marttunen, M., Helminen, M., & Kaltiala-Heino, R. (2011). Relationship between social phobia and depression differs between boys and

girls in mid-adolescence. *Journal of Affective Disorders, 133*, 97–104. doi:10.1016/j.jad.2011.03.036

Van Velzen, C. J. M., Emmelkamp, P. M. G., & Scholing, A. (1997). The impact of personality disorders on behavioral treatment outcome for social phobia. *Behavior Research and Therapy, 35*, 889–900. doi:10.1016/S0005-7967(97)00052-1

Van Velzen, C. J. M., Emmelkamp, P. M. G., & Scholing, A. (2000). Generalized social phobia versus avoidant personality disorder: Differences in psychopathology, personality traits, and social and occupational functioning. *Journal of Anxiety Disorders, 14*, 395–411. doi:10.1016/S0887-6185(00)00030-X

Versiani, M., Mundim, F., Nardi, A., & Liebowitz, M. R. (1988). Tranylcypromine in social phobia. *Journal of Clinical Psychopharmacology, 8*, 279–283. doi:10.1097/00004714-198808000-00008

Wells, J. C., Tien, A. Y., Garrison, R., & Eaton, W. W. (1994). Risk factors for the incidence of social phobia as determined by the Diagnostic Interview Schedule in a population-based study. *Acta Psychiatrica Scandinavica, 90*, 84–90. doi:10.1111/j.1600-0447.1994.tb01560.x

Wittchen, H. U., & Fehm, L. (2003). Epidemiology and natural course of social fears and social phobia. *Acta Psychiatrica Scandinavica, 108*, 4–18. doi:10.1034/j.1600-0447.108.s417.1.x

Wittchen, H. U., Fuetsch, M. M., Sonntag, H. H., Müller, N. N., & Liebowitz, M. M. (2000). Disability and quality of life in pure and comorbid social phobia: Findings from a controlled study. *European Psychiatry, 15*, 46–58. doi:10.1016/S0924-9338(00)00211-X

Zayfert, C., DeViva, J. C., & Hofmann, S. G. (2005). Comorbid PTSD and social phobia in a treatment-seeking population: An exploratory study. *Journal of Nervous and Mental Disease, 193*(2), 93–101. doi:10.1097/01.nmd.0000152795.47479.d9

Zvolensky, M. J., Baker, K., Yartz, A. R., Gregor, K., Leen-Feldner, E., & Feldner, M. T. (2005). Mental health professionals with a specialty in anxiety disorders: Knowledge, training, and perceived competence in smoking cessation practices. *Cognitive and Behavioral Practice, 12*, 312–318. doi:10.1016/S1077-7229(05)80053-7

Zvolensky, M. J., & Bernstein, A. (2005). Cigarette smoking and panic psychopathology. *Current Directions in Psychological Science, 14*, 301–305. doi:10.1111/j.0963-7214.2005.00386.x

11

Diversity Considerations in the Assessment and Treatment of Social Anxiety Disorder

Peter C. Meidlinger and Debra A. Hope
University of Nebraska-Lincoln, USA

Introduction

As research on social anxiety disorder (SAD) has expanded over the past 30+ years, so has our understanding of the importance of cultural diversity in the psychopathology, assessment, and treatment of mental disorders (American Psychological Association [APA], 2006). Social anxiety, by definition, involves a social context which is delineated by a variety of cultural variables, such as local social norms for verbal and nonverbal behavior, and expectations for certain social roles based on the demographic characteristics of the individuals involved in the social exchange. It is therefore somewhat surprising that the literature on SAD across diverse populations is still fairly modest. This chapter will first examine theories of SAD etiology and maintenance with cultural diversity in mind, followed by application of *minority stress theory* (e.g., Meyer, 2003). This theoretical work will then guide the review of the research and clinical literature on cultural diversity and SAD.

Before launching into the primary purpose of this chapter, one caveat must be considered. Although one might consider diversity to encompass almost any demographic variable, in research practice the focus has been on gender, race/ethnicity, sexual orientation, and, to a lesser extent, religion. Because of this, the bulk of information within this chapter will focus on these constructs. Going forward, it is important to note that our review of these constructs and categories comes with full recognition of the fundamental problems with them. While race/ethnicity information is reported in studies, and is important, it likely functions as a proxy for social and cultural issues rather than being a causal variable in its own right (e.g., Hoover, 2007). Binary construals of gender are also inadequate (e.g., Muehlenhard & Peterson, 2011) and gay/lesbian/bisexual/heterosexual labels for sexual orientation may mask important variation (e.g., Sell, 1997). Thus, our discussion of diversity in SAD is necessarily constrained by the limitations in the constructs used to define diversity in the clinical and scientific literature.

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

The core of SAD is often understood to be fear of negative and, more recently, positive evaluation (e.g., Weeks, Heimberg, & Rodebaugh, 2008; Weeks, Heimberg, Rodebaugh, & Norton, 2008) and is etiologically thought to frequently involve being made to feel different or victimized, often early in life (Rapee & Heimberg, 1997). Minority group membership may be linked to these etiological theories of SAD because members are more likely to experience discrimination and victimization, which has been documented among racial/ethnic minorities (e.g., Borrell et al., 2007; Pérez, Fortuna, & Alegria, 2008), women (e.g., Belle & Doucet, 2003), and sexual minorities (lesbian, gay, bisexual, and transgender individuals; e.g., Herek, 2009), as well as other groups. One might expect that this increased experience of discrimination would result in elevated perception of social threat among these groups as well as an elevated prevalence of SAD.

The reality of the occurrence of social anxiety across demographic variables is much more complex than what may be posited from conceptualizations of SAD and prevalence rates of discrimination (see Table 11.1 for illustrative SAD prevalence information). In terms of gender, research has shown that prevalence rates among women are higher than men (e.g., Grant et al., 2005) but that men are more likely to seek treatment (e.g., Rapee, Sanderson, & Barlow, 1988). This is particularly interesting because it indicates important factors operating on both genders. Prevalence rates among racial/ethnic minorities provide an even more complex picture, with African Americans, Latinos, and Asian Americans showing lower prevalence rates than European

Table 11.1 Illustrative Prevalence of Social Anxiety Disorder in Minority Groups

Group	*Lifetime prevalence rate (%)*	*Comparison prevalence rate (%) (Comparison group)*
Women	5.7[a]	4.2 (Men)[a]
	15.5[b]	11.1 (Men)[b]
African American	3.5[a]	5.5 (European American)[a]
	8.6[c]	12.6[c]
Hispanic American	3.2[a]	5.5 (European American)[a]
	8.2[c]	12.6 (European American)[c]
Asian American	3.3[a]	5.5 (European American)[a]
	5.3[c]	12.6 (European American)[c]
Native American	8.6[a]	5.5 (European American)[a]
LGB Women		
Lesbian	9.6[d]	7.9[d] (Heterosexual Women)
Bisexual	18.2[d]	7.9[d] (Heterosexual Women)
LGB Men		
Gay	12.4[d]	5.8[d] (Heterosexual Men)
Bisexual	14.2[d]	5.8[d] (Heterosexual Men)

[a]Grant et al. (2005).
[b]Kessler et al. (1994).
[c]Asnaani, Richey, Dimaite, Hinton, and Hofmann (2010).
[d]Bostwick, Boyd, Hughes, and McCabe (2010).
LBG, lesbian, gay, or bisexual.

Americans, but with Native Americans showing elevated incidences of SAD relative to European Americans (e.g., Grant et al., 2005). Interestingly, the lowest prevalence rates in some studies are shown among Asian Americans, who alternately show the highest scores on standard questionnaires measuring social anxiety (e.g., Hambrick et al., 2010).

The only well-researched group that shows prevalence rates in the predicted direction (regarding minority group membership) are individuals who identify as lesbian, gay, and bisexual (LGB), who have higher prevalence rates than heterosexual individuals (Bostwick et al., 2010), a difference that may be particularly large for gay men. Beyond meeting diagnostic criteria, there is some evidence that gay men report greater social evaluative concern than heterosexual men generally (e.g., Pachankis & Goldfried, 2006). Thus, the relationship between SAD and diversity appears to be particularly important for LGB individuals.

While there is no conclusive research indicating why LGB individuals differ from other minority groups regarding SAD prevalence, one salient possibility is the fact that LGB individuals can sometimes conceal their stigmatized identity (Pachankis, 2007). While other groups may similarly be able to alter the salience of their stigmatized identity by altering behaviors, appearance, or speech, the stigma itself is typically not as readily concealable as it is among LGB individuals. As shall be discussed further below, this capacity for avoidance, along with other issues, may provide theoretical reasons that LGB individuals report more social anxiety and exhibit higher rates of SAD.

Theories of SAD and Minority Stress

There have been a number of cognitive behavioral models proposed for SAD, and they share important commonalities such as the role of comparison to others and information processing biases (e.g., Rapee & Heimberg, 1997). These models hypothesize that audience factors cause the preferential allocation of attentional resources to both an internal mental representation of self as appraised by the audience and external indicators of social threat. Comparison of the internal representation with the excessive, expected audience standard results in elevated estimates of both the cost and probability of a negative outcome, which are additionally influenced by the cognitive biases of the individual. These estimates result in the cognitive (e.g., negative thoughts), physiological (e.g., increased heart rate), and behavioral (e.g., subtle avoidance, escape) aspects of SAD. These aspects then influence the internal representation of self and the increased attention to indicators of evaluation. They may also impair performance and affect the situational factors (e.g., audience reaction to impaired performance or escape/avoidance).

Minority stress models

A number of models similarly exist for understanding minority stress processes, typically focused on individual minority groups (e.g., Clark, Anderson, Clark, & Williams, 1999; Meyer, 2003). Moradi's (2013) pantheoretical framework of minority stress is an attempt to unify several specific models of minority stress in the literature on

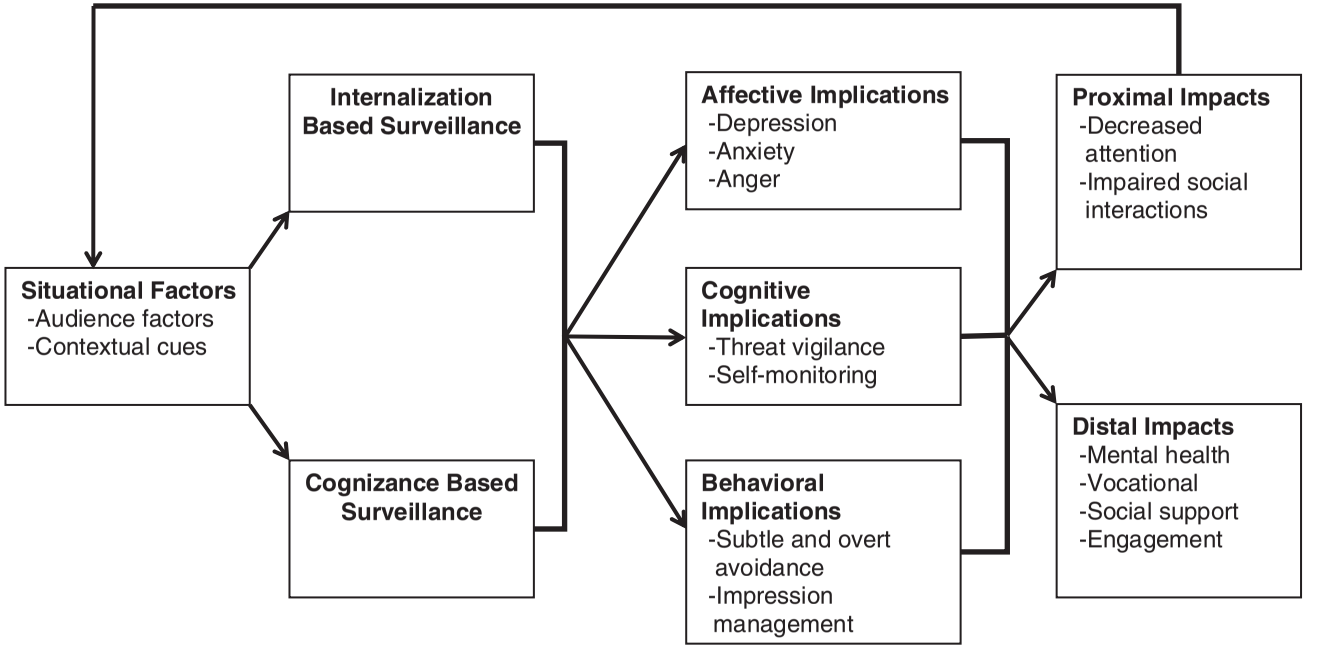

Figure 11.1 Moradi's pantheoretical model of minority stress applied to a specific situation for social anxiety.

ethnic/racial minorities, sexual minorities, and gender. As will be shown below, this model has marked similarity to cognitive behavioral theories of social anxiety. According to Moradi's framework, the experience of discrimination (e.g., racism, sexism, heterosexism) yields two separate types of *surveillance*, one based on the internalization of negative societal views and the other based in cognizance of potential discrimination. Surveillance, in this model, is vigilance toward potential threats and ongoing self-monitoring behaviors.

Cognizance-based surveillance is surveillance resulting from the awareness of potential threat, whereas internalization-based surveillance is based on internalization of negative societal views. The proximal result of both types of surveillance is that individuals may divert attention toward monitoring their social environment for potential indicators of interpersonal threat. Individuals may also focus on increased impression management by explicitly concealing their identity, managing aspects of it, or engaging in stigma-related monitoring (e.g., Buchanan, Fischer, Tokar, & Yoder, 2008). Some research also indicates that minority individuals engage in more general impression management than non-minorities (e.g., Bolino, Kacmar, Turnley, & Gilstrap, 2008).

Fitting Moradi's (2013) model into a cognitive behavioral framework yields a model compatible with our understanding of SAD (see Figure 11.1). Situational factors that elevate the salience of an individual's minority status (e.g., Vohs, Baumeister, & Ciarocco, 2005) or that increase the potential costs of negative evaluation may increase the degree of surveillance that an individual engages in. These situational factors are also likely to interact dynamically with both the degree of internalization and cognizance of an individual, which may influence basic information processing. The consequence of this is that individuals with high degrees of internalization or cognizance may be likely to view otherwise neutral or positive situations as more threatening. Both types of surveillance are also likely influenced by situational factors, with high-threat situations increasing both cognizance and internalization.

The perception of threat in a situation has cognitive, affective, and behavioral consequences through cognizance- and internalization-based surveillance. Cognitively, the primary result is surveillance, or increased vigilance toward external indicators of threat and internally focused self-monitoring of aspects of the threatened identity (Moradi, 2013). Heightened perception of threat is likely to result in increased negative affect. The behavioral implications are increased impression management and avoidance. In terms of concealable stigma, such as LGB identity, this process is more apparent and may involve avoiding mention of the stigmatized identity, avoidance of topics related to the stigma, and altering speech and behavioral patterns associated with the stigma (e.g., Pachankis & Goldfried, 2006). While these processes are often not discussed regarding nonconcealable stigma, there is evidence that these same sorts of impression management occur as attempts to decrease salience across all stigmatized identities (e.g., Ogbu, 2004). Minority individuals who are uncomfortable or threatened in certain situations may seek to escape from them quickly or avoid those situations altogether.

These cognitive, affective, and behavioral implications have both proximal and distal impacts. Proximally, they impair performance as the result of what Moradi (2013) calls "interrupted flow." This may take two separate routes. First, diverting

attentional resources to surveillance may use up resources needed to attend to the current social interaction, which may in turn impair performance (e.g., Vohs et al., 2005) and cause distress. Disrupted individual performance then further influences the situation, including the reactions of potential evaluators, which may serve to confirm expectations of negative evaluation or other social threat. As Moradi (2013) indicates, distally, the consequences may involve broader mental and physical health issues, educational and vocational issues, and decreased activism, especially if effective coping strategies are not employed. Research with LGB individuals has also indicated that concealment and self-monitoring may result in impaired social support, which may inhibit an individual's ability to cope adaptively with stressors (Potoczniak, Aldea, & DeBlaere, 2007).

Linking the models

There is significant conceptual overlap between models of SAD and Moradi's (2013) pantheoretical framework. Both models focus on the assessment of social threat through both self-focused and audience-focused attention. Both models have disrupted performance and avoidance as principal functional consequences. There are, however, significant differences between the two models, the most important one being that the model of SAD is a model of pathology, whereas the model of minority stress describes deficits resulting from psychosocial stressors.

In both models, the perception of interpersonal threat is initially cued by situational factors (e.g., audience variables, contextual variables) but is filtered through the individual's perceptions of social threat. In SAD, this interpretation is seen as occurring through the individual's information processing biases, which can be seen as similar to the degree of an individual's cognizance and internalization in Moradi's (2013) model. The two models differ, however, in the degree of realism involved in that threat assessment. In models of SAD, the increased sense of threat is the result of cognitive biases and is inherently maladaptive. However, racial/ethnic minorities (e.g., Borrell et al., 2007), sexual minorities (e.g., Herek, 2009), and women (e.g., Belle & Doucet, 2003) all face a realistically elevated threat of discrimination. This means that information processing tendencies that would be seen as biased and maladaptive in majority individuals may represent a realistic perception of social threat for some minority individuals.

In both models, the perception of social threat results in a shift of attentional resources toward situational indicators of social threat and internal modeling or surveillance to prevent negative social outcomes. According to both models, this shift in attentional resources leads to impaired performance within the situation. While this attention is inherently maladaptive in the model of SAD, it is not necessarily so in the minority stress model. While the diversion of attentional resources may impair the performance of a minority individual in social situations, if the presence of social threat is realistic then this impaired performance may represent the better of the two outcomes.

These models also have broader outcomes in terms of avoidance. In SAD models, individuals may avoid anxiety-provoking situations altogether, may escape early, or may engage in subtle avoidance within the situation (e.g., avoiding eye contact; Rapee & Heimberg, 1997). Similarly, the surveillance in Moradi's (2013) model

involves ongoing self-monitoring within the situation. Among individuals with nonconcealable stigmas, this may involve actions that may alter the salience of the stigmatized identity (e.g., avoiding topics associated with the identity), but among individuals with concealable stigmas, this may involve avoidance of the disclosure of the stigmatized identity completely (D'Augelli, 1992). Again, the major difference between these two models is that avoidance within the framework of minority stress is not necessarily pathological and may in fact represent adaptive functioning in some situations.

These two models both deal with perceptions of social threat and are broadly similar in terms of their function. The major difference between the two is that the perception of threat within the SAD model is inherently pathological, whereas the perception of threat within the framework of minority stress is nonpathological and may represent a realistic threat assessment. For example, a gay man who has not disclosed his sexual orientation to his family, and fears that they would disown him may, in fact, be correct. While this does not mean that it would not be beneficial for this client to disclose, it does indicate that more consideration should be given to helping the client weigh the costs, benefits, and potential risks of the decision. This also may blur the distinction between pathological and nonpathological behavior among minority clients. Fears of negative evaluation for some minority clients may represent realistic perception or, importantly, mildly exaggerated perception of realistic social threat. Moradi's (2013) model can be helpful in guiding treatment because it does not dismiss the feared outcomes as unrealistic, leaving room to utilize standard treatments for those fears which are unrealistic but also indicating options (e.g., improving social support) in those situations in which the fears are realistic.

Cultural Variation in Presentation

Culture-Specific Presentations

SAD is largely a social phenomenon and as such is subject to variation as a result of the cultural context in which it occurs. It is unsurprising then, given the variability of cultures and social norms across the world, that SAD appears to present differently in different cultures. The most well studied of these culturally specific presentations is *taijin kyofusho* (TKS), which appears predominantly among East Asian cultures, particularly Japan and Korea.

TKS has been subdivided into a neurotic subtype and an offensive subtype. The neurotic subtype is most similar to the presentation of SAD in Western cultures (e.g., Choy, Schneier, Heimberg, Oh, & Liebowitz, 2008). In the offensive subtype, fears focus more on offending or discomforting *others* than the traditional SAD fears of embarrassment or humiliation of the *self* (e.g., Takahashi, 1989). The fears of TKS are broadly thought of as being more allocentric than those in SAD, and in addition to the typical fears seen within SAD, include concern about offending others with one's body odor, intestinal gas, eye gaze, blushing, or stiff facial expressions. It is thought that this difference reflects the differences in individualism and collectivism between Western and European cultures and East Asian cultures, respectively (e.g., Schreier et al., 2010).

The exact relationship and amount of overlap between SAD and TKS remains unclear, however. Western individuals with SAD score higher on measures of TKS than nonanxious controls (e.g., Kim, Rapee, & Gaston, 2008). Research using a measure of TKS to compare US and Korean patient samples found that the US sample endorsed the allocentric fears as much or more than did the Korean sample (Choy et al., 2008). This study also found that both samples reported greater concern about embarrassment than offense, and concluded that TKS symptoms may be present to a greater degree in the United States than previously thought. Other research, however, has indicated that symptom overlap is not as complete, and individuals with either disorder meet criteria for the other often, but not always (e.g., Kim et al., 2008). Regardless of the overlap between SAD and TKS, it is worth considering that features of TKS may appear in Western individuals with SAD and be relevant to treatment.

A second, less extensively studied, cultural variation in the presentation of SAD is the report of *aymat zibur*, or "fear of the community," among ultra-orthodox Jewish men in Jerusalem (Greenberg, Stravynski, & Bilu, 2004). In this syndrome, men report fearing speaking about religious matters or leading religious rites, whereas other social concerns are absent. This absence is attributed to the emphasis placed on the centrality of religion to all interaction in this community. Interestingly, Greenberg and colleagues (2004) reported there were no women who suffered from *aymat zibur* due to women being discouraged from participation in these roles. Although *aymat zibur* may not be widespread, it is intriguing as an illustration of the influence that culture can have on the presentation of SAD.

Cultural Influences on Presentation

At the core of SAD, the fear of negative evaluation is embedded in the context of the individual. The exact presentation of an individual SAD case is, then, partially a product of the cultural environment. Our understanding of SAD within any specific cultural context, even with the relatively well-studied example of TKS, is moderate at best and applies to only a relatively small number of cases. Further examination of cultural variables relevant to SAD should enhance the understanding of a broader range of cases.

One important factor in understanding SAD across cultures and individuals is collectivistic versus individualistic cultures. Research indicates that cultures such as Japan and Korea, which are linked to TKS, are cultures that broadly view individuals as interdependent on one another and that value cooperative relationships; individualistic countries, like the United States, place greater value on individual achievement (e.g., Kitayama, Markus, Matsumoto, & Norasakkunkit, 1997). Individuals from collectivistic cultures (especially East Asian cultures) tend to score higher on self-report measures of social anxiety, but this elevated social concern becomes problematic when there is a cultural mismatch in which the collectivistic individual is placed in an individualistic culture (e.g., Cross, 1995). In contrast, people from collectivist Latin American countries score lower on measures of SAD compared with people from individualistic cultures (e.g., Schreier et al., 2010). The reason for this variation among

collectivistic cultures is unknown, although Schreier et al. (2010) posited that social norms may play a role.

Self-construal is thought to be one of the constructs underlying the differences between individualistic and collectivist cultures. Independent self-construals view the individual as a separate and unique actor operating outside of the social environment, whereas interdependent self-construals view the self as an integrated part of the social context (e.g., Markus & Kitayama, 1991). Interestingly, there is variation in self-construal within cultural boundaries, particularly across gender. Women reported more interdependent and men reported more independent self-construal (Cross & Madson, 1997). This relationship between gender and self-construal is particularly important because they appear to interact in predicting social anxiety, with low interdependence and high independence being associated with social anxiety in women but showing the opposite relationship in men (Moscovitch, Hofmann, & Litz, 2005).

Self-construal is related to self-report of social anxiety (e.g., Okazaki, 1997) and ease of embarrassment (e.g., Singelis & Sharkey, 1995). Researchers sampling from both Japan and the United States found that independent self-construal was associated with more self-focused aspects of SAD (e.g., embarrassment), whereas interdependent self-construal was associated with more other-focused aspects of SAD (e.g., fears of offending others; Norasakkunkit, Kitayama, & Uchida, 2012). Understanding self-construal may be clinically relevant because it may influence the degree of anxiety an individual self-reports and also the social outcomes they fear in a given situation.

Research relating to social and cultural norms has indicated that cultural differences in ease of embarrassment and the use and function of shame may be important factors in predicting social anxiety, and that these constructs may play a more important role in East Asian cultures, which tend to focus on shame more than Western European cultures (e.g., Singelis & Sharkey, 1995). Other research has indicated that other cultural norms are important, particularly the acceptability and reaction to social distress and withdrawal. Collectivistic cultures tend to be more accepting of social distress and withdrawal than individualistic cultures (Heinrichs et al., 2006; Schreier et al., 2010), which may explain why social anxiety may not be a problem for some individuals until they immigrate from collectivist to individualistic cultures (e.g., Cross, 1995). Gender roles may play a role also, with endorsement of more traditionally masculine gender within the United States predicting less social anxiety for both men and women (e.g., Moscovitch et al., 2005).

As shown in this brief review, a full understanding of the nature of SAD in a given individual requires consideration of his or her cultural context, including the relationship of the individual to society and the cultural norms for expression of and response to social anxiety, as well as related emotions such as shame. This is particularly important if a therapist or researcher does not share the same cultural background as the client or research participant.

Assessment

Relatively little research has examined issues related to assessment of SAD with various minority groups. The bulk of what has been conducted has focused largely on

self-report measures and on populations of Asian origin with relatively little research examining assessment among Latinos, African Americans, and sexual minorities.

Research comparing the responses of East Asians and East Asian Americans with those of European Americans on standard self-report measures of social anxiety has consistently shown higher anxiety among East Asians and those of East Asian descent (e.g., Hambrick et al., 2010; Okazaki, 2002). Interestingly, the tendency of Asian Americans to report more social anxiety than European Americans on self-report measures does not appear to translate into differences in behavioral indicators of anxiety (Okazaki, Liu, Longworth, & Minn, 2002) or other differences observable by their peers (Okazaki, 2002). Other research has indicated that bicultural identity may be a factor, as bicultural individuals reported significantly more anxiety than monocultural individuals from Canada or East Asia (Hsu et al., 2012).

Research on the measurement of social anxiety among African Americans has been sparse, but what has been found has indicated that African Americans generally report less social anxiety on self-report measures compared with European Americans (e.g., Beard et al., 2011; Gillis, Hagga, & Ford, 1995), although at least one study has found the opposite (Melka, Lancaster, Adams, Howarth, & Rodriguez, 2010). African Americans showed significantly lower scores than European Americans on a measure of social anxiety symptoms (Gillis et al., 1995), although in this sample the two comparison groups differed significantly on economic variables also. Hambrick et al. (2010) found that individual items in the Social Interaction Anxiety Scale (SIAS; Mattick & Clarke, 1998) functioned differently between African Americans and European Americans, although they drew no conclusions about those differences. Similarly, Melka et al. (2010) found that African Americans obtained lower average scores than European Americans on the Fear of Negative Evaluation Scale (FNE; Watson & Friend, 1969), but conversely higher scores on the Social Avoidance and Distress Scale (SADS; Watson & Friend, 1969), perhaps due to differing factor structures and/or differences in the functioning of specific scale items across the two groups. The Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987) also has shown a factor structure that differs by ethnicity in African American and European American samples, although the meaning of this difference is unclear (Beard et al., 2011).

Among Latinos, there has been less research into potential issues with self-report measures for social anxiety. One study conducted with English-speaking Latinos in the United States found that the LSAS showed a similar factor structure to that found among European Americans, indicating psychometric similarity (Beard, Rodriguez, Weisberg, Perry, & Keller, 2012). In comparing across ethnic groups, Norton and Weeks (2009) found that English-speaking Latino and African Americans scored lower than Asian and European Americans on a measure of fear of negative evaluation but not a measure of fear of positive evaluation. Research examining the psychometric properties of Spanish language versions of the SIAS and Social Phobia Scale (Mattick & Clarke, 1998) with a population in Spain replicated factor structures found in the measures in the United States (Olivares, García-López, & Hidalgo, 2001), although the meaning of these results for a Latino American population is unclear given the cultural differences between Spain and Latin America.

Several studies have shown that LGB individuals, particularly gay men, report greater social anxiety on self-report measures than do similar heterosexual controls (e.g.,

Pachankis & Goldfried, 2006; Safren & Pantalone, 2006) but these same studies raise the possibility that these scores are influenced by the presence of realistic interpersonal threat (e.g., Herek, 2009). The elevated self-report scores are consistent with elevated SAD prevalence rates of LGB samples discussed earlier. However, the diagnostic interviews may not have distinguished between excessive and realistic social fears. The clinical challenge then is to assess which fears are maladaptive and which situations are to be targets of clinical intervention. There is, of course, no well-researched means of doing this, but a clinician who is well-informed about the climate for LGB individuals in the community is essential. Broad techniques such as motivational interviewing to examine the pros and cons of addressing the feared situation versus those of not facing it (Hope, Heimberg, & Turk, 2010) may be helpful in aiding the client to examine the source of fears and decide on a course of action.

Assessment surrounding LGB individuals is further complicated by the heterocentric language present in many measures of social anxiety through the presumption of heterosexuality. Many measures of SAD ask about dating-related anxiety and do so by indicating the sex of the other person (e.g., "I have difficulty talking to attractive persons of the opposite sex"; Mattick & Clarke, 1998). Weiss, Hope, and Capozzoli (2012) found that alternate but nongendered wording (e.g., "I have difficulty talking to someone I'm attracted to") yielded psychometrically equivalent ratings compared with the standard wording for several self-report measures among a sample of largely heterosexual, European American college students. Although this wording is not yet validated on an LGB sample, Weiss et al. (2012) recommend its usage for ethical reasons.

Overall, the indication is that the scores obtained from self-report measures of SAD, and to some extent the measures themselves, may differ across different cultural groups. African Americans and Latinos tend to show lower scores than European Americans on most measures, which is consistent with prevalence rate information. Asian Americans show the highest scores on SAD measures but are less likely to be diagnosed with SAD than European Americans, possibly indicating that cultural differences result in greater social concern but less impairment and anxiety. Native Americans have higher prevalence rates of SAD in comparison with other racial/ethnic groups, but there are no published data on self-report measures of social anxiety. Finally, LGB individuals show elevated SAD prevalence rates and scores on self-report measures of social anxiety, but both may be influenced by realistic fears of negative evaluation due to heterosexism. It is also notable that the bulk of the research conducted on the assessment of social anxiety has been conducted with self-report measures, and there is no information regarding other assessment techniques such as behavioral assessment or even structured interviews.

Treatment

Clinical Trials of Cognitive Behavioral Treatment

Cognitive behavioral treatment (CBT) for SAD is the treatment modality that has received the most consistent research support in meta-analyses (e.g., Acarturk,

Cuijpers, van Straten, & de Graaf, 2009; Hofmann & Smits, 2008). These treatments typically involve the use of cognitive restructuring to help clients challenge maladaptive thoughts combined with graduated exposure to the feared situations (see **Chapter 22** for a thorough outline). Meta-analytic research has indicated that behavioral exposure is a key component, and that the addition of cognitive restructuring does not appear to alter outcomes (Feske & Chambless, 1995). CBT is also the most widely used of treatments for SAD (Rodebaugh, Holaway, & Heimberg, 2004) and much of the research evidence for treatment effects across different minority groups comes from randomized controlled trials (RCTs) conducted using this approach, so exposure-based treatments will be the focus of this review.

The most recent meta-analysis (Acarturk et al., 2009) included 30 RCTs for treatment of SAD; 7 of these trials were excluded from the review below because meeting diagnostic criteria was not used as inclusion criteria and 7 others were excluded because they used techniques different from cognitive restructuring and exposure (e.g., applied relaxation) or utilized a different therapeutic modality (i.e., internet administered therapy). The addition of five newer RCTs that were published following the publication of the meta-analysis, and two older RCTs excluded from the meta-analysis yield a total of 23 trials included in this review. This includes some studies with a pharmacotherapy condition, which are included primarily here rather than in the next section (see *Clinical Pharmacotherapy Trials* section). On the whole, these studies presented little information relating to the diversity of their samples, with 18 reporting only participant gender, only 4 reporting race/ethnicity, and no studies reporting participant sexual orientation. Details of the studies that reported analyses by these demographic variables appear in Table 11.2.

Two studies reported that gender did not appear to impact attrition (Hofmann, 2004; Oosterbaan, van Balkom, Spinhoven, van Oppen, & van Dyck, 2001) and others reported that gender did not predict treatment response (e.g., Clark et al., 2003; Davidson et al., 2004; Otto et al., 2000). These data appear to indicate that gender variability in presentation of SAD can be taken into account in existing treatments, even within the constraints of an RCT. It should be noted, however, that the sample sizes of most of these trials are modest, and lack of gender effects could be the result of statistical power issues.

Relatively fewer studies assessed sample race and ethnicity, with the reported results often being described as a white/nonwhite dichotomy (e.g., Bjornsson et al., 2011; Davidson et al., 2004). Those studies that tested for the effects of ethnicity found that it did not significantly predict treatment outcomes (Davidson et al., 2004; Hofmann, 2004). Again, this indicates that racial and ethnic background may not affect treatment or may affect it in ways that can be effectively accounted for within conventional protocols. These studies have small samples of ethnic and racial minorities and suffer from even greater statistical power issues than the gender comparisons.

There is a fair amount of evidence for the transportability of these treatments across nationalities. Exposure-based CBT has been shown to be effective in the treatment of SAD in trials in Great Britain (e.g., Clark et al., 2003), Australia (e.g., Mattick, Peter, & Clarke, 1989), Germany (e.g., Stangier, Schramm, Heidenreich, Berger, & Clark, 2011), Sweden (e.g., Blomhoff et al., 2001), Norway (e.g., Blomhoff et al., 2001), the Netherlands (e.g., Oosterbaan et al., 2001), and Spain (e.g., Salaberria &

Table 11.2 Sample Diversity and Differential Outcomes in Randomized Controlled Trials of *Cognitive-Behavioral* Therapy

Study	*Methodology*	*Participants*	*Treatment outcome by subgroup*
Blomhoff et al. (2001)	ET vs. sertraline vs. ET + sertraline	$N = 387$; 60.5% women	No interaction between gender and outcome
Clark et al. (2003)	CT vs. fluoxetine + SE vs. placebo vs. SE	$N = 60$; no demographics reported	Gender was not a predictor of treatment response
Davidson et al. (2004)	CBT + social skills vs. CBT + social skills + fluoxetine vs. CBT + social skills + placebo	$N = 295$; 47.1 % women; 22.8% nonwhite	Sex and ethnicity had no significant effect on treatment outcome
Hofmann (2004)	CBGT vs. ET vs. waitlist	$N = 90$; 46% women; 10% nonwhite	Race and gender did not affect attrition
Oosterbaan et al. (2001)	CT vs. moclobemide vs. placebo	$N = 82$; 42% women	No gender differences between completers and noncompleters
Otto et al. (2000)	CBGT vs. clonazepam	$N = 45$; 40.0% women	Gender did not predict treatment outcome
Pharmacotherapy trials			
Stein et al. (1998)	Paroxetine vs. placebo	57% women; 81% white, 12% African American, 2% Asian, 5% other	No main effect for gender
Davidson et al. (2004)	Fluvoxamine CR vs. placebo	36% women; 78% Caucasian, 8% black, 4% Asian, 6% Hispanic, <1% American Indian, 4% other	Report that neither race nor gender were determinants of outcome but no analyses reported
Kasper, Stein, Loft, and Nil (2005)	Escitalopram vs. placebo	45% women; race/ethnicity not reported	Gender did not interact with outcome
Lader, Stender, Bürger, and Nil (2004)	Escitalopram vs. placebo	53% women; 99% Caucasian	Gender did not interact with outcome

(*continued*)

Table 11.2 (*Continued*)

Study	*Methodology*	*Participants*	*Treatment outcome by subgroup*
Liebowitz, Mangano, Bradwejn, and Asnis (2005)	Venlafaxine vs. placebo	45% women; 80% white, 6% black, 6% Hispanic, 5% Asian, 3% other	No demographic differences between remitters and nonremitters
Stein, Kasper, Andersen, Nil, and Lader (2004)	Two combined trials of escitalopram	50.9% women; race/ethnicity not reported	Gender did not predict outcome
Stein, Stein, Pitts, Kumar, and Hunter (2002)	Three combined trials of paroxetine vs. placebo	Not reported	Gender did not predict outcome
Van Ameringen et al. (2001)	Sertraline vs. placebo	44% women; 93% Caucasian, 1% African American, 3% Asian, 2% other	No gender main effect or interaction

Note: Racial/ethnic groupings listed in this table reflect the terminology used in the individual study. ET, exposure therapy; CT, cognitive therapy; CBT, cognitive behavioral therapy; CBGT, cognitive behavioral group therapy.

Echeburua, 1998). While these studies all come from Western cultures, there has recently been some research indicating that CBT may be effective in treating TKS, the East Asian variant of SAD (Chen et al., 2007). Chen et al. (2007) conducted an open trial of cognitive behavioral group therapy (CBGT) for SAD in Japan, and found effect sizes comparable to those in most Western trials. These studies bolster the evidence that CBT for SAD may be broadly effective across cultural boundaries. They are, however, studies conducted with individuals within their own cultural context and may not be representative of the experiences of cultural minorities.

While it is likely that some LGB individuals were present in the samples for these RCTs, sexual orientation was not recorded/reported as a demographic variable in any study. While this is not surprising, given that reporting participant sexual orientation is not standard in research, it is a highly relevant concern given the high SAD prevalence rates and unique risk factors that LGB individuals possess.

The limited available evidence indicates that the variability of diverse populations can likely be accounted for within the framework of exposure-based treatments. There is, however, a paucity of research examining how cultural diversity might influence treatment outcomes. In part, this is likely driven by the small sample sizes of most

treatment studies, but it is also the result of the failure to report these data in most research studies. While the reporting of these data does seem to be increasing in the literature, the sample sizes remain too small to adequately test for group differences.

Clinical Pharmacotherapy Trials

The frontline pharmacotherapy treatments for SAD are generally considered to be selective serotonin reuptake inhibitors (SSRI) or serotonin and norepinephrine reuptake inhibitors (SNRI; e.g., Ravindran & Stein, 2010; see also **Chapter 24**). Focusing on these two drug classes, a review of the literature returned 18 SSRI RCTs and 5 SNRI RCTs. Similar to trials of CBT, there is little evidence for differential outcomes across gender, sexual orientation, or race/ethnicity in pharmacological trials for SAD, but the presence of these effects are seldom tested within the research. Details of the studies that reported analyses by these demographic variables appear in Table 11.2. The results appear to indicate that gender does not predict treatment outcomes in drug trials (e.g., Stein et al., 2002, 2004; van Ameringen et al., 2001). There is less information regarding race or ethnicity effects; with only one study (Davidson et al., 2004) reporting that race did not determine outcome but failing to report the relevant statistics, this study also included psychotherapy conditions, so the results are even less clear. The lack of racial effects is supported, however, by studies indicating that medication can be effective in cross-cultural treatment, including treatment of TKS (e.g., Stein, 2009).

On the other hand, a meta-analysis that examined ethnic minority effects in response to paroxetine (an SSRI) treatment across anxiety disorders, including SAD, found differential response rates by ethnic group (Roy-Byrne, Perera, Pitts, & Christi, 2005). In particular, response rates were lower for participants of Hispanic or Asian background. This is especially relevant for SAD because paroxetine is indicated for its treatment (e.g., Ravindran & Stein, 2010). Potentially, the racial/ethnic effects in this meta-analysis are due to disorders other than SAD; however, it is notable that both the sample size and diversity of the sample were greater in this study than in individual SAD RCTs. It is not known whether similar effects are present with other medications, including even other SSRIs.

Single Case CBT Studies

While they cannot be taken as conclusive evidence of the efficacy of CBT for SAD with diverse populations, case studies provide useful insight into the utility of these treatments and the ways in which they may be adapted to meet varying client needs. Fink, Turner, and Beidel (1996) reported decreases in scores on a self-report measure of social anxiety when using CBT to treat an African American woman for SAD whose anxiety was provoked by interacting with European American men. Both imaginal and *in vivo* exposures were used. Weiss, Singh, and Hope (2010) reported using individual CBT for two immigrants to the United States—a man from Central America and a woman from China. Both reported decreases in self-report measures and hierarchy ratings at the end of treatment. Social norms from the country of origin influenced

therapist–client interactions, and both clients reported that cognitive work, especially later in treatment, was more effective when done in the language of origin.

Staley and Lawyer (2010) explicitly addressed the ethnic identity concerns in the treatment of a biracial Japanese/European American man for comorbid SAD and major depressive disorder. Combined exposure and cognitive restructuring for SAD and behavioral activation for depression (Lejuez, Hopko, & Hopko, 2001) were used concurrently to treat his symptoms, with some cognitive work to address his racial identity issues by integrating them with his broader identity (e.g., gender, age). They reported improvement across multiple self-report measures of depression and social anxiety.

Although there is no information on the treatment of sexual minorities from controlled trials, two case studies are encouraging. Walsh and Hope (2010) discussed the treatment of a socially anxious man in the process of identifying as gay. Once the client identified as gay to himself, therapy focused on the process of disclosing his sexual orientation to others, addressing increased rejection sensitivity, and integrating his identity as a gay man with the rest of his life. Interestingly, they used tools from traditional CBT for SAD, but also incorporated realistic assessments of interpersonal risk (e.g., social rejection) associated with LGB status. At least one other case study found the process of disclosing sexual orientation relevant to anxiety treatment (Glassgold, 2009).

As a whole, these case studies seem to indicate that CBT for SAD is effective in addressing anxiety in various minority groups. Across groups, it appears that generally more attention may be paid to the integration of minority identity with other aspects of the client's broader identity if it is relevant to their fear content. The case studies suggest this may require a less directive approach than may be taken in more traditional CBT, where decreasing avoidance and facing all feared situations is typically an overarching goal of treatment.

Case Conceptualization

Evidence-based practice guidelines (EBP; APA, 2006) call for the integration of client characteristics, including both preferences and diversity issues, along with research evidence and the judgment of the clinician in case conceptualization, assessment, and treatment planning. This EBP approach requires the integration of nomothetic data and idiographic information. Doing this requires that clinicians have competency in relevant areas of diversity when working with clients whose race/ethnicity, gender, or sexual orientation may differ from the therapist. *Diversity competency* has been conceptualized as both therapist self-awareness of their own biases and knowledge concerning diverse groups (Daniel, Roysircar, Abeles, & Boyd, 2004). The actual content of this knowledge is beyond the scope of the present chapter, but there are a number of publications and books about working with individual minority groups, including understanding the role of acculturation for recent immigrants (e.g., Bernal, Trimble, Burlew, & Leong, 2003; Martell, Safren, & Prince, 2004).

When integrating the nomethetic and ideographic information about a client's background into a case conceptualization, Moradi's (2013) model of minority stress as

applied to SAD may be a useful framework. Following this model, social anxiety among diverse clients may involve both cognizance- and internalization-based surveillance and adaptive and maladaptive anxiety. Cognizance-based surveillance is conceptually similar to more superficial automatic thoughts (e.g., Beck, 2011) that are based on judgments of the likelihood and severity of the consequences of negative evaluation (e.g., "My interviewer will realize I'm gay and I won't get the job"). Similar to superficial automatic thoughts, cognizance-based surveillance also varies in terms of accuracy and how adaptive it is. Internalization-based surveillance may be seen as more inherently maladaptive, as it involves the internalization of negative societal views, and may be conceptually analogous to core beliefs or schemata in cognitive therapy (e.g., "My accent makes me different and I'll never really be as competent or get the best opportunities"). This information is relevant to both conceptualizing the case properly and determining the targets for intervention as therapy progresses.

The direct relationship of diversity to case conceptualization may vary substantially from individual to individual but may broadly represent factors in three related areas: distal factors relevant to etiology of SAD, those factors proximal to current social concerns, and/or factors relevant to the sociocultural context of SAD. In terms of distal factors, peer victimization and isolation are thought to play an etiological role in SAD. In this sense, individuals may experience discrimination as a result of their minority status, which may contribute to the development of SAD, regardless of whether it is relevant to current social concerns. Diversity may also play a distal role in development of SAD by shaping an individual's view of the social world while not directly contributing to pathology. A man raised by East Asian immigrants, for instance, may develop an interdependent self-construal that affects his later development of SAD and its symptoms. There is variability in how relevant these factors are to individual cases, however, and it is important to note that, for many minority individuals, their cultural background will not be relevant and/or they will not have experienced discrimination.

Diversity may also play a more proximal role in shaping some aspects of a client's social fears. For some clients, important variation in these fears may be due to concern about interaction with majority group members, resulting from cognizance- and/or internalization-based surveillance. This surveillance may result in behavioral changes that would otherwise be conceptualized as subtle avoidance strategies such as the alteration of mannerisms, speech patterns, or the content of speech related to the minority identity. For LGB individuals or individuals with other concealable identities, this may result in the overt concealment of the minority identity. In conceptualizing the relationship of these proximal factors to SAD, it is important for the clinician to discuss the role of these fears with the client and to consider whether they are adaptive in certain contexts. The potential complexity of these interactions means that it is especially important to determine the therapeutic targets on a collaborative basis.

Diversity may also be seen as a component of the selection of treatment goals. A client's cultural identity and environment may determine social norms and the socially appropriate target situations for intervention. In this sense, diversity may represent the bounds of what thoughts and behaviors are considered pathological. For instance, clients with an East Asian background may not be inclined to make direct eye contact as the result of cultural norms, rather than the avoidance of anxiety. This contextual element extends beyond the traditional bounds of diversity (e.g., race/ethnicity,

gender) and may include broader categories such as social class, religious affiliation, education, and any other dimension on which individuals may vary.

It is also important to recognize that the demographic categories associated with diversity serve as proxies for individual differences associated with them. Understanding these underlying differences may still be important in understanding clients who do not fit under even the broad definition of diversity. This is because the variables that diversity categories are proxies for (e.g., self-construal, concealment of stigma) are likely to vary substantially within both majority and minority groups. These constructs may then be highly relevant to some members of the majority group and irrelevant to some members of the minority group.

Furthermore, diversity in the context of most discussions is artificially narrowed in scope to race, gender, ethnicity, sexual orientation, and culture. Individuals who are members of a religious minority (e.g., Islam, Jehovah's Witnesses) likely face stressors that are both unique to their own subculture and similar to those of other marginalized groups. Equally as important may be conceptualizing the role that economic class and education play, particularly when one considers that treatments are created by individuals who are highly educated and represent limited economic diversity. Taken to their logical conclusion, these same theoretical frameworks used to understand diversity may be informative for almost any client within individualized case formulations (e.g., Persons, 1989). This practice of integrating minority stress models with CBT models of SAD may prove beneficial, because it helps to integrate the idiographic issues inherent to conceptualization with the nomothetic research base.

Conclusion

Despite the inherent role that social context plays in understanding social anxiety, there is surprisingly little clinical or research literature on cultural diversity and SAD. The literature that does exist, however, demonstrates the importance of culture on the presentation of social anxiety, including related culture-bound phenomena and the influence of the threat of discrimination on where one draws a boundary between realistic fears and pathological social anxiety. Moradi's (2013) pantheoretical model offers surprising parallels to prominent theoretical models of SAD, and integration of these models may help to conceptualize cases within a multicultural context. There is little evidence to date of differential effectiveness across diverse groups for the first-line treatments for SAD, but this literature is quite limited and studies are often underpowered to detect differences. Similar problems are seen in the assessment literature. More research with diverse samples is clearly needed.

The basic synthesis is that group membership may importantly shape the ways that individuals see themselves as social actors, their understanding of and relationship to social norms, and their experiences with other groups. These individual differences are then relevant to understanding the treatment of SAD because they may shape the severity, expression, and form of an individual's fears. In some situations, these fears of negative evaluation may be warranted. Appropriately conceptualizing and treating a specific case involves understanding these factors, understanding models of minority stress and SAD, and integrating them into an individualized treatment plan. These

same factors, which are so salient for minority groups in the literature, and this same approach to conceptualization and treatment may prove beneficial in the treatment of majority clients, particularly when the scope of diversity and individual differences are considered more broadly.

References

Acarturk, C. C., Cuijpers, P. P., van Straten, A. A., & de Graaf, R. R. (2009). Psychological treatment of social anxiety disorder: A meta-analysis. *Psychological Medicine, 39*(2), 241–254. doi:10.1017/S0033291708003590

American Psychological Association. (2006). Evidence-based practice in psychology. *American Psychologist, 61*(4), 271–285. doi:10.1037/0003-066X.61.4.271

Asnaani, A., Richey, J., Dimaite, R., Hinton, D. E., & Hofmann, S. G. (2010). A cross-ethnic comparison of lifetime prevalence rates of anxiety disorders. *Journal of Nervous and Mental Disease, 198*(8), 551–555. doi:10.1097/NMD.0b013e3181ea169f

Beard, C., Rodriguez, B. F., Moitra, E., Sibrava, N. J., Bjornsson, A., Weisberg, R. B., & Keller, M. B. (2011). Psychometric properties of the Liebowitz Social Anxiety Scale (LSAS) in a longitudinal study of African Americans with anxiety disorders. *Journal of Anxiety Disorders, 25*(5), 722–726. doi:10.1016/j.janxdis.2011.03.009

Beard, C., Rodriguez, B. F., Weisberg, R. B., Perry, A., & Keller, M. B. (2012). Psychometric properties of the Liebowitz Social Anxiety Scale in a longitudinal study of Latinos with anxiety disorders. *Hispanic Journal of Behavioral Sciences, 34*(2), 269–278. doi:10.1177/0739986312436660

Beck, J. S. (2011). *Cognitive behavior therapy: Basics and beyond* (2nd ed.). New York, NY: Guilford Press.

Belle, D., & Doucet, J. (2003). Poverty, inequality, and discrimination as sources of depression among U.S. women. *Psychology of Women Quarterly, 27*(2), 101–113. doi:10.1111/1471-6402.00090

Bernal, G., Trimble, J. E., Burlew, A. K., & Leong, F. T. L. (Eds.). (2003). *Handbook of racial and ethnic minority psychology.* Thousand Oaks, CA: Sage.

Bjornsson, A. S., Bidwell, L., Brosse, A. L., Carey, G., Hauser, M., Mackiewicz Seghete, K. L., . . . Craighead, W. (2011). Cognitive-behavioral group therapy versus group psychotherapy for social anxiety disorder among college students: A randomized controlled trial. *Depression and Anxiety, 28*(11), 1034–1042. doi:10.1002/da.20877

Blomhoff, S., Haug, T. T., Hellstrom, K., Holme, I., Humble, M., Madsbu, H. P., & Wold, J. E. (2001). Randomised controlled general practice trial of sertraline, exposure therapy and combined treatment in generalized social phobia. *British Journal of Psychiatry, 179*, 23–30. doi:10.1192/bjp.179.1.23

Bolino, M. C., Kacmar, K., Turnley, W. H., & Gilstrap, J. (2008). A multi-level review of impression management motives and behaviors. *Journal of Management, 34*(6), 1080–1109. doi:10.1177/0149206308324325

Borrell, L. N., Jacobs, D. R., Williams, D. R., Pletcher, M. J., Houston, T. K., & Kiefe, C. I. (2007). Self-reported racial discrimination and substance use in the Coronary Artery Risk Development in Adults Study. *American Journal of Epidemiology, 166*(9), 1068–1079. doi:10.1093/aje/kwm180

Bostwick, W. B., Boyd, C. J., Hughes, T. L., & McCabe, S. (2010). Dimensions of sexual orientation and the prevalence of mood and anxiety disorders in the United States. *American Journal of Public Health, 100*(3), 468–475. doi:10.2105/AJPH.2008.152942

Buchanan, T. S., Fischer, A. R., Tokar, D. M., & Yoder, J. D. (2008). Testing a culture-specific extension of objectification theory regarding African American women's body image. *The Counseling Psychologist, 36*(5), 697–718. doi:10.1177/0011000008316322

Chen, J., Nakano, Y., Ietzugu, T., Ogawa, S., Funayama, T., Watanabe, N., . . . Furukawa, T. A. (2007). Group cognitive behavior therapy for Japanese patients with social anxiety disorder: Preliminary outcomes and their predictors. *BMC Psychiatry, 7*, 1–10. doi:10.1186/1471-244X-7-69

Choy, Y., Schneier, F. R., Heimberg, R. G., Oh, K., & Liebowitz, M. R. (2008). Features of the offensive subtype of Taijin-Kyofu-Sho in US and Korean patients with DSM-IV social anxiety disorder. *Depression and Anxiety, 25*(3), 230–240. doi:10.1002/da.20295

Clark, D. M., Ehlers, A., McManus, F., Hackmann, A., Fennell, M., Campbell, H., . . . Louis, B. (2003). Cognitive therapy versus fluoxetine in generalized social phobia: A randomized placebo-controlled trial. *Journal of Consulting and Clinical Psychology, 71*(6), 1058–1067. doi:10.1037/0022-006X.71.6.1058

Clark, R., Anderson, N. B., Clark, V. R., & Williams, D. R. (1999). Racism as a stressor for African Americans: A biopsychosocial model. *American Psychologist, 54*(10), 805–816. doi:10.1037/0003-066X.54.10.805

Cross, S. E. (1995). Self-construals, coping, and stress in cross-cultural adaptation. *Journal of Cross-Cultural Psychology, 26*(6), 673–697. doi:10.1177/0022022195026006105

Cross, S. E., & Madson, L. (1997). Models of the self: Self-construals and gender. *Psychological Bulletin, 122*(1), 5–37. doi:10.1037/0033-2909.122.1.5

Daniel, J. H., Roysircar, G., Abeles, N., & Boyd, C. (2004). Individual and cultural-diversity competency: Focus on the therapist. *Journal of Clinical Psychology, 60*, 755–770. doi:10.1002/jclp.20014

D'Augelli, A. R. (1992). Lesbian and gay male undergraduates' experiences of harassment and fear on campus. *Journal of Interpersonal Violence, 7*(3), 383–395. doi:10.1177/088626092007003007

Davidson, J., Yaryura-Tobias, J., DuPont, R., Stallings, L., Barbato, L. M., van der Hoop, R., & Li, D. (2004). Fluvoxamine-controlled release formulation for the treatment of generalized social anxiety disorder. *Journal of Clinical Psychopharmacology, 24*(2), 118–125. doi:10.1097/01.jcp.0000106222.36344.96

Feske, U., & Chambless, D. L. (1995). Cognitive behavioral versus exposure only treatment for social phobia: A meta-analysis. *Behavior Therapy, 26*(4), 695–720. doi:10.1016/S0005-7894(05)80040-1

Fink, C. M., Turner, S. M., & Beidel, D. C. (1996). Culturally relevant factors in the behavioral treatment of social phobia: A case study. *Journal of Anxiety Disorders, 10*, 201–209.

Gillis, M. M., Haaga, D. F., & Ford, G. T. (1995). Normative values for the Beck Anxiety Inventory, Fear Questionnaire, Penn State Worry Questionnaire, and Social Phobia and Anxiety Inventory. *Psychological Assessment, 7*(4), 450–455. doi:10.1037/1040-3590.7.4.450

Glassgold, J. M. (2009). The case of Felix: An example of gay-affirmative, cognitive-behavioral therapy. *Pragmatic Case Studies in Psychotherapy, 5*(4), 1–21.

Grant, B. F., Hasin, D. S., Blanco, C., Stinson, F. S., Chou, S., Goldstein, R. B., . . . Huang, B. (2005). The epidemiology of social anxiety disorder in the United States: Results from the National Epidemiologic Survey on alcohol and related conditions. *Journal of Clinical Psychiatry, 66*(11), 1351–1361. doi:10.4088/JCP.v66n1102

Greenberg, D., Stravynski, A., & Bilu, Y. (2004). Social phobia in ultra-orthodox Jewish males: Culture-bound syndrome or virtue? *Mental Health, Religion & Culture, 7*(4), 289–305. doi:10.1080/13674670310001606496

Hambrick, J. P., Rodebaugh, T. L., Balsis, S., Woods, C. M., Mendez, J. L., & Heimberg, R. G. (2010). Cross-ethnic measurement equivalence of measures of depression, social anxiety, and worry. *Assessment, 17*(2), 155–171. doi:10.1177/1073191109350158

Heinrichs, N., Rapee, R. M., Alden, L. A., Bögels, S., Hofmann, S. G., Oh, K., & Sakano, Y. (2006). Cultural differences in perceived social norms and social anxiety. *Behaviour Research and Therapy, 44*(8), 1187–1197. doi:10.1016/j.brat.2005.09.006

Herek, G. M. (2009). Hate crimes and stigma-related experiences among sexual minority adults in the United States: Prevalence estimates from a national probability sample. *Journal of Interpersonal Violence, 24*(1), 54–74. doi:10.1177/0886260508316477

Hofmann, S. G. (2004). Cognitive mediation of treatment change in social phobia. *Journal of Consulting and Clinical Psychology, 72*(3), 392–399. doi:10.1037/0022-006X.72.3.392

Hofmann, S. G., & Smits, J. J. (2008). Cognitive-behavioral therapy for adult anxiety disorders: A meta-analysis of randomized placebo-controlled trials. *Journal of Clinical Psychiatry, 69*(4), 621–632. doi:10.4088/JCP.v69n0415

Hoover, E. L. (2007). There is no scientific rationale for race-based research. *Journal of the National Medical Association, 99*, 690–692.

Hope, D. A., Heimberg, R. G., & Turk, C. L. (2010). *Managing social anxiety: A cognitive-behavioral therapy: Therapist guide* (2nd ed.). New York, NY: Oxford University Press.

Hsu, L., Woody, S. R., Lee, H., Peng, Y., Zhou, X., & Ryder, A. G. (2012). Social anxiety among East Asians in North America: East Asian socialization or the challenge of acculturation? *Cultural Diversity and Ethnic Minority Psychology, 18*(2), 181–191. doi:10.1037/a0027690

Kasper, S., Stein, D. J., Loft, H., & Nil, R. (2005). Escitalopram in the treatment of social anxiety disorder: Randomised, placebo-controlled, flexible-dosage study. *The British Journal of Psychiatry, 186*(3), 222–226. doi:10.1192/bjp.186.3.222

Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C. B., Hughes, M., Eshleman, S., … Kendler, K. (1994). Lifetime and 12-month prevalence of DSM-III—R psychiatric disorders in the United States: Results from the National Comorbidity Study. *Archives of General Psychiatry, 51*(1), 8–19. doi:10.1001/archpsyc.1994.03950010008002

Kim, J., Rapee, R. M., & Gaston, J. E. (2008). Symptoms of offensive type Taijin-Kyomsho among Australian social phobics. *Depression and Anxiety, 25*(7), 601–608. doi:10.1002/da.20345

Kitayama, S., Markus, H., Matsumoto, H., & Norasakkunkit, V. (1997). Individual and collective processes in the construction of the self: Self-enhancement in the United States and self-criticism in Japan. *Journal of Personality and Social Psychology, 72*(6), 1245–1267. doi:10.1037/0022-3514.72.6.1245

Lader, M., Stender, K., Bürger, V., & Nil, R. (2004). Efficacy and tolerability of escitalopram in 12- and 24-week treatment of social anxiety disorder: Randomised, double-blind, placebo-controlled, fixed-dose study. *Depression and Anxiety, 19*(4), 241–248. doi:10.1002/da.20014

Lejuez, C. W., Hopko, D. R., & Hopko, S. D. (2001). A brief behavioral activation treatment for depression: Treatment manual. *Behavior Modification, 25*(2), 255–286. doi:10.1177/0145445501252005

Liebowitz, M.R. (1987). Social phobia. *Modern Problems of Pharmacopsychiatry, 22*, 141–173.

Liebowitz, M. R., Mangano, R. M., Bradwejn, J., & Asnis, G. (2005). A randomized controlled trial of venlafaxine extended release in generalized social anxiety disorder. *Journal of Clinical Psychiatry, 66*(2), 238–247. doi:10.4088/JCP.v66n0213

Markus, H. R., & Kitayama, S. (1991). Culture and the self: Implications for cognition, emotion, and motivation. *Psychological Review, 98*(2), 224–253. doi:10.1037/0033-295X.98.2.224

Martell, C. R., Safren, S. A., & Prince, S. E. (2004). *Cognitive-behavioral therapies with lesbian, gay, and bisexual clients.* New York, NY: Guilford Press.

Mattick, R. P., & Clarke, J. (1998). Development and validation of measures of social phobia scrutiny fear and social interaction anxiety. *Behaviour Research and Therapy, 36*(4), 455–470. doi:10.1016/S0005-7967(97)10031-6

Mattick, R. P., Peters, L., & Clarke, J. (1989). Exposure and cognitive restructuring for social phobia: A controlled study. *Behavior Therapy, 20*(1), 3–23. doi:10.1016/S0005-7894(89)80115-7

Melka, S. E., Lancaster, S. L., Adams, L. J., Howarth, E. A., & Rodriguez, B. F. (2010). Social anxiety across ethnicity: A confirmatory factor analysis of the FNE and SAD. *Journal of Anxiety Disorders, 24*(7), 680–685. doi:10.1016/j.janxdis.2010.04.011

Meyer, I. H. (2003). Prejudice, social stress, and mental health in lesbian, gay, and bisexual populations: Conceptual issues and research evidence. *Psychological Bulletin, 129*(5), 674–697. doi:10.1037/0033-2909.129.5.674

Moradi, B. (2013). Discrimination, objectification, and dehumanization: Toward a pantheoretical framework. In S. J. Gervais (Ed.), *Objectification and (de)humanization*. New York, NY: Springer.

Moscovitch, D. A., Hofmann, S. G., & Litz, B. T. (2005). The impact of self-construals on social anxiety: A gender-specific interaction. *Personality and Individual Differences, 38*(3), 659–672. doi:10.1016/j.paid.2004.05.021

Muehlenhard, C., & Peterson, Z. (2011). Distinguishing between sex and gender: History, current conceptualizations, and implications. *Sex Roles, 64*, 791–803. doi: 10.1007/s11199-011-9932-5

Norasakkunkit, V., Kitayama, S., & Uchida, Y. (2012). Social anxiety and holistic cognition: Self-focused social anxiety in the United States and other-focused social anxiety in Japan. *Journal of Cross-Cultural Psychology, 43*(5), 742–757. doi:10.1177/0022022111405658

Norton, P. J., & Weeks, J. W. (2009). A multi-ethnic examination of socioevaluative fears. *Journal of Anxiety Disorders, 23*(7), 904–908. doi:10.1016/j.janxdis.2009.05.008

Ogbu, J. U. (2004). Collective identity and the burden of "acting White" in Black history, community, and education. *The Urban Review, 36*(1), 1–35.

Okazaki, S. (1997). Sources of ethnic differences between Asian American and White American college students on measures of depression and social anxiety. *Journal of Abnormal Psychology, 106*(1), 52–60. doi:10.1037/0021-843X.106.1.52

Okazaki, S. (2002). Self-other agreement on affective distress scales in Asian Americans and White Americans. *Journal of Counseling Psychology, 49*(4), 428–437. doi:10.1037/0022-0167.49.4.428

Okazaki, S., Liu, J. F., Longworth, S. L., & Minn, J. Y. (2002). Asian American–White American differences in expressions of social anxiety: A replication and extension. *Cultural Diversity and Ethnic Minority Psychology, 8*(3), 234–247. doi:10.1037/1099-9809.8.3.234

Olivares, J., García-López, L., & Hidalgo, M. (2001). The Social Phobia Scale and the Social Interaction Anxiety Scale: Factor structure and reliability in a Spanish-speaking population. *Journal of Psychoeducational Assessment, 19*(1), 69–80. doi:10.1177/073428290101900105

Oosterbaan, D. B., van Balkom, A. M., Spinhoven, P., van Oppen, P., & van Dyck, R. (2001). Cognitive therapy versus moclobemide in social phobia: A controlled study. *Clinical Psychology & Psychotherapy, 8*(4), 263–273. doi:10.1002/cpp.291

Otto, M. W., Pollack, M. H., Gould, R. A., Worthington, J., McArdle, E. T., Rosenbaum, J. F., & Heimberg, R. G. (2000). A comparison of the efficacy of clonazepam and cognitive-behavioral group therapy for the treatment of social phobia. *Journal of Anxiety Disorders, 14*(4), 345–358. doi:10.1016/S0887-6185(00)00027-X

Pachankis, J. E. (2007). The psychological implications of concealing a stigma: A cognitive-affective-behavioral model. *Psychological Bulletin, 133*(2), 328–345. doi:10.1037/0033-2909.133.2.328

Pachankis, J. E., & Goldfried, M. R. (2006). Social anxiety in young gay men. *Journal of Anxiety Disorders, 20*(8), 996–1015. doi:10.1016/j.janxdis.2006.01.001

Pérez, D., Fortuna, L., & Alegria, M. (2008). Prevalence and correlates of everyday discrimination among U.S. Latinos. *Journal of Community Psychology, 36*(4), 421–433. doi:10.1002/jcop.20221

Persons, J. B. (1989). *Cognitive therapy in practice: A case formulation approach.* New York, NY: W. W. Norton & Co.

Potoczniak, D. J., Aldea, M. A., & DeBlaere, C. (2007). Ego identity, social anxiety, social support, and self-concealment in lesbian, gay, and bisexual individuals. *Journal of Counseling Psychology, 54*(4), 447–457. doi:10.1037/0022-0167.54.4.447

Rapee, R. M., & Heimberg, R. G. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy, 35*(8), 741–756. doi:10.1016/S0005-7967(97)00022-3

Rapee, R. M., Sanderson, W. C., & Barlow, D. H. (1988). Social phobia features across the DSM-III—R anxiety disorders. *Journal of Psychopathology and Behavioral Assessment, 10*(3), 287–299. doi:10.1007/BF00962552

Ravindran, L. N., & Stein, M. B. (2010). The pharmacological treatment of anxiety disorders: A review of progress. *Journal of Clinical Psychiatry, 71*(7), 839–854. doi:10.4088/JCP.10r06218blu

Rodebaugh, T. L., Holaway, R. M., & Heimberg, R. G. (2004). The treatment of social anxiety disorder. *Clinical Psychology Review, 24*(7), 883–908. doi:10.1016/j.cpr.2004.07.007

Roy-Byrne, P. P., Perera, P., Pitts, C. D., & Christi, J. A. (2005). Paroxetine response and tolerability among ethnic minority patients with mood or anxiety disorders: A pooled analysis. *Journal of Clinical Psychiatry, 66*, 1228–1233.

Safren, S. A., & Pantalone, D. W. (2006). Social anxiety and barriers to resilience among lesbian, gay, and bisexual adolescents. In A. M. Omoto & H. S. Kurtzman (Eds.), *Sexual orientation and mental health: Examining identity and development in lesbian, gay, and bisexual people* (pp. 55–71). Washington, DC: American Psychological Association. doi:10.1037/11261-003

Salaberria, K., & Echeburua, E. (1998). Long-term outcome of cognitive therapy's contribution to self-exposure *in vivo* to the treatment of generalized social phobia. *Behavior Modification, 22*, 262–284. doi:10.1177/01454455980223003

Schreier, S., Heinrichs, N., Alden, L., Rapee, R. M., Hofmann, S. G., Chen, J., … Bögels, S. (2010). Social anxiety and social norms in individualistic and collectivistic countries. *Depression and Anxiety, 27*(12), 1128–1134. doi:10.1002/da.20746

Sell, R. L. (1997). Defining and measuring sexual orientation: A review. *Archives of Sexual Behavior, 26*(6), 643–658. doi:10.1023/A:1024528427013

Singelis, T. M., & Sharkey, W. F. (1995). Culture, self-construal, and embarrassability. *Journal of Cross-Cultural Psychology, 26*(6), 622–644. doi:10.1177/002202219502600607

Staley, C. S., & Lawyer, S. R. (2010). Behavioral activation and CBT as an intervention for coexistent major depression and social phobia for a biracial client with diabetes. *Clinical Case Studies, 9*, 63–73. doi:10.1177/1534650109355187

Stangier, U., Schramm, E., Heidenreich, T., Berger, M., & Clark, D. M. (2011). Cognitive therapy vs interpersonal psychotherapy in social anxiety disorder: A randomized controlled trial. *Archives of General Psychiatry, 68*(7), 692–700. doi:10.1001/archgenpsychiatry.2011.67

Stein, D. J. (2009). Social anxiety disorder in the West and in the East. *Annals of Clinical Psychiatry, 21*(2), 109–117.

Stein, D. J., Kasper, S., Andersen, E., Nil, R., & Lader, M. (2004). Escitalopram in the treatment of social anxiety disorder: Analysis of efficacy for different clinical subgroups and symptom dimensions. *Depression and Anxiety, 20*(4), 175–181. doi:10.1002/da.20043

Stein, D. J., Stein, M. B., Pitts, C. D., Kumar, R., & Hunter, B. (2002). Predictors of response to pharmacotherapy in social anxiety disorder: An analysis of 3 placebo-controlled paroxetine trials. *Journal of Clinical Psychiatry, 63*(2), 152–155. doi:10.4088/JCP.v63n0211

Stein, M. B., Liebowitz, M. R., Lydiard, R. B., Pitts, C. D., Bushnell, W., & Gergel, I. (1998). Paroxetine treatment of generalized social phobia (social anxiety disorder). *Journal of the American Medical Association, 280*, 708–713.

Takahashi, T. (1989). Social phobia syndrome in Japan. *Comprehensive Psychiatry, 30*(1), 45–52. doi:10.1016/0010-440X(89)90117-X

Van Ameringen, M. A., Lane, R. M., Walker, J. R., Bowen, R. C., Chokka, P. R., Goldner, E. M., … Swinson, R. P. (2001). Sertraline treatment of generalized social phobia: A 20-week, double-blind, placebo-controlled study. *American Journal of Psychiatry, 158*, 275–281.

Vohs, K. D., Baumeister, R. F., & Ciarocco, N. J. (2005). Self-regulation and self-presentation: Regulatory resource depletion impairs impression management and effortful self-presentation depletes regulatory resources. *Journal of Personality and Social Psychology, 88*(4), 632–657. doi:10.1037/0022-3514.88.4.632

Walsh, K., & Hope, D. A. (2010). LGB-affirmative cognitive behavioral treatment for social anxiety: A case study applying evidence-based practice principles. *Cognitive and Behavioral Practice, 17*, 56–65. doi:10.1016/j.cbpra.2009.08.002

Watson, D., & Friend, R. (1969). Measurement of social-evaluative anxiety. *Journal of Consulting and Clinical Psychology, 33*(4), 448–457. doi:10.1037/h0027806

Weeks, J. W., Heimberg, R. G., & Rodebaugh, T. L. (2008). The Fear of Positive Evaluation: Assessing a proposed cognitive component of social anxiety. *Journal of Anxiety Disorders, 22*, 44–55. doi:10.1016/j.janxdis.2007.08.002

Weeks, J. W., Heimberg, R. G., Rodebaugh, T. L., & Norton, P. J. (2008). Exploring the relationship between fear of positive evaluation and social anxiety. *Journal of Anxiety Disorders, 22*(3), 386–400. doi:10.1016/j.janxdis.2007.04.009

Weiss, B. J., Hope, D. A., & Capozzoli, M. C. (2012). Heterocentric language in commonly used measures of social anxiety: Recommended alternate wording. *Behavior Therapy, 44*, 1–11. doi:10.1016/j.beth.2012.07.006

Weiss, B. J., Singh, J. S., & Hope, D. A. (2011). Cognitive behavioral therapy for immigrants presenting with social anxiety disorder: Two case studies. *Clinical Case Studies, 10*, 324–342. doi:10.1177/1534650111420706

12

Heterogeneity Within Social Anxiety Disorder

Megan E. Spokas and LeeAnn Cardaciotto
La Salle University, USA

Like many psychological disorders, social anxiety disorder (SAD) is marked by heterogeneity. Individuals with SAD can present quite differently in terms of the number and type of feared situations, focus of their fears, associated personality traits, and/or the domains of functional impairment. Failing to consider such heterogeneity may lead to inaccurate assumptions among clinicians, and may potentially influence treatment efficacy. Furthermore, better understanding dimensions of heterogeneity may shed light on the numerous etiological pathways to SAD. The purpose of this chapter is to review a number of dimensions that reflect heterogeneity within SAD. The presentation of SAD in understudied contexts and groups will also be discussed, highlighting unique aspects of the disorder in these subgroups.

Diagnostic Subtypes of Social Anxiety Disorder

Generalized Social Anxiety Disorder

The current version of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR; American Psychiatric Association [APA], 2000) includes a specifier for a generalized form of SAD. The *generalized* specifier is used when an individual reports fears in *most* social situations. In research studies, those not meeting such criteria are often grouped together as a *nongeneralized*, *specific*, or *limited* subgroup. This latter group can include individuals with more exclusive performance fears, such as speaking in public. A bi-dimensional subgrouping was supported using epidemiologic data from the first National Comorbidity Survey (NCS); the estimated lifetime prevalence of generalized social anxiety disorder (GSAD), defined as endorsing some other social fear in addition to a public speaking fear, was 8.5%, whereas the lifetime prevalence for those with exclusive public speaking fears was 2.9% (Kessler, Stein, & Berglund, 1998). Among outpatients with SAD, approximately 50–60% of individuals

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

meet criteria for GSAD (e.g., Mannuzza, Schneier, Chapman, & Liebowitz, 1995). Establishing the generalized specifier in DSM-III-R (APA, 1987) was the first way in which heterogeneity of SAD was officially recognized. However, this classification structure and its utility have not been without question.

Heimberg, Holt, Schneier, and Spitzer (1993) instead proposed a three-part classification system. The first group of *generalized* SAD patients describes individuals with social anxiety in most situations. The *nongeneralized* subtype characterizes individuals with significant anxiety in social interactions, but who also demonstrate one domain of social functioning in which they do not experience clinical levels of anxiety. The third, *circumscribed* (or specific) form of social anxiety would characterize those whose anxiety is only apparent in a limited number of specific situations, such as public speaking or eating in public. An advantage of such a tripartite model is a more specific classification for those with circumscribed fears, such as public speaking (see below). However, due to insufficient empirical evidence at the time, there was little basis to support the adoption of this model in DSM-IV (APA, 1994).

Ruscio and colleagues (2008) also questioned the utility of a generalized specifier. They investigated the number of feared situations endorsed by respondents in the National Comorbidity Survey—Replication (NCS-R). When using a detailed assessment of social fears, they found very few individuals met criteria for SAD with only one or two feared situations (3.4%). In fact, 71% of respondents with lifetime SAD reported eight or more social fears. They argued that with a broader range of assessed social fears, there is an increased detection of multiple fears among those who may be misclassified as nongeneralized cases.

An additional concern is the lack of a specific operational definition of the generalized subtype. The criterion of *most* situations has been interpreted differently by various research groups (see Hofmann, Heinrichs, & Moscovitch, 2004). Therefore, comparison across studies is difficult. Nonetheless, the current body of research seems to support the fact that those diagnosed with GSAD are different from those with more limited social fears in several ways. Compared to those with nongeneralized SAD, individuals with GSAD report more severe social fears (see Hofmann et al., 2004), with studies showing linear increases in symptom severity as the number of social fears increases (e.g., Stein, Torgrud, & Walker, 2000). The GSAD subgroup also appears to have more psychiatric comorbidity than the nongeneralized subgroup (e.g., Kessler et al., 1998). Further, endorsing generalized social fears may explain variance in functional impairment, particularly in the domains of interpersonal and occupational functioning and life satisfaction (reviewed below). Social skills in interaction situations also appear to be more impaired among those with GSAD (e.g., Beidel, Rao, Scharfstein, Wong, & Alfano, 2010; see also **Chapter 17** for expanded discussion on social skills and SAD). Finally, although those with GSAD show comparable benefits from cognitive behavioral treatments, they report more severe symptoms at the end of treatment (see Rodebaugh, Holaway, & Heimberg, 2004).

In considering the proposed revisions to the DSM-5, the classification of a generalized subgroup has again been criticized, and the task force has recommended removing this specifier (APA, 2012). They argue that those with more generalized fears are best considered a more severe form of the disorder, rather than categorically distinct from

those with nongeneralized fears (see Bögels et al., 2010). Given the preponderance of evidence supporting quantitative differences between the generalized and nongeneralized forms of SAD, the task force argues that the use of a continuous severity rating is more appropriate (Bögels et al., 2010). However, other recent reviews (Hofmann et al., 2004; Hook & Valentiner, 2002) have proposed quantitative *and qualitative* differences between the subtypes.

Hook and Valentiner (2002) conducted a review of the literature and suggested that some differences between those with generalized and more specific forms of SAD cannot be attributed to purely quantitative differences in severity. For example, those with GSAD versus nongeneralized SAD appear to differ in the course of the disorder, and there may also be different etiological contributions. Those with nongeneralized SAD report a later age of onset (approximate age 16 years) as compared with a typical age of onset before the age of 10 years for those with GSAD (Mannuzza et al., 1995). In terms of genetic contributions, the first-degree relatives of those with GSAD are significantly more likely to have GSAD than nongeneralized SAD (Stein et al., 1998), whereas individuals with nongeneralized SAD were no more likely than nonanxious controls to have first-degree relatives with SAD (Mannuzza et al., 1995). Such research suggests more prominent genetic contributions for GSAD.

Additionally, when using behavioral approach tests, there appear to be different physiological response profiles among the subtypes. As Hook and Valentiner (2002) review, four of the five studies measuring physiological reactivity differences suggest that those with specific performance fears show *greater* physiological responses to a speech task than those with GSAD. However, one would expect the generalized subgroup to show higher reactivity given that those with more fears show greater severity and impairment. Yet, as Hofmann and colleagues (2004) noted, the evidence for these qualitative differences does not necessarily support the distinction between subgroups based on number of social fears; instead, it may be the types of feared situations that better distinguish these subgroups (see below).

Comorbid Avoidant Personality Disorder

SAD shares several key features with avoidant personality disorder (AVPD), namely pervasive social inhibition and hypersensitivity to negative evaluation. Given the substantial overlap between the diagnostic criteria for SAD and AVPD, several researchers have critiqued the distinction between these two disorders. A thorough review of this research is beyond the scope of this chapter. However, some important points about subtyping are relevant. First, the highest comorbidity rates exist between AVPD and the generalized subtype of SAD (see Alden, Laposa, Taylor, & Ryder, 2002). Further, among those with GSAD, those with comorbid AVPD appear to report the most severe symptoms (see Bögels et al., 2010), as well as more social skill deficits (e.g., Chambless, Fydrich, & Rodebaugh, 2008). Taken together, these findings suggest that AVPD is a more severe form of SAD. However, an alternative possibility is that current DSM criteria for AVPD do not fully describe the disorder (see Alden et al., 2002); therefore, studies relying on these criteria will continue to be limited. For example, a recent review suggests that individuals with comorbid GSAD and AVPD

may have unique characteristics, such as greater likelihood for comorbid eating disorder pathology and comorbid personality disorders (Bögels et al., 2010). Further, the authors suggest that there is preliminary support linking AVPD with schizophrenia spectrum disorders. Therefore, more investigations are necessary before accepting AVPD as a more severe form of SAD.

Types of Feared Social Situations

Alternative conceptualizations of SAD subtypes make distinctions based on *types of feared situations* rather than quantity of feared situations. Studies have varied in their designs, including the use of factor analysis (Baker, Heinrichs, Kim, & Hofmann, 2002; Cox, Clara, Sareen, & Stein, 2008; Knappe et al., 2011; Perugi et al., 2001; Ruscio et al., 2008; Safren et al., 1999; Vriends, Becker, Meyer, Michael, & Margraf, 2007), cluster analysis (Eng, Heimberg, Coles, Schneier, & Liebowitz, 2000; Furmark, Tillfors, Stattin, Ekselius, & Fredrikson, 2000; Iwase et al., 2000), latent class analysis (Kessler et al., 1998), and examining indices of similarity for pairs of feared situations (Stein & Deutsch, 2003). Collectively, these studies have offered slightly different models, yet a number of groupings are similar across studies, and include fears of *performance*, *interactions*, and *observation*.

Performance Fears

Some variation of performance-based fears, such as fears of public speaking, emerged in all but three of the studies noted above. Regarding the latter three studies, Ruscio and colleagues (2008) failed to reveal a specific subgroup reporting performance fears. They examined possible subtypes in the NCS-R and found evidence for a single factor of feared situations. However, as noted by Blöte, Kint, Miers, and Westenberg (2009), these analyses contained data from the entire sample, including those without SAD, whereas the majority of the other studies only utilized data from respondents with SAD. The same may be true for the results offered by Knappe and colleagues (2011), as they only found evidence for a single factor of social fears in a large, community sample. Finally, Furmark and colleagues (2000) found evidence for the subgroups of generalized, nongeneralized, and specific forms of SAD. The specific group was the most common subtype, and among this subgroup, around 41% of the respondents reported public speaking fears only. Despite these discrepant findings, the majority of studies support the notion of a distinct class of social fears focusing on performance-based situations.

Performance fears appear to be common among most individuals with SAD (Eng et al., 2000). Further, *only* endorsing clinically significant public speaking fears is fairly common among a subgroup of individuals seeking outpatient treatment for SAD, with estimates ranging from 17% to 19% (Eng et al., 2000; Perugi et al., 2001). Such a group is comparable to the circumscribed subtype proposed by Heimberg and colleagues (1993) in that they endorse minimal social anxiety in interactions and in other situations under observation. In comparison to those reporting both interaction and performance fears, individuals with performance-only fears seem to

be less avoidant, experience less impairment, and have lower rates of comorbidity (Knappe et al., 2011).

Relatedly, Blöte and colleagues (2009) note some initial evidence for qualitative differences between those with pure public speaking anxiety versus GSAD. In their review, one study found evidence that those with only public speaking fears report higher anxiety in a speech task than those with GSAD (Tran & Chambless, 1995), yet two other studies failed to find such a difference (Hofmann, Gerlach, Wender, & Roth, 1997; Levin, Saoud, Strauman, & Gorman, 1993). In another study, Boone and colleagues (1999) found greater heart reactivity among those with circumscribed public speaking phobia, as compared with those with GSAD, during a speech task, even though they failed to report higher state anxiety. Interestingly, the same physiological differences were not apparent when using a social interaction task.

Although Boone and colleagues (1999) did not find significant differences between the subgroups in social skills, voice level, intonation, or gaze behaviors during the conversation and speech tasks, a more recent investigation of social skill deficits revealed important differences. Beidel and colleagues (2010) investigated differences among those with generalized and nongeneralized SAD, with 95% of those in the nongeneralized group endorsing only public speaking fears. Group differences in social skills emerged only when the participants engaged in social interaction tasks, with those with GSAD performing less well than those with nongeneralized SAD. There were no differences between these subgroups when examining social skills, distress, and degree of avoidance in an impromptu public speaking task. In addition, many of the qualitative differences noted by Hook and Valentiner (2002; described above) may apply to this subgroup of individuals with sole performance fears, as Hook and Valentiner defined their specific SAD subgroup as those who fear one type or a limited number of social situations.

Finally, those with only performance fears appear to have a unique etiological history. For instance, they are more likely to report traumatic performance situations, suggestive of a conditioning history (Stemberger, Turner, Beidel, & Calhoun, 1995). However, an important caveat was offered by Hofmann, Ehlers, and Roth (1995), who found that traumatic events most often occurred *after* the onset of the performance fears. In fact, a significant proportion of their speech phobic respondents (33%) identified the occurrence of a panic attack as the primary reason for the development of their phobia. This is interesting in light of a recent study involving a caffeine challenge, where those with panic disorder and performance-limited SAD showed greater physiological reactivity in comparison with those with GSAD and nonanxious controls (Nardi et al., 2009).

In summary, performance fears are common among most individuals with SAD, yet there is a subgroup of individuals who solely report performance fears. As Eng and colleagues (2000) noted, the group with predominant performance fears "is not best distinguished by its severity of anxiety in any one area, but rather, its lack of anxiety across several [areas]" (p. 1353). Thus, the extant literature largely supports the recent recommendation to replace the generalized specifier with a *predominantly performance* specifier in DSM-5 (Bögels et al., 2010). However, a clear definition of this specifier and adequate assessment measures are necessary in order to avoid the problems encountered with the vague generalized specifier.

Fears of Social Interactions and Observation

Several investigations have supported a unique factor reflecting fear in social interactions (Baker et al., 2002; Cox et al., 2008; Eng et al., 2000; Iwase et al., 2000; Perugi et al., 2001; Safren et al., 1999; Stein & Deutsch, 2003; Vriends et al., 2007). However, in comparison to performance fears, it appears to be more difficult to discretely isolate social interaction fears, as these fears seem to overlap significantly with fears about being observed (Cox et al., 2008; Ruscio et al., 2008). Further, those who endorse significant social interaction fears also report significant fears of performance and/or observation (Cox et al., 2008). For example, among 209 community respondents diagnosed with SAD, 96.2% reported a performance-related fear, whereas 64.5% reported an interaction-related fear (Knappe et al., 2011). With almost the entire group endorsing performance fears, it is clear that there were very few respondents only endorsing fears of social interaction. Overall, in contrast to the literature on performance fears, there appears to be less evidence for a subgroup who reports only social interaction fears. Similar to the research on social interaction fears, several investigations have identified a factor distinguished by fears of being observed (Cox et al., 2008; Eng et al., 2000; Perugi et al., 2001; Safren et al., 1999; Vriends et al., 2007). Examples include fears of being observed while writing, eating, or drinking in public, or using a public restroom. However, there is the same difficulty in distinguishing this area of concern from other social fears, and individuals reporting observation fears often endorse fears across several social domains (Cox et al., 2008).

Variance in the Focus of Social Fears

A cardinal feature of SAD is an excessive fear of acting in a way that will result in negative evaluation. However, some have discussed the importance of considering variations in this core fear. For example, research has examined how fears of being evaluated due to displays of anxiety (rather than due to how one acts) and fears of offending others (rather than being scrutinized) might represent distinct types of SAD.

Fears of Displaying Physical Signs of Social Anxiety

Individuals with SAD often note prominent fears of showing signs of anxiety, such as blushing, trembling, or sweating, which are observable to others and cannot easily be controlled. At least one study noted that these fears are common among those seeking treatment for SAD, with approximately 43% of Dutch patients reporting fears of showing symptoms such as blushing, trembling, or sweating as their primary source of fear (Bögels & Reith, 1999). Further, when examining social behaviors among SAD patients, visible anxiety signs may be a distinct factor from skilled behaviors such as good eye contact and smiling (Bögels, Rijsemus, & De Jong, 2002).

Almost all of the limited research in this area has focused on blushing. Specifically, SAD patients with prominent fears of blushing showed greater blushing responses in social tasks than SAD patients without these fears, reflecting a greater propensity to blush (Voncken & Bögels, 2009). These fears may also be associated a history of

traumatic experiences, such as being teased for blushing (Mulkens & Bögels, 1999). Given that there is evidence suggesting that individuals with SAD have fears about physical symptoms due to comorbid medical conditions such as hyperhidrosis (excessive sweating), essential tremor, and rosacea (see Bögels et al., 2010), more research in this area is necessary.

Fears of Offending Others

Fear of offending others is marked by a prominent focus on how one's self influences other people versus the typical preoccupation with negative evaluation of the self. Recently, greater attention has been paid to these fears of offending or causing discomfort to others, and they are central in some cultural-specific descriptions of SAD, such as taijin kyofusho (TKS) in Japan. The offensive subtype of TKS consists of fears of making another person uncomfortable (Iwase et al., 2000; see also **Chapter 11** for expanded discussion) and is thought to relate to collectivist cultural norms which emphasize the importance of interdependence and social harmony (Rector, Kocovski, & Ryder, 2006). Such cultural contributions would explain the higher rates of these fears in collectivist cultures, yet it is important to note that these fears are not unique to these cultures. Although Dinnel, Kleinknecht, and Tanaka-Matsumi (2002) found higher endorsements of TKS symptoms among Japanese students as compared with American students, they also found that the American students who rated themselves as high on interdependence and low on independence reported greater TKS symptoms. Further, in a recent examination of TKS symptoms in American and Korean patients diagnosed with SAD, moderate to severe fears of offending others were quite common among the American sample (Choy, Schneier, Heimberg, Oh, & Liebowitz, 2008).

Some have questioned whether fears of offending others are truly unique. For instance, fear of offending others may only be distressing to the extent that it reflects increased risk for negative evaluation (Magee, Rodebaugh, & Heimberg, 2006). Further, although prominent fears of offending others may call for treatment modifications (Rector et al., 2006), cognitive behavioral treatments with their focus on individualized case conceptualizations may easily address this area of concern (Ledley, 2006; Magee et al., 2006). Regardless, further assessment of such fears is useful. As Choy and colleagues (2008) noted, among the American clients with the highest endorsements of TKS fears, there had been no previous clinical documentation of such concerns, suggesting that clinicians are not typically assessing such fears and American clients are not spontaneously reporting them. Even if the fear ultimately relates back to concern over negative evaluation, thorough assessment will allow for adequate coverage of these concerns in treatment.

Personality Considerations

Personality traits have also been examined to better understand the heterogeneity within SAD. Although it is unclear whether individual variations represent differences in degree or kind of SAD, these traits are hypothesized to be related to one's behavioral

tendencies in response to social situations (Hofmann et al., 2004). This review focuses on the traits of impulsivity and aggression (see Hofmann et al. 2004; **Chapter 6** for thorough discussions of other relevant personality factors).

Although behavioral inhibition and overregulated behavior are commonly associated with SAD, a growing body of literature notes individuals with SAD who demonstrate significant impulsivity, risk-taking behaviors, and aggression. For example, using the NCS-R dataset, Kashdan, McKnight, Richey, and Hofmann (2009) found a subgroup of individuals with SAD who show an atypical pattern of anger and aggression and moderate to high impulsivity in sexual behaviors and substance use. This group constituted 21% of the SAD sample. Similarly, Kashdan and Hofmann (2008) found that 41.5% of a treatment-seeking GSAD sample was characterized by high novelty-seeking.

Compared to those with the typical presentation of low aggression and low risk-taking, those in the atypical class tended to be younger, male, and report an earlier age of onset; they also reported poorer global health, a greater number of feared situations, greater functional impairment, and were at increased risk for most impulse control and bipolar disorders (Kashdan et al., 2009). Individuals in the impulsive/aggressive subgroup also appear to have more severe comorbid substance use disorders (Kashdan & Hofmann, 2008). Further, in nonclinical samples, those in a disinhibited, socially anxious subgroup showed poorer quality of life, less social support, reduced psychological flexibility, and greater problems managing their negative emotions; they also reported more frequent risky sexual behaviors, aggression, and substance abuse in a 3-month follow-up period (Kashdan, Elhai, & Breen, 2008).

The presence of impulsivity and aggression has implications for treatment. Specifically, those with significant anger were found to be less likely to complete a group cognitive behavioral treatment for SAD (Erwin, Heimberg, Schneier, & Liebowitz, 2003). In another study, compared with those characterized by friendly–submissive behaviors, those in the hostile–submissive group had poorer treatment outcomes, as evidence by: higher posttreatment anxiety scores; higher fears of failure, critique, and social contact; more difficulties saying "no"; greater interpersonal distress; as well as lower scores on measures of self-value, well-being, and optimism, despite being comparable on these measures at pretreatment (Cain, Pincus, & Holtforth, 2010). Higher attrition and poorer treatment outcomes may suggest that treatment methods are less effective for this subgroup, or it may be that therapists have difficulty forming positive therapeutic alliances with these patients, thereby reducing treatment effects.

In summary, a mounting body of evidence supports a subgroup of individuals with SAD who have significant difficulties with risk-taking and aggression. As Kashdan and McKnight (2010) note, such behaviors may be adaptive in the short run by "regulating anxiety (substance abuse), preventing rejection (e.g., preemptive aggression), and producing pleasurable moments of belonging (e.g., sex with prostitutes)" (p. 49). They also suggest that those with hostile and impulsive behaviors may be overrepresented in the clients who fail to respond to standard treatments, yet this speculation awaits further empirical investigation. Greater research and consideration of these factors will likely lead to more complete conceptualizations of SAD, and possibly improved treatments.

Heterogeneity in Functioning

Numerous studies have demonstrated that those with SAD report impairment in many life domains. Individuals with SAD are more likely to have fewer friends and romantic relationships (Alden & Taylor, 2004), lower levels of social support (Ham, Hayes, & Hope, 2005), and greater impairment in work productivity (Patel, Knapp, Henderson, & Baldwin, 2002). Fewer studies have examined the heterogeneity in quality of life among those with SAD to understand why some individuals with SAD report more satisfaction in various life domains. Much of the research examining individual differences in quality of life has focused on symptom severity or diagnostic subtype.

Symptom Severity and Diagnostic Subtype

Interpersonal functioning. When interpersonal functioning is examined across the continuum of diagnostic severity, some differences emerge (see also **Chapter 8**). Specifically, the quality of relationships is poorer for individuals with more severe SAD symptoms, as those with GSAD show more interpersonal distress than those diagnosed with the nongeneralized subtype (Kachin, Newman, & Pincus, 2001). Those with higher levels of social anxiety perceive relationships with friends, family, and romantic partners to be less close and supportive (Montgomery, Haemmerlie, & Edwards, 1991). Further, individuals who are highly socially anxious seem to be more sensitive to both helpful and harmful effects of relationships, as well as show more concern for how their partner is being evaluated by others (Gordon, Heimberg, Montesi, & Fauber, 2012).

Research also suggests that SAD severity predicts how one behaves in relationships. Higher levels of SAD are associated with a range of dysfunctional behaviors, including being less assertive and emotionally expressive and more conflict avoidant and interpersonally dependent (Davila & Beck, 2002; Grant, Beck, Farrow, & Davila, 2007). A few studies have shown that those with higher SAD tend not to volunteer information about themselves, and when information is conveyed, it tends to be less personally revealing (e.g., Cuming & Rapee, 2010). Restricting one's self-disclosures seems to bring comfort, as women with high levels of SAD report feeling closer to their partners when they withhold their own negative emotions (Kashdan, Volkmann, Breen, & Han, 2007).

The severity of SAD symptoms also predicts the extent to which one is in a relationship and/or interacting with others. Specifically, higher levels of social anxiety were related to wanting to be alone when with less familiar and trusted individuals (Brown, Silvia, Myin-Germeys, & Kwapil, 2007). Further, the proportion of single patients with SAD seems to increase in a stepwise fashion when going from limited social anxiety (nongeneralized), to pervasive social anxiety (generalized), to pervasive and severe social anxiety (AVPD; Hart, Turk, Heimberg, & Liebowitz, 1999). Similarly, Safren, Heimberg, Brown, and Holle (1997) found that those with comorbid GSAD and AVPD were less likely to be married than those with nongeneralized SAD.

Occupational functioning. The occupational impairment experienced by those with SAD is well documented (e.g., Patel et al., 2002); however, only a few studies have

examined differences in work impairment based on diagnostic subtype and symptom severity. Specifically, those with clinically elevated subthreshold symptoms, compared with those that meet diagnostic criteria for SAD, report only minor reductions in work productivity (Wittchen, Fuetsch, Sonntag, Müller, & Liebowitz, 2000). On the other hand, those with GSAD reported higher levels of anxiety than the nongeneralized group when they started work in their current job (Bruch, Fallon, & Heimberg, 2003). Higher levels of SAD also predicted lower hourly wages as well as a lower probability of being in a managerial, technical, or professional occupation (Katzelnick & Greist, 2001). Further, those with comorbid GSAD and AVPD were shown to earn a lower income (Safren et al., 1997).

Life satisfaction. There is some evidence to suggest that symptom severity may account for why some individuals with SAD report better quality of life, as patients with GSAD and AVPD had lower quality of life scores than those with the nongeneralized subtype (Eng, Coles, Heimberg, & Safren, 2001; Safren et al., 1997). Thus, functional impairment seems to vary based on the extent of SAD symptoms and SAD subtype, with a comorbid diagnosis of AVPD predicting the poorest global functioning.

To examine quality of life beyond a global functioning score, Eng, Coles, Heimberg, and Safren (2005) examined specific domains of life satisfaction among individuals with SAD, considering the relationships between these domains and symptom severity. Those with SAD were not characterized by global dissatisfaction, but instead were specifically dissatisfied with the quality of their achievement (i.e., occupational activities, economic achievement) and social functioning (i.e., social activities, quality of one's current relationships). On the other hand, participants reported feeling satisfied with their personal growth (i.e., activities and pursuits that provide self-fulfillment and intrinsic reward) and surroundings (i.e., one's physical environment and community). Notably, all four domains of life satisfaction were inversely correlated with the severity of SAD symptoms, indicating that individuals with more severe symptomatology are more dissatisfied across various domains of their life.

Demographic Variables

Although the majority of research has focused on the influence of SAD symptom severity and diagnostic subtype on functioning, some research has examined how demographic variables, including gender and relationship status, may relate to an individual's functioning. Although males and females with SAD show similar levels of perceived social support quantity and satisfaction (e.g., Ham et al., 2005), gender differences emerge in relation to how men and women behave in their relationships. For example, women with SAD adopt a communication style characterized by a paucity of self-disclosure and limited emotional expression in friendships and romantic relationships; this was not found to be true for men (Cuming & Rapee, 2010). This research is limited, though, not only by the lack of studies including both male and female participants, but also by the fact that the samples of males tend to be smaller, potentially influencing statistical power (Cuming & Rapee, 2010). Gender differences have also been examined in terms of occupational functioning. Specifically, shy men were found to be more likely to have a delayed entry into a stable career, to underachieve in their career, and to manifest greater career instability, whereas shy

women were more likely to not have a work history at all or terminate it after entering a conventional path of marriage, childbearing, and homemaking (Caspi, Elder, & Bem, 1988).

Another demographic variable that has been examined is relationship status. Although differences have not been found between single and married patients with SAD in their level of education or employment, single patients with SAD reported greater fear and avoidance of social interaction situations than performance situations (Hart et al., 1999). Similarly, being married was significantly related to higher life satisfaction after controlling for symptom severity (Safren et al., 1997). Married women with SAD also showed larger social support networks and were more satisfied with their social support networks than single or divorced women with SAD (Ham et al., 2005). It seems that married women with SAD may benefit more than married men with SAD from the support of their spouse and spouse's social network.

Even though greater life satisfaction is reported by those with partners, this may nonetheless be accompanied by marital distress (Heinrichs, 2003, as cited in Alden & Taylor, 2004). Future research could examine how the interpersonal styles of those with high levels of SAD (e.g., less assertive, and more conflict avoidant and interpersonally dependent) predict marital distress, especially since being overly reliant on others has been found to mediate the relationship between social anxiety and interpersonal stress (Davila & Beck, 2002). Taken together, not being in a committed relationship seems to be related to poorer functioning. However, research is still needed to understand whether SAD symptoms decrease the likelihood of being in a committed romantic relationship, or if being single is a risk factor for more severe pathology (and thus poorer functioning).

Social Anxiety Disorder in Understudied Contexts and Populations

Much of the research on SAD focuses on individuals seeking treatment, respondents from the community, or student samples. In this final section, we consider the unique presentation of SAD in understudied contexts and groups.

SAD in Medical Settings

SAD is highly prevalent in the primary-care setting (i.e., 1 month prevalence rate of 7%; Stein, McQuaid, Laffaye, & McCahill, 1999). This may be an underestimate, as individuals with SAD have long delays between symptom onset and the time they seek help, and are unlikely to seek medical attention for social anxiety symptoms (Stein et al., 1999; Wagner, Silove, Marnane, & Rouen, 2006). They may not seek help because they see themselves as just being shy or having a social problem, or they may fear being ridiculed after revealing their symptoms (Wagner et al., 2006). Instead of seeking help for SAD, visits to primary care are likely for health concerns or a comorbid mental health disorder such as depression or substance abuse (Stein et al., 1999). Given the high rates of help-seeking in medical settings by those with SAD for

non-SAD symptoms, there is an increased opportunity for physicians to recognize and treat the disorder. However, research suggests that primary-care physicians are least skilled in making a diagnosis of SAD compared with other anxiety disorders (Wagner et al., 2006).

Not only is SAD relevant in the medical setting as a primary condition, it may also develop secondary to a medical condition. Several medical conditions have been associated with social anxiety, including stuttering (Stein, Baird, & Walker, 1996), and movement disorders (Ozel-Kizil, Akbostanci, Ozguven, & Atbasoglu, 2008). Conditions with observable symptoms or features may lead to increased avoidance and isolation. For example, research suggests that those with facial disfiguration show similar social phobic avoidance as patients diagnosed with SAD (Newell & Marks, 2000). Many patients with disfigurement report feeling stigmatized, being concerned about their appearance, and fear others' reactions, which can result in psychological distress and/or isolation (e.g., Vardy et al., 2002). Additional factors may need to be considered, though, as Hagedoorn and Molleman (2006) found that the degree of facial disfigurement was positively related to distress only when patients did not feel self-efficacious in social encounters.

For patients presenting with SAD and a primary medical condition, the DSM-IV requires that social anxiety not be related to a medical condition; therefore, these patients receive a diagnosis of anxiety disorder NOS. However, research supporting secondary SAD as a separate clinical entity from SAD as defined in the DSM is scarce, and some studies suggest that the clinical characteristics of SAD secondary to a medical condition might be similar to or even more severe than SAD as defined in the DSM (Ozel-Kizil et al., 2008). Therefore, screening for SAD in the primary-care setting seems well justified, since SAD symptoms prevent individuals from getting needed services, and those with SAD may not receive adequate care (Beard, Moitra, Weisberg, & Keller, 2010).

SAD Among Veterans

Herman (1992) discusses how social disconnectedness is one of the most profound results of trauma, especially events that involve interpersonal trauma such as combat, rape, and incest. After an interpersonal trauma, basic assumptions about relationships are threatened, and an individual's sense of connection with the community can be shattered. High levels of social support can offer resilience for posttrauma recovery (e.g., Pietrzak et al., 2010). However, many veterans report social withdrawal, isolation, and decreased social support (e.g., Keane, Scott, Chavoya, Lamparski, & Fairbank, 1985). One explanation for the high levels of social avoidance is that veterans are concerned about being evaluated by others in social situations, resulting in less positive social activity, less interaction with others, and, ultimately, impairment in social functioning.

Numerous studies have shown that veterans with posttraumatic stress disorder (PTSD) show high levels of SAD (e.g., Hofmann, Litz, & Weathers, 2003; Orsillo, Heimberg, Juster, & Garrett, 1996). For instance, 3.6% of veterans in primary-care clinics met criteria for SAD, and veterans with PTSD showed a greater rate of SAD than those without PTSD (22.0% vs. 1.1%; Kashdan, Frueh, Knapp, Hebert, & Magruder,

2006). Further, after controlling for major depressive disorder symptoms, the presence of SAD predicted PTSD severity, and the relationship between SAD and psychiatric comorbidity and suicidal risk seen in this sample was not attributable to PTSD severity or the presence of major depression.

As noted, many veterans report avoiding social situations. However, only those diagnosed with SAD report that they would be fearful of being evaluated by others; others report concerns related to danger or bodily harm, interpersonal failure, expectations of invalidation, or a general feeling of not caring about or enjoying the company of others (Hofmann et al., 2003; Orsillo et al., 1996). Some research suggests that the presence of PTSD and mood symptoms is the "driving force" behind the overall negative affect and impairment in social interactions reported by combat veterans. For example, Hofmann and colleagues (2003) found that social problems experienced by veterans with PTSD seem to be closely related to symptoms of depression and not social anxiety.

Given the high levels of social withdrawal and isolation reported by veterans, especially those who have been exposed to combat and interpersonal trauma, it is important to assess impairment in veterans' social interactions. However, the literature suggests that avoidance of social situations may be due to various reasons. Further, those who have avoided social situations for a long period of time may not be able to validly predict their emotional response or may exhibit secondary emotions. For example, anxiety about being rejected may be masked by the presence of anger (Orsillo et al., 1996). Therefore, it is important to assess the function and reasons behind the social withdrawal and isolation in this population.

SAD and the Internet

Over the past 10 years, more attention has been paid to how the presence of social anxiety relates to communicating and connecting with others through the Internet. One study reported that shyness is correlated with greater Facebook usage (Orr et al., 2009), and other research suggests that those with SAD who spend more hours online spend less time interacting with others in person (Erwin, Turk, Heimberg, Fresco, & Hantula, 2004). Therefore, individuals with SAD may be using the Internet as a way to get their interpersonal needs met.

Erwin and colleagues (2004) recruited individuals to complete an Internet survey about social anxiety, and concluded that Internet users with SAD represent a population of individuals with very severe SAD that are not well represented in clinical and epidemiological studies of SAD. Participants from their study reported experiencing anxiety, distress, and/or impairment in all of the situations surveyed except for eating in public, writing in public, and using public restrooms. Seventy-five percent reported that they socialize in-person with others less than 5 hr per week, one-third reported no significant friendships, and one-half reported no significant romantic relationships. Further, these individuals were twice as likely to have never married compared with individuals in the community assessed in the NCS (Kessler et al., 1998). Finally, Internet users with SAD were not likely to have received adequate treatment, as only one-third of the sample reported receiving psychotherapy and one-third receiving pharmacotherapy.

Given that there are so many uses for the Internet and ways to communicate with others electronically, as well as the fact that much of the research in this area was conducted several years ago and technology is continuously evolving, it is difficult to ascertain how those with SAD are utilizing the Internet. However, there is not much evidence to suggest that those with SAD are using the Internet to form relationships. For example, in the study by Erwin and colleagues (2004), Internet users with SAD reported interacting actively with others less than 20% of the time that they were online; more than 50% of the time spent online involved passive observance. Not only are those with SAD not going online with the purpose of interacting with others, it is unclear as to whether the formation of close relationships is enhanced or inhibited in this context. Some studies have found that when people communicate online, they engage in deeper questions and disclosures sooner, and develop closeness more quickly than in face-to-face relationships (e.g., Tidwell & Walther, 2002). However, when online communication is examined in the context of symptoms of social anxiety, different results emerge. In university students, shyness was associated with greater inhibition in online relationships (Ward & Tracey, 2004), and those with higher levels of social anxiety had fewer Facebook friends (Fernandez, Levinson, & Rodebaugh, 2012). Similarly, adolescents with SAD were found to be more likely to communicate online with individuals with whom they were not in close relationships, and were less likely to discuss intimate topics (Gross, Juvonen, & Gable, 2002).

Even though those with SAD may not be using the Internet to form close relationships, more time spent online has been found to be related to more active participation online (Erwin et al., 2004), suggesting that those with SAD are benefiting from the Internet in other ways. In the study by Erwin and colleagues (2004), Internet users with higher levels of SAD endorsed the development of new friendships, and reported receiving encouragement from others after self-disclosing their difficulties with social anxiety, via their online activity. Therefore, even though the initial intention is not to form a relationship with someone else, developing social support may be a byproduct of online activities. Further, among those with SAD, spending more time online also has been related to greater comfort with Internet-based interactions (Mazalin & Klein, 2008), and increased confidence when interacting with others in person (Erwin et al., 2004).

In addition to the benefits reported above, several additional reasons have been proposed as to why individuals with SAD may prefer online to in-person interactions. Individuals report feeling less shy online than in person (Knox, Daniels, Sturdivant, & Zusman, 2001), as online interactions limit the chance of negative evaluation because the amount of information displayed (especially visible signs of anxiety) is limited. Interacting online may also help those with SAD feel more comfortable because it gives the possibility of rehearsing or presenting oneself in a specific way (e.g., Fernandez et al., 2012). Therefore, for those with SAD, going online may be perceived as a safe and controlled way in which to fulfill one's social needs, providing a sense of connectedness to others.

Despite the greater comfort and support, though, there appear to be costs to spending time online. Introverted adults who used the Internet extensively reported increased loneliness and negative affect and decreased self-esteem (Kraut et al., 2002). College students with SAD who communicated frequently online showed higher

levels of depression and decreased quality of life (Weidman et al., 2012), and as time spent playing online games increased, SAD symptoms increased among Taiwanese college-age game players (Lo, Wang, & Fang, 2005). Further, not only does increased Internet use relate to decreased time spent in the social environment, it may increase the likelihood that in-person relationships deteriorate and become less fulfilling (Lo et al., 2005). These findings are consistent with research suggesting that relationships developed and maintained online are typically not as close as those formed offline (Cummings, Butler, & Kraut, 2002). Therefore, even though individuals with the most severe SAD may gain comfort by going online, this seems to be at the cost of more in-person isolation and psychological symptoms, and relationships of poorer quality.

Summary

There can be a great deal of variance in the presentation of SAD. Further, many of the differences have treatment implications. Unfortunately, the current DSM-IV diagnostic system, which includes the generalized specifier and the comorbid diagnosis of AVPD, has failed to provide the most useful categorization of important differences. We look forward to further investigation of different models of subtyping (e.g., based on feared situations) and descriptions of the disorder (e.g., focus of fears, personality traits). In addition, greater attention to the presentation of SAD in understudied contexts and populations will also aid in our increased understanding of the various presentations of SAD.

References

Alden, L. E., Laposa, J. M., Taylor, C. T., & Ryder, A. G. (2002). Avoidant personality disorder: Current status and future directions. *Journal of Personality Disorders, 16*(1), 1–29. doi:10.1521/pedi.16.1.1.22558

Alden, L. E., & Taylor, C. T. (2004). Interpersonal processes in social phobia. *Clinical Psychology Review, 24*(7), 857–882. doi:10.1016/j.cpr.2004.07.006

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., revised). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

American Psychiatric Association. (2012). *Social anxiety disorder (social phobia). DSM-5 development*. Retrieved from http://www.dsm5.org/ProposedRevision/Pages/proposedrevision.aspx?rid=163 [accessed August 30, 2012]

Baker, S. L., Heinrichs, N., Kim, H.-J., & Hofmann, S. G. (2002). The Liebowitz social anxiety scale as a self-report instrument: A preliminary psychometric analysis. *Behaviour Research and Therapy, 40*(6), 701–715. doi:10.1016/s0005-7967(01)00060-2

Beard, C., Moitra, E., Weisberg, R. B., & Keller, M. B. (2010). Characteristics and predictors of social phobia course in a longitudinal study of primary-care patients. *Depression and Anxiety, 27*(9), 839–845.

Beidel, D. C., Rao, P. A., Scharfstein, L., Wong, N., & Alfano, C. A. (2010). Social skills and social phobia: An investigation of DSM-IV subtypes. *Behaviour Research and Therapy, 48*(10), 992–1001. doi:10.1016/j.brat.2010.06.005

Blöte, A. W., Kint, M. J. W., Miers, A. C., & Westenberg, P. M. (2009). The relation between public speaking anxiety and social anxiety: A review. *Journal of Anxiety Disorders, 23*(3), 305–313. doi:10.1016/j.janxdis.2008.11.007

Bögels, S. M., Alden, L., Beidel, D. C., Clark, L. A., Pine, D. S., Stein, M. B., & Voncken, M. (2010). Social anxiety disorder: Questions and answers for the DSM-V. *Depression and Anxiety, 27*(2), 168–189. doi:10.1002/da.20670

Bögels, S. M., & Reith, W. (1999). Validity of two questionnaires to assess social fears: The Dutch Social Phobia and Anxiety Inventory and the Blushing, Trembling and Sweating Questionnaire. *Journal of Psychopathology and Behavioral Assessment, 21*(1), 51–66. doi:10.1023/a:1022812227606

Bögels, S. M., Rijsemus, W., & De Jong, P. J. (2002). Self-focused attention and social anxiety: The effects of experimentally heightened self-awareness on fear, blushing, cognitions, and social skills. *Cognitive Therapy and Research, 26*(4), 461–472. doi:10.1023/a:1016275700203

Boone, M. L., McNeil, D. W., Masia, C. L., Turk, C. L., Carter, L. E., Ries, B. J., & Lewin, M. R. (1999). Multimodal comparisons of social phobia subtypes and avoidant personality disorder. *Journal of Anxiety Disorders, 13*(3), 271–292. doi:10.1016/s0887-6185(99)00004-3

Brown, L. H., Silvia, P. J., Myin-Germeys, I., & Kwapil, T. R. (2007). When the need to belong goes wrong: The expression of social anhedonia and social anxiety in daily life. *Psychological Science, 18*(9), 778–782. doi:10.1111/j.1467-9280.2007.01978.x

Bruch, M. A., Fallon, M., & Heimberg, R. G. (2003). Social phobia and difficulties in occupational adjustment. *Journal of Counseling Psychology, 50*(1), 109–117. doi:10.1037/0022-0167.50.1.109

Cain, N. M., Pincus, A. L., & Holtforth, M. G. (2010). Interpersonal subtypes in social phobia: Diagnostic and treatment implications. *Journal of Personality Assessment, 92*(6), 514–527. doi:10.1080/00223891.2010.513704

Caspi, A., Elder, G. H., & Bem, D. J. (1988). Moving away from the world: Life-course patterns of shy children. *Developmental Psychology, 24*(6), 824–831. doi:10.1037/0012-1649.24.6.824

Chambless, D. L., Fydrich, T., & Rodebaugh, T. L. (2008). Generalized social phobia and avoidant personality disorder: Meaningful distinction or useless duplication? *Depression and Anxiety, 25*(1), 8–19. doi:10.1002/da.20266

Choy, Y., Schneier, F. R., Heimberg, R. G., Oh, K.-S., & Liebowitz, M. R. (2008). Features of the offensive subtype of Taijin-Kyofu-Sho in US and Korean patients with DSM-IV social anxiety disorder. *Depression and Anxiety, 25*(3), 230–240. doi:10.1002/da.20295

Cox, B. J., Clara, I. P., Sareen, J., & Stein, M. B. (2008). The structure of feared social situations among individuals with a lifetime diagnosis of social anxiety disorder in two independent nationally representative mental health surveys. *Behaviour Research and Therapy, 46*(4), 477–486. doi:10.1016/j.brat.2008.01.011

Cuming, S., & Rapee, R. M. (2010). Social anxiety and self-protective communication style in close relationships. *Behaviour Research and Therapy, 48*, 87–96. doi:10.1016/j.brat.2009.09.010

Cummings, J. N., Butler, B., & Kraut, R. (2002). The quality of online social relationships. *Communications of the ACM, 45*(7), 103–108.

Davila, J., & Beck, J. G. (2002). Is social anxiety associated with impairment in close relationships? A preliminary investigation. *Behavior Therapy, 33*(3), 427–446. doi:10.1016/s0005-7894(02)80037-5

Dinnel, D. L., Kleinknecht, R. A., & Tanaka-Matsumi, J. (2002). A cross-cultural comparison of social phobia symptoms. *Journal of Psychopathology and Behavioral Assessment, 24*(2), 75–84. doi:10.1023/a:1015316223631

Eng, W., Coles, M. E., Heimberg, R. G., & Safren, S. A. (2001). Quality of life following cognitive behavioral treatment for social anxiety disorder: Preliminary findings. *Depression and Anxiety, 13*(4), 192–193. doi:10.1002/da.1037.abs

Eng, W., Coles, M. E., Heimberg, R. G., & Safren, S. A. (2005). Domains of life satisfaction in social anxiety disorder: Relation to symptoms and response to cognitive-behavioral therapy. *Journal of Anxiety Disorders, 19*(2), 143–156. doi:10.1016/j.janxdis.2004.01.007

Eng, W., Heimberg, R. G., Coles, M. E., Schneier, F. R., & Liebowitz, M. R. (2000). An empirical approach to subtype identification in individuals with social phobia. *Psychological Medicine, 30*(6), 1345–1357. doi:10.1017/s0033291799002895

Erwin, B. A., Heimberg, R. G., Schneier, F. R., & Liebowitz, M. R. (2003). Anger experience and expression in social anxiety disorder: Pretreatment profile and predictors of attrition and response to cognitive-behavioral treatment. *Behavior Therapy, 34*(3), 331–350. doi:10.1016/s0005-7894(03)80004-7

Erwin, B. A., Turk, C. L., Heimberg, R. G., Fresco, D. M., & Hantula, D. A. (2004). The Internet: Home to a severe population of individuals with social anxiety disorder? *Journal of Anxiety Disorders, 18*(5), 629–646. doi:10.1016/j.janxdis.2003.08.002

Fernandez, K. C., Levinson, C. A., & Rodebaugh, T. L. (2012). Profiling: Predicting social anxiety from Facebook profiles. *Social Psychological and Personality Science, 3*, 706–713. doi:10.1177/1948550611434967

Furmark, T., Tillfors, M., Stattin, H., Ekselius, L., & Fredrikson, M. (2000). Social phobia subtypes in the general population revealed by cluster analysis. *Psychological Medicine, 30*(6), 1335–1344. doi:10.1017/s0033291799002615

Gordon, E. A., Heimberg, R. G., Montesi, J. L., & Fauber, R. L. (2012). Romantic relationships: Do socially anxious individuals benefit? *Cognitive Behaviour Therapy, 41*(2), 140–151. doi:10.1080/16506073.2012.656275

Grant, D. M., Beck, J. G., Farrow, S. M., & Davila, J. (2007). Do interpersonal features of social anxiety influence the development of depressive symptoms? *Cognition and Emotion, 21*(3), 646–663. doi:10.1080/02699930600713036

Gross, E. F., Juvonen, J., & Gable, S. L. (2002). Internet use and well-being in adolescence. *Journal of Social Issues, 58*(1), 75–90. doi:10.1111/1540-4560.00249

Hagedoorn, M., & Molleman, E. (2006). Facial disfigurement in patients with head and neck cancer: The role of social self-efficacy. *Health Psychology, 25*(5), 643–647. doi:10.1037/0278-6133.25.5.643

Ham, L., Hayes, S. A., & Hope, D. A. (2005). Gender differences in social support for socially anxious individuals. *Cognitive Behaviour Therapy, 34*(4), 201–206. doi:10.1080/16506070510008470

Hart, T. A., Turk, C. L., Heimberg, R. G., & Liebowitz, M. R. (1999). Relation of marital status to social phobia severity. *Depression and Anxiety, 10*, 28–32.

Heimberg, R. G., Holt, C. S., Schneier, F. R., & Spitzer, R. L. (1993). The issue of subtypes in the diagnosis of social phobia. *Journal of Anxiety Disorders, 7*(3), 249–269. doi:10.1016/0887-6185(93)90006-7

Heinrichs, N. (2003, September). *Does a partner matter*? Paper presented at the European Congress of Behavioural and Cognitive Therapy, Prague, Czechoslovakia.

Herman, J. L. (1992). *Trauma and recovery*. New York, NY: Basic Books.

Hofmann, S. G., Ehlers, A., & Roth, W. T. (1995). Conditioning theory: A model for the etiology of public speaking anxiety? *Behaviour Research and Therapy, 33*(5), 567–571. doi:10.1016/0005-7967(94)00072-r

Hofmann, S. G., Gerlach, A. L., Wender, A., & Roth, W. T. (1997). Speech disturbances and gaze behavior during public speaking in subtypes of social phobia. *Journal of Anxiety Disorders, 11*(6), 573–585. doi:10.1016/s0887-6185(97)00040-6

Hofmann, S. G., Heinrichs, N., & Moscovitch, D. A. (2004). The nature and expression of social phobia: Toward a new classification. *Clinical Psychology Review, 24*(7), 769–797. doi:10.1016/j.cpr.2004.07.004

Hofmann, S. G., Litz, B. T., & Weathers, F. W. (2003). Social anxiety, depression, and PTSD in Vietnam veterans. *Journal of Anxiety Disorders, 17*(5), 573–582. doi:10.1016/s0887-6185(02)00227-x

Hook, J. N., & Valentiner, D. P. (2002). Are specific and generalized social phobias qualitatively distinct? *Clinical Psychology: Science and Practice, 9*(4), 379–395. doi:10.1093/clipsy/9.4.379

Iwase, M., Nakao, K., Takaishi, J., Yorifuji, K., Ikezawa, K., & Takeda, M. (2000). An empirical classification of social anxiety: Performance, interpersonal and offensive. *Psychiatry and Clinical Neurosciences, 54*(1), 67–76. doi:10.1046/j.1440-1819.2000.00639.x

Kachin, K. E., Newman, M. G., & Pincus, A. L. (2001). An interpersonal problem approach to the division of social phobia subtypes. *Behavior Therapy, 32*(3), 479–501. doi:10.1016/s0005-7894(01)80032-0

Kashdan, T. B., Elhai, J. D., & Breen, W. E. (2008). Social anxiety and disinhibition: An analysis of curiosity and social rank appraisals, approach-avoidance conflicts, and disruptive risk-taking behavior. *Journal of Anxiety Disorders, 22*(6), 925–939. doi:10.1016/j.janxdis.2007.09.009

Kashdan, T. B., Frueh, B. C., Knapp, R. G., Hebert, R., & Magruder, K. M. (2006). Social anxiety disorder in veterans affairs primary care clinics. *Behaviour Research and Therapy, 44*(2), 233–247.

Kashdan, T. B., & Hofmann, S. G. (2008). The high-novelty-seeking, impulsive subtype of generalized social anxiety disorder. *Depression and Anxiety, 25*(6), 535–541. doi:10.1002/da.20382

Kashdan, T. B., & McKnight, P. E. (2010). The darker side of social anxiety: When aggressive impulsivity prevails over shy inhibition. *Current Directions in Psychological Science, 19*(1), 47–50. doi:10.1177/0963721409359280

Kashdan, T. B., McKnight, P. E., Richey, J. A., & Hofmann, S. G. (2009). When social anxiety disorder co-exists with risk-prone, approach behavior: Investigating a neglected, meaningful subset of people in the National Comorbidity Survey—Replication. *Behaviour Research and Therapy, 47*(7), 559–568. doi:10.1016/j.brat.2009.03.010

Kashdan, T. B., Volkmann, J. R., Breen, W. E., & Han, S. (2007). Social anxiety and romantic relationships: The costs and benefits of negative emotion expression are context-dependent. *Journal of Anxiety Disorders, 21*(4), 475–492. doi:10.1016/j.janxdis.2006.08.007

Katzelnick, D. J., & Greist, J. H. (2001). Social anxiety disorder: An unrecognized problem in primary care. *Journal of Clinical Psychiatry, 62*(Suppl. 1), 11–16.

Keane, T. M., Scott, W. O., Chavoya, G. A., Lamparski, D. M., & Fairbank, J. A. (1985). Social support in Vietnam veterans with posttraumatic stress disorder: A comparative analysis. *Journal of Consulting and Clinical Psychology, 53*(1), 95–102. doi:10.1037/0022-006x.53.1.95

Kessler, R. C., Stein, M. B., & Berglund, P. (1998). Social phobia subtypes in the National Comorbidity Survey. *American Journal of Psychiatry, 155*(5), 613–619.

Knappe, S., Beesdo-Baum, K., Fehm, L., Stein, M. B., Lieb, R., & Wittchen, H.-U. (2011). Social fear and social phobia types among community youth: Differential clinical features and vulnerability factors. *Journal of Psychiatric Research, 45*(1), 111–120. doi:10.1016/j.jpsychires.2010.05.002

Knox, D., Daniels, V., Sturdivant, L., & Zusman, M. E. (2001). College student use of the internet for mate selection. *College Student Journal, 35*(1), 158–160.

Kraut, R., Kiesler, S., Boneva, B., Cummings, J. N., Helgeson, V., & Crawford, A. (2002). Internet paradox revisited. *Journal of Social Issues, 58*, 49–74.

Ledley, D. R. (2006). Social anxiety and fear of causing discomfort to others: Conceptualization and treatment. *Journal of Social and Clinical Psychology, 25*(8), 919–928. doi:10.1521/jscp.2006.25.8.919

Levin, A. P., Saoud, J. B., Strauman, T., & Gorman, J. M. (1993). Responses of "generalized" and "discrete" social phobics during public speaking. *Journal of Anxiety Disorders, 7*(3), 207–221. doi:10.1016/0887-6185(93)90003-4

Lo, S.-K., Wang, C.-C., & Fang, W. (2005). Physical interpersonal relationships and social anxiety among online game players. *CyberPsychology & Behavior, 8*(1), 15–20. doi:10.1089/cpb.2005.8.15

Magee, L., Rodebaugh, T. L., & Heimberg, R. G. (2006). Negative evaluation is the feared consequence of making others uncomfortable: A response to Rector, Kocovski, and Ryder. *Journal of Social and Clinical Psychology, 25*(8), 929–936. doi:10.1521/jscp.2006.25.8.929

Mannuzza, S., Schneier, F. R., Chapman, T. F., & Liebowitz, M. R. (1995). Generalized social phobia: Reliability and validity. *Archives of General Psychiatry, 52*(3), 230–237. doi:10.1001/archpsyc.1995.03950150062011

Mazalin, D., & Klein, B. (2008). Social anxiety and the Internet: Positive and negative effects. *E-Journal of Applied Psychology, 4*(2), 43–50.

Montgomery, R. L., Haemmerlie, F. M., & Edwards, M. (1991). Social, personal, and interpersonal deficits in socially anxious people. *Journal of Social Behavior & Personality, 6*(4), 859–872.

Mulkens, S., & Bögels, S. M. (1999). Learning history in fear of blushing. *Behaviour Research and Therapy, 37*(12), 1159–1167. doi:10.1016/s0005-7967(99)00022-4

Nardi, A. E., Lopes, F. L., Freire, R. C., Veras, A. B., Nascimento, I., Valença, A. M., … Zin, W. A. (2009). Panic disorder and social anxiety disorder subtypes in a caffeine challenge test. *Psychiatry Research, 169*(2), 149–153. doi:10.1016/j.psychres.2008.06.023

Newell, R., & Marks, I. (2000). Phobic nature of social difficulty in facially disfigured people. *The British Journal of Psychiatry, 176*(2), 177–181. doi:10.1192/bjp.176.2.177

Orr, E. S., Sisic, M., Ross, C., Simmering, M. G., Arseneault, J. M., & Orr, R. R. (2009). The influence of shyness on the use of Facebook in an undergraduate sample. *Cyberpsychology & Behavior, 12*(3), 337–340. doi:10.1089/cpb.2008.0214

Orsillo, S. M., Heimberg, R. G., Juster, H. R., & Garrett, J. (1996). Social phobia and PTSD in Vietnam veterans. *Journal of Traumatic Stress, 9*(2), 235–252. doi:10.1002/jts.2490090207

Ozel-Kizil, E. T., Akbostanci, M. C., Ozguven, H. D., & Atbasoglu, E. C. (2008). Secondary social anxiety in hyperkinesias. *Movement Disorders, 23*(5), 641–645.

Patel, A., Knapp, M., Henderson, J., & Baldwin, D. (2002). The economic consequences of social phobia. *Journal of Affective Disorders, 68*(2–3), 221–233. doi:10.1016/s0165-0327(00)00323-2

Perugi, G., Nassini, S., Maremmani, I., Madaro, D., Toni, C., Simonini, E., & Akiskal, H. S. (2001). Putative clinical subtypes of social phobia: A factor-analytical study. *Acta Psychiatrica Scandinavica, 104*(4), 280–288. doi:10.1034/j.1600-0447.2001.00128.x

Pietrzak, R. H., Johnson, D. C., Goldstein, M. B., Malley, J. C., Rivers, A. J., Morgan, C. A., & Southwick, S. M. (2010). Psychosocial buffers of traumatic stress, depressive symptoms, and psychosocial difficulties in veterans of operations enduring freedom and Iraqi freedom:

The role of resilience, unit support, and postdeployment social support. *Journal of Affective Disorders, 120*(1–3), 188–192. doi:10.1016/j.jad.2009.04.015

Rector, N. A., Kocovski, N. L., & Ryder, A. G. (2006). Social anxiety and the fear of causing discomfort to others: Conceptualization and treatment. *Journal of Social and Clinical Psychology, 25*(8), 906–918. doi:10.1521/jscp.2006.25.8.906

Rodebaugh, T. L., Holaway, R. M., & Heimberg, R. G. (2004). The treatment of social anxiety disorder. *Clinical Psychology Review, 24*(7), 883–908. doi:10.1016/j.cpr.2004.07.007

Ruscio, A. M., Brown, T. A., Chiu, W. T., Sareen, J., Stein, M. B., & Kessler, R. C. (2008). Social fears and social phobia in the USA: Results from the National Comorbidity Survey Replication. *Psychological Medicine, 38*(1), 15–28. doi:10.1017/s0033291707001699

Safren, S. A., Heimberg, R. G., Brown, E. J., & Holle, C. (1997). Quality of life in social phobia. *Depression and Anxiety, 4*(3), 126–133.

Safren, S. A., Heimberg, R. G., Horner, K. J., Juster, H. R., Schneier, F. R., & Liebowitz, M. R. (1999). Factor structure of social fears: The Liebowitz Social Anxiety Scale. *Journal of Anxiety Disorders, 13*(3), 253–270. doi:10.1016/s0887-6185(99)00003-1

Stein, M. B., Baird, A., & Walker, J. R. (1996). Social phobia in adults with stuttering. *American Journal of Psychiatry, 153*(2), 278–280.

Stein, M. B., Chartier, M. J., Hazen, A. L., Kozak, M. V., Tancer, M. E., Lander, S., . . . Walker, J. R. (1998). A direct-interview family study of generalized social phobia. *American Journal of Psychiatry, 155*(1), 90–97.

Stein, M. B., & Deutsch, R. (2003). In search of social phobia subtypes: Similarity of feared social situations. *Depression and Anxiety, 17*(2), 94–97. doi:10.1002/da.10093

Stein, M. B., McQuaid, J. R., Laffaye, C., & McCahill, M. E. (1999). Social phobia in the primary care medical setting. *The Journal of Family Practice, 48*(7), 514–519.

Stein, M. B., Torgrud, L. J., & Walker, J. R. (2000). Social phobia symptoms, subtypes, and severity: Findings from a community survey. *Archives of General Psychiatry, 57*(11), 1046–1052. doi:10.1001/archpsyc.57.11.1046

Stemberger, R. T., Turner, S. M., Beidel, D. C., & Calhoun, K. S. (1995). Social phobia: An analysis of possible developmental factors. *Journal of Abnormal Psychology, 104*(3), 526–531. doi:10.1037/0021-843x.104.3.526

Tidwell, L. C., & Walther, J. B. (2002). Computer-mediated communication effects on disclosure, impressions, and interpersonal evaluations: Getting to know one another a bit at a time. *Human Communication Research, 28*(3), 317–348. doi:10.1111/j.1468-2958.2002.tb00811.x

Tran, G. Q., & Chambless, D. L. (1995). Psychopathology of social phobia: Effects of subtype and avoidant personality disorder. *Journal of Anxiety Disorders, 9*(6), 489–501. doi:10.1016/0887-6185(95)00027-l

Vardy, D., Besser, A., Amir, M., Gesthalter, B., Biton, B., & Buskila, D. (2002). Experiences of stigmatization play a role in mediating the impact of disease severity on quality of life in psoriasis patients. *British Journal of Dermatology, 147*(4), 736–742.

Voncken, M. J., & Bögels, S. M. (2009). Physiological blushing in social anxiety disorder patients with and without blushing complaints: Two subtypes? *Biological Psychology, 81*(2), 86–94. doi:10.1016/j.biopsycho.2009.02.004

Vriends, N., Becker, E. S., Meyer, A., Michael, T., & Margraf, J. (2007). Subtypes of social phobia: Are they of any use? *Journal of Anxiety Disorders, 21*(1), 59–75. doi:10.1016/j.janxdis.2006.05.002

Wagner, R., Silove, D., Marnane, C., & Rouen, D. (2006). Delays in referral of patients with social phobia, panic disorder and generalized anxiety disorder attending a specialist

anxiety clinic. *Journal of Anxiety Disorders, 20*(3), 363–371. doi:10.1016/j.janxdis.2005.02.003

Ward, C. C., & Tracey, T. J. G. (2004). Relation of shyness with aspects of online relationship involvement. *Journal of Social and Personal Relationships, 21*(5), 611–623. doi:10.1177/0265407504045890

Weidman, A. C., Fernandez, K. C., Levinson, C. A., Augustine, A. A., Larsen, R. J., & Rodebaugh, T. L. (2012). Compensatory internet use among individuals higher in social anxiety and its implications for well-being. *Personality and Individual Differences, 53*, 191–195.

Wittchen, H. U., Fuetsch, M., Sonntag, H., Müller, N., & Liebowitz, M. (2000). Disability and quality of life in pure and comorbid social phobia: Findings from a controlled study. *European Psychiatry, 15*(1), 46–58. doi:10.1016/s0924-9338(00)00211-x

III

Optimizing Assessment Approaches

How to Best Target Social Anxiety Symptoms

13

Clinical Interviews

Empirical Overview and Procedural Recommendations

Daniel W. McNeil and Laura L. Quentin

West Virginia University, USA

Introduction

Clinical interviews involving patients with social anxiety disorder (SAD; i.e., social phobia) or high degrees of social anxiety are essential but potentially quite anxiety-provoking assessments, given the nature of the disorder or condition and the social aspects of the situation. As the interview itself is a social interaction, the situation may evoke anxiety for such patients. At the same time, clinical interviews have the potential to be therapeutic for these patients, and can begin a relationship that provides support and allows the patient to learn and grow in the treatment process. The social aspects of interviews with SAD patients are of critical importance, in that, among other reasons, affected individuals may be so anxious about being negatively evaluated that they have delayed seeking treatment, for years or even decades.

Interviews elicit information about SAD symptomatology, yielding data about different patient presentations depending on the subtype of SAD, whether there is comorbid avoidant personality disorder (APD) (or other disorders), and whether there are skill/performance deficits. Individuals presenting with the generalized subtype of SAD, who fear a wide range of social situations (American Psychiatric Association [APA], 2000), and/or APD may be more challenging to engage during the interview because of the degree and extent of the social anxiety. It is wise to assess for both SAD and APD if high degrees of social anxiety, skill/performance deficits, or both are present.

Social (and other) anxieties exist along a continuum or spectrum; they range from no anxiety, to "normal" shyness and commonplace social anxieties, to high degrees of social anxiety, and ultimately to SAD and APD (McNeil, 2010). Determining where on the anxiety continuum the individual falls will allow the clinician to determine if SAD and/or APD in fact are diagnosable. Because of the nature of the disorder, SAD typically is chronic, particularly when generalized, and can negatively affect multiple domains of an individual's life (Buckner, 2009).

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

SAD may accompany a multitude of disorders, which frequently are comorbid, such as other anxiety disorders, depression, or substance abuse; even schizophrenia can be co-existing with high degrees of social anxiety (Lysaker et al., 2010; Yoon & Joormann, 2012; see also **Chapter 10**). Consequently, interviews should be broad. Ideally, interviews for SAD are part of a comprehensive functional assessment that allows not only for exploration of problem areas but determines strengths, and broadly considers various areas of life-health functioning.

While SAD may not be the presenting concern of the patient, the presence of SAD symptomatology still may need to be addressed in the interview as well as in treatment for successful outcomes and improved quality of life. SAD-related issues can impede or obstruct the process of gathering information in a clinical interview. Patients may be reluctant to readily share information due to embarrassment, lack of interpersonal skills to relate information, or being unaware of the social contingencies that affect one's behaviors and socially related cognitions. Interviews may be best if they are iterative; they set the occasion to help patients think about, analyze, and learn more about themselves and their problems.

Due to the complex social nature and chronicity of SAD, individuals presenting with possible symptoms or the disorder itself pose unique challenges to clinicians when conducting clinical interviews. Nevertheless, the interview still is the most vital assessment for this disorder. This chapter focuses primarily on adults; other chapters cover SAD relative to children and adolescents (see **Chapter 9**). Nevertheless, the adult–child/adolescent distinction certainly is not the only developmental consideration in assessing SAD, as will be later noted. This chapter focuses on interviewing with patients who can be diagnosed with SAD, and so will be referred to as "SAD patients." Nevertheless, these same principles apply to those for whom SAD is not the principal diagnosis, and to individuals with a high degree of distress on the continuum or spectrum of social anxiety (McNeil, 2010) whose problems are sub-syndromal and do not meet criteria for SAD.

Conceptualizing the SAD Interview

The clinical interview is the most commonly utilized form of assessment by clinicians in psychotherapy and other clinical endeavors (Cormier, Nurius, & Osborn, 2013; Herbert, Rheingold, & Brandsma, 2010), including SAD. The clinical interview is an essential procedure to determine the patient's concerns, to establish diagnoses, and to develop a functional assessment (Griest, Kobak, Jefferson, Katzelnic, & Chene, 1995). At the heart of the clinical interview is the therapeutic alliance, which is especially important to develop with SAD patients due to the nature of the clinical syndrome. As with any disorder, the quality of the therapeutic alliance is important, and is directly and indirectly associated with treatment outcome (Krause, Altimir, & Horvath, 2011).

Individuals with SAD may believe that their fearful reactions are highly unique and "abnormal" (Herbert et al., 2010). With unstructured interviewing, the assessor may wish to take a psychoeducational approach, helping the patient to learn about SAD as a disorder. Such an approach may help to normalize and validate the patient, allowing

the person to realize that s/he is not so different from others who suffer from SAD, to instill hope, and to communicate that there are evidence-based treatments available. Importantly, the interview can be conducted in such a way as to help the patient understand what to expect from treatment, how treatment may progress, and the role for the patient (e.g., homework).

SAD symptoms vary widely, as the stimulus situations that evoke anxiety differ greatly across patients. As such, there are pitfalls that may be encountered during interviewing. If the interviewer is perceived as an "authority figure," patients who fear those sorts of interactions may have difficulty answering questions and otherwise interacting with the clinician. For some patients with SAD, the interpersonal closeness of a one-on-one interview may be extremely anxiety-provoking. Again, the evocative social situations differ extensively across patients; other SAD patients find individual interviews comforting and less anxiety-provoking than interacting with multiple individuals simultaneously.

Marks' (1987) conceptualization of four types of fear behaviors (i.e., withdrawal, immobility, submission, aggression) provides a basis to consider manifestations of SAD during interviews. Certainly, at least some avoidance is at the core of SAD (and other phobic disorders). Individuals with SAD may *escape* by leaving social situations early or, in interviews, by providing short, minimal answers. They also may *freeze* when giving speeches, in speaking with potential romantic partners, or even in interviews. Camouflage is another possible strategy for patients with SAD who avoid attire or social behavior that may draw attention toward them. Obsequiousness and overpoliteness may function to lessen the possibility of negative social reactions from others, particularly authority figures. Finally, social rudeness or aloofness may be unique manifestations of some forms of high social anxiety, even in interviews (e.g., Kachin, Newman, & Pincus, 2001).

Patients with SAD also may experience difficulty expressing and understanding their own emotions, a potential reflection of emotion-regulation deficits (Glick & Orsillo, 2011; Werner, Goldin, Ball, Heimberg, & Gross, 2011). Adults with SAD generally are considered accurate informants in providing accurate and complete information regarding their symptomatology (Herbert et al., 2010). Children and adolescents with SAD, however, may have a tendency to under-report the severity of their problems (Herbert et al., 2010).

Because of the high degree of fear of negative evaluation by others that typically is associated with SAD (e.g., see Heimberg, Brozovich, & Rapee, 2010), it is important for the therapist to be empathetic, warm, nonjudgmental, and accepting to the individual. At the same time, however, it also is appropriate for the clinician to communicate his/her expertise as an expert, authoritative (as opposed to authoritarian) therapeutic figure (Cormier et al., 2013; Krause et al., 2011). Sometimes SAD patients (and others) are particularly sensitive to clinicians' facial expressions and other nonverbal behaviors, so clinicians' awareness about such issues is well advised (Herbert et al., 2010). Building rapport and trust is important with virtually all patients, but perhaps more so for patients with SAD-related issues due to the nature of their problems (Fluckiger et al., 2012; Johansson & Jansson, 2010). Developing a therapeutic alliance with a patient with SAD is critical, as it provides a learning opportunity for the patient to develop and maintain a relationship with another person, acknowledging, of

course, the unique nature of the therapeutic relationship. Just as learning contributes to the development and maintenance of SAD (McNeil, Lejeuz, & Sorrell, 2010), learning new skills and strategies is inherent in treatment; this new learning can be primed or even initiated during initial interviews.

Another concern with treatment-seeking individuals suffering from SAD is that they may cancel or not show up for initial or subsequent sessions (Griest et al., 1995). First and early sessions may be especially difficult for the patient due to the social novelty of the situation. If an individual with SAD does not attend the intake session, it may not be due to an absence of desire to seek assistance, but rather to an intense fear of being negatively evaluated or judged (Griest et al., 1995). In fact, many individuals who suffer from SAD never access treatment due to the intensity of their fears and anxieties (Amir & Taylor, 2012). Therefore, when SAD patients do appear for intake appointments, it is imperative that clinical interviews be completed in such a way that they not only elicit information and help to establish diagnoses but also help the patient to become engaged in and receptive to treatment.

Interviewing and psychotherapy involve (sometimes intense) social interactions. Thus, it may be helpful to ask early in the first meeting how the patient feels while participating in the interview, and what thoughts and physiological reactions s/he is experiencing, in part to assess and in part to acknowledge the interview as a potentially anxiety-provoking situation. An appropriate follow-up may be to ask what the clinician might do to help reduce the anxiety during those early contacts.

Conducting SAD Interviews

Interview Environment

The physical environment for the interview should be arranged so as to help patients with SAD feel safe and secure in a novel setting since they may experience distress in communicating with the clinician (Beidel & Turner, 1998). In relation to setting, Griest et al. (1995) recommend that interviews with SAD patients be in a room that allows the clinician and patient to sit as comfortably apart as necessary, with muted lighting to decrease level of arousal. They also suggest that there be no disturbances during the clinical interview. Allotting adequate time for the session (e.g., 60–90 min), especially initially, to build rapport with the patient also is important, although the duration of such appointments likely is dictated by clinic policy or third-party payers.

At clinics with observational windows or one-way mirrors, the scrutiny involved in such an environment may be difficult for patients with SAD. Audio or videotaping of sessions in training clinic situations may evoke anxiety in SAD patients. Having to interact with more than one interviewer, or an interviewer and his/her supervisor, also may elicit anxiety. All of these approaches may be both necessary and appropriate to insure high-quality clinical care and to meet other needs (e.g., clinical training); nevertheless, SAD patients would be well served in working with clinicians who are sensitive to the anxiety that may be associated with such procedures.

Approaches to Interviewing

There are various suggestions regarding the balance and sequencing of open-ended and closed-ended questions in initial interviews with SAD patients (Beidel & Turner, 1998; Griest et al., 1995; Herbert et al., 2010). Some advocate that frequent use of closed-ended questions helps to decrease arousal level, because there is less demand on the patient to create a response. Alternately, open-ended questions are recommended to elicit greater information and to highlight the importance of the patient's feelings and thoughts. Regardless, every patient is unique, and the best practice in the case of unstructured interviews is for the session to be conducted in a way that is ideographic and fits well with the individual patient.

One of the strategies that often is used by some highly socially anxious individuals to avoid being the center of attention (either with or without awareness) is keeping the focus on the other person in a conversation, perhaps by repeatedly asking questions. In an interview, such a strategy is impossible for the patient. Thus, the exclusive focus on the patient that is inherent to interviews may be highly anxiety-provoking for patients with SAD. Even "small talk" early in an interview can be extremely difficult for some patients. While clinicians may engage in casual conversation to try to help patients feel more comfortable (or may use it as a way to assess the patient's social skills), there should be awareness that it may be quite anxiety-provoking. Ideally, there will be "give and take" across an interview session, avoiding interrogating or "staccato" questioning (i.e., question after question, repeatedly and quickly) of the patient, who may revert to yes-or-no answers, particularly to closed-ended questions.

Self-report instruments are an important part of assessment for SAD; their use is covered in elsewhere in this volume (see **Chapter 14**). Clinicians may wish to use them during or alongside interviews, to provide structure to the social interaction, which may be comforting to patients, particularly initially in treatment. Review of the items of a questionnaire with a patient after s/he completes it may further elucidate the nature and function of symptomatology, particularly if the interviewer asks the patient to expand upon, or to describe situations, that led to a response to a particular item (E. Krackow, personal communication, November 2012).

Liberal but not overwhelming use of reassurance, validation, and praise for overcoming anxiety to participate in an interview may be helpful for many patients with SAD. *Labeled praises* (e.g., McNeil & Hembree-Kigin, 2010) can be utilized effectively to specifically note the positive behaviors of the person participating in the interview. For example, the clinician may say "I've asked you a lot of questions today, and I think some of them made you feel uncomfortable. But, you've done a great job sharing just as much as you possibly can, and I appreciate that, because you're helping yourself."

Motivational Interviewing: SAD Interviewing and Preparation for Treatment

As already noted, bombarding SAD patients with constant questions, even if they are open-ended, is less than ideal in an interview. Taking the pressure off the patient by not continuing to ask question after question for long stretches of time may help

him/her feel more comfortable. Thus, it is wise to incorporate other approaches such as *reflection, paraphrasing, summarization, affirmation*, and *self-disclosure* to assist in building rapport and reducing the patient's arousal. Using reflections, as in motivational interviewing (MI; Miller & Rollnick, 2013), can be a helpful way of responding to patients in trying to build rapport. The use of *reflections* during the interview displays empathy and a degree of understanding for the patient's concerns and difficulties associated with SAD (Cormier et al., 2013). Particularly early on, the reflections may fall flat because the patient is cognitively "busy" and/or physiologically aroused, and may genuinely not know how to respond. Reflections also can be viewed as helping individuals with SAD to manage, discriminate, and effectively communicate their feelings more effectively to enhance treatment (Cormier et al., 2013). *Paraphrasing* can be used as a type of reflection to transmit understanding to, and allow for prompt elaboration by, the patient. In addition, the use of paraphrasing can assist the clinician in focusing on a particular component of what the patient has communicated about social- and performance-based anxieties. Typically used prior to moving along to a new topic, the clinician also can incorporate a *summarizing* statement which highlights the themes of the patient's concerns and indicates that the clinician understands (Miller & Rollnick, 2013).

Affirmation, utilized in MI, is a method for acknowledging a patient's positive or effective behavior in hopes of building his or her confidence (Arkowitz & Westra, 2009; Miller & Rollnick, 2013). As an example, an affirmation may be reasonably expressed after a patient with SAD expresses that he or she was able to approach another individual and start a conversation despite being anxious about being negatively evaluated. Also, limited use of *self-disclosure* on the part of the clinician can enhance the therapeutic bond with a SAD patient. Self-disclosures may best be used cautiously, particularly early in therapeutic contacts, and then incorporating them a bit more once the initial therapeutic alliance has been established (Krause et al., 2011). Properly and sparingly used, self-disclosures can enhance the affective bond between the clinician and the patient, thus increasing comfort and building trust (Krause et al., 2011).

Allowing some silence, and then filling in the space and transitioning the patient into the next part of the conversation, can be helpful for patients with SAD. The interviewer should be prepared to allow some silences but, particularly early on, may wish to avoid long pauses, as they may put undue pressure on the patient. Later in treatment, such pauses can be used therapeutically, as a form of exposure, to prompt the patient to take some responsibility for keeping the conversation going. In later interviews, a discussion about silences may be productive, both in terms of the process of psychotherapy, and as a potential area for social skills training, if needed (see *Behaviors to Consider During Interviews*, below, for additional details on this issue). Patients with SAD, in particular, may need silent times during interviews and therapy to collect their thoughts, given the social demands of the interaction with the clinician.

Since treatment for SAD involves direct contact with potentially anxiety-evoking (social) situations, even with the clinician, the patient understandably may be vulnerable to discontinuing treatment after an intake interview. Learning that therapy involves at least some exposure to social situations also may engender anxiety that could lead to avoidance of treatment. This internal struggle within a patient of

determining if treatment is necessary and if it likely will be beneficial can be compared to a state of *ambivalence* as conceptualized in MI. Using an MI approach, the patient's ambivalence about initiating and maintaining treatment can be explored, exploring both sides of the argument. The clinician can help guide the patient through this process, which may yield a decision that treatment is (or is not) necessary and a worthwhile investment of the anxiety that will be produced as a result of change. MI added to various treatment methodologies such as cognitive behavior therapy has been found to be successful with individuals who have SAD-related disorders (Westra, 2012). Specifically, MI has been found to improve treatment-seeking behaviors as well as SAD symptomatology (Buckner, 2009). When MI has been added as a supplement to CBT, SAD symptomatology has improved significantly (Buckner, 2009; Westra, 2012).

Behaviors to Consider During Interviews

Interview behaviors serve as samples of the patient's overall behavioral repertoire and, importantly, can provide information about social skill and performance in one-on-one interactions. Clinicians should note the paralinguistic qualities (e.g., volume, pitch, rate of speech, length of pauses, and fluency) of the patient's communications (Cormier et al., 2013; Lewin, McNeil, & Lipson, 1996). Low vocal volume can be an issue; furthermore, individuals with SAD may have long and/or frequent pauses when speaking (Lewin et al., 1996). As noted previously, pauses and silences are important components of the interaction between the patient and the clinician (Sharpley, Munro, & Elly, 2005). Nonverbal communication also should be noted during the interview, including such behaviors as eye contact, trembling or shaking, blushing, and rigidity or fluidity of posture (Angelico, Crippa, & Loureiro, 2010; Bogels et al., 2010; Cormier et al., 2013).

Interviews provide an important basis for initial social skills and performance assessment. There has been controversy in the literature about skill- and performance-based deficits regarding social anxiety (Hopko, McNeil, Zvolensky, & Eifert, 2001; see also **Chapter 17** for extended discussion of this issue). Most important to note is that not everyone who is diagnosed with SAD has social skill deficits, as is sometimes assumed. Skill deficits *may* be a factor in the development as well as the maintenance of SAD, which is why social skills training may be beneficial to *some* SAD patients (Hopko et al., 2001). Many individuals with various forms of SAD, however, are quite adept in social interactions. Some SAD individuals with circumscribed public speaking phobia (McNeil et al., 1995), for example, are adroit in *delivering* speeches, yet are extremely troubled by their physiological and cognitive reactions in anticipation of, during, and while reflecting after the speech. Alternately, an individual with SAD may have deficits in the ability to *perform* in social settings due to intense physiological or cognitive arousal which may not be related to an actual skill deficit (Hopko et al., 2001). When avoidance or escape from social or performance settings is not possible for individuals with SAD, performance deficits can result, which may be a product of lack of skill (in that the individual never learned it) or high anxiety (Hopko et al., 2001).

A significant degree of social anxiety may prevent an individual from acquiring the necessary skills to perform effectively in social settings (Hopko et al., 2001). Degree of social skill can be classified on a continuum, much like the degree of anxiety (Hopko et al., 2001; McNeil, 2010). For an individual to perform effectively in social settings, s/he must have an appropriate level of social perception, which is inclusive of an understanding of social expectations in specific social situations and knowledge of conversational and contextual cues (Hopko et al., 2001). It should be noted that some individuals with SAD are socially hyper-aware, attending and reacting (sometimes excessively) to very subtle social cues—perhaps even to cues that they expected to occur but did not, or which they only imagined. An appropriate level of social awareness may be difficult to achieve given that individuals with SAD typically have inaccurate perceptions of their social skills and interactions with others (Hopko et al., 2001). The interview provides a means to begin to discover how well or poorly a patient performs in one-on-one social interactions, whether deficits are related to a lack of skill, and if a high degree of anxiety is present and whether it precludes the patient from interacting skillfully.

Examining the Subtype and Pervasiveness of SAD

Most treatment-seeking individuals with SAD will be of the generalized subtype, as the pervasiveness of the condition affects quality of life to the extent that it drives the individual to seek help. Nevertheless, specific or circumscribed subtypes of SAD also can be so severe that they prompt the affected person to present for treatment. Individuals with specific public speaking fear (which is a type of SAD, but may be better conceptualized as a specific phobia; McNeil, 2010) typically can avoid that situation, but may seek professional care when their career situation changes (e.g., a promotion) that disallows continued avoidance. Similarly, a former patient with a specific social phobia of using public restrooms (including single stalls on airplanes) had been able to cope for years by restricting liquid intake, and renting motel rooms adjacent to airports whenever he traveled, specifically to use the restroom immediately before or after an airplane flight. Even that strategy became infeasible with increased family demand to travel, thus requiring the individual to seek treatment.

Careful interviewing, perhaps incorporating selected self-report instruments, can provide an adequate functional assessment, which allows a determination of the type and level of social anxiety/fear, and whether SAD is diagnosable. Interviewing the patient about the number and types of social situations that cause significant distress is a critical component in determining the subtype of SAD, and also whether APD may be present.

Rapee and Sanderson (1998) discussed the importance of elucidating details about distressing social situations, as well as the degree to which the individual's quality of life is impaired due to social restriction or other aspects of the condition. Herbert et al. (2010) stressed the necessity of assessing the individual's unique pattern of symptoms in social situations. Identifying common elements of anxiety evocation across situations (e.g., fear of negative evaluation) and typical reactions by the patient

(e.g., escape) will help to formulate a treatment plan. One approach that the clinician may take in an interview is to ask the patient to imagine an assortment of social situations, and then to talk about each one, reporting subjective units of distress and associated cognitions at various points. In addition, the clinician also could ask the patient to report on the most recent instance of a social situation and to recount what happened. To fully capture the patient's experience of the situation, the clinician also may inquire about strengths, such as identifying comfortable social situations and/or ones in which the patient believes s/he performs well.

Common Conditions to be Assessed and Differential Diagnosis

Conditions that frequently are comorbid with SAD include psychoactive substance abuse or dependence, other anxiety disorders, mood disorders, and APD (see also **Chapter 10**). SAD also is highly associated with suicidal ideation (Buckner, 2009; Chartrand, Cox, El-Gabalawy, & Clara, 2011; Nepon, Flett, Molnar, & Hewitt, 2011). Some individuals who suffer with SAD resort to the consumption of alcohol or other drugs to relieve or decrease the physiological and cognitive arousal that they experience in social- or performance-based settings (Herbert et al., 2010; O'Grady, Cullum, Armeli, & Tennen, 2011). It may be beneficial for the clinician to ask patients with SAD how they cope with their social anxiety in an open-ended way. If alcohol or other drug use is not mentioned, the clinician could discuss with the patient how some individuals cope with anxiety by consuming alcohol or other substances before, during, or even after a social or performance-related event to reduce arousal, and then ask if the patient has ever had to depend on such coping strategies.

SAD and depression are highly comorbid; SAD confers a three times greater risk of developing subsequent depression (Horn & Wuyek, 2010). SAD with major depressive disorder (MDD), relative to MDD alone, is associated with greater suicide risk, perhaps related to a sense of disconnection from others (Davidson, Wingate, Grant, Judah, & Mills, 2011). In addition, individuals with comorbid SAD and depression have greater rates of rumination after social events, leading to an exacerbation of symptomatology (Kocovski, MacKenzie, & Rector, 2011; Nepon et al., 2011). Consequently, assessing for mood disorders and conducting a suicide risk assessment are essential components of intake interviews in cases of suspected SAD.

Individuals meeting criteria for generalized SAD also often meet criteria for APD as well (Hofmann, 2010; Rodebaugh, Gianoli, Oltmanns, & Turkheimer, 2010). According to one estimate, approximately 89% of individuals with the generalized subtype of SAD also meet diagnostic criteria for APD (Huppert, Strunk, Ledley, Davidson, & Foa, 2008). Others report that the comorbidity between APD and SAD ranges from 21% to 90% (Boone et al., 1999). APD is highly similar to the generalized subtype of SAD (Boone et al., 1999; Hofmann, 2007; Huppert et al., 2008), and is distinct from circumscribed social phobias. APD and its relation to SAD is the source of much controversy (Herbert et al., 2010). It has been suggested that APD may simply represent a more severe form of SAD (Boone et al., 1999; Huppert et al., 2008; McNeil, 2010; Rodebaugh et al., 2010). Both SAD and APD are characterized

by inhibition and avoidance associated with social situations (Rodebaugh et al., 2010). Both disorders can be diagnosed concurrently due to ambiguous distinctions between the diagnostic criteria (Herbert et al., 2010).

It is important to determine whether personality disorder(s) are present in addition to SAD. Along with APD, Cluster A (i.e., schizoid, schizotypal, and paranoid) personality disorders, as categorized by the *Diagnostic and Statistical Manual of Mental Disorders, fourth edition* (APA, 2000), should be particularly considered during interviews and continuing into treatment. As with most Axis II disorders, being given a diagnosis of APD has been associated with poorer treatment outcomes (Huppert et al., 2008). Individuals who have been diagnosed with both SAD and APD have been found to have greater impairment than individuals with only one of these diagnoses (Huppert et al., 2008). Another important factor to note is that APD is more commonly found in individuals with depression than SAD, and also is frequently comorbid with a multitude of other anxiety disorders such as obsessive-compulsive disorder and panic disorder (Huppert et al., 2008).

In terms of differential diagnosis, it sometimes is challenging to distinguish the generalized subtype of SAD from agoraphobia, if the distressing situations are primarily or solely social ones. Anxiety about and avoidance of crowds, for example, may be manifested in both SAD and agoraphobia. With SAD, the concern about crowds likely is related to one having to engage in social interactions and to deal with social demands, while in agoraphobia, not being able easily to get to a "safe" place away from the crowd is the more likely focus. Panic attacks can be manifested in SAD; if situationally bound or predisposed to social events, these are distinct from panic disorder. Social hypersensitivity, and even suspiciousness about others, may be observed in SAD and APD, and may need to be distinguished from paranoia seen in other personality disorders and even psychotic disorders by the level and pervasiveness of such concerns.

Cultural and Developmental Considerations

Hofmann, Asnaani, and Hinton (2010) describe SAD as being culturally dependent and a function of social roles and expectations. Similarly, Schreier et al. (2010) emphasize the role of cultural norms and values in the development of SAD. In assessing for SAD and related disorders, the clinician should attempt to understand the culture(s) in which the patient developed (or is developing) and in which he or she has lived or is living (see also **Chapter 11** for expanded discussion of this issue). While a discussion of cultural competencies in the assessment and treatment of psychological problems is beyond the scope of this chapter, there are helpful resources in this area (e.g., Comas-Díaz, 2012). Hofmann et al. (2010) recommend that clinicians conceptualize SAD in relation to the individual's reference group, inclusive of culture, for the most accurate assessment. Social behaviors are, in part, culturally determined, so an understanding of patients' past and present cultural influences is important during interviewing. As an example, what could be perceived as a lack of assertiveness may in fact represent the patient adhering to the norms of his/her culture.

For instance, historically in some American Indian plains tribes, adult males did not speak directly with their mother-in-law due to a cultural taboo (Kuiper, 2011). While this restriction may currently not be formalized, the cultural roots still may influence social behavior today.

Clinicians would be well advised to consider their own cultural background(s) and how they may impact interviews with patients, who bring to the table their own cultural histories and influences. Nonverbal aspects of communication are partly determined by culture. For example, in some groups, children do not maintain eye contact with adults, as doing so would be too bold, and even rude. Interviewers can misattribute such behavior, assuming that it reflects SAD or shyness or social anxiety in children, when it may be normative for the individual's culture.

For the interviewer, considering whether his/her own background is more individualistic or more collectivistic, and assessing that dimension for the patient, is one basic distinction. In collectivistic cultures, embarrassing someone else is more distressing than embarrassing oneself; this concept may be thought of as producing shame for others through one's actions (Hofmann et al., 2010). According to Schreier et al. (2010), the prevalence of SAD is higher in collectivistic cultures than in individualistic cultures; self-criticism is greater in individuals with SAD from more collectivistic cultures.

Another distinction between Western cultures (more individualistic) and Eastern cultures (more collectivistic) is a specific disorder, *Taijin Kyofusho* (TKS), which is diagnostically similar to SAD, but predominately diagnosed in Eastern cultures (Chapman, Mannuzza, & Fyer, 1995). Specifically, TKS involves a fear of being watched or observed in social settings (Hofmann et al., 2010), and fears relating to offending another individual through excessive blushing or even body odor (Chapman et al., 1995). Interviews with individuals who have a collectivistic background should address the possibility of TKS.

A variety of demographic and cultural factors affect manifestations of SAD. While SAD certainly is evidenced differently in childhood and adolescence relative to adulthood, there are indications that it varies across the adult lifespan as well (Ciliberti, Gould, Smith, Chorney, & Edelstein, 2011). Interviews should be structured so as to be sensitive to a patient's station and stage in life. Older adults, for example, may have more social concerns about asking for help, looking incompetent, healthcare visits, and being noticed when one forgets information (Ciliberti et al., 2011). Males and females may differentially experience certain social situations (e.g., asking for directions), thus affecting social fears. SAD appears to be more common in rural than in urban areas (McNeil, 2010). Other factors to consider are social status, gender role, and sexual orientation identification (Hofmann et al., 2010). In addition to developing cultural knowledge and other cultural competencies generally, one option is for the clinician to openly discuss the role of culture in relation to the disorder, gaining feedback from the patient throughout treatment (Berg-Cross & So, 2011). When conducting a functional assessment of SAD-related concerns, issues such as cultural identity, acculturation, worldview, values, potential stigmas, and cultural meaning of the presenting problem(s) all are important to consider (Cormier et al., 2013).

Structured and Semi-Structured Clinical Interviews and Symptom Assessment

While unstructured interviews are the most common type of SAD interview, and can be very useful in assessment, structured approaches offer certain advantages (e.g., empirically demonstrated reliability) but limitations (e.g., rigidity) as well. Consistent with the varied responses to the stimulus aspects of social situations among patients with SAD, the use of these more structured approaches may be comforting to some, who find the highly structured format reassuring in that it clearly demarcates how and when they should respond. These approaches, however, may be off-putting to others, who find the repeated questioning demanding and restricting.

Diagnostic Interviews

While contemporary comprehensive interview assessments all assess for SAD to one degree or another, only those commonly used in SAD research or practice for adults will be presented here.

Anxiety Disorders Interview Schedule for DSM-IV The Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV) is a semi-structured assessment that is designed to be consistent with DSM-IV criteria and to differentiate among anxiety and mood disorders (Brown, DiNardo, & Barlow, 1994; Herbert et al., 2010; Hook, Hodges, Whitney, & Segal, 2008; Majdandžić, de Vente, & Bögels, 2010). The ADIS-IV also assesses for other disorders such as substance abuse and dependence, and additional disorders that have commonalities with various anxiety disorders (Hart, Jack, Turk, & Heimberg, 1999). The ADIS module on SAD is composed of 2 initial questions, with multiple sub-questions pertaining to negative evaluation/judgment by others, feeling uncomfortable in social settings, and separate fear and avoidance ratings of 13 specific social situations (with an option to identify other troubling events). If there is evidence of clinically severe social fear or avoidance, the clinician continues to ask the patient another series of eight questions composed of multiple subquestions which are designed to assess the severity of SAD, physiological symptomatology, and past experiences that may have led to the development of SAD (Brown et al., 1994).

An advantage of the ADIS-IV for diagnosing SAD is that it provides detailed information regarding anxiety and avoidance patterns associated with social interaction- and performance-based settings (Herbert et al., 2010; Hook et al., 2008). Specifically, the ADIS-IV examines behaviors, cognitions, degree of avoidance, and the individual's past experiences with the disorder (Griest et al., 1995). There also are parent (ADIS-P) and child (ADIS-C) versions of the ADIS-IV for use with children and adolescents (Beidel & Turner, 1998; Herbert et al., 2010). The ADIS-IV has good inter-rater reliability across diagnoses ($.60 < \kappa < .86$) (Codd, Twohig, Crosby, & Enno, 2011), with a κ of .79 for the diagnosis of SAD (Beidel & Turner, 1998). A lifetime version, the ADIS-IV-L, also is available (Hart et al., 1999). In regard to diagnosing SAD, the ADIS-IV-L has reasonable reliability ($\kappa = .64$) (Hart et al.,

1999). The ADIS-IV-L is highly regarded for its coverage of the spectrum of anxiety disorders as well as its user-friendly format (Hart et al., 1999).

Structured Clinical Interview for DSM-IV The Structured Clinical Interview for DSM-IV (SCID-IV) is semi-structured, hierarchical in nature, and designed to assess for Axis I and II disorders from the DSM (First, Spitzer, Gibbon, & Williams, 2005; Hook et al., 2008; Sanchez-Villegas et al., 2008). It covers a broad range of psychological disorders, inclusive of SAD (Herbert et al., 2010). The SAD module of the SCID consists of items that evaluate whether the individual experiences discomfort doing tasks in the presence of others, feels anxiety, and/or actively avoids social/performance situations, and has an awareness of the irrationality of the anxiety, as well as the impact that SAD has on his/her life. The duration of the disorder also is assessed. There are various versions of the SCID-IV for clinical and research purposes (First et al., 2005).

The SCID-IV has been found to provide an accurate and reliable diagnosis of SAD (Crippa et al., 2008). This instrument has moderate inter-rater reliability ($.47 < \kappa < .57$) for current and lifetime diagnoses of SAD (Beidel & Turner, 1998). The SCID-IV has been criticized for not providing the depth of information that other assessments (e.g., the ADIS-IV) of SAD provide in relation to symptomatology (Hart et al., 1999).

Schedule for Affective Disorders and Schizophrenia The Schedule for Affective Disorders and Schizophrenia (Endicott & Spitzer, 1978) is historically important, but has been criticized for not eliciting sufficient information for making accurate differential diagnoses among the anxiety disorders (Hart et al., 1999; Rogers, Jackson, Salekin, & Neumann, 2003). This instrument has good inter-rater reliability ($.70 < r < .94$) for diagnoses and individual symptomatology (Beidel & Turner, 1998; Rogers et al., 2003), with average test-retest reliability ($r = .67$; Beidel & Turner, 1998). The interview has been criticized for limited assessment of anxiety disorders (Beidel & Turner, 1998); its general focus may impede the specific determination of SAD and other anxiety-related disorders.

A lifetime version of the Schedule for Affective Disorders and Schizophrenia modified for anxiety disorders (Mannuzza, Fyer, Klein, & Endicott, 1986) is available and assesses symptomatology based on DSM criteria (Beidel & Turner, 1998; Griest et al., 1995; Hart et al., 1999). In relation to SAD, the Schedule for Affective Disorders and Schizophrenia—Lifetime Anxiety version assesses social anxiety in 10 different social settings (Beidel & Turner, 1998). This instrument has adequate inter-rater reliability ($.68 < \kappa < .71$) for both current and lifetime diagnosis of SAD (Mannuzza et al., 1986).

There also are present and lifetime versions of the Schedule for Affective Disorders and Schizophrenia for youth ages 6–18 years, the "Kiddie SADS" (K-SADS); these tools have been found to be effective in assessing anxiety disorders in children (Beidel & Turner, 1998). The K-SADS begins with two screening items to determine if the child is uncomfortable in or anxious about social settings. If either item suggests problems, then the clinician proceeds to inquire about the child's comfort level around individuals with whom the child interacts frequently. The clinician also

inquires about impairment and avoidance across a variety of interactions with peers and adults outside of the family, and settings such as school and home. The K-SADS has adequate to strong inter-rater reliability ($\kappa = .88$) for the diagnosis of affective and anxiety disorders in children (Beidel & Turner, 1998). Present and lifetime versions of the K-SADS have been found to have strong psychometric properties (Birmaher et al., 2009).

Structured Clinical Interview for Social Anxiety Spectrum The Structured Clinical Interview for Social Anxiety Spectrum (SCI-SHY 3.0) provides an assessment of a broad spectrum of symptomatology associated with social anxiety, shyness, and SAD (Dell'Osso et al., 2002). This instrument allows evaluation of individuals who may fall below clinical diagnosis levels based on DSM criteria (Dell'Osso et al., 2002). The design of the SCI-SHY (and other spectrum-based assessments) is more aligned with a dimensional approach to psychopathology than a categorical approach (Dell'Osso et al., 2002). This quality is noteworthy, in that SAD may be somewhat more difficult to delimit along a continuum or spectrum (Dell'Osso et al., 2002), given the unique social nature of the disorder, among human beings who are social creatures.

The SCI-SHY 3.0 consists of 168 items that assess multiple domains of SAD. It includes items that address shyness in childhood, social interactions with individuals of varying social status, behavioral and physiological characteristics of SAD, performance fears or concerns, and escape behaviors (e.g., use of various substances to reduce arousal) (Cassano et al., 2002). A self-report version of the SCI-SHY, the SHY-SR, has been developed, consisting of 164 items that cover 4 domains associated with SAD, which are *social phobic traits during childhood and adolescence*, *interpersonal sensitivity*, *behavior inhibition and somatic symptoms*, and *specific anxieties and phobic features* (Dell'Osso et al., 2002, p. 83). Generally, the self-report version and clinician-administered interview have been shown to be comparable overall, although it was suggested that the interview version may be associated with symptom minimization by the patient (Dell'Osso et al., 2002). The SCI-SHY has strong psychometric qualities, including discriminant validity, concurrent validity, and inter-rater reliability (Dell'Osso et al., 2002).

Symptom Severity Assessments

Liebowitz Social Anxiety Scale The Liebowitz Social Anxiety Scale (LSAS) is a 24-item clinician-administered assessment of fear and avoidance associated with SAD based on various social settings (Liebowitz, 1987). The LSAS is unique in its assessment of SAD, focusing on situations or settings rather than symptomatology (Beard et al., 2011; Heeren et al., 2012). Requiring 20–30 min to administer, items on the LSAS are divided fairly evenly between social ($n = 11$) and performance ($n = 13$) settings (Hart et al., 1999; Herbert et al., 2010). Sample items include being observed while completing various tasks (e.g., consuming food, performing), being the center of attention in a room or area, and approaching individuals in social settings. The LSAS yields four scores (i.e., social fear, social avoidance, performance fear, and performance avoidance), as well as an overall severity rating (Liebowitz,

1987); total avoidance and total fear subscale scores also can be calculated (Hart et al., 1999).

The LSAS has been validated in a sample of 382 individuals presenting with SAD (Orsillo, 2001). It has been shown to have adequate to strong internal consistency ($.81 < \alpha < .96$), sensitivity, and concurrent validity with other scales that assess SAD (Hart et al., 1999; Hedman et al., 2011; Herbert et al., 2010; Safir, Wallach, & Bar-Zvi, 2012). Test-retest reliability over 1 week is strong ($r = .97$; Hedman et al., 2011). The LSAS also has adequate divergent validity in relation to measures of depression ($.52 < r < .56$; Orsillo, 2001). The LSAS Social Fear subscale has been found to differentiate between generalized and nongeneralized subtypes of SAD (Hart et al., 1999). The factor structure of the LSAS has been questioned due to a high degree of overlap between the performance- and social-based item domains (Griest et al., 1995; Hart et al., 1999; Herbert et al., 2010).

Besides the clinician-administered version of the LSAS, there is a self-report version. The Liebowitz Social Anxiety Scale—Self-Report (LSAS-SR) consists of 24 items that also assess fear and avoidance associated with various social settings (Beard & Amir, 2010; Hedman et al., 2011; Herbert et al., 2010). The LSAS-SR has been found to be psychometrically similar to the clinician-administered version (Beard & Amir, 2010). A version modified from the adult instrument also has been developed for youth from ages 7–18 years, the Liebowitz Social Anxiety Scale for Children and Adolescents (LSAS-CA; Masia-Warner et al., 2003). It has been administered to children and adolescents with SAD and has been found to have good internal consistency ($.83 < \alpha < .95$; Storch et al., 2006). The LSAS-CA also has good test-retest reliability over a 1-week period, with coefficients ranging from .89 to .94; there is evidence for the discriminant validity of the LSAS-CA (Storch et al., 2006).

Brief Social Phobia Scale Davidson et al. (1997) developed the Brief Social Phobia Scale (BSPS), which contains 18 items and assesses SAD in relation to three domains (i.e., *avoidance*, *fear*, and *physiological symptoms*) within the last week (Griest et al., 1995; Herbert et al., 2010; Orsillo, 2001; Osorio, Crippa, & Loureiro, 2010). Scores can range from 0 to 72 with higher scores indicative of greater impairment; a score of 20 or greater is considered clinically significant (Kelly, Walters, & Phillips, 2010). Sample BSPS items include anxiety or avoidance in approaching other individuals of varying social status, attending social gatherings, and being negatively evaluated (Orsillo, 2001). BSPS administration is simple and takes only 5–10 min to complete; it has been recommended that this instrument be integrated after the initial interview (Orsillo, 2001). According to Orsillo (2001), the BSPS has been administered to multiple samples, one of which consisted of 275 individuals presenting with SAD (mean total score on the BSPS = 41.6). The BSPS has sound psychometric properties, including inter-rater reliability ($r = .99$), internal consistency ($.60 < \alpha < .90$), test-retest reliability of 1 week ($.91 < r < .98$), and concurrent validity with other scales assessing SAD and social anxiety, such as the LSAS ($.70 < r < .72$) (Griest et al., 1995; Osorio et al., 2010). According to Herbert et al. (2010) and Hart et al. (1999), the strengths of the BSPS are its straightforward administration protocol, brevity, and inclusion of physiological assessment. It should be noted, however, that the "physiological" assessment is only the patient's perception of such responses;

moreover, it includes only symptoms that may be noticed by others, such as blushing or sweating (Hart et al., 1999; Orsillo, 2001). Similar to the LSAS, the factor structure of the BSPS has been questioned, as several items load on multiple factors (Hart et al., 1999).

Summary and Recommendations

Interviewing is the primary and most important assessment method for SAD (and other psychological disorders). Both unstructured and structured interview formats, in addition to multiple symptom severity assessments, can be used to gather necessary information about patients and their disorders, backgrounds, and lives.

Interviews with patients who have SAD, high social anxieties, or related personality disorders (e.g., APD) present both opportunities and challenges. One of the first priorities and recommendations regarding initiating treatment with a patient who presents with a high degree of social anxiety is establishing and maintaining rapport. Creating a therapeutic relationship may require (extra) time, sensitivity, and clinical skill in work with patients with SAD-related disorders and conditions. From the first contact, there is a need to make a "connection" with the patient, although perhaps not "too close," as that may be uncomfortable for him or her. Careful consideration is needed regarding the clinical environment (e.g., the arrangement of furniture, lighting, minimization of interruptions) as well as therapeutic techniques (e.g., open vs. closed questions, reflections, affirmations). Motivational approaches (e.g., MI) may be particularly helpful in engaging the patient during interviews. Clinicians should utilize reassurance, praise, and validation of the client's disclosures, thus reinforcing social interaction and reinforcing trust. Trying to understand patients in (functional) context, and the environments in which they live, work, and play, is a crucial component of clinical assessments, but is only possible if there is a sufficient relationship that allows the patient to be comfortable in communicating this information to the clinician. Therefore, the development of trust and a working alliance is crucial. Interviews involving individuals with SAD (and other disorders) have tremendous potential in being psychoeducational for the patient. Taking a psychoeducational approach with an individual with SAD assists in building rapport as well as improving treatment through greater understanding of the individual's disorder.

SAD is a complex composition of physiological arousal, negative cognitions, and overt behaviors (e.g., avoidance). Interviews should assess all of these areas, and ideally will be supplemented with questionnaires as part of a multi-method multi-trait approach. Also ideally, comprehensive assessment includes psychophysiological assessment and behavior tests. Although highly important, interviews are inherently limited in that they rely on verbal report, with all the attendant issues of such an approach, including that of retrospective reporting. The fact that there are shortcomings with interviews and self-report assessments further highlights the need for additional assessment methods (e.g., see **Chapter 16** for additional details on behavioral assessment of SAD).

Nevertheless, interviews allow clinicians to observe certain social behaviors of the patient in real time. In addition to serving as an observation of social behavior and

gathering information about the patient, interviews also serve the important function of social exposure. Further, interaction with the clinician can serve to improve social skills if the patient presents with social-related behavioral deficits. Moreover, the clinical interview allows for differential diagnosis (e.g., from agoraphobia) and examination of other potential comorbid disorders (e.g., other anxiety disorders, depression, and APD). In addition to assessing for comorbid conditions, examination of coping mechanisms (e.g., alcohol or other drug-related use) also is pivotal. Potential cultural differences and other ideographic differences (e.g., sexual orientation, gender roles) also should be considered and addressed in the clinical interview to enhance sensitivity and to improve treatment.

The clinical interview, as highlighted in this chapter, has a pivotal role in assessment and treatment with SAD and related disorders. Carefully and positively conducted interviews can establish a strong foundation for later treatment with patients suffering from SAD and related conditions.

Acknowledgments

Thanks are extended to colleagues in the Anxiety, Psychophysiology, and Pain Research Laboratory in the Department of Psychology at West Virginia University, whose ideas helped shape this chapter.

References

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

Amir, N., & Taylor, C. T. (2012). Interpretation training in individuals with generalized social anxiety disorder: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 80*, 497–511.

Angelico, A. P., Crippa, J. A. S., & Loureiro, S. R. (2010). Social anxiety disorder and social skills: A critical review of the literature. *International Journal of Behavioral Consultation and Therapy, 6*, 95–110.

Arkowitz, H., & Westra, H. A. (2009). Introduction to the special series on motivational interviewing and psychotherapy. *Journal of Clinical Psychology, 65*, 1149–1155.

Beard, C., & Amir, N. (2010). Negative interpretation bias mediates the effect of social anxiety on state anxiety. *Cognitive Therapy and Research, 34*, 292–296.

Beard, C., Rodriguez, B. F., Moitra, E., Sibrava, N. J., Bjornsson, A., Weisberg, R. B., & Keller, M. B. (2011). Psychometric properties of the Liebowitz Social Anxiety Scale (LSAS) in a longitudinal study of African Americans with anxiety disorders. *Journal of Anxiety Disorders, 25*, 722–726.

Beidel, D. C., & Turner, S. M. (1998). *Shy children, phobic adults: Nature and treatment of social phobia*. Washington, DC: American Psychological Association.

Berg-Cross, L., & So, D. (2011). *Evidenced-based multicultural therapies: The start of a new era. National Register of Health Service Providers in Psychology, The Register Report: Fall 2011.* Retrieved from http://www.nationalregister.org/trr_fall11_bergcross.html

Birmaher, B., Ehmann, M., Axelson, D. A., Goldstein, B. I., Monk, K., Kalas, C., . . . , Brent, D. A. (2009). Schedule for affective disorders and schizophrenia for school-age children

(K-SADS-PL) for the assessment of preschool children—A preliminary psychometric study. *Journal of Psychiatric Research, 43*(7), 680–686.

Bogels, S. M., Alden, L., Beidel, D. C., Clark, L. A., Pine, D. S., Stein, M. B., & Voncken, M. (2010). Social anxiety disorder: Questions and answers for the DSM-V. *Depression and Anxiety, 27*, 168–189.

Boone, M. L., McNeil, D. W., Masia, C. L., Turk, C. L., Carter, L. E., Ries, B. J., & Lewin, M. R. (1999). Multimodal comparisons of social phobia subtypes and avoidant personality disorder. *Journal of Anxiety Disorders, 13*, 271–292.

Brown, T. A., DiNardo, P. A., & Barlow, D. H. (1994). *Anxiety disorders interview schedule for DSM-IV: Client interview schedule*. Oxford, UK: Oxford University Press.

Buckner, J. D. (2009). Motivation enhancement therapy can increase utilization of cognitive-behavioral therapy: The case of social anxiety disorder. *Journal of Clinical Psychology, 65*, 1195–1206.

Cassano, G. B., Frank, E., Milanfranchi, A. M., Shear, M. K., Maser, J. D., Dell'Osso, L., . . . , Grochocinski, V. J. (2002). *SCI-SHY: Structured clinical interview for social phobia Version 3.0.* Retrieved from http://www.spectrum-project.org/questionnaires/shy.html

Chapman, T. F., Mannuzza, S., & Fyer, A. J. (1995). Epidemiology and family studies of social phobia. In R. G. Heimberg, M. R. Liebowitz, D. A. Hope, & F. R. Schneier (Eds.), *Social phobia: Diagnosis, assessment, and treatment* (pp. 185–201). New York, NY: Guildford Press.

Chartrand, H., Cox, B., El-Gabalawy, R., & Clara, I. (2011). Social anxiety disorder subtypes and their mental health correlates in a nationally representative Canadian sample. *Canadian Journal of Behavioural Science, 43*, 89–98.

Ciliberti, C., Gould, C., Smith, M., Chorney, D., & Edelstein, B. (2011). A preliminary investigation of developmentally sensitive items for the assessment of social anxiety in late life. *Journal of Anxiety Disorders, 25*, 686–689.

Codd, R. T., Twohig, M. P., Crosby, J. M., & Enno, A. (2011). Treatment of three anxiety disorder cases with acceptance and commitment therapy in a private practice. *Journal of Cognitive Psychotherapy, 25*, 203–217.

Comas-Díaz, L. (2012). *Multicultural care: A clinician's guide to cultural competence*. Washington, DC: American Psychological Association.

Cormier, S., Nurius, P. S., & Osborn, C. J. (2013). *Interviewing and change strategies for helpers: Fundamental skills and cognitive-behavioral interventions* (7th ed.). Belmont, CA: Brooks/Cole Cengage Learning.

Crippa, J. S., De Lima Osório, F., Del-Ben, C., Filho, A., da Silva Freitas, M., & Loureiro, S. (2008). Comparability between telephone and face-to-face structured clinical interview for DSM–IV in assessing social anxiety disorder. *Perspectives in Psychiatric Care, 44*, 241–247.

Davidson, C. L., Wingate, L. R., Grant, D. M., Judah, M. R., & Mills, A. C. (2011). Interpersonal suicide risk and ideation: The influence of depression and social anxiety. *Journal of Social and Clinical Psychology, 30*, 842–855.

Davidson, J. R. T., Miner, C. M., de Veaugh-Geiss, J., Tupler, L. A., Colket, J. T., & Potts, N. L. S. (1997). The Brief Social Phobia Scale: A psychometric evaluation. *Psychological Medicine, 27*, 161–166.

Dell'Osso, L., Rucci, P., Cassano, G., Maser, J., Endicott, J., Shear, M., . . . , Frank, E. (2002). Measuring social anxiety and obsessive-compulsive spectra: Comparison of interviews and self-report instruments. *Comprehensive Psychiatry, 43*, 81–87.

Endicott, J., & Spitzer, R. L. (1978). A diagnostic interview: The schedule for affective disorders and schizophrenia. *Archives of General Psychiatry, 35*, 837–844.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (2005). *User's guide for the structured clinical interview for DSM-IV axis I disorders: Clinical version.* Washington, DC: American Psychiatric Press.

Fluckiger, C., Del, R. A. C., Wampold, B. E., Znoj, H., Caspar, F., & Jorg, U. (2012). Valuing patients' perspective and the effects on the therapeutic alliance: A randomized controlled study of an adjunctive instruction. *Journal of Counseling Psychology, 59*, 18–26.

Glick, D. M., & Orsillo, S. M. (2011). Relationships among social anxiety, self-focused attention, and experiential distress and avoidance. *Journal of Cognitive and Behavioral Psychotherapies, 11*, 1–12.

Griest, J. H., Kobak, K. A., Jefferson, J. W., Katzelnic, D. J., & Chene, R. L. (1995). The clinical interview. In R. G. Heimberg, M. R. Liebowitz, D. A. Hope, & F. R. Schneier (Eds.), *Social phobia: Diagnosis, assessment, and treatment* (pp. 185–201). New York, NY: Guilford Press.

Hart, T. A., Jack, M. S., Turk, C. L., & Heimberg, R. G. (1999). Issues for the measurement of social phobia. In H. G. M. Westenberg & J. A. den Boer (Eds.), *Social phobia: Recent trends and progress* (pp. 133–155). Amsterdam, the Netherlands: Syn-Thesis Publishers.

Hedman, E., Andersson, G., Ljotsson, B., Andersson, E., Ruck, C., Mortberg, E., & Lindefors, N. (2011). Internet-based cognitive behavior therapy vs. cognitive behavioral group therapy for social anxiety disorder: A randomized controlled non-inferiority trial. *PLoS One, 6*, e18001.

Heeren, A., Maurage, P., Rossignol, M., Vanhaelen, M., Peschard, V., Eeckhout, C., & Philippot, P. (2012). Self-report version of the Liebowitz Social Anxiety Scale: Psychometric properties of the French version. *Canadian Journal of Behavioural Science, 44*, 99–107.

Heimberg, R. G., Brozovich, F. A., & Rapee, R. M. (2010). A cognitive-behavioral model of social anxiety disorder: Update and extension. In S. G. Hofmann & P. M. DiBartolo (Eds.), *Social anxiety: Clinical, developmental, and social perspectives* (2nd ed., pp. 396–422). Burlington, MA: Academic Press/Elsevier.

Herbert, J. D., Rheingold, A. A., & Brandsma, L. L. (2010). Assessment of social anxiety and social phobia. In S. G. Hofmann & P. M. Dibartolo (Eds.), *Social anxiety: Clinical, developmental, and social perspectives* (2nd ed., pp. 23–64). Burlington, MA: Academic Press/Elsevier.

Hofmann, S. G. (2007). Treating avoidant personality disorder: The case of Paul. *Journal of Cognitive Psychotherapy, 21*, 346–352.

Hofmann, S. G. (2010). Recent advances in the psychosocial treatment of social anxiety disorder. *Depression and Anxiety, 27*, 1073–1076.

Hofmann, S. G., Asnaani, A., & Hinton, D. E. (2010). Cultural aspects in social anxiety and social anxiety disorder. *Depression and Anxiety, 27*, 1117–1127.

Hook, J. N., Hodges, E., Whitney, K., & Segal, D. L. (2008). Structured and semistructured interviews. In M. Hersen & J. C. Thomas (Eds.), *Handbook of clinical interviewing with adults* (pp. 24–37). Los Angeles, CA: Sage.

Hopko, D. R., McNeil, D. W., Zvolensky, M. J., & Eifert, G. H. (2001). The relation between anxiety and skill in performance-based anxiety disorders: A behavioral formulation of social phobia. *Behavior Therapy, 32*, 185–207.

Horn, P. J., & Wuyek, L. A. (2010). Anxiety disorders as a risk factor for subsequent depression. *International Journal of Psychiatry in Clinical Practice, 14*, 244–247.

Huppert, J. D., Strunk, D. R., Ledley, D. R., Davidson, J. R. T., & Foa, E. B. (2008). Generalized social anxiety disorder and avoidant personality disorder: Structural analysis and treatment outcome. *Depression and Anxiety, 25*, 441–448.

Johansson, H., & Jansson, J. A. (2010). Therapeutic alliance and outcome in routine psychiatric out-patient treatment: Patient factors and outcome. *Psychology and Psychotherapy: Theory, Research and Practice, 83*, 193–206.

Kachin, K. E., Newman, M. G., & Pincus, A. L. (2001). An interpersonal problem approach to the division of social phobia subtypes. *Behavior Therapy, 32*, 479–501.

Kelly, M. M., Walters, C., & Phillips, K. A. (2010). Social anxiety and its relationship to functional impairment in body dysmorphic disorder. *Behavior Therapy, 41*, 143–153.

Kocovski, N. L., MacKenzie, M. B., & Rector, N. A. (2011). Rumination and distraction periods immediately following a speech task: Effect on postevent processing in social anxiety. *Cognitive Behaviour Therapy, 40*(1), 45–56.

Krause, M., Altimir, C., & Horvath, A. (2011). Deconstructing the therapeutic alliance: Reflections on the underlying dimensions of the concept. *Clínica y Salud, 22*, 267–283.

Kuiper, K. (Ed.). (2011). *The native American sourcebook: Native American culture*. New York, NY: Britannica Educational Publishing.

Lewin, M. R., McNeil, D. W., & Lipson, J. L. (1996). Enduring without avoiding: Behavioral assessment of verbal dysfluencies in public speaking anxiety. *Journal of Psychopathology and Behavioral Assessment, 18*, 387–402.

Liebowitz, M. R. (1987). Social phobia. *Modern Problems of Pharmacopsychiatry, 22*, 141–173.

Lysaker, P. H., Salvatore, G., Grant, M. A., Procacci, M., Olesek, K. L., Buck, K. D., . . . , Dimaggio, G. (2010). Deficits in theory of mind and social anxiety as independent paths to paranoid features in schizophrenia. *Schizophrenia Research, 124*, 81–85.

Majdandžić, M., de Vente, W., & Bögels, S. (2010). Rearing histories of individuals with and without social anxiety who become first time parents. *Anxiety, Stress and Coping, 23*, 243–258.

Mannuzza, S., Fyer, A. J., Klein, D. F., & Endicott, J. (1986). Schedule for affective disorders and schizophrenia-lifetime version (modified for the study of anxiety disorders): Rationale and conceptual development. *Journal of Psychiatric Research, 20*, 317–325.

Marks, I. M. (1987). *Fears, phobias, and rituals: Panic, anxiety, and their disorders*. New York, NY: Oxford.

Masia-Warner, C., Storch, E. A., Pincus, D. B., Klein, R. G., Heimberg, R. G., & Liebowitz, M. R. (2003). The Liebowitz Social Anxiety Scale for children and adolescents: An initial psychometric investigation. *Journal of the American Academy of Child and Adolescent Psychiatry, 42*, 1076–1084.

McNeil, C. B., & Hembree-Kigin, T. L. (2010). *Parent–child interaction therapy* (2nd ed.). New York, NY: Springer.

McNeil, D. W. (2010). Evolution of terminology and constructs in social anxiety and its disorders. In S. G. Hofmann & P. M. DiBartolo (Eds.), *Social anxiety: Clinical, developmental, and social perspectives* (2nd ed., pp. 3–21). Burlington, MA: Academic Press/Elsevier.

McNeil, D. W., Lejeuz, C. W., & Sorrell, J. T. (2010). Basic behavioral mechanisms and processes in social anxieties and social anxiety disorders. In S. G. Hofmann & P. M. DiBartolo (Eds.), *Social anxiety: Clinical, developmental, and social perspectives* (2nd ed., pp. 347–372). Burlington, MA: Academic Press/Elsevier.

McNeil, D. W., Ries, B. J., Taylor, L. J., Boone, M. L., Carter, L. E., Turk, C. L., & Lewin, M. R. (1995). Comparison of social phobia subtypes using Stroop tests. *Journal of Anxiety Disorders, 9*, 47–57.

Miller, W. R., & Rollnick, S. (2013). *Motivational interviewing: Helping people change* (3rd ed.). New York, NY: Guilford Press.

Nepon, T., Flett, G. L., Molnar, D. S., & Hewitt, P. L. (2011). Perfectionism, negative social feedback, and interpersonal rumination in depression and social anxiety. *Canadian Journal of Behavioural Science, 43*, 297–308.

O'Grady, M. A., Cullum, J., Armeli, S., & Tennen, H. (2011). Putting the relationship between social anxiety and alcohol use into context: A daily diary investigation of drinking in response to embarrassing events. *Journal of Social and Clinical Psychology, 30*, 599–615.

Orsillo, S. M. (2001) Measures for social phobia. In M. M. Antony, S. M. Orsillo, & L. Roemer (Eds.), *AABT clinical assessment series practitioner's guide to empirically based measures of anxiety* (pp. 159–163). New York, NY: Kluwer Academic/Plenum Publishers.

Osorio, F. L., Crippa, J. A. S., & Loureiro, S. R. (2010). Study of the psychometric qualities of the Brief Social Phobia Scale (BSPS) in Brazilian university students. *European Psychiatry, 25*(3), 178–188.

Rapee, R. M., & Sanderson, W. C. (1998) *Social phobia: Clinical application of evidenced-based psychotherapy*. North Bergen, NJ: Jason Aronson.

Rodebaugh, T. L., Gianoli, M. O., Oltmanns, T. F., & Turkheimer, E. (2010). The interpersonal problems of the socially avoidant: Self and peer shared variance. *Journal of Abnormal Psychology, 119*, 331–340.

Rogers, R., Jackson, R. L., Salekin, K. L., & Neumann, C. S. (2003). Assessing axis I symptomatology on the SADS–C in two correctional samples: The validation of subscales and a screen for malingered presentations. *Journal of Personality Assessment, 81*, 281–290.

Safir, M. P., Wallach, H. S., & Bar-Zvi, M. (2012). Virtual reality cognitive-behavior therapy for public speaking anxiety: One-year follow-up. *Behavior Modification, 36*, 235–246.

Sanchez-Villegas, A., Schlatter, J., Ortuno, F., Lahortiga, F., Pla, J., Benito, S., & Martinez-Gonzalez, M. A. (2008). Validity of a self-reported diagnosis of depression among participants in a cohort study using the structured clinical interview for DSM-IV (SCID-I). *BMC Psychiatry, 8*, 43.

Schreier, S. S., Heinrichs, N., Alden, L., Rapee, R. M., Hofmann, S. G., Chen, J., . . . , Bogels, S. (2010). Social anxiety and social norms in individualistic and collectivistic countries. *Depression and Anxiety, 27*, 1128–1134.

Sharpley, C. F., Munro, D. M., & Elly, M. J. (2005). Silence and rapport during initial interviews. *Counselling Psychology Quarterly, 18*, 149–159.

Storch, E. A., Masia-Warner, C., Heidgerken, A. D., Fisher, P. H., Pincus, D. B., & Liebowitz, M. R. (2006). Factor structure of the Liebowitz Social Anxiety Scale for children and adolescents. *Child Psychiatry & Human Development, 37*, 25–37.

Werner, K. H., Goldin, P. R., Ball, T. M., Heimberg, R. G., & Gross, J. J. (2011). Assessing emotion regulation in social anxiety disorder: The emotion regulation interview. *Journal of Psychopathology and Behavioral Assessment, 33*, 346–354.

Westra, H. A. (2012). *Motivational interviewing in the treatment of anxiety*. New York, NY: Guilford Press.

Yoon, K., & Joormann, J. (2012). Stress reactivity in social anxiety disorder with and without comorbid depression. *Journal of Abnormal Psychology, 121*, 250–255.

14

Self-Report Assessment

The Status of the Field and Room for Improvement

Katya C. Fernandez, Marilyn L. Piccirillo, and Thomas L. Rodebaugh

Washington University in St. Louis, USA

Introduction

Our aim in this chapter is to help researchers and clinicians go beyond standard practice in selecting self-report social anxiety measurement tools, and focus on the specific characteristics of measures in the context in which they are being used. Accordingly, we will (a) discuss major issues in the evaluation of assessment modalities, focusing specifically on reliability and validity; (b) discuss the advantages and disadvantages of self-report tools related to social anxiety and social anxiety disorder (SAD); (c) describe popular social anxiety self-report assessment tools and their psychometric properties; and (d) provide a framework for future conceptualizations of social anxiety assessment.

The Road Ahead

We want to prepare the reader that much of this chapter concerns *principles* of assessment. Readers who want specific recommendations on assessment measures—a common feature in this kind of chapter—may refer to Table 14.1. However, we hope all readers will find our review of the principles of assessment valuable in interpreting that table and the literature more broadly. Also in regard to the road ahead, we would like to identify and discuss the key problem with self-report, or, the villain of the piece: Common method variance. Some readers may be familiar with this villain already, whereas others may be puzzled by the term or why we bring it up so early in the chapter. In short, one way to view this chapter is as the unmasking of this villain and a series of recommendations for thwarting it.

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

Table 14.1 Recommended Self-Report Assessment Tools at the Different Levels of the Broad Model of Social Anxiety and Reviews of their Statistical Properties

Construct(s) (Exemplar)	*Factor structure*	*Reliability*	*Convergent and discriminant validity*	*Measurement invariance*	*Treatment sensitivity*	*Notes*
Positive and Negative Affect (PANAS; Watson, Clark, & Tellegen, 1988)	Each subscale roughly unifactorial (Watson et al., 1988), but removal of items, intercorrelation of error terms, or more complex structures may be required for good fit (Crawford & Henry, 2004; Egloff, Schmukle, Burns, Kohlmann, & Hock, 2003; Villodas, Villodas, & Roesch, 2011)	Each subscale has excellent internal consistency (Crawford & Henry, 2004; Watson et al., 1988) and stability (trait: Watson et al., 1988)	Each subscale is good overall, where tests are available (Lucas, Diener, & Suh, 1996; Watson et al., 1988)	Neither subscale likely to be completely invariant across groups of interest; preliminary evidence for partial invariance for a short version of the scale across age, sex, education, marital status, and financial hardship, with the exception of the "excited" item for PA (Mackinnon et al., 1999)	Good preliminary evidence for NA subscale (Kring, Persons, & Thomas, 2007; Mohr et al., 2005); evidence slightly less clear for PA subscale (Kring et al., 2007; Mohr et al., 2005)	Consider bivalent affect measures (Barrett & Russell, 1998; Bradley & Lang, 1994)

(*continued*)

Table 14.1 (*Continued*)

Construct(s) (Exemplar)	*Factor structure*	*Reliability*	*Convergent and discriminant validity*	*Measurement invariance*	*Treatment sensitivity*	*Notes*
Extraversion and Neuroticism (NEO-PI-R; Costa & McCrae, 1992)	For each subscale, reasonable factorial validity when loadings relaxed (Marsh et al., 2010), no evidence of good factor structure with strict loadings (Borkenau & Ostendorf, 1990; Parker, Bagby, & Summerfeldt, 1993; Vassend & Skrondal, 1995, 1997)	Each subscale has very good to excellent internal consistency and test-retest reliability (Costa & McCrae, 1992)	Each subscale is generally good across tests; modest correlations between self- and peer report (Costa & McCrae, 1992)	Preliminary evidence of invariance across gender and time when strict loadings are relaxed for both subscales (in German version: Marsh et al., 2010)	Although not frequently used, there is preliminary evidence for each subscale (Glinski & Page, 2010)	Consider IPIP scales (Donnellan, Oswald, Baird, & Lucas, 2006)
Fear of Negative Evaluation (BFNE, reverse-scored items omitted; Leary, 1983)	Good evidence for the straightforward items being unifactorial (Carleton, Collimore, McCabe, & Antony, 2011; Duke, Krishnan, Faith, & Storch, 2006; Rodebaugh et al., 2004; Weeks et al., 2005)	Overall very good to excellent internal consistency and stability for straightforward items (Collins, Westra, Dozois, & Stewart, 2005; Duke et al., 2006; Rodebaugh et al., 2004; Weeks et al., 2005)	Good discriminant validity (Collins et al., 2005; Weeks et al., 2005)	Preliminary evidence of invariance across gender (Carleton et al., 2011)	Good across many tests (Collins et al., 2005; Rapee, Abbott, Baillie, & Gaston, 2007; Weeks et al., 2005)	

Fear of Positive Evaluation (FPES; Weeks, Heimberg, & Rodebaugh, 2008)	Unifactorial structure for scored items (Weeks, Heimberg, Rodebaugh, Goldin, & Gross, 2012); as hypothesized, a correlated two-factor scale fit best in combined CFA with BFNE (Fergus et al., 2009; Weeks, Heimberg, & Rodebaugh, 2008)	Good internal consistency and stability (Fergus et al., 2009; Weeks, Heimberg, & Rodebaugh, 2008; Weeks et al., 2012)	Good in tests so far; more tests needed (Fergus et al., 2009; Weeks, Heimberg, & Rodebaugh, 2008; Weeks, Heimberg, Rodebaugh, & Norton, 2008; Weeks et al., 2012)	Evidence needed; preliminary evidence of invariance across sample recruitment site (Weeks et al., 2012)	Good across two available tests (Fergus et al., 2009; Weeks et al., 2012)
Social Anxiety: Social Interaction Anxiety (SIAS, reverse-scored items omitted; Mattick & Clarke, 1998)	Straightforward items are unifactorial (Habke, Hewitt, Norton, & Asmundson, 1997; Mattick & Clarke, 1998; Osman, Gutierrez, Barrios, Kopper, & Chiros, 1998; Rodebaugh, Woods, & Heimberg, 2007; Rodebaugh, Woods, Heimberg, Liebowitz, & Schneier, 2006)	Overall high internal consistency and stability (Cox, Ross, Swinson, & Direnfeld, 1998; Mattick & Clarke, 1998; Mörtberg, Clark, Sundin, & Åberg Wistedt, 2007)	Good across many tests (Brown et al., 1997; Heimberg, Mueller, Holt, Hope, & Liebowitz, 1992; Mattick & Clarke, 1998; Peters, 2000), although reverse-scored items of scale are weak (Rodebaugh et al., 2007)	May not be invariant across ethnicity or college vs. clinical samples (Hambrick et al., 2010; Rodebaugh et al., 2006)	Good across many tests (Cox et al., 1998; Mattick & Clarke, 1998; Mörtberg et al., 2007; Rapee et al., 2007; Titov et al., 2010)

(*continued*)

Table 14.1 (*Continued*)

Construct(s) (Exemplar)	*Factor structure*	*Reliability*	*Convergent and discriminant validity*	*Measurement invariance*	*Treatment sensitivity*	*Notes*
Social Anxiety: Fear of scrutiny (SPS; Mattick & Clarke, 1998)	Unclear structure; up to three factors reported (Habke et al., 1997; Mattick & Clarke, 1998; Osman et al., 1998), although studies almost always score as if unifactorial	Good internal reliability, consistency, test-retest reliability, and some evidence for temporal stability (Cox et al., 1998; Mattick & Clarke, 1998; Mörtberg et al., 2007)	Overall good convergent and discriminant validity (Brown et al., 1997; Heimberg et al., 1992; Mattick & Clarke, 1998; Peters, 2000)	[a]	Good across many tests (Cox et al., 1998; Mattick & Clarke, 1998; Mörtberg et al., 2007; Rapee et al., 2007; Rytwinski et al., 2009; Titov et al., 2010)	

Social Anxiety: Social Anxiety Disorder symptoms (SPAI; Turner, Beidel, & Dancu, 1996)	Bifactorial structure, as hypothesized, with social anxiety symptoms loading on one factor (Clark et al., 1994; Osman, Barrios, Aukes, & Osman, 1995; Osman, Barrios, Haupt, & King, 1996; Turner, Stanley, Beidel, & Bond, 1989)	Very good to excellent internal reliability and test-retest reliability of total and subscale scores (Bunnell, Joseph, & Beidel, 2013; Clark et al., 1994; Cox et al., 1998; Mörtberg et al., 2007; Osman et al., 1995, 1996; Turner, Beidel, Dancu, & Stanley, 1989)	Good discriminant validity across most studies (Clark et al., 1994; Osman et al., 1995; Peters, 2000; Turner, Beidel, et al., 1989), but not all (Osman et al., 1996)	Preliminary evidence of invariance across gender in college, clinical, and nonclinical samples (Bunnell, et al., 2013; Osman et al., 1995)	Good across many tests (Beidel, Turner, & Cooley, 1993; Hoffart, Borge, Sexton, & Clark, 2009; Hofmann, 2004; Mörtberg et al., 2007; Moscovitch, Hofmann, Suvak, & In-Albon, 2005)	Consider LSAS-SR if positive evidence continues to accrue (Fresco et al., 2001; Moscovitch et al., 2005; Oakman, Van Ameringen, Mancini, & Farvolden, 2003; Rytwinski et al., 2009)

Note. Constructs are presented in order from top to bottom of the Broad Model of Personality's Influence on Social Anxiety in the Levinson et al., **Chapter 6**. PANAS, Positive and Negative Affect Schedule; PA, positive affect; NA, negative affect; NEO-PI-R, Neuroticism Extraversion Openness-Personality Inventory—Revised; IPIP, International Personality Item Pool; BFNE, Brief Fear of Negative Evaluation; FPES, Fear of Positive Evaluation Scale; SIAS, Social Interaction Anxiety Scale; SPS, Social Phobia Scale; LSAS-SR, Liebowitz Social Anxiety Scale Self Report; SPAI, Social Phobia and Anxiety Inventory; SAAS, Social Appearance Anxiety Scale; CFA, confirmatory factor analysis.

[a]We could not locate any clear evidence. Notably, measurement invariance is difficult to test without a clear CFA structure.

Social Anxiety versus *kinds* of Social Anxiety

Let us begin by clarifying what we mean by the *assessment of social anxiety*. Previous studies have suggested that social anxiety is not a single construct. These studies have typically suggested that social anxiety may have different properties when it is related to specific types of situations. For example, it has been theorized that social anxiety consists of both social interaction anxiety and performance anxiety (Mattick & Clarke, 1998). We take the basic position that, although social anxiety may be multifaceted, available assessment measures have generally not been designed based on a replicated model of its multifaceted dimensions. Given this situation, writing about social anxiety as if it is generally a single thing seems as defensible as other proposed divisions in types of social anxiety.

The lack of a measure based on a replicated multifaceted model is largely attributable to studies not converging on such a model except in the most general way. Using epidemiological data, Ruscio et al. (2008) found that, among people who had ever had trouble with social anxiety, both performance and interactional fears loaded onto a single factor: There was little evidence for distinct types of social anxiety. However, other studies have found evidence for multiple factors; for example, Cox, Clara, Sareen, and Stein (2008) used the same epidemiologic data as Ruscio et al., but focused on people who met diagnostic criteria for SAD. Cox et al. found evidence for three types of social anxiety (social interaction fears, observation fears, and public speaking fears). Some measures have been created under the assumption that social interaction anxiety is meaningfully different from performance anxiety (e.g., Liebowitz Social Anxiety Scale; Liebowitz, 1987). One might therefore argue that an interaction versus performance distinction should be focused on in assessment. However, measures of social interaction anxiety and performance anxiety are often highly correlated, and although social interaction anxiety seems potentially distinguishable from other social anxieties, factor analytic studies have not generally found much support for performance situations all loading on a single factor (Perugi et al. 2001). Given all of the above, we focus in this chapter on the assessment of social anxiety broadly, but we will specify the precise construct that a measure is designed to assess (when available) rather than simply subsume all measures under the broad construct of *social anxiety*.

A Guide to Evaluating Assessment Tools

A good starting point for discussing the evaluation of assessment tools is the *Standards for Educational and Psychological Testing* (SEPT; American Educational Research Association, 1999). The SEPT was developed jointly by the American Educational Research Association, the American Psychological Association, and the National Council on Measurement in Education, and consists of a series of guidelines for conceptualizing test construction, evaluation, documentation, fairness, and applications. The SEPT clearly defines *reliability and validity*, and provides standards for how researchers should use these psychometric terms; we will use the definitions in the SEPT as a starting point for our discussion on reliability and validity.

Reliability

An important consideration when choosing an assessment tool is the degree to which the assessment tool is a *reliable* method of assessing a particular construct. According to the SEPT, reliability can be defined as "the consistency of [such] measurements when the testing procedure is repeated on a population of individuals or groups" (American Educational Research Association, 1999, p. 25). In other words, reliability simply refers to whether or not an assessment tool consistently produces equivalent results under similar conditions. Below we will review two major reliability-related concepts relevant to testing measures of psychological constructs; although these concepts may be familiar, we will note frequent misunderstandings of their meaning.

Internal consistency Internal consistency basically refers to the degree to which items within a measure, or subscale, correlate with one another. If five items are thought to assess a particular construct, then these five items should correlate highly with each other. Although showing internal consistency is a good first step in demonstrating reliability, it is not a good estimate of overall reliability for a number of reasons. For example, internal consistency values will overestimate reliability if responses are affected by extraneous commonalities across questions (e.g., the respondent answering all items when in a certain mood even though the measure is not meant to assess mood).

Test-retest reliability Test-retest reliability refers to the degree to which a measure yields similar scores when administered under similar conditions across time. In other words, a measure with high test-retest reliability, when administered 1 week apart, should yield similar test scores for each respondent. As typically assessed in this literature, test-retest reliability is also a relatively weak assessment of reliability. Typically, test-retest reliability is assessed by repeating an identical measure. Although this is an intuitively compelling test of repeatability, any simple correlation between two administrations is an inextricable combination of true repeatable assessment of the same construct, and various other factors that could lead to higher or lower correlations across time. For example, repeating an identical measure raises, and has no power to dismiss, the possibility that respondents might remember their initial answers and repeat them. These and other concerns about typical test-retest methods are recounted by Nunnally and Bernstein (1994). Ideal tests of reliability are rarely conducted in research related to SAD. Such tests include tests of multiple forms, or at least partially nonoverlapping items, whereas research more often concentrates entirely on predefined measures for which there is only one form.

Such a brief summary is merely an introduction to issues surrounding reliability. Our primary point is that no measure of social anxiety that we are aware of has been tested using a method involving multiple forms or multiple subsets of items. We recommend that enterprising researchers address this shortcoming in the future.

Validity

Another important consideration when choosing an assessment tool is the degree to which the assessment tool is a *valid* method of assessing a particular construct.

Validity can be defined as "the degree to which evidence and theory support the interpretations of test scores entailed by proposed uses of tests" (American Educational Research Association, 1999, p. 9). In other words, validity refers to whether an assessment tool measures what it is intended to measure. Validity is often expressed as being comprised of a variety of specific types of validity that are closely linked to statistical properties of scores derived from measures, such as external/ecological validity, internal validity, construct validity, and factorial validity, among others. It is tempting for researchers to consider validity a one-dimensional or even binary construct, but such a simplification is misleading. Similarly, it is tempting for researchers to seek to *validate* a particular assessment tool, which presumes that once such validation is complete, the tool as a whole is considered valid. According to the SEPT, "the process of validation involves accumulating evidence to provide a sound scientific basis for the proposed score interpretations" (American Educational Research Association, 1999, p. 9), which highlights that test *scores* are validated in specific situations, not the test or measure itself.

The notion that any given measure as a whole can be validated is understandably tempting; for example, it would simplify the search for appropriate measures and eliminate the use of measures that are not considered (or have not been demonstrated to be) valid. Unfortunately, however, there is no universally accepted process for determining whether a measure is valid for all populations (e.g., across age, ethnic groups, levels of education) and all purposes. In other words, if a particular test score is used in a new context, the new context should ideally be validated. To date, very few assessment tools of interest in this chapter have evidence of validity across more than a handful of different contexts.

Issues in the definition of validity Several types of validity are important to consider when evaluating a measure. At the most basic level, *construct validity* describes whether an assessment tool measures the construct that it is designed to measure (Cronbach & Meehl, 1955). Construct validity is familiar to most psychologists, and is measured with tools such as the multitrait multimethod matrix and other tests related to the concept of a nomological network, or system of laws, that governs the behavior of a measure (Campbell & Fiske, 1959). This depiction of validity is relatively complex and embedded in a specific historical context, but is still the concept of validity most heavily referred to by psychological researchers. In short, this conception of validity assumes that, *because psychological constructs cannot be assessed directly, measures of such constructs can only be judged based on the associations they show with other measures or data*. The nomological network, then, is the system of lawful associations with other data that the measure of interest demonstrates (and validity within a given context can be supported or not supported by such relationships).

Our impression is that the idea of construct validity is often misinterpreted, such that many researchers assume that ideally a measure should correlate highly with all similar measures. For example, a new measure of social anxiety might be considered the best measure if it correlates most highly with all available measures of social anxiety. We will deal further with this issue below, but here it is important to note that the concept of the nomological network is not based primarily on correlations being high or significant, but rather on correlations *being of the strength they are hypothesized to be*.

Thus, a finding that a correlation is .20 may be just as much evidence as a correlation of .85: The question is what hypotheses have been generated about the nomological network. In fact, *not all psychological measures should necessarily demonstrate correlations with related measures.* As pointed out by Embretson (1983), for some measures, a primary concern in determining whether a measure possesses construct validity is determining *the components* of a particular measure or set of measures (referred to as construct representation). As Embretson also notes, components can be determined even when those components have no meaningful variation among individuals. For example, a researcher could use statistical techniques to determine that two tests measure reading ability, yet those tests could measure such basic reading ability that in most samples individuals will not vary (even if these individuals show variation on other constructs). If individuals cannot vary, the measures will not correlate with anything else, including each other, although they can still be good measures of basic reading ability. In practice, most measures related to SAD will almost always show variation across individuals, largely because measures are typically constructed with the intention of measuring the continuum of a particular construct. For our purposes, the concept of construct representation serves as a reminder that higher correlations are not always better.

Another important alternate conception of validity is both simpler in concept and more complex in execution than the nomological network conception. Borsboom, Mellenbergh, and Heerden (2004) point out that the idea of construct validity is highly influenced by its historical context and the need of researchers to conform to the most influential philosophical attitudes in science at the time that clinical psychology began to struggle with issues of assessment. Skepticism regarding apparently metaphysical entities (e.g., beliefs, attitudes, and opinions) led to attempts to measure things like beliefs while nevertheless asserting that beliefs themselves did not exist: They were merely a hypothetical construct, or a sort of place holder until a real, underlying thing could be identified (e.g., the output of a particular set of neurons). Borsboom, Mellenbergh, and Heerden suggest that attempts to address validity of instruments while at the same time denying that the instruments measure something that exists is nonsensical and self-defeating. In this view of validity, *a measure is valid if it measures something that exists such that variations in that thing lead to variations on the measure.* In other words, a measure of social anxiety is valid if social anxiety exists and higher levels of social anxiety cause higher scores on the measure. We find the observations of Borsboom and colleagues compelling, but it is important to note that the strongest tests of this conception of validity (e.g., experimental designs) are particularly difficult to develop for constructs such as those addressed in this chapter.

Having introduced some general validity concepts, we will now discuss some of the more frequently noted types of validity. We review these not because they encompass the entire set of validity terms that have been used in this literature, but rather because they appear to be some of the most frequently used. Because we essentially accept Borsboom et al.'s (2004) arguments regarding validity, we wish to note that we discuss these validity concepts not as a part of an essential checklist for validity: We agree with their contention that no list of validity tests will replace a precise theory of how a particular assessment tool relates to the constructs being measured in specific contexts.

Convergent and discriminant validity Convergent and divergent validity are two commonly used validity terms in the social anxiety literature. Convergent validity is the extent to which a particular assessment tool correlates with other assessment tools that it theoretically should correlate with. As a very broad example, if social anxiety is known to correlate with constructs such as depression, neuroticism, and behavioral inhibition, then any individual measure of social anxiety should also correlate with the same constructs. Divergent (or discriminant) validity is the extent to which a particular assessment tool does *not* correlate with other assessment tools that it theoretically should not correlate with. As a simple example, we have little theoretical reason to expect that a measure of social anxiety should correlate with political affiliation. At the heart of convergent and divergent validity is the idea of *specificity*; namely, if a measure is thought to measure a specific construct, then in addition to demonstrating convergent and divergent validity, it should predict expected outcomes above and beyond more general measures. For example, in a social interaction, we expect that measures of social interaction anxiety would relate more strongly to self-reported anxiety than broad measures of neuroticism or extraversion. When measures fail to specifically predict outcomes over broader measures, one should ask "Am I just measuring X (where X is a broad temperament factor, such as neuroticism)?" We would like to gently suggest that researchers concerned with mental disorders would be better off if they asked this kind of question more frequently.

Factorial validity An important statistical technique to keep in mind when considering validity is that of factor analysis techniques, namely exploratory factor analysis (EFA) and confirmatory factor analysis (CFA). EFA is an exploratory statistical technique used to discern the number of factors that might be present in a given measure; CFA is a confirmatory statistical technique utilized to confirm the number of factors already hypothesized to be present in a given measure. EFA and CFA are powerful tools that help researchers understand the exact construct that a given measure is assessing. If more than one factor is present in a measure, then researchers are able to better understand what each factor is, and what the theoretical role of each factor is. On a more practical note, knowing the factor structure is key to correctly calculating the total or subscale scores of measures. For example, if a given measure of social anxiety contains two factors, then a researcher should be conscious of totaling and analyzing each factor separately, as opposed to summing all the items in the questionnaire and treating the measure as though it only measured one construct. Similarly, when deciding whether or not to use a measure, researchers should pay close attention to the *content* of items in each factor, and not just the factor names assigned by the creators of the measure. Additionally, the validity of factors is dependent upon empirical demonstrations that different factors behave as expected in crucial tests. A thing a researcher called *Duck* because it had *down*, was *soft*, and had *feathers*, but which does not fly or swim might just be a feather bed.

Going beyond the traditional nomological net Assessment of correlations between measures, as well as item structures, are familiar concerns to many psychologists, but current methods and findings in assessment make it clear that such correlations provide only a relatively basic assessment of validity. For example, as detailed below

(see Reliability and Validity), current statistical methods can help determine whether a given set of items provides good measurement of a latent construct at varying levels of that construct. In an intuitive example, the question "Have you ever been bothered by anxiety in social situations at least a little bit?" will capture some information about relatively low versus extremely low levels of social anxiety. In contrast, the question "Have you ever been nearly completely paralyzed with fear in a social situation?" will capture some information about relatively high levels of social anxiety. These particular questions are exaggerated for illustrative purposes, but similar differences in thresholds for items can exist in measures developed in earnest. We encourage readers who want to better understand how the concept of item thresholds affects concepts of validity to start with the introductory text by Embretson and Reise (2000).

For our current purposes, the primary reason to go beyond the traditional nomological net is to discuss the issue of *measurement invariance*. Measurement invariance, or, the ability of a measure to retain validity across conditions of concern, is an often overlooked but important psychometric property (Borsboom, 2006). Measurement invariance means that the measure performs similarly regardless of a particular characteristic of the sample. Characteristics frequently of concern are gender and ethnicity, but there may be many others. Take, for example, a measure of social anxiety that is invariant across gender. Such invariance would mean that a woman with a given level of social anxiety would respond in the same way to the measure's items as a man with that same level of social anxiety. In contrast, failure of measurement invariance could mean that women have higher or lower scores on the measure compared with men who actually have the same underlying level of social anxiety. At its worst, failure of measurement invariance could even mean that women respond to the items as if they are all related to social anxiety, whereas men might respond as if some items are related to social anxiety and others are related to another construct altogether. Measurement invariance in its varying forms can be assessed using a variety of statistical techniques, including multiple group categorical factor analysis and Item Response Theory (IRT) analyses.

A major threat to validity: Common method variance (CMV) CMV, already tagged above as the villain of this chapter, is the "systematic error variance shared among variables measured with and introduced as a function of the same method and/or source" (Richardson, Simmering, & Sturman, 2009, p. 763). In other words, CMV is variance attributable to the measurement method itself (e.g., self-report) rather than to the constructs being measured. CMV can encompass issues ranging from scale types and response formats (e.g., whether the scale is dichotomous or Likert-type) to response biases such as the ones discussed above (e.g., social desirability). Findings regarding CMV remind us that a measure's validity is limited by the features that comprise the measure. In an extreme example, a self-report measure of something that individuals do not actually know about themselves is likely to be misleading.

A Practical Guide to Assessment Selection

Thus far, we have discussed the evaluation of assessment tools, and hinted that self-report measures of anything, including social anxiety, are likely to be imperfect tools

subject to many biases. More specifically, we have established that measures cannot be validated in the general sense; in other words, no assessment tool will carry out its intended purpose in every possible circumstance. However, despite the potential imperfections associated with the assessment of social anxiety, we assert that a thorough, valid, and reliable assessment of social anxiety is possible in most contexts, and offer guidelines for how to achieve this goal. We will begin by briefly describing the broad model of social anxiety outlined in **Chapter 6**.

The Assessment of Related Constructs

As outlined in **Chapter 6**, available research thus far highlights the key role of two broad temperamental factors—approach and avoidance tendencies—in better understanding social anxiety. Levinson et al. (**Chapter 6**) outline a broad model of social anxiety whereby approach and avoidance tendencies each relate to specific vulnerabilities (e.g., fear of negative evaluation), which in turn lead to trait social anxiety symptoms, and thereby result in an increased likelihood of the development of SAD. SAD itself, or separate effects of the same factors, may then lead to correlated conditions (e.g., depressive symptoms).

Recommended Assessments at Different Levels of the Model

Table 14.1 is a focused review of recommended self-report assessment tools at the different levels of the broad model of social anxiety. This table contains some of the most widely used measures of social anxiety, affect, and personality. We present these recommended assessment tools as a table, and we provide references for all assessment tools. Readers are encouraged to investigate these references should they desire more specific information on any particular assessment tool.

Some readers might prefer an exhaustive discussion of measures and clearer recommendations regarding the one best measure for the various constructs at issue. We propose, however, that the current state of the field does not lend itself to such a review. One factor here is that the overall evidence for psychometric properties of measures assessing social anxiety is generally modest by psychometric standards, even for the most frequently used measures. As an example, although the Social Interaction Anxiety Scale (Mattick & Clarke, 1998) has more psychometric data to recommend it than many other self-report measures of social anxiety, we still harbor concerns that it may not be acceptably invariant across ethnic groups (Hambrick et al., 2010; see also below). We are hopeful that this state of affairs will continue to improve, in which case it would be far more beneficial for the reader to use our overall guide to assess the current literature in further detail, rather than trust any recommendation that we might currently make on single best measures.

More generally, however, we do not want to encourage thinking of assessment tools as if they are uniformly good or bad. We see our recommendations as, at best, a good start: Measures that we might recommend *in general* might be of very limited use to a specific researcher or clinician, despite the fact that psychometric evidence might be abundant for that particular measure. Our hope is that we can provide the reader with a way of thinking about assessment that will help in evaluating the literature and lead

to the selection of the most appropriate tools for a given research question. Of note, we often have decided to depart from our specific recommendations. For example, we have often used an alternative measure of personality with less psychometric support because it is very brief and clearly free for research use (Donnellan et al., 2006). Similarly, because of concerns about the wisdom of only measuring *activated* positive and negative affect (see **Chapter 6**), we have been investigating alternative affective measures, such as the Self-Assessment Manikin (Bradley & Lang, 1994).

Sample Measure Analysis

Our suggestion that readers review the literature further on specific instruments is not made lightly. The decision of whether or not to use a certain assessment tool can certainly be daunting. To illustrate how such a decision may be carried out, we will walk through the example of a researcher who wishes to assess social interaction anxiety, and is contemplating whether to use the Social Interaction Anxiety Scale (SIAS; Mattick & Clarke, 1998), which is a measure we generally recommend, but is not without problems. The SIAS was originally introduced as a companion measure to the Social Phobia Scale (SPS; Mattick & Clarke, 1998), and its main purpose is to assess social interaction anxiety (i.e., anxiety experienced when an individual meets and talks with others). The original scale consisted of 19 or 20 items employing a 0 (*not at all*) to 4 (*extremely*) Likert-type scale. The number of items is a matter of some confusion because, although initial studies that appeared in the literature reported on 20 items (Heimberg et al., 1992), the eventual appearance of the formal psychometric validation of the SIAS suggested that only 19 items were in the scale (Mattick & Clarke, 1998). Most subsequent studies reported on 20 items.

Initial validation of the SIAS (Mattick & Clarke, 1998) indicated that the scale showed high levels of internal consistency and test-retest reliability, in addition to being able to discriminate between individuals with different anxiety disorders (SAD, specific phobia, and agoraphobia) and between individuals with and without SAD. Additionally, the authors demonstrated that SIAS scores lowered with treatment of SAD. Although an exploratory factor analysis was promising, it was noted that at least two items did not load as well as most, indicating a need for further analysis of the measure's structure.

A later study analyzed the factor structure and screening utility of the SIAS, with the primary goals of assessing (a) whether the one-dimensional factor structure originally reported held in a replication sample, and (b) whether the SIAS could serve as a screener for discriminating between individuals with and without SAD in undergraduate and clinical samples (Rodebaugh et al., 2006). The authors found that the SIAS was not unifactorial, and that instead it appeared to have items that loaded onto two factors, a factor with the straightforwardly worded items and a factor with the reverse-scored items; this multidimensional factor structure was found in both undergraduate students and individuals with SAD. Additionally, the authors found that undergraduate students approached the items on the SIAS differently than individuals with SAD, such that researchers using the SIAS as a screening measure may end up excluding students who had levels of social anxiety consistent with SAD; in other words, the SIAS cut-off might inappropriately exclude students who would meet criteria for SAD more so than inappropriately include students who would not meet criteria. Two

previous studies had suggested a screening cut-off of 34 for the SIAS (Brown et al., 1997; Heimberg et al., 1992), and these studies suggested that the cut-off would hold among undergraduates as well as members of the community. Additional support for the notion that the reverse-scored items required some special handling to achieve good factor fit has been found (Carleton et al., 2009). Further, the reverse-scored items have shown validity issues such that they were more strongly influenced by extraversion and less effective at measuring social anxiety than the straightforwardly worded items (Rodebaugh et al., 2007).

Finally, a recent study continued to examine the responses to straightforwardly worded items (vs. the reverse-scored items) in the SIAS, and found that responses to the reverse-scored items were moderated by age, such that the reverse-scored questions showed a weaker relationship with the rest of the scale for older individuals (Rodebaugh et al., 2011). The authors of that study speculated that reverse-scored items are more difficult to interpret, lowering their validity for people with cognitive challenges, such as those more common among older than younger adults. The authors concluded that, although response bias is a concern for a measure that exclusively includes straightforwardly worded items, it appears as though the use of reverse-scored items introduces other assessment problems, namely that the use of these items would lead to selective exclusion of older adults. The authors strongly encouraged the use of straightforwardly worded items (and the omission of the reverse-scored items), and also recommended a cut-off score of 28 for the straightforwardly worded items as an indication of clinical levels of social anxiety.

As illustrated above, even a generally good measure can have a complicated history that might lead to concern about its use. Only a thorough investigation of the available data on the SIAS would uncover that its reverse-scored items should not be scored, even if they are administered. Although much of the above describes general properties of the SIAS that would be of concern to most clinicians and researchers, there are also more application-specific issues. For example, one might prefer to use one of the published shortened versions of the SIAS (e.g., the SIAS-6; Peters, Sunderland, Andrews, Rapee, & Mattick, 2012). However, several of the items included in the shortened measure were found to be particularly problematic in terms of measurement invariance across ethnicity in a test of the straightforwardly worded items of the SIAS (Hambrick et al., 2010). This finding suggests that a shorter measure consisting largely of items that fail tests of measurement invariance across ethnicity would make comparisons across ethnic groups difficult. A researcher or clinician who is compelled (e.g., due to time limitations) to use a short inventory of social interaction anxiety might prefer the shortened SIAS, yet a researcher who expects a diverse sample might find it inadvisable to do so. Again, the situation at hand should drive a careful consideration of the evidence for the measure to be used.

Self-Report's Real Problems, Illusory Problems, and Problematic Solutions

The current chapter focuses on self-report measures. We will now delineate the limits of self-report assessment, discuss potential solutions to these limits, and review

assessment tools that do not rely on self-report that might be useful in the assessment of social anxiety.

Limits of Self-Report

Although self-report is the most widely used form of assessment, self-report carries serious limitations (see Stone, Shiffman, Atienza, & Nebeling, 2007, for a review). These limitations are particularly evident when self-report measures are assessed at a single time point, which is the most typical method in research and clinical work as well, because even when clinicians intend to give a measure twice or more, their clients may not comply.

Assessment at a single point in time leaves measurement particularly vulnerable to biases and extraneous factors. For example, measuring trait social anxiety immediately following an embarrassing social situation (e.g., giving a poorly received speech in front of an audience) may yield different results than measuring trait social anxiety immediately following a successful social situation (e.g., delivering a highly praised speech); it is nearly impossible to be certain that a construct measured at a single time point is measured without such biases. Similar considerations often apply to even multiple measurements. Because most laboratory and clinical settings are relatively unfamiliar to respondents, it is difficult to discern whether results from assessments in these contexts are equivalent to those gathered in a more naturalistic setting (e.g., the respondent's home). Self-report as typically used is therefore particularly vulnerable to problems of *external validity* (i.e., being transferrable to real-world environments and situations).

Another consideration is that many self-report questions ask the respondent to think about certain attitudes, thoughts, and behaviors *in general*; the respondent is required to retrospectively analyze themselves across a variety of situations and times in their life and provide a single response that accurately depicts the particular construct. Such an evaluation is so complex that it stretches credulity to expect that humans can always execute it accurately. A final point concerns the fact that, although researchers are often interested in how constructs relate over time, they often measure constructs as traits at a single time point. This practice is problematic for several reasons; for example, studies have shown that two constructs can be negatively correlated at the trait level, but positively correlated at the state level, or vice versa (Affleck, Zautra, Tennen, & Armeli, 1999). When measuring constructs at one point in time, it is important to acknowledge that relationships between variables may change depending on when and how they are being measured.

The villain of the piece When all of these specific limitations of self-report are considered together, we come to an important question: What is the key problem with self-report assessment? The simplest answer is CMV. At its core, CMV reflects a serious problem with self-report measurements as typically used: If researchers do not consider all possible influences on an individual's responses to a measure, then the researcher is drawing conclusions based on analyses of responses that may not reflect the theorized construct being measured. It is important to note that it is not actually the self-report

aspect of self-report assessment as it is typically used that makes it most vulnerable to CMV. More troublesome are single measurements, measurements in a single location, and requests for the respondent to simultaneously introspect and retrospect. Further, it is important to note that self-report is not the only type of assessment vulnerable to CMV issues; for example, the implicit association test (described more fully below) is susceptible to validity issues stemming from method variance (Klauer, Voss, Schmitz, & Teige-Mocigemba, 2007). Defeating the villainy of CMV is not simple. At first glance, it may seem as though a solution is to create an assessment tool that is CMV-free. However, such a measure is impossible to create, as it is impossible to assess all the possible sources of method variance in a given assessment tool. A more feasible approach to minimizing the CMV problem is a combination of tools that differ in assessment modalities; we elaborate on this point later in this chapter.

Some Solutions for Self-Report

Some potential solutions to the problems posed by self-report (and many other types of assessments reviewed) are (a) the use informant/observer report and (b) ecological momentary assessment, or experience sampling. Because many of the concerns about using self-report revolve around the associated limits in insight and biases, obtaining data from other individuals offers researchers the opportunity to examine those limits and biases. For example, in an analysis of avoidant personality disorder traits, Rodebaugh, Gianoli, Turkheimer, and Oltmanns (2010) found differences between self- and informant-report of interpersonal problems, and that the two reports together gave a more complete picture of the interpersonal problems of individuals higher in avoidant personality traits than either report on its own.

Ecological momentary assessment (EMA; Stone & Shiffman, 1994) is most simply defined as the collection of multiple measurements over time in the course of an individual's everyday life. Although in its infancy EMA largely consisted of paper-and-pencil diaries, advances in computer technology have allowed researchers to take real-time data collection one step further, and today many EMA-based studies involve the use of electronic handheld devices such as personal digital assistants and interactive voice response systems in obtaining momentary assessments of behaviors and psychological constructs such as social anxiety and affect (Courvoisier, Eid, Lischetzke, & Schreiber, 2010; Stone et al., 2007; Trull et al., 2008). In the field of social anxiety, the use of EMA research has already provided insight into the relationship between social anxiety and constructs such as positive affect (Kashdan & Collins, 2010) and sexuality (Kashdan et al., 2011).

Could Interviews Solve the Problem?

Another potential way to address some of the problems with self-report measures is to have a trained interviewer ask the respondent a series of questions pertaining to certain psychological constructs; a common interview used for the measurement of anxiety disorders is the Anxiety Disorders Interview Schedule (ADIS; DiNardo, Brown, & Barlow, 1994). The use of interviews certainly carries advantages: For example, if the respondent does not understand a particular question, the interviewer is able to clarify

the question and provide helpful examples. Furthermore, if a particular response is complex or requires a verbal explanation, an interviewer might capture the response and document it in a way that a self-report measure might not. Finally, interviewers are typically thought to be able to capture nonverbal cues that cannot be captured in self-report measures. For example, an interviewer can notice behaviors that are consistent with individuals with higher levels of social anxiety, such as gaze avoidance, blushing, sweating, trembling, and other physiological reactions. See also **Chapter 13** for expanded discussion of clinical interviews.

These potential advantages, however, are in practice offset by serious limitations, such that interviews generally fail to demonstrate validity in excess of self-report assessments of the same construct. First, interviews are primarily still a form of self-report data; the respondent is still providing responses that may be biased or stem from limited insight. Second, the interviewer is potentially an additional source of error. The interviewer as a source of error might be of particular concern if the interviewer is not well-trained or not familiar with the construct being assessed, unable to clarify certain questions appropriately, or limited in ability to assess nonverbal cues. At the same time, the voluminous literature on clinical judgment suggests that individual clinicians will too frequently make adjustments to decision rules, apparently due to their conviction that their experience trumps the wisdom of decision rules (Dawes, Faust, & Meehl, 1989; Grove, Zald, Lebow, Snitz, & Nelson, 2000). In this sense, it might actually be preferable for interviewers to be trained not to exercise judgment but to follow protocol.

Finally, most interviews require a certain rapport to be established between the interviewer and the respondent. If this rapport is not well-established, the respondent may be more inclined to limit his or her self-disclosure, or respond in a negative way to the interviewer; in both cases, researchers and clinicians should be concerned with the potential biases in reporting. Given the potential advantages and disadvantages of using interviews, it is important to note that many of the concerns with interviews, in particular those involving the interviewer, might be ameliorated through rigorous interviewer training. Even if this is accomplished, however, one might find only a slight improvement over a self-report measure. For example, the Liebowitz Social Anxiety Scale (when administered as an interview) has been shown to correlate highly with a self-report form of the same measure (Fresco et al., 2001), which suggests that self-report measures of social anxiety might be a more effective route in terms of time and resources.

Behavioral Measures

Behavioral measures are another type of assessment commonly used to address issues that arise with self-report measures. Behavioral measures are methodological tools aimed at assessing behavior; in the study of social anxiety, a common example of a behavioral measure is giving speeches in the lab (Beidel, Turner, Jacob, & Cooley, 1989) and having participants engage in laboratory-based social interactions with confederates (Weeks, Heimberg, & Heuer, 2011). Although behavioral measures overcome many of the limitations of self-report assessment tools (e.g., they do not rely on insight or retrospection), behavioral measures also carry certain disadvantages.

First, behavioral measures rarely correlate more than modestly with internal experience (Mauss, Wilhelm, & Gross, 2004); in the social anxiety literature, this phenomenon is best illustrated with available data on self- versus observer-rated performance in social situations. For example, a consistent finding in the social anxiety literature is that individuals with SAD underestimate their actual performance in social interactions when compared with observer ratings of their performance; these findings extend to both one-on-one interactions as well as public speaking (Rapee & Lim, 1992; Stopa & Clark, 1993). Researchers should be aware of this discrepancy, since blindly utilizing behavioral measures as a proxy for internal experience of anxiety or self-perception in these cases would lead to incorrect inferences.

A second limitation of behavioral assessment tools pertains more to practicality and utility of such measures, namely that behavioral tools are often inefficient for translation into real-world applications. Behavioral assessment often requires laborious coding procedures in which a set of individuals (i.e., coders) are recruited and asked to rate behaviors on a variety of dimensions. Some behavioral measures can be obtained with minimal coding, such as vocal pitch or body collapse (e.g., submissive slouch), which Weeks et al. (2011) demonstrated were correlated with social anxiety under certain conditions. Such highly calibrated behavioral measures, however, typically require extensive preparation or special equipment or software. For research purposes, behavioral measures can be very informative, but it is unlikely that such laborious behavioral coding or special preparation would be efficient in most treatment settings.

A relatively recent proposed solution to these limitations of behavioral assessment tools is the use of what we will refer to as *quasi-behavioral* measures (i.e., non-naturalistic behavior that can be numerically captured by a computer). Common examples of these quasi-behavioral measures include the implicit association test (IAT; Greenwald, McGhee, & Schwartz, 1998), the dot probe task (Mansell, Clark, Ehlers, & Chen, 1999), and the flexible, iterated prisoner's dilemma (Rodebaugh et al., 2011). In these measures, respondents are instructed to complete a task at the computer that can be translated into a quantifiable score that in turn is believed to be indicative of a particular psychological construct. In the case of the IAT, if a respondent more quickly pairs a particular set of categories together then it is inferred that the respondent holds strong implicit associations between those categories; for example, Westberg, Lundh, and Jonsson (2007) found that individuals who self-reported as higher in social anxiety were more likely to pair *self* words with *anxiety* words (e.g., afraid, anxious) than they were to pair *self* words with *social-relaxation* words (e.g., calm, safe). Such quasi-behavioral measures are less obviously related to real-world behavior than are traditional laboratory behavioral tasks (e.g., giving a speech) and therefore have much to prove regarding external validity. However, they do show promise as a way to supplement typical usage of self-report measures. See also **Chapter 16** for expanded discussion of behavioral measures of SAD.

Psychophysiological Measures

Psychophysiological assessment tools are those in which physiological indices (e.g., heart rate, skin conductance) are measured, usually in a particular context or situation and usually in an attempt to capture the presence or effects of emotions and

internal thoughts. Studies on social anxiety using psychophysiological measures have found limited support. Overall, the literature provides some straightforward results with intuitive relationships; for example, there is evidence of increased heart rate, higher blood pressure, increased skin conductance, and greater frontal EEG asymmetry among individuals with higher social anxiety (Beidel, Turner, & Dancu, 1985; Dimberg, Fredrikson, & Lundquist, 1986; Heimberg, Gansler, Dodge, & Becker, 1987; Schmidt et al., 2012; Shimizu, Seery, Weisbuch, & Lupien, 2011; see also **Chapter 5** for a review). Although these studies demonstrate correlational evidence between self-reported social anxiety and certain physiological indices, there are many studies that have not found such expected differences (Anderson & Hope, 2009; Puigcerver, Martínez-Selva, García-Sánchez, & Gómez-Armor, 1989). Our interpretation of the literature is that an ongoing search for physiological indicators of social anxiety is worthwhile, yet at the present time, we are aware of no evidence that any physiological indicator offers a particular advantage above and beyond self-report in the assessment of SAD such that self-report can be dismissed. However, candidate physiological responses might be integrated with other measures, including self-report, to provide a clearer picture of social-anxiety-related processes.

The Future of Self-Report Social Anxiety Assessment: What Is the Next Step?

As indicated by our table of assessment tools (see Table 14.1), we believe that the self-report assessment of SAD should not stop with the assessment of social anxiety symptoms. Instead, related constructs such as approach and avoidance tendencies should be assessed concurrently when possible. As we describe below, the measurement of related constructs allows researchers who wish to draw conclusions about the effects of social anxiety to test whether such effects are better accounted for by related constructs. In other words, because SAD is related to multiple vulnerabilities, full assessment must take these relationships into account.

Similarly, researchers are encouraged to assess constructs in a specific and detailed way. For example, a thorough measure of affect may include affect activation (activated and deactivated affect; Feldman Barrett & Russell, 1998), intensity, and frequency (Diener, Larsen, Levine, & Emmons, 1985; Larsen & Diener, 1985). Similarly, researchers should consider the assessment of state-like mood in addition to trait-like tendencies. For example, the most commonly used form of the Positive and Negative Affect Schedule (Watson et al., 1988) is useful for many different purposes, but it is not useful for assessing anger, joy, or boredom. Additionally, because affect is dynamic in nature, researchers would ideally measure affect over time.

Use Multiple Types of Assessment Tools

An ideal approach to measuring social anxiety consists of a combination of assessment tools that differ in approach. Ideally, a researcher or clinician who is assessing social anxiety would do so utilizing a combination of the different assessment types discussed

above (e.g., self-report, observer-report, interviews, behavioral measures, and ecological momentary assessment). Such an approach would allow for a multidimensional study of social anxiety. Using this approach, a researcher can also be more certain of the existence of certain hypothesized effects (e.g., the possibility that the hypothesized effect might only be present due to CMV can be ruled out). However, this approach carries some practical limitations such as increased participant or client burden and research costs.

Conduct Thorough Psychometric Analyses

When reporting on findings, we strongly encourage the use of rigorous tests to demonstrate the performance of chosen measures in specific contexts. In the event that the chosen measures have not been tested in the sample or context that a researcher is using them, such psychometric tests of reliability and validity are even more necessary. We particularly encourage tests of measurement invariance, especially if the measure being utilized has not undergone tests of invariance in demographic categories such as gender and ethnicity.

Conclusions

Our goals in this chapter were to: discuss major issues in the evaluation of assessment tools, focusing specifically on reliability and validity; discuss the advantages and disadvantages of self-report tools related to social anxiety and SAD; provide the reader with a detailed list of major self-report assessment tools of or relating to social anxiety and their respective psychometric properties; and lay groundwork for future conceptualizations of self-report social anxiety assessment. Additionally, we discussed assessment tools that do not rely on self-report, and reviewed their respective strengths and limitations. Overall, although the field has come a long way in terms of psychometric understanding of a variety of assessment measures, there is still much room for improvement in how researchers conceptualize and assess social anxiety and related constructs. Our recommendations for researchers wishing to assess social anxiety are to assess constructs related to social anxiety, and not simply social anxiety alone; whenever possible, use multiple assessment tools, and conduct thorough psychometric analyses on measures being used in specific contexts.

References

Affleck, G., Zautra, A., Tennen, H., & Armeli, S. (1999). Multilevel daily process designs for consulting and clinical psychology: A preface for the perplexed. *Journal of Consulting and Clinical Psychology, 67*, 746–754. doi:10.1037/0022-006x.67.5.746

American Educational Research Association. (1999). *Standards for educational and psychological testing*. Washington, DC: American Psychological Association.

Anderson, E. R., & Hope, D. A. (2009). The relationship among social phobia, objective and perceived physiological reactivity, and anxiety sensitivity in an adolescent population. *Journal of Anxiety Disorders, 23*, 18–26. doi:10.1016/j.janxdis.2008.03.011

Barrett, L. F., & Russell, J. A. (1998). Independence and bipolarity in the structure of current affect. *Journal of Personality and Social Psychology, 74*, 967–984. doi:10.1037/0022-3514.74.4.967

Beidel, D. C., Turner, S. M., & Cooley, M. R. (1993). Assessing reliable and clinically significant change in social phobia: Validity of the Social Phobia and Anxiety Inventory. *Behaviour Research and Therapy, 31*, 331–337. doi:10.1016/0005-7967(93)90033-q

Beidel, D. C., Turner, S. M., & Dancu, C. V. (1985). Physiological, cognitive and behavioral aspects of social anxiety. *Behavioral Research Therapy, 23*, 109–117. doi:10.1016/0005-7967(85)90019-1

Beidel, D. C., Turner, S. M., Jacob, R. G., & Cooley, M. R. (1989). Assessment of social phobia: Reliability of an impromptu speech task. *Journal of Anxiety Disorders, 3*, 149–158. doi:10.1016/0887-6185(89)90009-1

Borkenau, P., & Ostendorf, F. (1990). Comparing exploratory and confirmatory factor analysis: A study on the 5-factor model of personality. *Personality and Individual Differences, 11*, 515–524. doi:10.1016/0191-8869(90)90065-y

Borsboom, D. (2006). When does measurement invariance matter? *Medical Care, 44*, S176–S181. doi:10.1097/01.mlr.0000245143.08679.cc

Borsboom, D., Mellenbergh, G. J., & Heerden, J. (2004). The concept of validity. *Psychological Review, 111*, 1061–1071. doi:10.1037/0033-295X.111.4.1061

Bradley, M. M., & Lang, P. J. (1994). Measuring emotion: The Self-Assessment Manikin and the Semantic Differential. *Journal of Behavior Therapy and Experimental Psychiatry, 25*, 49–59. doi:10.1016/0005-7916(94)90063-9

Brown, E. J., Turovsky, J., Heimberg, R. G., Juster, H. R., Brown, T. A., & Barlow, D. H. (1997). Validation of the Social Interaction Anxiety Scale and the Social Phobia Scale across the anxiety disorders. *Psychological Assessment, 9*, 21–27. doi:10.1037/1040-3590.9.1.21

Bunnell, B. E., Joseph, D. L., & Beidel, D. C. (2013). Measurement invariance of the Social Phobia and Anxiety Inventory. *Journal of Anxiety Disorders, 27*, 84–91.

Campbell, D. T., & Fiske, D. W. (1959). Convergent and discriminant validation by the multitrait-multimethod matrix. *Psychological Bulletin, 56*, 81–105. doi:10.1037/h0046016

Carleton, R. N., Collimore, K. C., Asmundson, G. J. G., McCabe, R. E., Rowa, K., & Antony, M. M. (2009). Refining and validating the Social Interaction Anxiety Scale and the Social Phobia Scale. *Depression and Anxiety, 26*, 71–81. doi:10.1002/da.20480

Carleton, R. N., Collimore, K. C., McCabe, R. E., & Antony, M. M. (2011). Addressing revisions to the Brief Fear of Negative Evaluation scale: Measuring fear of negative evaluation across anxiety and mood disorders. *Journal of Anxiety Disorders, 25*, 822–828. doi:10.1016/j.janxdis.2011.04.002

Clark, D. B., Turner, S. M., Beidel, D. C., Donovan, J. E., Kirisci, L., & Jacob, R. G. (1994). Reliability and validity of the Social Phobia and Anxiety Inventory for adolescents. *Psychological Assessment, 6*, 135–140. doi:10.1037/1040-3590.6.2.135

Collins, K. A., Westra, H. A., Dozois, D. J. A., & Stewart, S. H. (2005). The validity of the brief version of the Fear of Negative Evaluation Scale. *Journal of Anxiety Disorders, 19*, 345–359. doi:10.1016/j.janxdis.2004.02.003

Costa, P. T., & McCrae, R. R. (1992). *NEO PI-R professional manual.* Odessa, FL: Psychological Assessment Resources.

Courvoisier, D. S., Eid, M., Lischetzke, T., & Schreiber, W. H. (2010). Psychometric properties of a computerized mobile phone method for assessing mood in daily life. *Emotion, 10*, 115–124. doi:10.1037/a0017813

Cox, B. J., Clara, I. P., Sareen, J., & Stein, M. B. (2008). The structure of feared social situations among individuals with a lifetime diagnosis of social anxiety disorder in two independent nationally representative mental health surveys. *Behaviour Research and Therapy, 46*, 477–486. doi:10.1016/j.brat.2008.01.011

Cox, B. J., Ross, L., Swinson, R. P., & Direnfeld, D. M. (1998). A comparison of social phobia outcome measures in cognitive-behavioral group therapy. *Behavior Modification, 22*, 285–297. doi:10.1177/01454455980223004

Crawford, J. R., & Henry, J. D. (2004). The Positive and Negative Affect Schedule (PANAS): Construct validity, measurement properties and normative data in a large non-clinical sample. *British Journal of Clinical Psychology, 43*, 245–265. doi:10.1348/0144665031752934

Cronbach, L., & Meehl, P. (1955). Construct validity in psychological tests. *Psychological Bulletin, 52*, 281–302. doi:10.1037/h0040957

Dawes, R. M., Faust, D., & Meehl, P. E. (1989). Clinical versus actuarial judgment. *Science, 243*, 1668–1674. doi:10.1126/science.2648573

Diener, E., Larsen, R. J., Levine, S., & Emmons, R. A. (1985). Intensity and frequency: Dimensions underlying positive and negative affect. *Journal of Personality and Social Psychology, 48*, 1253–1265. doi:10.1037/0022-3514.48.5.1253

Dimberg, U, Fredrikson, M., & Lundquist, O. (1986). Autonomic reactions to social and neutral stimuli in subjects high and low in public speaking fear. *Biological Psychology, 23*, 223–233. doi:10.1016/0301-0511(86)90001-3

DiNardo, P. A., Brown, T. A., & Barlow, D. H. (1994). *Anxiety disorders interview schedule for DSM-IV*. Albany, NY: Graywind.

Donnellan, M. B., Oswald, F. L., Baird, B. M., & Lucas, R. E. (2006). The Mini-IPIP Scales: Tiny-yet-effective measures of the big five factors of personality. *Psychological Assessment, 18*, 192–203. doi:10.1037/1040-3590.18.2.192

Duke, D., Krishnan, M., Faith, M., & Storch, E. A. (2006). The psychometric properties of the Brief Fear of Negative Evaluation Scale. *Journal of Anxiety Disorders, 20*, 807–817. doi:10.1016/j.janxdis.2005.11.002

Egloff, B., Schmukle, S. C., Burns, L. R., Kohlmann, C., & Hock, M. (2003). Facets of dynamic positive affect: Differentiating joy, interest, and activation in the Positive and Negative Affect Schedule (PANAS). *Journal of Personality & Social Psychology, 85*, 528–540.

Embretson, S. E. (1983). Construct validity: Construct representation versus nomothetic span. *Psychological Bulletin, 93*, 179–197. doi:10.1037/0033-2909.93.1.179

Embretson, S. E., & Reise, S. P. (2000). *Item response theory for psychologists*. Mahwah, NJ: Lawrence Erlbaum.

Feldman Barrett, L., & Russell, J. A. (1998). Independence and bipolarity in the structure of affect. *Journal of Personality and Social Psychology, 74*, 967–984. doi:10.1037/0022-3514.74.4.967

Fergus, T. A., Valentiner, D. P., McGrath, P. B., Stephenson, K., Gier, S., & Jencius, S. (2009). The Fear of Positive Evaluation Scale: Psychometric properties in a clinical sample. *Journal of Anxiety Disorders, 23*, 1177–1183. doi:10.1016/j.janxdis.2009.07.024

Fresco, D. M., Coles, M. E., Heimberg, R. G., Liebowitz, M. R., Hami, S., Stein, M. B., & Goetz, D. (2001). The Liebowitz Social Anxiety Scale: A comparison of the psychometric properties of self-report and clinical-administered formats. *Psychological Medicine, 31*, 1025–1035. doi:10.1017/S0033291701004056

Glinski, K., & Page, A. C. (2010). Modifiability of neuroticism, extraversion, and agreeableness by group cognitive behaviour therapy for social anxiety disorder. *Behaviour Change, 27*, 42–52. doi:10.1375/bech.27.1.42

Greenwald, A. G., McGhee, D. E., & Schwartz, J. L. K. (1998). Measuring individual differences in implicit cognition: The implicit association test. *Journal of Personality and Social Psychology, 74*, 1464–1480. doi:10.1037/4153–2200.6.47.4641

Grove, W. M., Zald, D. H., Lebow, B. S., Snitz, B. E., & Nelson, C. (2000). Clinical versus mechanical prediction: A meta-analysis. *Psychological Assessment, 12*, 19–30. doi:10.1037/1040-3590.12.1.19

Habke, A. M., Hewitt, P. L., Norton, G. R., & Asmundson, G. (1997). The Social Phobia and Social Interaction Anxiety Scales: An exploration of the dimensions of social anxiety and sex differences in structure and relations with pathology. *Journal of Psychopathology and Behavioral Assessment, 19*, 21–39.

Hambrick, J. P., Rodebaugh, T. L., Balsis, S., Woods, C. M., Mendez, J. L., & Heimberg, R. G. (2010). Cross-ethnic measurement equivalence of measures of depression, social anxiety, and worry. *Assessment, 17*, 155–171. doi:10.1177/1073191109350158

Heimberg, R. G., Gansler, D., Dodge, C. S., & Becker, R. E. (1987). Convergent and discriminant validity of the Cognitive-Somatic Anxiety Questionnaire in a social phobic population. *Behavioral Assessment, 9*, 379–388.

Heimberg, R. G., Mueller, G. P., Holt, C. S., Hope, D. A., & Liebowitz, M. R. (1992). Assessment of anxiety in social interaction and being observed by others: The Social Interaction Anxiety Scale and the Social Phobia Scale. *Behavior Therapy, 23*, 53–73. doi:10.1016/S0005-7894(05)80308-9

Hoffart, A., Borge, F.-M., Sexton, H., & Clark, D. M. (2009). Change processes in residential cognitive and interpersonal psychotherapy for social phobia: A process-outcome study. *Behavior Therapy, 40*, 10–22. doi:10.1016/j.beth.2007.12.003

Hofmann, S. G. (2004). Cognitive mediation of treatment change in social phobia. *Journal of Consulting and Clinical Psychology, 72*, 392–399.

Kashdan, T. B., Adams, L., Savostyanova, A., Ferssiczidis, P., McKnight, P. E., & Nezlek, J. B. (2011). Effects of social anxiety and depressive symptoms on the frequency and quality of sexual activity: A daily process approach. *Behaviour Research and Therapy, 49*, 352–360. doi:10.1016/j.brat.2011.03.004

Kashdan, T. B., & Collins, R. L. (2010). Social anxiety and the experience of positive emotion and anger in everyday life: An ecological momentary assessment approach. *Anxiety, Stress and Coping: An International Journal, 23*, 259–272. doi:10.1080/10615800802641950

Klauer, K. C., Voss, A., Schmitz, F., & Teige-Mocigemba, S. (2007). Process components of the implicit association test: A diffusion-model analysis. *Journal of Personality and Social Psychology, 93*, 353–368. doi:10.1037/0022-3514.93.3.353

Kring, A. M., Persons, J. B., & Thomas, C. (2007). Changes in affect during treatment for depression and anxiety. *Behaviour Research and Therapy, 45*, 1753–1764. doi:10.1016/j.brat.2007.02.001

Larsen, R. J., & Diener, E. (1985). A multitrait-multimethod examination of affect structure: Hedonic level and emotional intensity. *Personality and Individual Differences, 6*, 631–636. doi:10.1016/0191-8869(85)90013-3

Leary, M. R. (1983). A brief version of the Fear of Negative Evaluation Scale. *Personality and Social Psychology Bulletin, 9*, 371–375. doi:10.1177/0146167283093007

Liebowitz, M. R. (1987). Social phobia. *Modern Problems in Pharmacopsychiatry, 22*, 141–173.

Lucas, R. E., Diener, E., & Suh, E. (1996). Discriminant validity of well-being measures. *Journal of Personality and Social Psychology, 71*, 616–628. doi:10.1037/0022-3514.71.3.616

Mackinnon, A., Jorm, A. F., Christensen, H., Korten, A. E., Jacomb, P. A., & Rodgers, B. (1999). A short form of the Positive and Negative Affect Schedule: Evaluation of factorial

validity and invariance across demographic variables in a community sample. *Personality and Individual Differences, 27*, 405–416. doi:10.1016/s0191-8869(98)00251-7

Mansell, W., Clark, D. M., Ehlers, A., & Chen, Y. (1999). Social anxiety and attention away from emotional faces. *Cognition and Emotion, 13*, 673–690. doi:10.1080/026999399379032

Marsh, H. W., Lüdtke, O., Muthén, B., Asparouhov, T., Morin, A. J. S., Trautwein, U., & Nagengast, B. (2010). A new look at the big five structure through exploratory structural equation modeling. *Psychological Assessment, 22*, 471–491. doi:10.1037/a0019227

Mattick, R. P., & Clarke, J. C. (1998). Development and validation of measures of social phobia scrutiny fear and social interaction anxiety. *Behaviour Research and Therapy, 36*, 455–470. doi:10.1016/S0005-7967(97)10031-6

Mauss, I. B., Wilhelm, F. H., & Gross, J. J. (2004). Is there less to social anxiety than meets the eye? Emotion experience, expression, and bodily responding. *Cognition and Emotion, 18*, 631–662. doi:10.1080/02699930341000112

Mohr, D. C., Hart, S. L., Julian, L., Catledge, C., Honos-Webb, L., Vella, L., & Tasch, E. T. (2005). Telephone-administered psychotherapy for depression. *Archives of General Psychiatry, 62*, 1007–1014. doi:10.1001/archpsyc.62.9.1007

Mörtberg, E., Clark, D. M., Sundin, O., & Åberg Wistedt, A. (2007). Intensive group cognitive treatment and individual cognitive therapy versus treatment as usual in social phobia: A randomized controlled trial. *Acta Psychiatrica Scandinavica, 115*, 142–154. doi:10.1111/j.1600-0447.2006.00839.x

Moscovitch, D. A., Hofmann, S. G., Suvak, M. K., & In-Albon, T. (2005). Mediation of changes in anxiety and depression during treatment of social phobia. *Journal of Consulting and Clinical Psychology, 73*, 945–952. doi:10.1037/0022-006x.73.5.945

Nunnally, J. D., & Bernstein, I. H. (1994). *Psychometric theory* (3rd ed.). New York, NY: McGraw-Hill.

Oakman, J., Van Ameringen, M., Mancini, C., & Farvolden, P. (2003). A confirmatory factor analysis of a self-report version of the Liebowitz Social Anxiety Scale. *Journal of Clinical Psychology, 59*, 149–161. doi:10.1002/jclp.10124

Osman, A., Barrios, F. X., Aukes, D., & Osman, J. R. (1995). Psychometric evaluation of the Social Phobia and Anxiety Inventory in college students. *Journal of Clinical Psychology, 51*, 235–243. doi:10.1002/1097-4679(199503)51:23.0.co;2-r

Osman, A., Barrios, F. X., Haupt, D., & King, K. (1996). The Social Phobia and Anxiety Inventory: Further validation in two nonclinical samples. *Journal of Psychopathology and Behavioral Assessment, 18*, 35–47. doi:10.1007/bf02229101

Osman, A., Gutierrez, P. M., Barrios, F. X., Kopper, B. A., & Chiros, C. E. (1998). The Social Phobia and Social Interaction Anxiety Scales: Evaluation of psychometric properties. *Journal of Psychopathology and Behavioral Assessment, 20*, 249–264. doi:10.1023/a:1023067302227

Parker, J. D., Bagby, R. M., & Summerfeldt, L. J. (1993). Confirmatory factor analysis of the revised NEO personality inventory. *Personality and Individual Differences, 15*, 463–466. doi:10.1016/0191-8869(93)90074-d

Perugi, G., Nassini, S., Maremmani, I., Madaro, D., Toni, C., Simonini, E., & Akiskal, H. S. (2001). Putative clinical subtypes of social phobia: A factor-analytical study. *Acta Psychiatrica Scandinavica, 104*, 280–288. doi:10.1111/j.1600-0447.2001.00128.x

Peters, L. (2000). Discriminant validity of the Social Phobia and Anxiety Inventory (SPAI), the Social Phobia Scale (SPS), and the Social Interaction Anxiety Scale (SIAS). *Behaviour Research and Therapy, 38*, 943–950. doi:10.1016/S0005-7967(99)00131-X

Peters, L., Sunderland, M., Andrews, G., Rapee, R. M., & Mattick, R. P. (2012). Development of a short form Social Interaction Anxiety (SIAS) and Social Phobia Scale (SPS) using nonparametric item response theory: The SIAS-6 and the SPS-6. *Psychological Assessment, 24*, 66–76. doi:10.1037/a0024544

Puigcerver, A., Martínez-Selva, J. M., García-Sánchez, F. A., & Gómez-Armor, J. (1989). Individual differences in psychophysiological and subjective correlates of speech anxiety. *Journal of Psychophysiology, 3*, 75–81.

Rapee, R. M., Abbott, M. J., Baillie, A. J., & Gaston, J. E. (2007). Treatment of social phobia through pure self-help and therapist-augmented self-help. *The British Journal of Psychiatry, 191*, 246–252. doi:10.1192/bjp.bp.106.028167

Rapee, R. M., & Lim, L. (1992). Discrepancy between self- and observer ratings of performance in social phobics. *Journal of Abnormal Psychology, 101*, 728–731. doi:10.1037/0021-843X.101.4.728

Richardson, H. A., Simmering, M. J., & Sturman, M. C. (2009). A tale of three perspectives: Examining post hoc statistical techniques for detection and correction of common method variance. *Organizational Research Methods, 12*, 762–800. doi:10.1177/1094428109332834

Rodebaugh, T. L., Gianoli, M. O., Turkheimer, E., & Oltmanns, T. F. (2010). The interpersonal problems of the socially avoidant: Self and peer shared variance. *Journal of Abnormal Psychology, 119*, 331–340. doi:10.1037/a0019031

Rodebaugh, T. L., Heimberg, R. G., Brown, P. J., Fernandez, K. C., Blanco, C., Schneier, F. R., & Liebowitz, M. R. (2011). More reasons to be straightforward: Findings and norms for two scales relevant to social anxiety. *Journal of Anxiety Disorders, 25*, 623–630. doi:10.1016/j.janxdis.2011.02.002

Rodebaugh, T. L., Woods, C. M., & Heimberg, R. G. (2007). The reverse of social anxiety is not always the opposite: The reverse-scored items of the Social Interaction Anxiety Scale do not belong. *Behavior Therapy, 38*, 192–206. doi:10.1016/j.beth.2006.08.001

Rodebaugh, T. L., Woods, C. M., Heimberg, R. G., Liebowitz, M. R., & Schneier, F. R. (2006). The factor structure and screening utility of the Social Interaction Anxiety Scale. *Psychological Assessment, 18*, 231–237. doi:10.1037/1040-3590.18.2.231

Rodebaugh, T. L., Woods, C. M., Thissen, D., Heimberg, R. G., Chambless, D. L., & Rapee, R.M. (2004). More information from fewer questions: The factor structure and item properties of the original and brief fear of negative evaluation scales. *Psychological Assessment, 16*, 169–181. doi:10.1037/1040-3590.16.2.169

Ruscio, A. M., Brown, T. A., Chiu, W. T., Sareen, J., Stein, M. B., & Kessler, R. C. (2008). Social fears and social phobia in the USA: Results from the National Comorbidity Survey Replication. *Psychological Medicine, 38*, 15–28. doi:10.1017/S0033291707001699

Rytwinski, N. K., Fresco, D. M., Heimberg, R. G., Coles, M. E., Liebowitz, M. R., Cissell, S., Stein, M. B., & Hofmann, S. G. (2009). Screening for social anxiety disorder with the self-report version of the Liebowitz Social Anxiety Scale. *Depression and Anxiety, 26*, 34–38. doi:10.1002/da.20503

Schmidt, L. A., Santesso, D. L., Miskovic, V., Mathewson, K. J., McCabe, R. E., Antony, M. M., & Moscovitch, D. A. (2012). Test-retest reliability of regional electroencephalogram (EEG) and cardiovascular measures in social anxiety disorder (SAD). *International Journal of Psychophysiology, 84*, 65–73. doi:10.1016/j.ijpsycho.2012.01.011

Shimizu, M., Seery, M. D., Weisbuch, M., & Lupien, S. P. (2011). Trait social anxiety and physiological activation: Cardiovascular threat during social interaction. *Personality and Social Psychology Bulletin, 37*, 94–106. doi:10.1177/0146167210391674

Stone, A. A., & Shiffman, S. (1994). Ecological momentary assessment (EMA) in behavioral medicine. *Annals of Behavioral Medicine, 16*, 199–202.

Stone, A. A., Shiffman, S., Atienza, A., & Nebeling, L. (Eds.). (2007). *The science of real-time data capture: Self-reports in health research*. New York, NY: Oxford University Press.

Stopa, L., & Clark, D. M. (1993). Cognitive processes in social phobia. *Behaviour Research and Therapy, 31*, 255–267. doi:10.1016/0005-7967(93)90024-O

Titov, N., Andrews, G., Schwencke, G., Robinson, E., Peters, L., & Spence, J. (2010). Randomized controlled trial of internet cognitive behavioural treatment for social phobia with and without motivational enhancement strategies. *Australian and New Zealand Journal of Psychiatry, 44*, 938–945. doi:10.3109/00048674.2010.493859

Trull, T. J., Solhan, M. B., Tragesser, S. L., Jahng, S., Wood, P. K., Piasecki, T. M., & Watson, D. (2008). Affective instability: Measuring a core feature of borderline personality disorder with ecological momentary assessment. *Journal of Abnormal Psychology, 117*, 647–661. doi:10.1037/a0012532

Turner, S. M., Beidel, D. C., & Dancu, C. V. (1996). *Social phobia and anxiety inventory: Manual*. Toronto, ON: Multi-health Systems.

Turner, S. M., Beidel, D. C., Dancu, C. V., & Stanley, M. A. (1989). An empirically derived inventory to measure social fears and anxiety: The Social Phobia and Anxiety Inventory. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 1*, 35–40. doi:10.1037/1040-3590.1.1.35

Turner, S. M., Stanley, M. A., Beidel, D. C., & Bond, L. (1989). The Social Phobia and Anxiety Inventory: Construct validity. *Journal of Psychopathology and Behavioral Assessment, 11*, 221–234. doi:10.1007/bf00960494

Vassend, O., & Skrondal, A. (1995). Factor analytic studies of the NEO personality inventory and the five-factor model: The problem of high structural complexity and conceptual indeterminacy. *Personality and Individual Differences, 19*, 135–147. doi:10.1016/0191-8869(95)00041-4

Vassend, O., & Skrondal, A. (1997). Validation of the NEO personality inventory and the five-factor model. Can findings from exploratory and confirmatory factor analysis be reconciled? *European Journal of Personality, 11*, 147–166. doi:10.1002/(sici)1099-0984(199706)11:23.0.co;2-e

Villodas, F., Villodas, M. T., & Roesch, S. (2011). Examining the factor structure of the positive and negative affect schedule (PANAS) in a multiethnic sample of adolescents. *Measurement and Evaluation in Counseling and Development, 44*, 193–203. doi:10.1177/0748175611414721

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*, 1063–1070. doi:10.1037/0022-3514.54.6.1063

Weeks, J. W., Heimberg, R. G., Fresco, D. M., Hart, T. A., Turk, C. L., Schneier, F. R., & Liebowitz, M. R. (2005). Empirical validation and psychometric evaluation of the Brief Fear of Negative Evaluation Scale in patients with social anxiety disorder. *Psychological Assessment, 17*, 179–190. doi:10.1037/1040-3590.17.2.179

Weeks, J. W., Heimberg, R. G., & Heuer, R. (2011). Exploring the role of behavioral submissiveness in social anxiety. *Journal of Social and Clinical Psychology, 30*, 217–249. doi:10.1521/jscp.2011.30.3.217

Weeks, J. W., Heimberg, R. G., & Rodebaugh, T. L. (2008). The Fear of Positive Evaluation Scale: Assessing a proposed cognitive component of social anxiety. *Journal of Anxiety Disorders, 22*, 44–55. doi:10.1016/j.janxdis.2007.08.002

Weeks, J. W., Heimberg, R. G., Rodebaugh, T. L., Goldin, P. R., & Gross, J. J. (2012). Psychometric evaluation of the Fear of Positive Evaluation Scale in patients with social anxiety disorder. *Psychological Assessment, 24*, 301–312. doi:10.1037/a0025723

Weeks, J. W., Heimberg, R. G., Rodebaugh, T. L., & Norton, P. J. (2008). Exploring the relationship between fear of positive evaluation and social anxiety. *Journal of Anxiety Disorders, 22*, 386–400. doi:10.1016/j.janxdis.2007.04.009

Westberg, P., Lundh, L., & Jonsson, P. (2007). Implicit associations and social anxiety. *Cognitive Behaviour Therapy, 36*, 43–51. doi:10.1080/08037060601020401

IV

Symptomological Manifestations

15

Cognitive Biases among Individuals with Social Anxiety

Shari A. Steinman, Eugenia I. Gorlin, and Bethany A. Teachman

University of Virginia, USA

Why do individuals with social anxiety believe that they are incompetent in social situations, sometimes even in the absence of negative feedback from others? Even more puzzling, why does positive feedback often fail to ameliorate social anxiety? In this chapter, we review empirical evidence for cognitive biases in individuals diagnosed with social anxiety disorder (SAD) and individuals with high levels of social anxiety (HSA, typically determined by exceeding a cut point on a questionnaire measure of social anxiety symptoms). These biases are posited to maintain, and potentially cause, social anxiety (e.g., Beck, Emery, & Greenberg, 1985; Clark & Wells, 1995; Heimberg, Brozovich, & Rapee, 2010). Before evaluating the evidence for specific categories of cognitive bias, we provide a brief, general overview of cognitive models of social anxiety to lay the theoretical groundwork for the biases reviewed in this chapter (see also **Chapter 1** of this text and Musa & Lépine, 2000).

According to cognitive models (e.g., Beck et al., 1985; Clark & Wells, 1995), social anxiety is characterized by negative beliefs about the self in social situations. These beliefs include very high standards for the self ("I must never say the wrong thing"), conditional beliefs related to negative evaluation ("If others notice I am sweating, then they will reject me"), and unconditional beliefs about the self ("I am not interesting"). Consequently, socially anxious individuals experience anxiety prior to, during, and often following social situations. In their influential model, Clark and Wells emphasized that when individuals with social anxiety fear negative evaluation, they shift their attention inward, such that they focus on their emotions, including anxiety, and their bodily sensations, such as blushing or heart pounding. This shift can prevent socially anxious individuals from processing external cues, including others' actual behavior, leaving socially anxious individuals to rely on their own feelings and cognitive biases to determine how others view them. For example, someone with social anxiety may miss potentially corrective social feedback, such as someone smiling at them, and may instead think, "I feel anxious, so everyone must know I am anxious."

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

Additionally, Clark and Wells (1995) theorized that socially anxious individuals tend to interpret ambiguous social cues in a negative manner and to engage in behaviors designed to reduce potential rejection, such as mentally rehearsing what to say prior to speaking aloud. These behaviors both preclude the opportunity for socially anxious individuals to learn that what they fear does not actually occur and can have detrimental effects on social performance. Finally, following a social situation, socially anxious individuals tend to ruminate over perceived negative aspects of the interaction, reinforcing their negative beliefs about themselves in social situations. Building on Clark and Wells' (1995) model, Rapee and Heimberg (1997) added that individuals with social anxiety create distorted mental representations of how they appear to others, based on images in memory of how they appear and on their perception of internal (e.g., bodily sensations) and external (e.g., feedback from others) cues, all of which can be biased, as we outline below.

Our goal in this chapter is to review the empirical evidence for many of the key cognitive biases as they occur in social anxiety. Note that we use the term "bias" to refer to a relative difference between groups, rather than to an absolute level of accuracy (see Mathews & MacLeod, 2005). The literature on interpretation and judgment, unconscious and conscious selective attention, post-event processing (PEP) and memory, and implicit associations is reviewed. For each bias, when available, evidence for known group differences, predictive validity, and malleability is discussed. As will become evident, there are mostly data indicating group differences, rather than longitudinal or experimental studies testing the role of these biases in predicting or causing critical social anxiety outcomes, leaving many questions for future research.

Interpretations and Judgment

Social situations are often ambiguous, such that signs of approval or disapproval from others are not always readily apparent. For instance, during a public speech, audience members rarely give wide smiles and thumbs-up. Cognitive models of anxiety posit that individuals with social anxiety are more likely than nonanxious individuals to interpret this ambiguity in a threatening manner and to judge themselves negatively in social situations (e.g., Beck & Clark, 1997; Beck et al., 1985; Clark & Wells, 1995). Moreover, these models suggest that these judgment and interpretive biases maintain, and potentially cause, social anxiety. In this section, we review the literature on how individuals with HSA and SAD judge the likelihood and catastrophic nature of negative social events, interpret ambiguous information, and appraise valenced information.

With regard to judgment biases, individuals with HSA (Clark & Arkowitz, 1975) and SAD (Rapee & Lim, 1992) tend to judge their own behavior more negatively in social situations, but tend to be more accurate when judging others' behavior, in comparison to independent observer ratings. Additionally, individuals with SAD overestimate the likelihood of negative social events occurring, as well as their related costs, compared to nonanxious individuals (e.g., Foa, Franklin, Perry, & Herbert,

1996). Importantly, reduction in the estimated costs associated with negative social events predicts level of social anxiety following treatment (Foa et al., 1996).

To assess how individuals interpret ambiguous information, Amir, Foa, and Coles (1998) presented participants with ambiguous scenarios and then had them rank the likelihood of disambiguated interpretations of the scenarios (i.e., positive, negative, or neutral interpretations). Individuals with SAD interpreted ambiguous social scenarios, but not nonsocial scenarios, negatively. Further, nonanxious individuals and individuals with obsessive-compulsive disorder did not show this bias, suggesting that it may be specific to social anxiety. The finding that individuals with SAD and HSA interpret social ambiguity negatively has been well replicated (e.g., Stopa & Clark, 2000) and extended to more ecologically valid measures, such as interpreting a confederate's ambiguous behaviors during a speech (Kanai, Sasagawa, Chen, Shimada, & Sakano, 2010) and interpreting videos of ambiguous social behavior (Amir, Beard, & Bower, 2005).

Rather than displaying an exaggerated negative bias, some studies have found that individuals with HSA or SAD lack a normative positive bias (e.g., Constans, Penn, Ihen, & Hope, 1999). Using a measure in which participants made interpretations about information as it was presented (i.e., a measure of online interpretation bias), Hirsch and Mathews (2000) had participants with SAD and nonanxious participants complete a lexical decision task at various points in an ambiguous text about job interviews. Results suggested that nonanxious participants had positively biased interpretations of ambiguous social events, while participants with SAD lacked this bias (see also studies using event-related potential methodology, e.g., Moser, Huppert, Foa, & Simons, 2012).

Not all social situations are ambiguous, so it is critical to also understand how socially anxious individuals appraise valenced social information. Questionnaire-based studies have found that individuals with HSA (Vassilopoulos, 2006) and SAD (Stopa & Clark, 2000) evaluate mildly negative social situations as catastrophic and tend to appraise positive events more negatively than nonanxious individuals (e.g., Alden, Taylor, Mellings, & Laposa, 2008). These findings may help explain why socially anxious individuals often disqualify positive social performance feedback (e.g., Kashdan, Weeks, & Savostyanova, 2011). Biased evaluations of bodily sensations, such as sweating and blushing, are also common, including the belief that bodily sensations are visible to others and likely to result in negative evaluations (Kanai et al., 2009; Roth, Antony, & Swinson, 2001; see also **Chapter 5**).

One of our most important and ubiquitous interpersonal activities is responding to others' facial expressions, and this is another area of bias for socially anxious persons. Several studies have shown that socially anxious individuals are likely to interpret ambiguous or neutral faces more negatively than nonanxious individuals (e.g., Mohlman, Carmin, & Price, 2007; Yoon & Zinbarg, 2007). Individuals with SAD also tend to rate happy or positive faces as less approachable (Campbell et al., 2009), more critical (Vassilopoulos, 2011), and more rejecting (Stevens, Gerlach, & Rist, 2008, although see also Gilboa-Schechtman, Foa, Vaknin, Marom, Hermesh, 2008) than do nonanxious control participants.

Despite the robustness of social-anxiety-linked interpretation and judgment biases, they appear to be malleable. Following cognitive behavioral therapy, Wilson and

Rapee (2005) found that participants with SAD had significant reductions in negative interpretations of social events (see also Voncken & Bögels, 2006). Additionally, social anxiety symptoms at a 3-month follow-up assessment were predicted by treatment-related change in the belief that negative social situations are indicative of negative characteristics about the self.

Further evidence for bias malleability stems from the emerging cognitive bias modification (CBM) literature. In CBM paradigms targeting interpretation bias, participants are trained to repeatedly generate benign or positive interpretations of ambiguous information. Murphy, Hirsch, Mathews, Smith, and Clark (2007) had participants with HSA imagine themselves to be in a series of ambiguous social scenarios. In the two active training conditions (a positive training and a nonnegative training condition), the scenarios were disambiguated in a benign manner, while in the control condition, the ambiguity was not resolved. For example, in a scenario related to giving a speech, participants in the active conditions received information about the speech going well, while those in the control condition were not given information about how the speech went. Relative to participants in the control condition, participants in the active CBM conditions subsequently interpreted novel ambiguous scenarios more positively and less negatively and reported less anticipatory anxiety about an expected future social situation. Similar results have been found using other interpretation-based CBM paradigms, and importantly, modifying interpretations has been shown to decrease social anxiety symptoms (e.g., Amir & Taylor, 2012; Beard & Amir, 2008). These studies highlight the malleability of interpretation bias and provide support for its hypothesized causal role in altering social anxiety symptoms.

In summary, converging research suggests that individuals with HSA and SAD (compared to nonsocially anxious individuals) judge themselves more negatively than they judge others and interpret social cues more negatively, whether the cues are ambiguous, slightly negative, or even positive. Note that sometimes this is seen as a bias toward threat, while at other times it is seen as a lack of a normative positive bias or even a fear of positive evaluation from others (see Weeks, Heimberg, Rodebaugh, Goldin, & Gross, 2012; see also **Chapter 20**). These biases appear to be malleable; and reducing these biases can decrease social anxiety, pointing to the importance of targeting biased interpretation and judgment in treatment.

Selective Attention Bias

Cognitive models of social anxiety emphasize that socially anxious persons have biased attention, such that they selectively attend to threatening information (e.g., Clark & Wells, 1995; Rapee & Heimberg, 1997). Further, they often do not attend to more positive or neutral information, and consequently miss potentially corrective social information. In this section, literature on attention biases in individuals with HSA and SAD is reviewed, focusing particularly on studies using the Dot probe (MacLeod, Mathews, & Tata, 1986) and emotional Stroop (modified from the classic task by Stroop, 1935) tasks. First, attentional biases that occur outside of conscious awareness, typically assessed through subliminal tasks that present masked material for very brief durations (usually no longer than 32 ms), are discussed. Next, attention biases that

occur within conscious awareness (assessed through the same tasks, except that the material is presented for longer durations and is not masked) are outlined.

Unconscious Selective Attention

Given the adaptive nature of detecting threats quickly, many theorists have noted that survival is likely enhanced by rapidly identifying potential signs of danger, even preconsciously (see Öhman & Mineka, 2001). Specific to social anxiety, threatening facial expressions are thought to be important indicators of social dominance that should be selectively processed because of their significant consequences for determining one's position on the social hierarchy (e.g., Dimberg & Öhman, 1996).

In fact, even nonanxious individuals have been found to detect threatening faces more rapidly than friendly faces among a set of distractors (Öhman, Lundqvist, & Esteves, 2001). Moreover, evidence of selective processing endured in healthy individuals even when threatening faces, such as angry facial expressions, were backward masked (i.e., presented very briefly and then followed with an alternate image, so that the person was not aware of what expression was briefly flashed; e.g., Parra, Esteves, Flykt, & Öhman, 1997).

In this section, we consider whether this normative tendency to preferentially process social threat cues outside conscious awareness operates differently among persons with SAD or HSA, relative to nonanxious individuals. Cognitive models of anxiety point to the importance of automatic processing of disorder-relevant information (see Teachman, Joormann, Steinman, & Gotlib, 2012), leading to an expectation that socially anxious persons will be especially primed and motivated to orient toward potential threat cues. This would presumably result in faster responding to threatening (relative to nonthreatening) material in socially anxious versus nonanxious samples. At the same time, vigilance-avoidance models (e.g., Mogg & Bradley, 2004) suggest that this initial hypervigilance for threat cues will soon be followed by motivated avoidance of the cues because of the perceived danger of interacting with the threatening material. Thus, depending on the timing of the measurement, there are theoretical reasons to predict both greater attention toward *and* away from social threat cues by individuals with social anxiety.

The paradigms used most frequently to assess unconscious processing of social threat cues include the subliminal or masked versions of the Dot probe and emotional Stroop tasks. In these tasks, response latencies to name ink color of a picture or word (on the emotional Stroop) or to identify some feature of a probe (such as the probe's location or direction of an arrow on the Dot probe task) are compared when the stimulus has been linked to threat material, such as angry facial expressions, versus when the stimulus has been linked to nonthreatening material, such as neutral faces. The difference in response latency is used to infer selective attention to the threat stimuli. The tasks capture unconscious processing when the threat and neutral material are presented under conditions of restricted awareness, so that the person cannot report the specific facial expressions or words that were presented.

There is limited evidence for preferential attentional orienting toward masked angry versus neutral faces in individuals with HSA, compared to nonanxious individuals.

Notwithstanding, on a Dot probe task, Mogg and Bradley (2002) found that a sample with HSA was faster at responding to probes following masked threat (vs. neutral) stimuli when presented for 7–14 ms (see also Kim, Lundh, & Harvey, 2002). Interestingly, Mogg and Bradley also found that the threat orienting bias was more pronounced when threat cues appeared in the left (vs. right) visual field, which they interpreted as indicating that the right hemisphere plays a particularly important role in processing faces preconsciously. Similarly, there is little evidence for social anxiety group differences indicating preferential avoidance of masked threat cues, though Putman, Hermans, and van Honk (2004) found that higher scores on a social anxiety questionnaire among an unselected student sample were associated with greater avoidance of angry faces when presented for 25 ms.

The bulk of the evidence indicates null results when comparing persons with SAD versus nonanxious persons' response latency when presented with masked or briefly presented social threat cues (compared to neutral cues, e.g., LeMoult & Joormann, 2012; Mogg, Philippot, & Bradley, 2004; Mueller et al., 2009). There are, however, a number of challenges in interpreting these null findings. These include the very small number of studies that have directly examined unconscious processing in SAD, the frequent use of small sample sizes, and methodological concerns about whether repeated exposure to the threat cues leads to habituation (and a consequent decrement in the hypervigilance effect; Ononaiye, Turpin, & Reidy, 2007). Further, study designs have varied greatly with regard to paradigms and stimuli used, samples with confirmed SAD versus HSA, duration of stimulus and mask presentations, extent that a check for awareness was conducted, and so forth, making it difficult to compare results across studies. Moreover, it seems likely that a number of factors may moderate the selective processing effects, including the role of comorbidity (especially depression; see LeMoult & Joormann), influence of stress or state anxiety (see Ononaiye et al.; van Peer, Spinhoven, & Roelofs, 2010), and effects of alcohol (see Stevens, Rist, & Gerlach, 2009). Perhaps most importantly, there are studies suggesting that even when there are no group differences between SAD and nonanxious samples on the behavioral reaction time measures, other (perhaps more sensitive) markers suggest group differences in early processing of threat cues: for example, event-related potentials (e.g., Mueller et al.; van Peer et al.); eye gaze (e.g., Horley, Williams, Gonsalvez, & Gordon, 2003); and intensity of expression required to identify angry faces (e.g., Joormann & Gotlib, 2006).

Thus, at this stage, the clearest conclusion is that we do not yet know whether, or under what conditions, unconscious processing of social threat cues varies for individuals with SAD and HSA, relative to nonanxious individuals. Resolving this issue could help to determine just how early social anxiety leads to distortions in the stream of processing biases. It is perhaps not remarkable that evidence for social-anxiety-linked group differences has been mixed to date, given the evidence for normative, selective processing of social threat cues, the methodological challenges of presenting stimuli so that they are identified but do not reach conscious awareness, and the timing issues that arise in determining when to expect vigilance versus avoidance. Further, given that group differences in unconscious processing biases have not yet been consistently demonstrated, it is unsurprising that we also know little about the predictive validity of these biases or their malleability and treatment sensitivity.

Conscious Selective Attention

In contrast to the mixed results for unconscious selective attention, a compelling body of research shows that conscious selective attention to social threat cues occurs even at relatively early stages of conscious processing in samples with SAD and HSA (e.g., Amir, Freshman, & Foa, 2002; Hope, Rapee, Heimberg, & Dombeck, 1990; Mogg & Bradley, 1998). These findings are consistent with cognitive models suggesting that preferential attention to external threat cues (e.g., Williams, Watts, MacLeod, & Mathews, 1997) and negative self-focused attention (e.g., Clark & Wells, 1995) are central features of social anxiety. Notably, this self-focused attention can be both semantic and/or visual, given evidence that individuals with SAD and HSA experience negative self-imagery related to their social performance, such as exaggerated mental images of the extent to which they are blushing or sweating (e.g., Hirsch, Meynen, & Clark, 2004; Makkar & Grisham, 2011). The tendency to orient toward this negative, self-relevant social information is theorized to reinforce negative self-perceptions that maintain social anxiety.

Research on conscious threat processing in social anxiety largely relies on the same paradigms used in unconscious processing research, such as the emotional Stroop and the modified Dot probe task described above, except that the tasks use unmasked, or *supraliminal*, stimulus presentation, that is, stimuli are presented without subsequent masking and for a duration that allows for conscious identification (usually ≥500 ms, though this can be briefer; see Bar-Haim, Lamy, Pergamin, Bakermans-Krane, & van IJzendoorn, 2007). Numerous studies have shown that individuals with HSA (vs. nonanxious individuals) are slower to name the colors of socially threatening words, relative to neutral or positive words, on the unmasked emotional Stroop (referred to as an *interference effect;* e.g., Hope et al., 1990; Kim et al., 2002). Moreover, this effect appears specific to social threat, as opposed to physical threat or neutral stimuli (e.g., Lundh & Öst, 1996). Notwithstanding, some null findings have been reported, particularly when facial rather than word stimuli are used (e.g., Putman et al., 2004).

The majority of studies employing Dot probe paradigms have also found that individuals with HSA, relative to nonanxious participants, exhibit an early attentional bias toward threat, as evidenced by faster latencies when the target probe replaces a threat stimulus (usually an angry face) than when it replaces a neutral stimulus (usually a neutral face; e.g., Mogg & Bradley, 2002; Pishyar, Harris, & Menzies, 2004). However, the effect typically appears only at relatively brief stimulus durations (e.g., at 500 ms, but not at 1250 ms; Mogg et al., 2004). To clarify whether this bias reflects enhanced vigilance for threat or difficulty disengaging from threatening material once it has captured attention, Amir, Elias, Klumpp, and Przeworski (2003) used a modified Posner task, which is a variant of the Dot probe in which only one threat or neutral cue is presented per trial, thus allowing for disambiguation between these attentional processes. They found preliminary evidence for difficulty disengaging from threat, but not more rapid orienting to threat cues in SAD, compared to healthy control individuals. Studies using eye-tracking technology to assess visual attention by tracking direction and duration of eye fixations have lent further support to this finding (e.g. Buckner, Maner, & Schmidt, 2010; Schofield, Johnson, Inhoff, & Coles, 2012).

The nature of this attentional bias appears to vary with the particular task conditions, such as length of stimulus presentation. For instance, several Dot probe studies using longer stimulus durations, such as 1000 ms, have actually found that individuals with SAD selectively orient *away* from threat (e.g., Chen, Ehlers, Clark, & Mansell, 2002), consistent with the later phase of the vigilance-avoidance model. It also appears that the attentional bias can be suppressed under conditions of elevated state anxiety (e.g., Amir et al., 1996) or following affective priming with a social threat word (Helfinstein, White, Bar-Haim, & Fox, 2008). In fact, Mansell, Clark, Ehlers, and Chen (1999) found that the direction of attentional bias was reversed, with participants with HSA (relative to nonanxious participants) exhibiting attentional avoidance of threat stimuli on a 500 ms Dot probe task, but only when expecting to give a speech. Amir et al. (2002) speculated that the attenuation in Stroop interference and other cognitive biases when state anxiety is elevated may be due to increased effortful control when a social threat is imminent. Perhaps the alarming escalation in state anxiety motivates the anxious individual to mobilize his/her remaining controlled processing resources, which in turn enable him/her to shift his/her initial vigilance bias to one of motivated avoidance in an attempt to reduce exposure to the noxious stimulus and decrease state anxiety.

Attentional biases in social anxiety have also been studied using visual search and "face-in-the-crowd" paradigms (Byrne & Eysenck, 1995), which measure the speed at which participants detect faces of a given valence, such as "happy" or "angry," within an array of distracter faces. These tasks provide further evidence that individuals with SAD and HSA (vs. nonanxious individuals) selectively orient toward angry, rather than happy or neutral, faces (e.g., Gilboa-Schechtman, Foa, & Amir, 1999; Perowne & Mansell, 2002). Eastwood et al. (2005) further found that detection speed for participants with HSA was less impacted than nonanxious participants' speed by increasing the number of distracter faces when detecting a negative face, suggesting that individuals with HSA may exhibit more efficient detection of negative than positive stimuli. However, this effect requires replication (see Teachman et al., 2012).

Beyond this cross-sectional evidence showing a relationship between social anxiety status and attention biases, there is also some evidence that these biases are sensitive to treatment of, and even causally related to, social anxiety. For instance, several studies have shown significant reductions in Stroop interference for social threat words following cognitive behavioral therapy and/or pharmacotherapy for social anxiety, but only among treatment responders (e.g., Lundh & Öst, 2001; Mattia, Heimberg, & Hope, 1993). Further, recent findings indicate that CBM paradigms that directly train less threat-oriented attention biases (typically using a modified Dot probe paradigm to train attention away from threatening facial stimuli) can significantly reduce social anxiety symptoms (Amir et al., 2009; Li, Tan, Qian, & Liu, 2008; Schmidt, Richey, Buckner, & Timpano, 2009).

In sum, research on conscious attention biases in social anxiety suggests early engagement with social threat cues in socially anxious samples, with somewhat more mixed findings regarding the role of attentional disengagement at later stages of processing. Further research is needed to map the time course of these attentional processes more precisely and to disentangle the effects of moderating factors, such as

state anxiety, type of stimulus (such as face vs. word), and comorbidity (particularly with depression, e.g., LeMoult & Joormann, 2012). Finally, recent CBM findings provide evidence of a causal link between modification of these biases and changes in social anxiety symptoms.

Post-event Processing and Memory

In addition to the biased processing of threat cues *during* social situations, individuals with SAD and HSA also engage in repetitive, self-focused thought processes *following* social interactions, further distorting their self-perceptions in memory (e.g., Brozovich & Heimberg, 2008). According to Clark and Wells' (1995) model, this tendency to ruminate on the negative aspects of one's performance after a social interaction—known as *post-event processing*—should lead to negatively biased memory for social-evaluative situations, which in turn maintains and reinforces socially anxious individuals' negative beliefs about themselves in social situations. This section focuses on evidence for the role of PEP and negatively biased memory in social anxiety, though it should be noted that socially anxious samples also exhibit biased anticipatory processing (e.g., Mellings & Alden, 2000; Vassilopoulos, 2008).

There is strong support for the prediction that PEP is characteristic of individuals with HSA (e.g., Edwards, Rapee, & Franklin, 2003; Lundh & Sperling, 2002) and is associated with more negative self-evaluations of social performance (compared to nonanxious individuals, e.g., Abbott & Rapee, 2004; Dannahy & Stopa, 2007). Importantly, PEP following social interactions also predicts avoidance of similar, future social situations (Rachman, Grüter-Andrew, & Shafran, 2000, though this finding is in need of replication).

While the role of PEP in social anxiety is relatively well established, tests of the subsequent prediction that socially anxious individuals will exhibit a memory bias for socially threatening, relative to neutral or positive, information have yielded mixed results. Given the considerable variability in findings depending on the types of stimuli (pictorial vs. verbal), encoding procedure (implicit vs. explicit), and retrieval mode (recognition vs. recall) used, it is difficult to draw general conclusions about the nature of anxiety-related memory biases (see Mitte, 2008, for a meta-analysis). For instance, most studies using implicit memory and recognition bias paradigms have not found social anxiety group differences, with the exception of studies assessing recognition bias for threatening face stimuli (Mitte, 2008). On the other hand, a number of studies have demonstrated that participants with SAD and HSA show a recall bias for negative self-relevant information (e.g., Amir, Coles, Brigidi, & Foa, 2001; Edwards et al., 2003), though these findings have also been inconsistent (see Coles & Heimberg, 2002; Morgan, 2010).

Given the divergent results in this area, researchers have called for a focus on specific contextual factors that vary across studies, such as stimulus type, encoding procedure, and presence of state anxiety, as potential moderators of the link between social anxiety and memory bias (Mitte, 2008). Indeed, several studies have found that the manipulation of certain state-specific factors, including state anxiety, PEP, and performance feedback, differentially affects recall of autobiographical information based on social

anxiety status (e.g., Dannahy & Stopa, 2007). For instance, Morgan and Banerjee (2008) found that participants with HSA, but not nonanxious participants, instructed to focus on negative feedback following a social task later recalled more anxiety-relevant memories than those who focused on positive feedback. Relatedly, Cody and Teachman (2010) found an anxiety-linked recognition (though not recall) bias following the provision of performance feedback after a speech, such that participants with HSA remembered their negative feedback as worse than nonanxious participants and showed reduced recognition for their positive feedback over time. Further, the degree of PEP following the speech mediated the relationship between social anxiety status and negative recognition bias. This finding builds on earlier research suggesting that individuals with SAD and HSA remember their social performance more negatively over time, relative to nonanxious individuals, due to the influence of PEP (Abbott & Rapee, 2004; Mellings & Alden, 2000). There is also preliminary evidence that individuals with HSA preferentially remember more global ("I gave a terrible speech") versus local or specific ("I said 'um' too many times") social information (Cody & Teachman, 2011).

An important qualification to these findings is that the self-appraisals of individuals with SAD following a social task often do not become more negative over time, but rather remain stable, whereas healthy control participants become more positive in their self-appraisals (Abbott & Rapee, 2004). Akin to some of the other evidence reviewed in this chapter, this suggests that social anxiety may be characterized as much (or, in some cases, more) by the lack of a normative bias for positive social information as by the presence of a more negative bias. For instance, using a directed forgetting paradigm in which participants were shown positive, neutral, and negative social words and instructed to remember or forget each word, Liang, Hsu, Hung, Wang, and Lin (2011) found that participants with HSA were able to forget more positive social words when directed to do so than were nonanxious participants.

By contrast, still other studies have shown a memory bias *away* from negative stimuli in social anxiety, further attesting to the variability in findings depending on the study design and context. For instance, LeMoult and Joormann (2012) found that participants with SAD, relative to control participants, recognized fewer angry (but not happy, sad, or disgusted) faces that they had seen presented on an attentional Dot probe task. The authors attributed this finding to an avoidance bias during encoding of social threat stimuli. This interpretation is further supported by evidence from thought suppression studies in which participants with SAD and HSA show an enhanced ability to suppress socially threatening information, relative to control participants (Cougle, Smits, Lee, Powers, & Telch, 2005; Kingsep & Page, 2010). Moreover, using a judgment latency paradigm in which participants were instructed to suppress certain social threat and neutral prime words, Kingsep and Page found that participants with SAD showed more "successful" suppression of social threat primes than did control participants. The authors posited that this enhanced suppression of social threat may prevent individuals with SAD from fully engaging with (and disconfirming) their socially threatening thoughts, which may lead to greater anxiety and more negatively distorted self-evaluations in the long run.

To date, there has been minimal research examining the malleability of PEP and memory biases in social anxiety, although some promising initial findings suggest that

PEP is reduced following 12 weeks of cognitive behavioral therapy (Abbott & Rapee, 2004). However, as PEP was only assessed at the start and end of treatment, it is unclear whether reduction in PEP preceded or followed changes in other clinical symptoms, such as avoidance or negative self-appraisals. Even fewer studies have directly examined the treatment sensitivity of social anxiety-relevant memory biases, although preliminary findings from a nonclinical sample suggest that CBM training for interpretation biases can lead to a subsequent reduction in the corresponding memory bias (Tran, Hertel, & Joormann, 2011). These results highlight the need for further research aimed at shifting these post-event and memory biases more directly, as well as examining their interactive roles in the maintenance of social anxiety.

Taken together, these findings suggest that intrusive self-focused thought following social situations, such as PEP, is a hallmark of social anxiety and that biased memory for anxiety-relevant information occurs under certain circumstances in socially anxious individuals—perhaps especially when recalling social threat material under conditions of elevated state anxiety and increased PEP and/or when processing more ecologically valid types of stimuli, such as threatening faces versus words. However, there are many open questions about when socially anxious individuals will show enhanced memory for, versus avoidance of, self-relevant social information.

Implicit Associations

In this section, evidence for implicit associations related to social anxiety is reviewed. These studies reflect *uncontrollable processing*, a key feature of pathological anxiety centering on the inability to stop or modify processing of disorder-relevant material once it has begun (see McNally, 1995; Teachman et al., 2012). In typical implicit association tasks, participants are asked to rapidly categorize disorder-relevant stimuli into superordinate categories. For example, in the Implicit Association Test (IAT; Greenwald, McGhee, & Schwartz, 1998), participants view stimuli from four superordinate categories (e.g., "self," "others," "good," "bad"), which are paired together in ways that match or contradict participants' hypothesized implicit associations in memory (i.e., "self" paired with "bad" reflecting negative self-esteem, vs. "self" paired with "good" reflecting positive self-esteem). By comparing reaction times for classification of stimuli under different category pairing conditions, the strength of implicit associations can be inferred. See **Chapter 16** for a discussion of implicit approach/avoidance tendencies, which provide further evidence for biased associations among socially anxious individuals (e.g., using the Approach/Avoidance Task; Rinck & Becker, 2007).

The first study to apply implicit association measures to a socially anxious sample found that, relative to nonanxious individuals, individuals with HSA implicitly associate social cues ("date") with negative outcomes ("rejection"; de Jong, Pasman, Kindt, & van den Hout, 2001, though, see also Sasaki, Iwanaga, Kanai, & Seiwa, 2010, for null results using a similar measure). Using the Single Target IAT (stIAT; Wigboldus, Holland, & van Knippenberg, 2005), a variant of the IAT that does not require an explicitly labeled, relative comparison category, Glashouwer, Vroling, de Jong, Lange, and de Keijser (2012) found converging evidence that individuals with

SAD have stronger associations between social cues and negative outcomes, relative to both nonclinical control participants and individuals diagnosed with panic disorder (see also de Hullu, de Jong, Sportel, & Nauta, 2011, for similar results comparing HSA and nonanxious adolescent samples). The Glashouwer et al. finding suggests that these social outcome associations may be SAD specific, rather than related to anxiety more generally.

Individuals with HSA and SAD also implicitly associate the self and bodily sensations with negative evaluations. For instance, individuals with HSA have significantly lower implicit self-esteem than nonanxious individuals (e.g., de Jong, 2002; Tanner, Stopa, & De Houwer, 2006). Moreover, self-anxious and self-depressed associations have been shown to significantly predict the onset of SAD between initial testing and a 2-year follow-up (Glashouwer, de Jong, & Penninx, 2011). Additionally, individuals with SAD and a fear of blushing have stronger implicit associations between blushing and negative outcomes, relative to nonanxious individuals (Glashouwer, de Jong, Dijk, & Buwalda, 2011). Similarly, participants with HSA have stronger implicit associations between terms linked to anxiety reactions (mostly bodily sensations, such as "blush" or "sweating") and negative evaluations (such as "rejection" or "criticism"), relative to nonanxious individuals (Sasaki et al., 2010). However, in an unselected adolescent sample, Teachman and Allen (2007) found no significant relationship between an IAT measuring associations with the self as rejected versus liked and an explicit measure of social anxiety symptoms, suggesting perhaps that the implicit association measures are more sensitive within adult samples and/or require selection of highly symptomatic individuals to show effects.

Social-anxiety-linked implicit associations appear to be malleable following interventions (though malleability following state anxiety manipulations has mixed supporting evidence; see Schmukle & Egloff, 2004; Westberg, Lundh, & Jönsson, 2007). For example, following cognitive behavioral therapy, the self-anxious implicit associations of HSA individuals significantly decreased (Gamer, Schmukle, Luka-Krausgrill, & Egloff, 2008). Similarly, *cognitive preparation* (Harvey, Clark, Ehlers, & Rapee, 2000) before listening to their own tape-recorded speech led to lower associations between the self and social-anxiety-relevant words ("embarrassed"), relative to a condition that did not receive cognitive preparation (Nilsson, Lundh, Faghini, & Roth-Andersson, 2011). Further evidence for the malleability of implicit associations stems from CBM research. Clerkin and Teachman (2010) had individuals with HSA complete a conditioning paradigm in which participants repeatedly paired pictures of themselves giving a speech with smiling faces and pictures of others with disgusted or neutral faces. Relative to two control conditions, participants who underwent positive training had lower implicit rejection associations and were more likely to complete a speech task, highlighting the causal relationship between change in implicit associations and anxious behavior. However, there were no effects of CBM condition on subjective anxiety during the speech.

Overall, relative to nonanxious individuals, individuals with HSA and SAD often uncontrollably associate social cues and bodily sensations with negative outcomes and have lower levels of implicit self-esteem. Moreover, these associations may be malleable via treatment, but future research is needed to establish the causal link between changes in implicit associations and reduction in social anxiety symptoms.

Conclusions and Call for Future Research

Taken together, the evidence supports cognitive models of social anxiety, which suggest that individuals with HSA and SAD experience cognitive biases, particularly when processing cues related to social threat. Of note, the level of support varies for different cognitive biases. Strong empirical evidence suggests that individuals with HSA and SAD interpret social cues in a threatening manner (which sometimes manifests as a less positive interpretation bias), judge themselves more negatively than they judge others, and have a selective attention bias toward threat (especially a selective self-focus and a hypervigilance bias at early stages of conscious processing). Moreover, converging lines of research suggest that these biases are malleable, have predictive validity, and are causally related to social anxiety. Further, strong evidence supports the existence of intrusive PEP following social interactions. However, while initial evidence suggests that PEP is malleable and has predictive validity, replication is needed, and further research is required to determine if this specific bias is causally related to social anxiety.

Other aspects of cognitive models of social anxiety are less clearly supported by the available data. Specifically, findings are mixed for conscious attention biases in later stages of processing, as well as for memory biases. Additionally, existing evidence for unconscious processing of threat cues prohibits clear conclusions at this time. Consequently, it is unsurprising that the predictive validity and malleability of these biases are not yet well understood. Finally, converging evidence suggests that individuals with HSA and SAD have biased implicit associations, which appear to have predictive validity and to be malleable, although these findings are in need of replication. These implicit association studies highlight the uncontrollability of processing in social anxiety, in line with theories of automaticity in anxiety (McNally, 1995; Teachman et al., 2012).

Although much is known about information processing biases in individuals with HSA and SAD, there is much left to discover. For instance, a better understanding of how different methodological factors, including types of stimuli, stimulus duration, and timing of state anxiety manipulations, affect bias presentation is likely to clarify some mixed findings. Additionally, more longitudinal and experimental studies are needed to clarify the predictive validity and causal role of cognitive biases in social anxiety, although initial research is promising, particularly for interpretation and conscious attention biases (see MacLeod & Mathews, 2012). Further, little is known about how demographic characteristics, such as gender, race, and age, and other individual difference factors (e.g., personality characteristics) alter the expression and experience of cognitive biases. Another interesting avenue for future research is to determine whether individuals with HSA and SAD exhibit biased perception, such that they may see themselves as blushing more than they actually are, or see a crowd as larger than it actually is (similar to perceptual biases found in height fearful individuals; Teachman, Stefanucci, Clerkin, Cody, & Proffitt, 2008). Finally, research on how various cognitive biases interrelate is called for, because experiments tend to focus on only one bias at a time (see Hirsch, Clark, & Mathews, 2006). Future integrative models that take into account a broader range of cognitive biases, and the ways they work independently and interactively to cause and maintain social anxiety

(see Teachman & Clerkin, 2013), are likely to lead to novel prevention and intervention efforts to more effectively relieve social anxiety.

Acknowledgments

The writing of this chapter was supported in part by an AG033033 grant from the National Institute of Health to Bethany Teachman.

References

Abbott, M. J., & Rapee, R. M. (2004). Post-event rumination and negative self-appraisal in social phobia before and after treatment. *Journal of Abnormal Psychology, 113*(1), 136–144. doi:10.1037/0021-843X.113.1.136

Alden, L. E., Taylor, C. T., Mellings, T. M. J. B., & Laposa, J. M. (2008). Social anxiety and the interpretation of positive social events. *Journal of Anxiety Disorders, 22*, 577–590. doi:10.1016/j.janxdis.2007.05.007

Amir, N., Beard, C., & Bower, E. (2005). Interpretation bias and social anxiety. *Cognitive Therapy and Research, 29*(4), 433–443. doi:10.1007/s10608-005-2834-5

Amir, N., Beard, C., Taylor, C. T., Klumpp, H., Elias, J., Burns, M., & Chen, X. (2009). Attention training in individuals with generalized social phobia: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 77*(5), 961–973. doi:10.1037/a0016685

Amir, N., Coles, M. E., Brigidi, B., & Foa, E. B. (2001). The effect of practice on recall of emotional information in individuals with generalized social phobia. *Journal of Abnormal Psychology, 110*, 713–720. doi:10.1037/0021-843X.110.1.76

Amir, N., Elias, J., Klumpp, H., & Przeworski, A. (2003). Attentional bias to threat in social phobia: Facilitated processing of threat or difficulty disengaging attention from threat?. *Behaviour Research and Therapy, 41*(11), 1325–1335. doi:10.1016/S0005-7967(03)00039-1

Amir, N., Foa, E. B., & Coles, M. E. (1998). Negative interpretation bias in social phobia. *Behaviour Research and Therapy, 36*, 945–957. doi:10.1016/S0005-7967(98)00060-6

Amir, N., Freshman, M., & Foa, E. (2002). Enhanced Stroop interference for threat in social phobia. *Journal of Anxiety Disorders, 16*, 1–9. doi:10.1016/S0887-6185(01)00084-6

Amir, N., McNally, R. J., Riemann, B. C., Burns, J., Lorenz, M., & Mullen, J. T. (1996). Suppression of the emotional Stroop effect by increased anxiety in patients with social phobia. *Behaviour Research and Therapy, 34*(11–12), 945–948. doi:10.1016/S0005-7967(96)00054-X

Amir, N., & Taylor, C. T. (2012). Interpretation training in individuals with generalized social anxiety disorder: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 80*(3), 497–511. doi:10.1037/a0026928

Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: A meta-analytic study. *Psychological Bulletin, 133*(1), 1–24. doi:10.1037/0033-2909.133.1.1

Beard, C., & Amir, N. (2008). A multi-session interpretation modification program: Changes in interpretation and social anxiety symptoms. *Behaviour Research and Therapy, 46*, 1135–1141. doi:10.1016/j.brat.2008.05.012

Beck, A. T., & Clark, D. A. (1997). An information processing model of anxiety: Automatic and strategic processes. *Behaviour Research and Therapy, 35*(1), 49–58. doi:10.1016/S0005-7967(96)00069-1

Beck, A. T., Emery, G., & Greenberg, R. I. (1985). *Anxiety disorders and phobias.* New York, NY: Basic Books.

Brozovich, F., & Heimberg, R. G. (2008). An analysis of post-event processing in social anxiety disorder. *Clinical Psychology Review, 28*(6), 891–903. doi:10.1016/j.cpr. 2008.01.002

Buckner, J. D., Maner, J. K., & Schmidt, N. B. (2010). Difficulty disengaging attention from social threat in social anxiety. *Cognitive Therapy and Research, 34*(1), 99–105. doi:10.1007/s10608-008-9205-y

Byrne, A., & Eysenck, M. W. (1995). Trait anxiety, anxious mood, and threat detection. *Cognition and Emotion, 9*, 549–562. doi:10.1080/02699939508408982

Campbell, D. W., Sareen, J., Stein, M. B., Kravetsky, L. B., Paulus, M. P., Hassard, S. T., & Reiss, J. P. (2009). Happy but not so approachable: The social judgments of individuals with generalized social phobia. *Depression and Anxiety, 26*, 419–424. doi:10.1002/da.20474

Chen, Y. P., Ehlers, A., Clark, D. M., & Mansell, W. (2002). Patients with generalized social phobia direct their attention away from faces. *Behaviour Research and Therapy, 40*(6), 677–687. doi:10.1016/S0005-7967(01)00086-9

Clark, D. M., & Wells, A. (1995). A cognitive model of social phobia. In R. G. Heimberg, M. R. Liebowitz, D. A. Hope, & F. R. Schneier (Eds.), *Social phobia: Diagnosis, assessment, and treatment* (pp. 69–93). New York, NY: Guilford Press.

Clark, J. V., & Arkowitz, H. (1975). Social anxiety and self-evaluation of interpersonal performance. *Psychological Reports, 36*, 211–221.

Clerkin, E. M., & Teachman, B. A. (2010). Training implicit social anxiety associations: An experimental intervention. *Journal of Anxiety Disorders, 24*, 300–308. doi:10.1016/j.janxdis.2010.01.001

Cody, M. W., & Teachman, B. A. (2010). Post-event processing and memory bias for performance feedback in social anxiety. *Journal of Anxiety Disorders, 24*, 468–479. doi:10.1016/j.janxdis.2010.03.003

Cody, M. W., & Teachman, B. A. (2011). Global and local evaluations of public speaking performance in social anxiety. *Behavior Therapy, 42*(4), 601–611. doi:10.1016/j.beth.2011.01.004

Coles, M. E., & Heimberg, R. G. (2002). Memory biases in the anxiety disorders: Current status. *Clinical Psychology Review, 22*, 587–627. doi:10.1016/S0272-7358(01)00113-1

Constans, J. I., Penn, D. L., Ihen, G. H., & Hope, D. A. (1999). Interpretive biases for ambiguous stimuli in social anxiety. *Behaviour Research and Therapy, 37*, 643–651. doi:10.1016/S0005-7967(98)00180-6

Cougle, J. R., Smits, J. A. J., Lee, H. J., Powers, M. B., & Telch, M. J. (2005). Singular and combined effects of thought suppression and anxiety induction on frequency of threatening thoughts: An experimental investigation. *Cognitive Therapy and Research, 29*, 525–539. doi:10.1007/s10608-005-2793-x

Dannahy, L., & Stopa, L. (2007). Post-event processing in social anxiety. *Behaviour Research and Therapy, 45*, 1207–1219. doi:10.1016/j.brat.2006.08.017

de Hullu, E., de Jong, P. J., Sportel, B. E., & Nauta, M. H. (2011). Threat-related automatic associations in socially anxious adolescents. *Behaviour Research and Therapy, 49*, 518–522. doi:10.1016/j.brat.2011.05.008

de Jong, P. J. (2002). Implicit self-esteem and social anxiety: Differential self-favouring effects in high and low anxious individuals. *Behaviour Research and Therapy, 40*, 501–508. doi:10.1016/S0005-7967(01)00022-5

de Jong, P. J., Pasman, W., Kindt, M., & van den Hout, M. A. (2001). A reaction time paradigm to assess (implicit) complaint-specific dysfunctional beliefs. *Behaviour Research and Therapy, 39*, 101–113. doi:10.1016/S0005-7967(99)00180-1

Dimberg, U., & Öhman, A. (1996). Behold the wrath: Psychophysiological responses to facial stimuli. *Motivation and Emotion, 20*(2), 149–182. doi:10.1007/BF02253869

Eastwood, J. D., Smilek, D., Oakman, J. M., Farvolden, P., van Ameringen, M., Mancini, C., & Merikle, P. M. (2005). Individuals with social phobia are biased to become aware of negative faces. *Visual Cognition, 12*(1), 159–179. doi:10.1080/13506280444000175

Edwards, S. L., Rapee, R. M., & Franklin, J. A. (2003). Postevent rumination and recall bias for a social performance event in high and low socially anxious individuals. *Cognitive Therapy and Research, 27*(6), 603–617. doi:10.1023/A:1026395526858

Foa, E. B., Franklin, M. E., Perry, K. J., & Herbert, J. D. (1996). Cognitive biases in generalized social phobia. *Journal of Abnormal Psychology, 105*(3), 433–439. doi:10.1037/0021-843X.105.3.433

Gamer, J., Schmukle, S. C., Luka-Krausgrill, U., & Egloff, B. (2008). Examining the dynamics of the implicit and the explicit self-concept in social anxiety: Changes in the Implicit Association Test-anxiety and the social phobia anxiety inventory following treatment. *Journal of Personality Assessment, 90*(5), 476–480. doi:10.1080/00223890802248786

Gilboa-Schechtman, E., Foa, E., Vaknin, Y., Marom, S., & Hermesh, H. (2008). Interpersonal sensitivity and response bias in social phobia and depression: Labeling emotional expressions. *Cognitive Therapy and Research, 32*, 605–618. doi:10.1007/s10608-008-9208-8

Gilboa-Schechtman, E., Foa, E. B., & Amir, N. (1999). Attentional biases for facial expressions in social phobia: The face-in-the-crowd paradigm. *Cognition and Emotion, 13*(3), 305–318. doi:10.1080/026999399379294

Glashouwer, K. A., de Jong, P. J., Dijk, C., & Buwalda, F. M. (2011). Individuals with fear of blushing explicitly and automatically associating blushing with social costs. *Journal of Psychopathology and Behavioral Assessment, 33*, 540–546. doi:10.1007/s10862-011-9241-x

Glashouwer, K. A., de Jong, P. J., & Penninx, B. W. (2011). Predictive validity of automatic self-associations for the onset of anxiety disorders. *Journal of Abnormal Psychology, 120*(3), 607–616. doi:10.1037/a0023205

Glashouwer, K. A., Vroling, M. S., de Jong, P. J., Lange, W.-G., & de Keijser, A. (2012). Low implicit self-esteem and dysfunctional automatic associations in social anxiety disorder. *Journal of Behavior Therapy and Experimental Psychiatry, 44*(2):262–270. doi:10.1016/j.jbtep.2012.11.005

Greenwald, A. G., McGhee, D. E., & Schwartz, J. L. K. (1998). Measuring individual differences in implicit cognition: The Implicit Association Test. *Journal of Personality and Social Psychology, 74*(6), 1464–1480. doi:10.1037/0022-3514.74.6.1464

Harvey, A. G., Clark, D. M., Ehlers, A., & Rapee, R. M. (2000). Social anxiety and self impression: Cognitive preparation enhances the beneficial effects of video feedback following a stressful social task. *Behaviour Research and Therapy, 38*(12), 1183–1192. doi:10.1016/S0005-7967(99)00148-5

Heimberg, R. G., Brozovich, F. A., & Rapee, R. M. (2010). A cognitive-behavioral model of social anxiety disorder: Update and extension. In S. G. Hofmann & P. M. DiBartolo (Eds.), *Social anxiety: Clinical, developmental, and social perspectives* (pp. 395–422). New York, NY: Elsevier. doi:10.1016/B978-0-12-375096-9.00028-6

Helfinstein, S. M., White, L. K., Bar-Haim, Y., & Fox, N. A. (2008). Affective primes suppress attention bias to threat in socially anxious individuals. *Behaviour Research and Therapy, 46*, 799–810. doi:10.1016/j.brat.2008.03.011

Hirsch, C. R., Clark, D. M., & Mathews, A. (2006). Imagery and interpretations in social phobia: Support for the combined cognitive biases hypothesis. *Behavior Therapy, 37*(3), 223–236. doi:10.1016/j.beth.2006.02.001

Hirsch, C. R., & Mathews, A. (2000). Impaired positive inferential bias in social phobia. *Journal of Abnormal Psychology, 109*(4), 705–712. doi:10.1037//002I-843X.109.4.705

Hirsch, C. R., Meynen, T., & Clark, D. M. (2004). Negative self-imagery in social anxiety contaminates social interactions. *Memory, 12*(4), 496–506. doi:10.1080/09658210444000106

Hope, D. A., Rapee, R. M., Heimberg, R. G., & Dombeck, M. J. (1990). Representations of the self in social phobia: Vulnerability to social threat. *Cognitive Therapy and Research, 14*(2), 177–189. doi:10.1007/BF01176208

Horley, K., Williams, L. M., Gonsalvez, C., & Gordon, E. (2003). Social phobics do not see eye to eye: A visual scanpath study of emotional expression processing. *Journal of Anxiety Disorders, 17*, 33–44. doi:10.1016/S0887-6185(02)00180-9

Joorman, J., & Gotlib, I. H. (2006). Is this happiness I see? Biases in the identification of emotional facial expressions in depression and social phobia. *Journal of Abnormal Psychology, 115*(4), 705–714. doi:10.1037/0021-843X.115.4.705

Kanai, Y., Sasagawa, S., Chen, J., Shimada, H., & Sakano, Y. (2010). Interpretation bias for ambiguous social behavior among individuals with high and low levels of social anxiety. *Cognitive Therapy and Research, 34*, 229–240. doi:10.1007/s10608-009-9273-7

Kanai, Y., Sasagawa, S., Chen, J., Suzuki, S., Shimada, H., & Sakano, Y. (2009). Negative interpretation of bodily sensations in social anxiety. *International Journal of Cognitive Therapy, 2*(3), 292–307. doi:10.1521/ijct.2009.2.3.292

Kashdan, T. B., Weeks, J. W., & Savostyanova, A. A. (2011). Whether, how, and when social anxiety shapes positive experiences and events: A self-regulatory framework and treatment implications. *Clinical Psychology Review, 31*(5), 786–799. doi:10.1016/j.cpr. 2011.03.012

Kim, H.-Y., Lundh, L.-G., & Harvey, A. (2002). The enhancement of video feedback by cognitive preparation in the treatment of social anxiety. A single-session experiment. *Journal of Behavior Therapy and Experimental Psychiatry, 33*, 19–37. doi:10.1016/S0005-7916(02)00010-1

Kingsep, P., & Page, A. (2010). Attempted suppression of social threat thoughts: Differential effects for social phobia and healthy controls? *Behaviour Research and Therapy, 48*(7), 653–660. doi:10.1016/j.brat.2010.03.018

LeMoult, J., & Joormann, J. (2012). Attention and memory biases in social anxiety disorder: The role of comorbid depression. *Cognitive Therapy and Research, 36*, 47–57. doi:10.1007/s10608-010-9322-2

Li, S., Tan, J., Qian, M., & Liu, X. (2008). Continual training of attentional bias in social anxiety. *Behaviour Research and Therapy, 46*(8), 905–912. doi:10.1016/j.brat. 2008.04.005

Liang, C.-W., Hsu, W.-Y., Hung, F.-C., Wang, W.-T., & Lin, C.-H. (2011). Absence of a positive bias in social anxiety: The application of a directed forgetting paradigm. *Journal of Behavior Therapy and Experimental Psychiatry, 42*, 204–210. doi:10.1016/j.jbtep.2010.12.0 02

Lundh, L.-G., & Öst, L.-G. (1996). Stroop interference, self-focus and perfectionism in social phobics. *Personality and Individual Differences, 20*(6), 725–731. doi:10.1016/0191-8869(96)00008-6

Lundh, L.-G., & Öst, L.-G. (2001). Attentional bias, self-consciousness and perfectionism in social phobia before and after cognitive behavioral therapy. *Scandinavian Journal of Behavior Therapy, 30*(1), 4–16. doi:10.1080/028457101300140428

Lundh, L.-G., & Sperling, M. (2002). Social anxiety and the post-event processing of socially distressing events. *Cognitive Behaviour Therapy, 31*(3), 129–134. doi:10.1080/165060702320338004

MacLeod, C., & Mathews, A. (2012). Cognitive bias modification approaches to anxiety. *Annual Review of Clinical Psychology, 8*, 189–217. doi:10.1146/annurev-clinpsy-032511-143052

MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology, 95*, 15–20. doi:10.1037/0021-843X.95.1.15

Makkar, S. R., & Grisham, J. R. (2011). Social anxiety and the effects of negative self-imagery on emotion, cognition, and post-event processing. *Behaviour Research and Therapy, 49*(10), 654–664. doi:10.1016/j.brat.2011.07.004

Mansell, W., Clark, D. M., Ehlers, A., & Chen, Y.-P. (1999). Social anxiety and attention away from emotional faces. *Cognition and Emotion, 13*(6), 673–690. doi:10.1080/026999399379032

Mathews, A., & MacLeod, C. (2005). Cognitive vulnerability to emotional disorders. *Annual Review of Clinical Psychology, 1*(1), 167–195. doi:10.1146/annurev.clinpsy.1.102803.143916

Mattia, J. I., Heimberg, R. G., & Hope, D. A. (1993). The revised Stroop color-naming task in social phobics. *Behaviour Research and Therapy, 31*(3), 305–313. doi:10.1016/0005-7967(93)90029-T

McNally, R. J. (1995). Automaticity and the anxiety disorders. *Behaviour Research and Therapy, 33*(7), 747–754. doi:10.1016/0005-7967(95)00015-P

Mellings, T. M. B., & Alden, L. E. (2000). Cognitive processes in social anxiety: The effects of self-focus, rumination and anticipatory processing. *Behaviour Research and Therapy, 38*, 243–257. doi: 10.1016/S0005-7967(99)00040-6

Mitte, K. (2008). Memory bias for threatening information in anxiety and anxiety disorders: A meta-analytic review. *Psychological Bulletin, 134*(6), 886–911. doi:10.1037/a0013343

Mogg, K., & Bradley, B. P. (1998). A cognitive-motivational analysis of anxiety. *Behaviour Research and Therapy, 36*(9), 809–848. doi:10.1016/S0005-7967(98)00063-1

Mogg, K., & Bradley, B. P. (2002). Selective orienting of attention to masked threat faces in social anxiety. *Behaviour Research and Therapy, 40*, 1403–1414. doi:10.1016/S0005-7967(02)00017-7

Mogg, K., & Bradley, B. P. (2004). A cognitive-motivational perspective on the processing of threat information and anxiety. In J. Yiend (Ed.), *Cognition, emotion and psychopathology* (pp. 68–85). New York, NY: Cambridge University Press. doi:10.1017/CBO9780511521263.005

Mogg, K., Philippot, P., & Bradley, B. P. (2004). Selective attention to angry faces in clinical social phobia. *Journal of Abnormal Psychology, 113*(1), 160–165. doi:10.1037/0021-843X.113.1.160

Mohlman, J., Carmin, C. N., & Price, R. B. (2007). Jumping to interpretations: Social anxiety disorder and the identification of emotional facial expressions. *Behaviour Research and Therapy, 45*, 591–599. doi:10.1016/j.brat.2006.03.007

Morgan, J. (2010). Autobiographical memory biases in social anxiety. *Clinical Psychology Review, 30*, 288–297. doi:10.1016/j.cpr.2009.12.003

Morgan, J., & Banerjee, R. (2008). Post-event processing and autobiographical memory in social anxiety: The influence of negative feedback and rumination. *Journal of Anxiety Disorders, 22*, 1190–1204. doi:10.1016/j.janxdis.2008.01.001

Moser, J. S., Huppert, J. D., Foa, E. B., & Simons, R. F. (2012). Interpretation of ambiguous social scenarios in social phobia and depression: Evidence from event-related brain potentials. *Biological Psychology, 89*, 387–397. doi:10.1016/j.biopsycho.2011.12.001

Mueller, E. M., Hofmann, S. G., Santesso, D. L., Meuret, A. E., Bitran, S., & Pizzagalli, D. A. (2009). Electrophysiological evidence of attentional biases in social anxiety disorder. *Psychological Medicine, 39*(7), 1141–1152. doi:10.1017/S0033291708004820

Murphy, R., Hirsch, C. R., Mathews, A., Smith, K., & Clark, D. M. (2007). Facilitating a benign interpretation bias in a high socially anxious population. *Behaviour Research and Therapy, 45*, 1517–1529. doi:10.1016/j.brat.2007.01.007

Musa, C. Z., & Lépine, J. P. (2000). Cognitive aspects of social phobia: A review of theories and experimental research. *European Psychiatry, 15*(1), 59–66. doi:10.1016/S0924-9338(00)00210-8

Nilsson, J.-E., Lundh, L.-G., Faghihi, S., & Roth-Andersson, G. (2011). The enhancement of beneficial effects following audio feedback by cognitive preparation in the treatment of social anxiety: A single-session experiment. *Journal of Behavior Therapy and Experimental Psychiatry, 42*, 497–503. doi:10.1016/j.jbtep.2011.05.004

Öhman, A., Lundqvist, D., & Esteves, F. (2001). The face in the crowd revisited: A threat advantage with schematic stimuli. *Journal of Personality and Social Psychology, 80*(3), 381–396. doi:10.1037/0022-3514.80.3.381

Öhman, A., & Mineka, S. (2001). Fears, phobias, and preparedness: Toward an evolved module of fear and fear learning. *Psychological Review, 108*(3), 483–522. doi:10.1037/0033-295X.108.3.483

Ononaiye, M. S. P., Turpin, G., & Reidy, J. G. (2007). Attentional bias in social anxiety: Manipulation of stimulus duration and social-evaluative anxiety. *Cognitive Therapy and Research, 31*(6), 727–740. doi:10.1007/s10608-006-9096-8

Parra, C., Esteves, F., Flykt, A., & Öhman, A. (1997). Pavlovian conditioning to social stimuli: Backward masking and the dissociation of implicit and explicit cognitive processes. *European Psychologist, 2*(2), 106–117. doi:10.1027/1016-9040.2.2.106

Perowne, S., & Mansell, W. (2002). Social anxiety, self-focused attention, and the discrimination of negative, neutral and positive audience members by their non-verbal behaviours. *Behavioural and Cognitive Psychotherapy, 30*, 11–23. doi:10.1017/S1352465802001030

Pishyar, R., Harris, L. M., & Menzies, R. G. (2004). Attentional bias for words and faces in social anxiety. *Anxiety, Stress & Coping: An International Journal, 17*(1), 23–36. doi:10.1080/10615800310001601458

Putman, P., Hermans, E., & van Honk, J. (2004). Emotional Stroop performance for masked angry faces: It's BAS, not BIS. *Emotion, 4*(3), 305–311. doi:10.1037/1528-3542.4.3.305

Rachman, S., Grüter-Andrew, J., & Shafran, R. (2000). Post-event processing in social anxiety. *Behaviour Research and Therapy, 38*, 611–617. doi:10.1016/S0005-7967(99)00089-3

Rapee, R. M., & Heimberg, R. G. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy, 35*(8), 741–756. doi:10.1016/S0005-7967(97)00022-3

Rapee, R. M., & Lim, L. (1992). Discrepancy between self- and observer ratings of performance in social phobics. *Journal of Abnormal Psychology, 101*(4), 728–731. doi:10.1037/0021-843X.101.4.728

Rinck, M., & Becker, E. S. (2007). Approach and avoidance in fear of spiders. *Journal of Behavior Therapy and Experimental Psychiatry, 38*(2), 105–120. doi:10.1016/j.jbtep.2006.10.001

Roth, D., Antony, M. M., & Swinson, R. P. (2001). Interpretations for anxiety symptoms in social phobia. *Behaviour Research and Therapy, 39*, 129–138. doi:10.1016/S0005-7967(99)00159-X

Sasaki, S., Iwanaga, M., Kanai, Y., & Seiwa, H. (2010). Implicit and explicit associations in the fear structure of social anxiety. *Perceptual and Motor Skills, 110*(1), 19–32. doi:10.2466/PMS.110.1.19-32

Schmidt, N. B., Richey, J. A., Buckner, J. D., & Timpano, K. R. (2009). Attention training for generalized social anxiety disorder. *Journal of Abnormal Psychology, 118*(1), 5–14. doi:10.1037/a0013643

Schmukle, S. C., & Egloff, B. (2004). Does the Implicit Association Test for assessing anxiety measure trait and state variance? *European Journal of Personality, 18*, 483–494. doi:10.1002/per.525

Schofield, C. A., Johnson, A. L., Inhoff, A. W., & Coles, M. E. (2012). Social anxiety and difficulty disengaging threat: Evidence from eye-tracking. *Cognition and Emotion, 26*(2), 300–311. doi:10.1080/02699931.2011.602050

Stevens, S., Gerlach, A. L., & Rist, F. (2008). Effects of alcohol on ratings of emotional facial expressions in social phobics. *Journal of Anxiety Disorders, 22*, 940–948. doi:10.1016/j.janxdis.2007.09.007

Stevens, S., Rist, F., & Gerlach, A. L. (2009). Influence of alcohol on the processing of emotional facial expressions in individuals with social phobia. *British Journal of Clinical Psychology, 48*(2), 125–140. doi:10.1348/014466508×368856

Stopa, L., & Clark, D. M. (2000). Social phobia and interpretation of social events. *Behaviour Research and Therapy, 38*, 273–283. doi:10.1016/S0005-7967(99)00043-1

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology, 18*, 643–662. doi:10.1037/h0054651

Tanner, R. J., Stopa, L., & De Houwer, J. (2006). Implicit views of the self in social anxiety. *Behaviour Research and Therapy, 44*, 1397–1409. doi:10.1016/j.brat.2005.10.007

Teachman, B. A., & Allen, J. P. (2007). Development of social anxiety: Social interaction predictors of implicit and explicit fear of negative evaluation. *Journal of Abnormal Child Psychology, 35*, 63–78. doi:10.1007/s10802-006-9084-1

Teachman, B. A., & Clerkin, E. M. (2013). *The interactive model of anxious processing: A transdiagnostic framework integrating automatic and strategic cognitive processing biases.* Manuscript submitted for publication.

Teachman, B. A., Joormann, J., Steinman, S. A., & Gotlib, I. H. (2012). Automaticity in anxiety disorders and major depressive disorder. *Clinical Psychology Review, 32*, 575–603. doi:10.1016/j.cpr.2012.06.004

Teachman, B. A., Stefanucci, J. K., Clerkin, E. M., Cody, M. W., & Proffitt, D. R. (2008). A new mode of fear expression: Perceptual bias in height fear. *Emotion, 8*(2), 296–301. doi:10.1037/1528-3542.8.2.296

Tran, T., Hertel, P. T., & Joormann, J. (2011). Cognitive bias modification: Induced interpretive biases affect memory. *Emotion, 11*, 145–152. doi:10.1037/a0021754

van Peer, J. M., Spinhoven, P., & Roelofs, K. (2010). Psychophysiological evidence for cortisol-induced reduction in early bias for implicit social threat in social phobia. *Psychoneuroendocrinology, 35*(1), 21–32. doi:10.1016/j.psyneuen.2009.09.012

Vassilopoulos, S. P. (2006). Interpretation of judgmental biases in socially anxious and nonanxious individuals. *Behavioural and Cognitive Psychotherapy, 34*, 243–254. doi:10.1017/S1352465805002687

Vassilopoulos, S. P. (2008). Coping strategies and anticipatory processing in high and low socially anxious individuals. *Journal of Anxiety Disorders, 22*, 98–107. doi:10.1016/S0005-7967(99)00089-3

Vassilopoulos, S. P. (2011). Interpretation bias for facial expressions in high and low socially anxious individuals: Effects of stimulus duration. *Hellenic Journal of Psychology, 8*, 44–65.

Voncken, M. J., & Bögels, S. M. (2006). Changing interpretation and judgmental bias in social phobia: A pilot study of a short, highly structured cognitive treatment. *Journal of Cognitive Psychotherapy, 20*(1), 59–73. doi:10.1891/jcop.20.1.59

Weeks, J. W., Heimberg, R. G., Rodebaugh, T. L., Goldin, P. R., & Gross, J. (2012). Psychometric evaluation of the Fear of Positive Evaluation Scale (FPES) in patients with social anxiety disorder. *Psychological Assessment, 24*, 301–312.

Westberg, P., Lundh, L.-G., & Jönsson, P. (2007). Implicit associations and social anxiety. *Cognitive Behaviour Therapy, 36*(1), 43–51. doi:10.1080/08037060601020401

Wigboldus, D. H. J., Holland, R. W., & van Knippenberg, A. (2005). *Single target implicit associations.* Unpublished manuscript.

Williams, J. M. G., Watts, F. N., MacLeod, C., & Mathews, A. (1997). *Cognitive psychology and emotional disorders* (2nd ed.). Chichester, UK: John Wiley & Sons.

Wilson, J. K., & Rapee, R. M. (2005). The interpretation of negative social events in social phobia: Changes during treatment and relationship to outcome. *Behaviour Research and Therapy, 43*, 373–389. doi:10.1016/j.brat.2004.02.006

Yoon, K. L., & Zinbarg, R. E. (2007). Threat is in the eye of the beholder: Social anxiety and the interpretation of ambiguous facial expressions. *Behaviour Research and Therapy, 45*, 839–847. doi:10.1016/j.brat.2006.05.004

16

Behavioral Deviations

Surface Features of Social Anxiety and What They Reveal

Wolf-Gero Lange, Mike Rinck, and Eni S. Becker

Radboud University Nijmegen, The Netherlands

The Evolution of Social Anxiety and Related Processing and Behavioral Biases

Social anxiety disorder (SAD), with its comparably high prevalence rates between 7% and 13% (e.g., Fehm, Pelissolo, Furmark, & Wittchen, 2005) in Western societies, has been predominantly conceptualized as a disorder of "distorted information processing" or, in other words, negatively biased cognitive processes (see **Chapter 1**). Herein, the models of Clark and Wells (1995), Rapee and Heimberg (1997), and that of Hofmann (2007) have been in focus for many years now. The basic (and scientifically founded) idea underlying all three models is that individuals suffering from SAD/highly socially anxious individuals (SAIs) have a tendency to quickly detect and focus their attention on putative signals of social devaluation (e.g., facial expressions; Staugaard, 2010), to evaluate or interpret signals in an anxiety-confirming way (Huppert, Pasupuleti, Foa, & Mathews, 2007) and to overestimate the potential costs of a (negative) social interaction (Schofield, Coles, & Gibb, 2007). In these models, observable behavior only plays a secondary role, as in overt avoidance behavior or in so-called safety behaviors (i.e., behaviors that SAIs display because it gives them the illusion of control over the situation and thereby reduces anxiety). In sum, according to the current view, SAD can be mainly understood as a tendency to see social danger (social devaluation and rejection) where in fact there is none. Yet, there is cumulative evidence that SAIs *are* truly evaluated in a negative way (Heerey & Kring, 2007; Voncken, Alden, Bogels, & Roelofs, 2008). Here, apart from occasionally exaggerated avoidance or safety behaviors, deviations in subtle behaviors that are normal in social interaction may contribute to true negative evaluation. In order to understand the suggested interplay of emotion, cognition, and behavior, and their sequence with respect to time, we need to have a short look at the evolution of anxiety in general, and social anxiety in particular.

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

Anxiety, in general, can be seen as a threefold set of responses to a threatening situation: Information processing (cognition), physiological activation, and behavior initiation/inhibition (Lang, 1985). Relevant information of context and environment must be analyzed and filtered, the body must be prepared to take action, and appropriate behavior must be launched—all within a fraction of time. It has been argued that automatic, quick evaluation of a situation and activation of an organism to freeze, fight, or flee increases its survival and consequently the likelihood of reproduction (Darwin, 1859; Öhman, 1993). Geary (2007) suggested that, as a result, preferential processing for survival-related cues, such as spiders, heights, or (threatening) facial expressions (Öhman, Dimberg, & Öst, 1985; Seligman, 1971) evolved. Facial expressions, for example, are highly communicative (e.g., Haxby, Hoffman, & Gobbini, 2000). Öhman et al. (1985) suggested that an enhanced detection of angry faces used to be (and still is) relevant for an individual in a group to show submissive behavior if demanded. By doing so, an individual sustains group structure and one's own membership. In the same line, ignorance of an emotional appeal may have led to disrupted social group coherence and rejection from the group (and still does). Ignorance of cues for alliances/mating or signals of threat may have minimized chances to survive (Gilbert, 2001) and to reproduce (Darwin, 1859).

Fridlund (1994) investigated the evolutionary relevance of social signals as a whole. He assumed that displaying, for instance, an angry face may have evolved as a means to spare an individual from "costly" fighting. At the same time, an attentional bias for the quick detection of angry faces in an observer of such a display, paired with a quick retreat, may have kept one from dangerous, tedious defense. The display of threat and its quick detection increase the chances of survival for both potential rivals in the context of agonistic encounters.

There is an evolutionary drawback, however: An excessive use of "threat displays" undermines its trustworthiness and may actually provoke attacks. Hypervigilance for such displays and excessive retreat, on the other hand, may have kept someone from survival-related resources (Fridlund, 1994). Even though this mechanism is much more complex than presented here (c.f., **Chapter 2**), it serves as a comprehensible analogy: If one responds hypersensitively to socio-evaluative stress, one may detect social threat where, in fact, there is none and become socially anxious. Consequently, the person retreats from any form of social contact and/or stops displaying prosocial behaviors. The result is severely detrimental: In addition to evaluating nonthreatening social situations as threatening, SAIs also display behaviors that actually make a negative evaluation more likely. The interpretation of social signals may thus be rather accurate and not negatively biased: People do evaluate SAIs more negatively.

In sum, the evolution of a "need to belong" (Baumeister & Leary, 1995), a threat-detection or preparation system specifically tuned to social cues and interaction, triggering behaviors apt to restore affiliation with others, appears very likely. This may, even today, explain general stress proneness in human beings when interacting in groups (Juth, Lundqvist, Karlsson, & Öhman, 2005). Thus, evolved responsiveness to socio-evaluative cues that communicate the states of others, and a certain degree of social anxiety that steers one's actions in a social context, is adaptive and favorable. Evolutionarily relevant structures that, when dysfunctional, give room for

hypersensitivity to social threat and disturbed (social) behaviors may lie at the very core of SAD (see also **Chapter 2** for an expanded discussion of this issue).

Models of Biased Processing and Behavior

In light of evolved "threat-detection systems," neurobiological research has repeatedly indicated that the amygdala, a central subcortical brain structure, plays a key role in emotion-related processes, including anxiety. It appears as if two neural pathways are involved in the processing of fear-relevant cues: A quick, direct route via thalamus and amygdala; and a slower, indirect route via thalamus, cortical regions, and amygdala (Vuilleumier, 2005). Along the former route, the perceptual information processing is rather rudimentary: Based on several key features, analysis of the emotional relevance of a stimulus takes place quickly and, if necessary, preparatory reflexive behavior patterns are initiated. Here, especially, attentional biases could take their course. Via the latter route, a more thorough analysis takes place in the cortex. Here, not only threat cues but also environment cues, earlier experiences, and (biased) knowledge gained over previous experiences are taken into consideration. Further action is guided by all available information, and continues or inhibits the behavior triggered via the quick route (LeDoux, 1996). Both routes contribute to alerting and protecting an individual from harm when functioning properly but can keep someone constantly vigilant and "on the flight" when the system is over-reactive.

In 1995, Peter J. Lang suggested a motivation-emotional approach for the understanding of emotion processing, physiological reactivity, and consecutive behavior. He hypothesized that emotions are "action disposition states of vigilant readiness that vary widely in reported affect, physiology, and behavior. They are driven, . . . by only 2 opponent motivational systems, appetitive and aversive-subcortical circuits that mediate reactions to primary reinforcers" (Lang, 1995, p. 372). Lang assumed that although all emotions differ quite dramatically, they can all be positioned in a two-dimensional space within the dimensions of arousal (low/high) and valence (negative/positive). Consecutive responsivity of an organism to a stimulus is highly related to its categorization in terms of valence and arousal. Lang based his work on the presumption that two motivational systems drive human behavior: an appetitive system and an aversive system (Dickinson & Dearing, 1979).

Based on Lang's (1995) idea, Mogg and Bradley (1998) introduced a cognitive-motivational model specifically for anxiety. In essence, they suggested that all stimuli entering any of the sensory modalities are evaluated by a valence evaluation system integrating both quick superficial feature analysis and slower detailed exploration of context, knowledge from experiences, and so on (e.g., LeDoux, 1996). Any given input receives a valence "tag," ranging from "no threat" to "high threat." According to this tag, a "goal engagement system" motivates the subsequent course of action: Interrupt current goal, possibly "freeze" in order to reorient and look for escape from danger (Lang, Davis, & Öhman, 2000), or pursue current goals because the stimuli do not warrant immediate action. The goal engagement system is thought to be responsible for the steering of subsequent cognitive processing such as attentional avoidance (with mild negative/threat valence) and attentional vigilance and dwelling

(with high threat valence) and may ignite overt behavioral responses later on. Mogg and Bradley further assume that the sensitivity of the valence evaluation system can be altered by biological predisposition, the situational context, experience, and character traits. As Mogg and Bradley's motivational model focuses on anxiety alone, these authors do not elaborate in detail upon how positive stimuli are processed by the valence evaluation system. More recently, Mogg and Bradley (2007) have endeavored to extend their model. They proposed that positive evaluations must feed into the goal engagement system too and that the valence evaluation system must be imagined as an entity predicting degrees of reward and punishment on a continuous scale (compare Lang, 1995). Consequently, an organism that is motivated to pursue goals that either keep it away from punishment/negative experiences or strive for reward/positive experiences will engage in initial orienting, but predominantly avoidance or approach behaviors.

It remains open to debate as to which, and how strongly, factors fine-tune the valence evaluation system. As Mogg and Bradley (1998) asserted in their original paper, stimulus properties as well as the situational context and state anxiety might play an important role. Before those factors come into play, though, biological preparedness (e.g., Seligman, 1971), but even more importantly, genetic predisposition (Stein, Jang, & Livesley, 2002) and prior learning experiences (Neal & Edelmann, 2003), may have shaped someone's personality and sensitized the valence evaluation system in a way that makes an individual more or less anxious in a specific context with specific stimuli. In line with this notion, there is some evidence of a correlation between temperament and attentional processing (Derryberry & Reed, 1994).

More generally, not only have genetic vulnerability, temperament as a child, parent–child interaction, and aversive/negative experiences been identified to contribute to the etiology of SAD, but also deficits in social skills, avoidance behavior, and especially cognitive styles (e.g., negative interpretation of ambiguous social events; for overview see, e.g., Mathew & Ho, 2006).

Two influential models have attempted to explain these cognitive phenomena. In 1995, Clark and Wells presumed that individuals suffering from SAD base their negative evaluations of social situations on a number of threatening assumptions (e.g., "If I am not an interesting "conversationist," they will not like me"). Accordingly, social cues are interpreted in a tendentiously negative way which serves to confirm their fears, instantiating negative images of themselves in the present and for future situations and triggering anxiety symptoms, safety behaviors, and anxious cognitions.

In addition to Clark and Wells' (1995) model, Rapee and Heimberg (1997) suggested a vigilance to and focus on external cues representing supposedly negative social evaluation of the anxious individual. In an update of their model, Heimberg, Brozovich, and Rapee (2010) suggest that even benign evaluation by others may make an SAI fear to disappoint others in the end. After detection of possible cues for (negative) evaluation in the environment, continuous comparison of the image that the audience must have of them/will eventually have and the image that others "should" have takes place. The observed social signals are interpreted as signs of imminent or future threat and serve as indicators that the audience's expectations are not or will not be fulfilled—consequently, anxiety increases.

In 2007, Hofmann adopted the basic ideas of these models and refined them. The most significant additions he made concerned the "anticipation of social mishaps" and "post-event rumination": The first stresses the fear of SAIs that they are not only about to behave socially inadequately but also that such a social mishap has disastrous consequences. The second refers to the fact that SAIs tend to keep ruminating about past social experiences and remember instances as being more negative than they really were.

Since the introduction of these models, a substantial body of research has evidenced the validity of (parts of) these models. However, although cognitive-behavioral therapy protocols that are based on these models are the "gold standard" in the treatment of SAD, treatment response and sustained treatment effects remain moderate (Heimberg, 2002). In light of the more general anxiety models (i.e., Lang, 1995; Mogg & Bradley, 1998), it appears as if the cognitive models may have underestimated the impact of anxiety on behavior. As depressed individuals are thought to have a rather realistic view of the world while it is the nondepressed that show a (positive) bias, it is possible that SAIs are not utterly wrong when perceiving negative evaluation and rejection in social situations.

The remainder of this chapter will summarize evidence to substantiate this claim and will provide a refined model of SAD, which will incorporate social behavior contributing to *true* negative evaluation in addition to the experience of putative negative judgments.

Behaviors That Provoke Social Devaluation

As mentioned above, SAIs have a tendency to overexaggerate possible cues of negative evaluation as is suggested by the cognitive models, but there is also cumulative evidence that they are objectively evaluated in a negative way (e.g., Heerey & Kring, 2007; Voncken et al., 2008). It is assumed that certain behaviors of SAIs contribute to these negative evaluations. Herein, avoidance behaviors, lack of prosocial behaviors or social skills, and anxious behaviors have been suggested to play a prominent role.

Avoidance During Social Interactions

Based on the previously mentioned cognitive models it has been argued that high degrees of social anxiety should be related to attentional vigilance (and subsequent avoidance) of social cues (e.g., facial expressions) portraying presumed social devaluation. A considerable body of research has substantiated that claim to a certain degree (for review, see Staugaard, 2010), though it is not completely clear which specific negative facial expressions should cause these attentional biases. Another approach, however, has been more straightforward. If particular faces were evaluated as threatening, they should initiate impulsive avoidance responses. With the introduction of the Approach-Avoidance Task (AAT; Rinck & Becker, 2007), it became possible to implicitly investigate attitudes or evaluations by means of associated behavior.

The underlying idea of this task is that human beings have a tendency to automatically approach pleasant stimuli, while avoiding unpleasant or threatening ones (e.g., Chen & Bargh, 1999). In the original version of the AAT, participants can "avoid" a stimulus on a computer screen by quickly pushing it away and "approach" it by pulling it closer by means of a joystick. An increase of stimulus size on the computer screen whenever the joystick is pulled, and a decrease in size when the joystick is pushed, intensifies the impression of actually moving the stimulus. Heuer, Rinck, and Becker (2007) used this task with SAIs and nonanxious control participants (NACs) and showed them neutral, angry, or smiling faces and puzzles. Participants were instructed to either push the faces and pull the puzzles or vice versa. It appeared that SAIs, compared to NACs, impulsively pushed (avoided) angry and smiling faces quicker or pulled (approached) them slower, when compared to neutral faces and puzzles.

Since this first publication, results have been remarkably consistent across stimuli, methods, and labs. Lange, Keijsers, Becker, and Rinck (2008), for example, used different ratios of neutral–angry and smiling–angry facial expressions combined in so-called crowds. Here, in SAIs, the degree of avoiding neutral–angry crowds increased with the ratio of angry faces in the crowd, while NACs did not show any preference for pulling or pushing. The smiling–angry crowds were consistently avoided by SAIs, independent of ratio, indicating that both expressions were perceived to be threatening.

In another variation of the task, Roelofs et al. (2010) used single faces and manipulated the gaze direction of the depicted individuals. The angry, neutral, or smiling faces either looked directly at the participant or gazed slightly to the left or to the right of the participant. With the direct gaze stimuli, the effects for angry and smiling faces were replicated. When the gazes were averted, however, only the smiling stimuli provoked avoidance impulses in SAIs. As smiling faces are much more common in social interaction, this is a remarkable result. It seems as if smiling, friendly people in a social context do not seem to radiate sympathy and encouragement in the eyes of SAIs. It is yet unclear whether the smile is seen as a sign of being ridiculed or whether SAIs also fear positive evaluation as suggested earlier (Weeks, Heimberg, & Rodebaugh, 2008; see also **Chapter 20** for a comprehensive review).

Interpersonal Distance and Personal Space

The idea that SAIs may show impulsive (subtle) avoidance in response to social cues is intriguing. If that happened in a *real* social interaction, it is plausible to assume that the interaction partner would somehow pick up on these subtle signals and interpret them as signs of not being liked. The partner, on his/her part, may then show subtle behaviors that signal devaluation which, in turn, will be sensed by the SAI. The SAI interprets these signals in a *correct* way and becomes more anxious, which results in more avoidance and so on.

The first hunch of how to translate the AAT results to real interaction would be to measure interpersonal distance in a social situation. The investigation of avoidance behavior has been taken to virtual reality labs recently. In a study by Rinck et al. (2010), participants with varying degrees of social anxiety were equipped with a head-mounted

display, and instructed to approach an avatar situated in a virtual supermarket, and to memorize a word attached to the chest and a number attached to the back of the avatar. It appeared that participants with higher degrees of social anxiety approached the avatar more slowly and kept a larger distance from it.

With a different approach, Wieser, Pauli, Grosseibl, Molzow, and Mühlberger (2010) investigated the behavioral and physiological responses of female SAIs when they *were* approached by others. Here, male and female avatars approached the participants and stopped at a distance of 50 cm or 1.5 m. Their gaze was either directed at the participant or averted. Wieser et al. (2010) observed that the SAIs, when compared to NACs, showed increased avoidance of eye contact of specifically male avatars already at 1.5 m and showed increased backward movements of the head at both distances.

Finally, Lange, Roelofs, and Becker (in prep.) positioned 87 individuals (68% female) with various degrees of social anxiety on a stabilometric force platform to measure body sway. In 50% of the cases, a male experimenter approached the participants in even-sized steps from 3 m to 20 cm, while a female approached the other half of the participants. In addition, participants were asked to indicate the distance that they would find comfortable in a conversation with the experimenter. The preliminary results revealed that degree of social anxiety was related to an increase of (uneasy?) shifting and repositioning beginning at 1.6 m from the participant and decreasing back to normal at about 80 cm. Interestingly, these results were unrelated to the expected "comfortable" distance that participants had reported.

Conclusively, it appears that SAIs have a tendency to respond with impulsive avoidance when being confronted with social cues. Consequently, it is reasonable to assume that even in "real" social situations, SAIs keep more interpersonal space; show other avoidance behavior; or respond with uneasy, twitchy behavior once their own personal space is violated. It is very likely that an interaction partner senses these behaviors, comes to his/her own conclusions, and responds accordingly.

Behavioral and Facial Mimicry

Presuming that SAIs unintendedly keep greater interpersonal distance in an interaction, or respond subtly avoidantly or nervously once their own personal space is entered, it is very likely that other automatized prosocial behaviors that are considered "normal" in an interaction are hampered as well. Behavioral mimicry is one of these behaviors. In social interactions, people often unconsciously and unintentionally mimic each other's behavior, that is, they change their behavior (e.g., voice, facial expressions, posture) to match that of the person they are interacting with (Dijksterhuis & Bargh, 2001). Chartrand and Bargh (1999) found that this similarity facilitates the smoothness of the interaction and makes the mimicker more likable. Bailenson and Yee (2005) found that, overall, a mimicking avatar was evaluated more positively than a nonmimicking avatar. In addition, affiliation, rapport, and feelings of comfort with a mimicking person are usually increased (Lakin, Jefferis, Cheng, & Chartrand, 2003).

Vrijsen, Lange, Becker, and Rinck (2010) assumed that, as SAIs do not feel comfortable in social interactions and keep a larger interpersonal distance, they would mimic less in a social situation than NACs would. In an immersive virtual environment, participants had to listen to the speech of an avatar for a bogus memory task. The avatar would show specific head movements at predefined instances while the participants' head movements were recorded. SAIs showed less mimicry than did NACs. In a second experiment, with comparable setup, the avatar either mimicked the participants' head movements or displayed arbitrary head movements whenever the participants moved (Vrijsen, Lange, Dotsch, Wigboldus, & Rinck, 2010). As would be expected, NACs evaluated the mimicking avatar to be more friendly than the nonmimicking avatar; SAIs however, evaluated a mimicking avatar not friendlier than a nonmimicking avatar.

Mimicking of facial expressions is less indicated to directly improve social interactions. It has been suggested to improve comprehending affective states of others. While this phenomenon has been largely explored in healthy subjects, only a few studies have assessed facial mimicry in the context of social anxiety. Facial responses to presentations of facial expressions are generally assessed by electromyography (EMG). Here, especially the muscle reactivity of the corrugator supercilii which furrows the eye brows in a frown and the zygomaticus major which lifts the cheeks in a smile is targeted (Hjortsjö, 1970). In addition to their possible role in facial mimicry, however, EMG of these muscles is also seen as a quick indicator of an automatic subjective evaluation of the presented stimuli (Dimberg & Thunberg, 2007). Consequently, it is difficult to disentangle research results as being either mimicry of the seen face or a simple reaction to evaluation. Vrana and Gross (2004), for example, found that high degrees of speech anxiety were associated with more corrugator and less zygomaticus muscle responses. Specifically, highly speech-anxious individuals, when compared to NACs, showed fewer smiles in response to any expressions, with the discrepancy being the greatest in reaction to smiles. As for the corrugator responses, SAIs only showed a nonsignificant trend to respond more negatively to angry faces, when compared to controls. These results were in line with Dimberg (1997), but in 1991, the same lab had found that SAIs, in contrast to NACs, showed an overall diminished responsiveness to happy and angry faces (Dimberg & Christmanson, 1991), while Dimberg and Thunberg (2007) could only partially replicate their own earlier results. They reported that speech-fearful individuals not only showed a stronger corrugator reaction difference when angry and happy faces were compared, but also exhibited a greater difference in zygomaticus reactions when comparing responses to happy and angry faces.

In sum, it appears as if SAIs respond subtly atypically when in a social interaction: They mimic others less, they appreciate being mimicked less, and, with respect to facial expressions, they presumably do not respond entirely appropriately. In addition, they seem to behave subtly differently than would be expected when, for example, being smiled at. Again, as these processes are supposedly unintended and uncontrolled, it is very likely that nonanxious interaction partners sense these subtle inadequacies and either come to the conclusion that the behavior of SAIs is "strange," or they themselves become nonsympathetic.

Social Skills and Social Behavior

Social skills are considered crucial for the appropriate adjustment and functioning of individuals in social situations. They are characterized by an interplay of cognitive as well as behavioral abilities. Deficits herein can lead to severe disruptions in psychosocial functioning and have been repeatedly linked to SAD. Social skills deficits can be broadly grouped into three dimensions: (1) Deficits of skill acquisition, which means that specific social skills were never acquired in the first place; (2) deficits in social performance, which relates to the acquired skill not being performed as frequently as would be necessary; (3) deficits of fluency, which describes the presence of specific skills but errors or lack of mastery in their execution (Angelico, Crippa, & Loureiro, 2010). The two subscales of the "Social Behavior and Anxious Appearance Rating Scale" (Voncken & Bögels, 2008) help to narrow down the long list of candidate behaviors that may be termed "social skills": (a) anxious appearance: fidgeting, blushing, laughing nervously, trembling, stuttering, and so on; and (b) social behavior: eye contact, smiling, completing sentences, coherence, adequate coping with silence and use of pauses, listening, showing interest, adequate responding, and self-disclosure (Bögels, Rijsemus, & De Jong, 2002). Nevertheless, it remains rather difficult to pinpoint an exact description of skills deficit, as it seems to differ considerably from individual to individual. Some studies, for example, describe the use of safety behaviors (e.g., as repeatedly asking questions) as social incompetence. When SAIs ask a lot of questions in a conversation, they aim to prevent the other from asking for "personal" information (which could lead to negative evaluation). This gives them a sense of safety and control. Unknown to them, however, they undermine the "normal" reciprocity of sharing personal information in an interaction which truly leads to negative evaluation. Other descriptions of the social behaviors of SAIs, which make it difficult to clearly define "social skills," are even more vague, such as SAIs' supposedly exaggerated or aggressive responses after being criticized or treated unfairly, being cold, and SAIs are experienced as dissimilar to oneself, or being disinterested in others, and so on.

Nevertheless, it is undeniable that SAIs show a number of behaviorisms that could be grouped under "disrupted social skills" which make them less liked by others (Angelico et al., 2010; Levitan & Nardi, 2009). To portray this interaction of evaluation expectation, behavior, and true evaluation, Curtis and Miller (1986) led healthy participants to believe that they were either liked or disliked by an interaction partner whom they would meet again after a short encounter. They found that the initial belief of the evaluation of the other led to behaviors that actually provoked the expected evaluation. Behaviors associated with positive changes in evaluation were more self-disclosure, more agreeing, expressing more similarity, having a more positive tone of voice, and a more positive attitude in general. Negative changes, on the other hand, were related to opposite behaviors.

In a number of studies, the lab group of Voncken confirmed these findings. In a study by Voncken and Bögels (2008), SAD patients and NACs had to predict their social performance in a speech and in an interaction with confederates. In addition, the confederates and video observers evaluated the videotaped performance of each participant. Apart from the previously reported underestimation of their skills, it also

became obvious that patients with SAD had actual social performance deficits in the conversation but not in the speech condition. During social interactions, they displayed distortions in social/communicative behaviors, such as finishing sentences, keeping eye contact, maintaining the conversation, replying coherently, expressing interest in others, or reacting reciprocally. In addition, they also showed anxious behaviors, such as trembling, sweating, blushing, fidgeting, laughing nervously, speaking fast, or stuttering (for comparable results, see, e.g., Baker & Edelmann, 2002).

In an extensive study by Beidel, Rao, Scharfstein, Wong, and Alfano (2010), social skills and subjective anxiety of a large sample of individuals diagnosed according to the DSM-IV criteria (American Psychiatric Association, 2000) as qualifying for either generalized SAD or nongeneralized SAD, or as nonanxious (i.e., controls), were investigated. In a battery of behavioral tests, they assessed the social skills of participants in eight structured roleplays with confederates. "The content of the structured interactions (expression of disapproval or criticism, social assertiveness, confrontation and anger expression, heterosexual contact, interpersonal warmth, conflict or rejection, interpersonal loss, receiving compliments)... tend to elicit social distress" (Beidel et al., 2010, p. 955). In a more unstructured interaction, participants were only provided with a more general scenario (e.g., first contact with new neighbor) and had to interact with confederates for about 3 min. Finally, the participants had to initiate an impromptu speech after a preparation of 3 min. Again, their skills were evaluated, subjective anxiety was rated, and signs of anxiety were evaluated. In all conversation tasks, structured or unstructured, it was confirmed that both SAD groups significantly lacked social skills, had higher degrees of subjective anxiety, and appeared more anxious when compared to nonanxious controls. There was also a difference between the two SAD groups: The generalized subtype reported significantly more anxiety and showed more social skill deficits than the nongeneralized type. In addition, it was found that the generalized subtype showed significant deficits in their transitions from one subject to another and in their memory of formerly discussed subjects. In contrast to Voncken et al. (2008), Beidel et al. (2010) also reported skill deficits during the speech task. Here, in general, the two SAD subgroups did not differ in terms of (self-reported and perceived) anxiety. Both groups ended their speech earlier than the NACs, but it was only the generalized subtype that was evaluated as less skilled, while the nongeneralized subtype was not evaluated differently from the NACs. Baker and Edelmann (2002) looked more specifically at communicative behaviors. They found that individuals diagnosed with SAD showed markedly inadequate gestures, fluency of speech, and overall behavior during a conversation. Their eye contact and clarity of speech were considered inadequate as well when compared to NACs, but not when compared to a control group with diagnosed anxiety disorders other than SAD.

In a study by Weeks, Heimberg, and Heuer (2011), submissive behaviors such as eye gaze and body posture, as well as anxiety-related behaviors such as pitch of voice of male SAIs and NACs were assessed during a conversation with one female confederate and another male confederate "intruding" later during the interaction and competing for the attention of the female confederate. They investigated body collapse (slumped, closed posture), vocal pitch peaks, gaze avoidance, and words spoken before and after the "intruder" entered. It was found that social anxiety was primarily related to increased submissiveness indexed by body collapse and vocal pitch after the intruder

entered but not before. Gaze behavior (as assessed by objective observers) and number of words spoken were not associated with social anxiety. Wieser et al. (2010), on the other hand, did find gaze avoidance in SAIs.

In a recent review, Angelico et al. (2010) confirmed that the available literature predominantly confirms social skill deficits in SAD. Yet, these findings are debated. In the first place, the definition of "skills" comprises a quite broad variety of observable and unobservable behaviors. Second, there are no validated measures that would allow correct comparisons between studies (Angelico et al., 2010). Third, and most importantly, it remains unclear whether SAIs are incapable of acquiring these behaviors—it is thinkable that SAIs are potentially capable of executing these skills but are reluctant to do so or are inhibited as a result of their anxiety (Furmark, 2000). Finally, it must be noted that results from studies evaluating the effects of social skills trainings in comparison, or addition, to, for example, cognitive-behavioral therapy are mixed. While some studies report a superior effect of an additional social skills training (e.g., Herbert et al., 2005), others suggest no additional value (Marzillier, Lambert, & Kellett, 1976). Again, some theoretical and methodological considerations may restrict the generalizability of either of these sets of findings. A thorough discussion of this controversy can be found in **Chapter 17**.

Anxious Behavior

One crucial element of socially anxious fear is the visibility of anxious behavior. SAIs fear that they will show symptoms of anxiety in a social interaction and that these signals will indisputably lead to negative evaluation by others. Regarding the literature cited so far, it must be made clear that safety behaviors, submissive behaviors, and lack of social skills and prosocial behaviors may all be seen as anxiety symptoms of some kind, which makes a clear-cut definition of "symptoms" quite difficult. Yet, some observable behaviors are much more clearly interpreted as anxiety related than others. One of these anxiety-related responses is blushing (c.f., **Chapter 5**). Drummond and Su (2012) have shown that objective measurement of facial blood flow (i.e., blushing responses) is positively correlated with degree of social anxiety as measured with the Fear of Negative Evaluation scale, although the sample was not particularly selected for fear of blushing. In addition, blushing propensity was not correlated with the degree of expectancy to blush or the subjective experience of blushing. Heerey and Kring (2007) reported that social anxiety was associated with other behaviors such as nervous fidgeting and reassurance seeking. Mauss, Wilhelm, and Gross (2004) have shown that rigid posture and fearful as well as rigid facial expressions were characteristic for SAIs but not for NACs. In the same line, Levitan et al. (2012) reported that SAIs tended toward "freeze-like" (reduced body sway and slower swaying) behavior when placed on a stabilometric force platform.

In an attempt to investigate whether showing anxiety symptoms is truly related to negative evaluation by others, Gee, Antony, Koerner, and Aiken (2012) showed typical anxious behavior captured in video fragments to SAI and NAC raters. In addition, two of the videos contained different degrees of self-disclosure about the anxiety that the portrayed individual experienced. The researchers reported that, without

self-disclosure, symptoms of anxiety were related to increased ratings of awkwardness, lack of social skills, and weakness. Self-disclosure about feeling anxious, however, led to decreases in negative ratings. Interestingly, the degree of social anxiety of the rater had no influence on the evaluations whatsoever.

The reviewed literature suggests that social anxiety has detrimental effects on social interaction behaviors in SAIs. In sum, they keep more interpersonal space, move nervously when someone approaches them, avoid eye contact, mimic less, show less social and conversational skills, maintain a rigid body posture, show inappropriate facial expressions, and show other submissive- and anxiety-related behaviors. It would be, beyond doubt, naïve to assume that these behavioral peculiarities remain unnoticed by (nonanxious) interaction partners. It is highly plausible that these behaviors influence the evaluation that others have of the person displaying these behaviors. Until now, cognitive models and treatment regimens have only peripherally integrated these kinds of behaviors, though some evidence has highlighted the extra value of specifically targeting safety behaviors or social skills in therapy, and its positive effects on subjective experience of anxiety and objective evaluation by others (e.g., Furukawa et al., 2009; McManus, Sacadura, & Clark, 2008). Based on the sum of this evidence, it is, in our eyes, negligent to make no use of this knowledge to improve the understanding and treatment of SAD. Therefore, in the last part of this chapter, we will suggest a model that readily incorporates this notion and possibly augments the common understanding of SAD.

Cognition and Behavior in SAD: A Refined Model

About 30 years ago, distortions in information processing were identified to be a key component of emotional disorders (Beck, 1976) and, as such, were integrated in a first model for treatment. Since then, it has been claimed that individuals suffering from anxiety disorders share a misconception about how threatening an object, situation, or sensation really is and that they preferentially process *putative* threat-relevant information. These cognitive biases are subdivided into a number of different phenomena: for example, negative interpretation bias, evaluation or judgmental biases, and attention bias.

In recent years, the cognitive processes underlying anxiety have come more into focus. Cognitive theories, such as Clark and Wells' (1995), have stressed that biases in the information processing of social cues in patients diagnosed with SAD might contribute to the maintenance, and maybe even etiology, of the disorder (Heinrichs & Hofmann, 2001). Presently there is cumulative experimental evidence to strengthen these claims (Hirsch & Clark, 2004). Furthermore, as reviewed above, there is also cumulative evidence that high degrees of social anxiety are related to disrupted behavior patterns that are suspected to lead to *true* negative evaluation. Based on these findings, it is plausible to assume that, whenever an SAI enters a situation in which (negative) evaluation by others is possible, a threat evaluation takes place. This may be accomplished by a kind of valence evaluation system comparable to that suggested by Mogg and Bradley (1998). This system is fine-tuned by situational context, prior learning, and biological preparedness (as discussed earlier). Once a general "threat

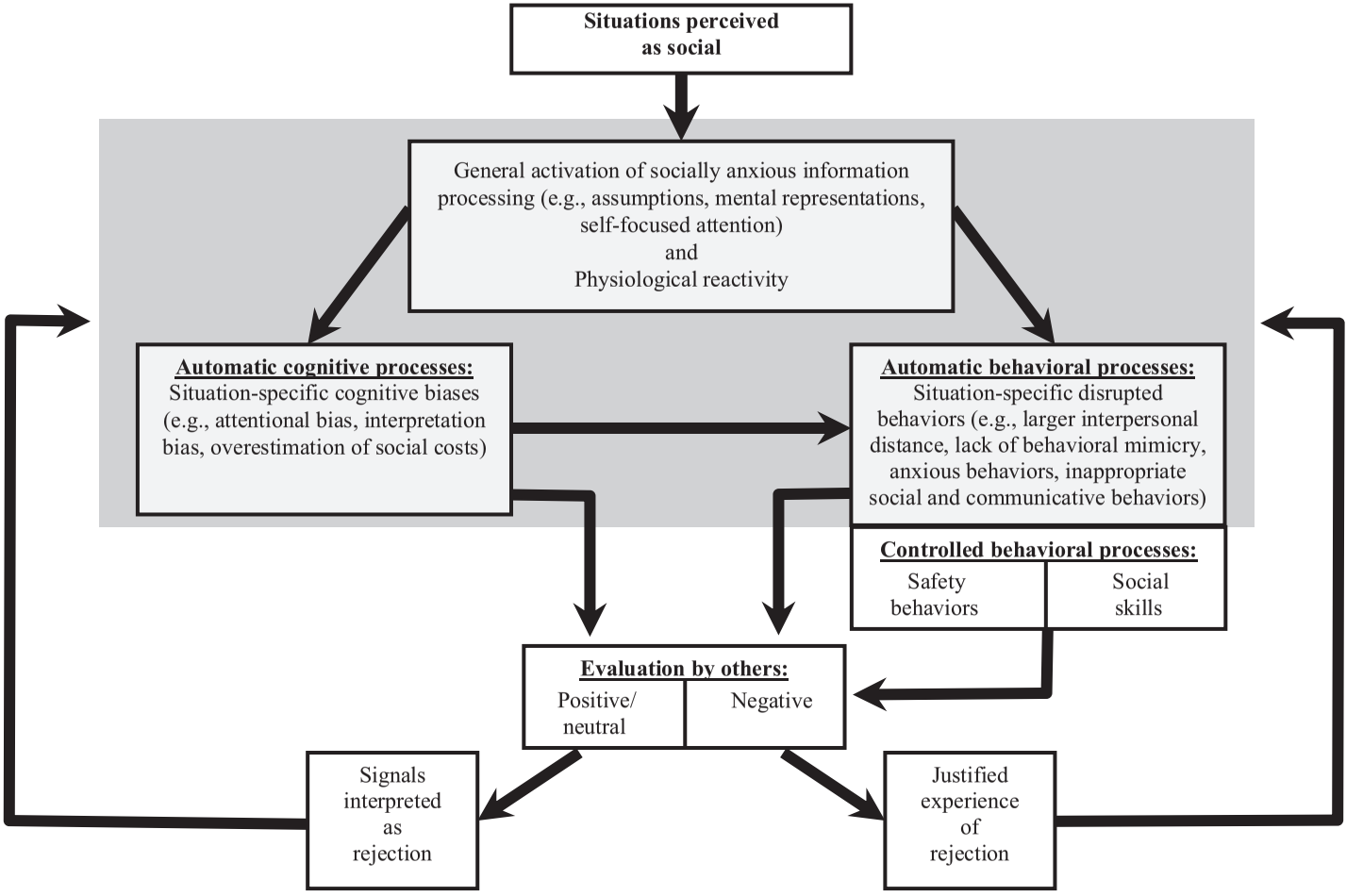

Figure 16.1 Refined cognitive-behavioral model of social anxiety disorder.

tag" has been given to the situation, a nonspecific activation of socially anxious information processing occurs (see Figure 16.1). Here, broad, threatening assumptions (e.g., "If they notice that I am anxious they will find me stupid"), self-focused attention to distorted mental representations of the self as seen by others, or to physiological symptoms of anxiety, are initiated (Clark & Wells, 1995; Heimberg et al., 2010). On the one hand, this initiates more situation-specific biased cognitive processes such as looking for external indicators of (negative) evaluation, as suggested by Rapee and Heimberg (1997): Hypervigilant attention is then directed to possible threat signals, negative interpretation of ambiguous behaviors, and estimation of the likelihood of and the consequences of negative evaluations, should they occur.

On the other hand, the activation of the more general threat assumptions leads not only to automatic situation-specific cognitive biases, but also to automatic situation-specific behavioral processes. These have been described as rather nonspecific "behavioral symptoms of anxiety" in the previous models and were basically acknowledged as safety behaviors. Rapee and Heimberg (1997) gave these some more credit by linking them with external indicators of negative evaluation, and evaluation in general in their updated model. In addition, they also suggested that focusing simultaneously inward, on external cues of evaluation, and on the task at hand, may considerably hamper adequate social behavior in that situation (Heimberg et al., 2010). The consequences of these behaviors with respect to true negative evaluation, however, remain largely unexplored. The proposed model (Figure 16.1) distinguishes automatic behaviors from strategic behaviors, such as safety behaviors, or behaviors that can be willingly acquired, such as social skills. The most prominent problem in the debate about lacking versus inhibited social skills in SAIs, and the effect of social skills training (c.f., **Chapter 17**), is the lack of a clear-cut definition for these skills. In the absence of such a definition, a "working definition" will be used based on Voncken and Bögels (2008). They consider observable *anxiety-related behaviors* (e.g., nervous fidgeting) and *communicative behaviors* (eye contact, smiling, completion of sentences, interest in subject, reciprocity, etc.) as target behaviors that reflect social skills.

The behaviors that we describe as "automatic" can be best understood in light of the social psychological concept of "embodied cognition" that eventually leads to social resonance. Embodied cognition or *embodiment* describes the idea that our attitudes toward our environment are visibly or imperceptibly reflected in our body movements and behaviors. In the same way, however, our body postures and behaviors reciprocally determine our attitudes too (Niedenthal, Barsalou, Winkielman, Krauth Gruber, & Ric, 2005). If, for example, we like someone or something, we tend to smile or lean forward. In turn, if we are forced to keep our mouth in a smile-like expression with the aid of, for example, a pen, while evaluating someone, our ratings are more positive than if we would have made our evaluations while standing straight and looking neutral (Glenberg, Havas, Becker, & Rinck, 2005). The same thing happens in a social interaction. Once Person 1 finds Person 2 amicable, Person 1 will move closer, smile more often, have more eye contact and show and imitate numerous other subtle mannerisms that he/she is not consciously aware of. At the same time, subsequent evaluations of Person 2 are likely to become more positive as these evaluations take place while one is already in a "positive" body state. The idea of social resonance can be taken even further. While Person 1 tries to *gain* sympathy from Person 2

by showing affiliative behaviors such as leaning forward or mimicking, he/she also expresses his/her own sympathy at the same time. Although unconsciously, this does not remain unnoticed by Person 2, who, in turn, shows these behaviors him-/herself. This normally leads to reciprocal behavior coordination or "rapport" between the two interacting parties (Kopp, 2010).

We believe that some of these automatic behaviors are triggered while others are inhibited when a situation is evaluated as socially "threatening." When the situation and the interaction partner are seen as threatening, or negative, associated "embodied" behaviors will be executed that reflect this attitude. At the same time, behaviors meant to increase affiliation, such as mimicry, or maintained eye contact, will be inhibited. If these were planned or readily controllable behaviors, one would assume that SAIs would express them frequently. Comparable to safety behaviors, they would hope that their anxiety could be tamed and that they can manage the impression they make on others to a certain degree. However, these behaviors are not readily learned by choice or training: The body "acts on its own accord," and so reflects the state of discomfort and fear that SAIs experience. No social skills training so far has incorporated modules of keeping the "correct interpersonal space" or "how to appropriately mimic" someone in a conversation, simply because these behaviors are, to a certain degree, biologically rooted and fine-tuned across a lifetime of experience and observation. In general, socially anxious thinking is based on threat inferences from the valence evaluation system, as well as on the automatic cognitive and behavioral processes. These are probably activated in quick succession or even simultaneously. This is difficult to disentangle, even more so as these processes are partially interrelated, as Hirsch, Clark, and Mathews (2006) suggested in their *combined cognitive bias hypothesis.* We assume that the degree of initial threat evaluation, as well as the rigidity of negative assumptions and mental representations, determines the speed and effort with which attention is focused on negative cues (or with which ambiguities are interpreted). This desperation to "find evidence" for one's (negative) presumptions probably determines the degree to which the automatic behaviors are executed or to which their "normal" execution is inhibited.

Even though the general activation of anxious cognitions is probably rapid and automatic, this does not mean that the contents of these assumptions or mental representations remain unconscious to the individual. Realizing that there is threat (i.e., that one is feeling anxious), and the realization that one possibly lacks the social skills to handle a given situation, leads to the execution of a repertoire of planned behaviors. These behaviors encompass prepared avoidance or, if not possible, strategies to regain control of the situation (e.g., impression management) and safety behaviors to reduce anxiety (Plasencia, Alden, & Taylor, 2011). Again, it is unclear how the sequence of these events unfolds, but it is very likely that the automatic and the more strategic behaviors are initiated in quick succession.

In addition to the incorporation of automatic as well as strategic behavioral deviations from normality, the factual evaluation of SAIs by others distinguishes this model from its predecessors. Earlier models have generally assumed that the fears of SAIs are ungrounded. The reviewed evidence, however, stresses the necessity to embrace the inherently true negative evaluation of SAIs. In our eyes, SAIs' behaviors in a social interaction—both automatic or strategic—can bring about these negative judgments

by others. Biased cognitive processing alone cannot account for this. This is crucial as, of course, SAIs *do* notice direct or indirect signs of negative evaluation in their environment, as they are tuned to look for these cues. Finding them confirms their initial fears, consolidates basic assumptions that they have, fine-tunes the valence evaluation system for the future, and reinforces these very same anxiety-related behaviors in the social situations to come.

Admittedly, it could be argued that it may not make a difference for an SAI whether he or she "sees" imagined signs of rejection versus true signs. For psychotherapy, however, this makes a world of a difference. As it is unclear as to how far cognitive and behavioral elements are causally related, it is very likely that the automatic behaviors develop into a stable behavioral repertoire triggered in any social situation, even when a patient may cognitively "know" that there should not be anything to be afraid of. This would mean that therapeutic interventions focused on challenging biased cognitions and strategic behaviors or social skills alone cannot lead to sufficient success if patients remain truly evaluated in a negative manner. To make matters worse, such patients probably lose confidence in the therapist and the suitability of cognitive-behavioral therapy.

In addition, there is an increasing body of evidence that SAIs respond differently from NACs in situations where they are truly rejected (ostracized). Mallott, Maner, DeWall, and Schmidt (2009), for example, have found that SAIs behaved less socially, were more anxious, and had decreased vocal and gaze quality after being experimentally excluded. Zadro, Boland, and Richardson (2006) found that SAIs did not differ from NACs in the effects of ostracism, such as evaluating the ostracizing individuals more negatively in terms of attractiveness and personality or interpreting ambiguous situations in a negative way. However, they took longer to recover from these effects than did NACs. In the same line, Oaten, Williams, Jones, and Zadro (2008) reported that SAIs still showed extensive self-regulation deficits 45 min after ostracism. Breen and Kashdan (2011) found that SAIs displayed more anger after imagining having been excluded. However, they used anger suppression techniques more than NACs to not experience the emotion. In the same line, we found that social anxiety is related to higher subjective anger when being socially excluded but not to expressed aggressive behavior (Lange & Becker, in prep.). In sum, there is a lot to be learned from these studies. Seemingly, SAIs do not, as would be expected, show more prosocial behavior after exclusion, in order to "reconnect" and "belong" again. To the contrary, they seem to withdraw from any (further) investment. It is possible that experimental exclusion is a more reliable way to evoke socially anxious response than merely setting up a conversation with confederates. This fairly new line of research could lead to a better understanding of the qualitative differences between various behaviors after imagined versus real rejection.

Taken together, both kinds of experiences (feared or true rejection) feed back into the automatically driven cognitive/behavioral circuitry: Fearful assumptions about the self and others as well as negative mental representations of the self are being confirmed, updated, and consolidated with every new experience. As automatic cognitive biases become more distinguished, automatic behaviors more pronounced, and strategic behavior more rigid in the experiences to come, the likelihood of true negative evaluation increases.

Conclusions

On the preceding pages, we have proposed a revised model for the maintenance of SAD based on the work of Clark and Wells (1995), Rapee and Heimberg (1997), and Hofmann (2007). The most crucial revisions concern the role of automatic and strategic behaviors and the incorporation of evaluations by others. We reviewed literature evidencing that SAIs do provoke negative evaluation from others rather than only fearing this event and that SAIs do differ in social behaviors bearing possible social (de-)evaluation. In turn, we assume that SAIs undoubtedly notice these reactions and thereby confirm their set of negative and anxious threat associations. Further, we assume that cognitive biases play an important additional role. Yet, behavior should play a more substantial role in a comprehensive model of SAD. Although automatic behaviors may be triggered by biased cognitive processes, they are probably not under direct control, nor are they easily overcome by changing the biases. They are the driving force in evoking *true* negative evaluation by others. If psychotherapy, as it does now, primarily tackles the faulty cognitions while neglecting that the fear of negative evaluation may be justified, chances of substantial and sustained recovery are undermined.

It still needs to be investigated as to how far change of automatic behavioral processes is mediated by threat associations, cognitive biases, or state social anxiety. Likewise, it is also crucial to differentiate behaviors in response to imagined versus true rejection. Based on treatment regimens and research results, we believe that this revised model provides a logical framework for an understanding of SAD and its maintaining factors. Even though models of psychopathology provide only simplified connotations of the "real" world and usually neglect approaches from other disciplines (e.g., medical), we hope that we have provided a comprehensive overview of factors and their intertwined relationships that contribute to new predictions, new research, better models, and a better understanding of SAD.

References

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders text revision* (4th ed.). Washington, DC: Author.

Angelico, A. P., Crippa, J. A., & Loureiro, S. R. (2010). Social anxiety disorder and social skills: A critical review of the literature. *International Journal of Behavioral Consultation and Therapy, 6*(2), 95–110.

Bailenson, J. N., & Yee, N. (2005). Digital chameleons: Automatic assimilation of nonverbal gestures in immersive virtual environments. *Psychological Science, 16*(10), 814–819. doi:10.1111/j.1467-9280.2005.01619.x

Baker, S. R., & Edelmann, R. J. (2002). Is social phobia related to lack of social skills? Duration of skill-related behaviours and ratings of behavioural adequacy. *British Journal of Clinical Psychology, 41*(3), 243–257.

Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychological Bulletin, 117*(3), 497–529. doi:10.1037/0033-2909.117.3.497

Beck, A. T. (1976). *Cognitive therapy and the emotional disorders*. New York, NY: International Universities Press.

Beidel, D. C., Rao, P. A., Scharfstein, L., Wong, N., & Alfano, C. A. (2010). Social skills and social phobia: An investigation of DSM-IV subtypes. *Behaviour Research and Therapy, 48*(10), 992–1001. doi:10.1016/j.brat.2010.06.005

Bögels, S. M., Rijsemus, W., & De Jong, P. J. (2002). Self-focused attention and social anxiety: The effects of experimentally heightened self-awareness on fear, blushing, cognitions, and social skills. *Cognitive Therapy and Research, 26*(4), 461–472. doi:10.1023/A:1016275700203

Breen, W. E., & Kashdan, T. B. (2011). Anger suppression after imagined rejection among individuals with social anxiety. *Journal of Anxiety Disorders, 25*(7), 879–887. doi:10.1016/j.janxdis.2011.04.009

Chartrand, T. L., & Bargh, J. A. (1999). The chameleon effect: The perception-behavior link and social interaction. *Journal of Personality and Social Psychology, 76*(6), 893–910. doi:10.1037/0022-3514.76.6.893

Chen, M., & Bargh, J. A. (1999). Consequences of automatic evaluation: Immediate behavioral predispositions to approach or avoid the stimulus. *Personality and Social Psychology Bulletin, 25*(2), 215–224.

Clark, D. M., & Wells, A. (1995). A cognitive model of social phobia. In R. Heimberg, M. Liebowitz, D. Hope, & F. Schneier (Eds.), *Social phobia diagnosis, assessment, and treatment* (pp. 69–112). New York, NY: Guilford Press.

Curtis, R. C., & Miller, K. (1986). Believing another likes or dislikes you: Behaviors making the beliefs come true. *Journal of Personality and Social Psychology, 51*(2), 284–290.

Darwin, C. R. (1859). *On the origin of species by means of natural selection, or the preservation of favoured races in the struggle for life* (1st ed.). London, UK: John Murray.

Derryberry, D., & Reed, M. A. (1994). Temperament and attention: Orienting toward and away from positive and negative signals. *Journal of Personality and Social Psychology, 66*(6), 1128–1139.

Dickinson, A., & Dearing, M. F. (1979). Appetitive-aversive interactions and inhibitory processes. In A. Dickinson & R. A. Boakes (Eds.), *Mechanisms of learning and motivation* (pp. 203–231). Hillsdale, NJ: Erlbaum.

Dijksterhuis, A., & Bargh, J. A. (2001). The perception-behavior expressway: Automatic effects of social perception on social behavior. In M. P. Zanna (Ed.), *Advances in experimental social psychology* (Vol. 33, pp. 1–40). San Diego, CA: Academic Press.

Dimberg, U. (1997). Social fear and expressive reactions to social stimuli. *Scandinavian Journal of Psychology, 38*(3), 171–174.

Dimberg, U., & Christmanson, L. (1991). Facial reactions to facial expressions in subjects high and low in public speaking fear. *Scandinavian Journal of Psychology, 32*(3), 246–253. doi:10.1111/j.1467-9450.1991.tb00875.x

Dimberg, U., & Thunberg, M. (2007). Speech anxiety and rapid emotional reactions to angry and happy facial expressions. *Scandinavian Journal of Psychology, 48*(4), 321–328.

Drummond, P. D., & Su, D. (2012). The relationship between blushing propensity, social anxiety and facial blood flow during embarrassment. *Cognition and Emotion, 26*(3), 561–567. doi:10.1080/02699931.2011.595775

Fehm, L., Pelissolo, A., Furmark, T., & Wittchen, H. U. (2005). Size and burden of social phobia in Europe. *European Neuropsychopharmacology, 15*(4), 453–462.

Fridlund, A. J. (1994). *Human facial expression: An evolutionary view*. San Diego, CA: Academic Press.

Furmark, T. (2000). *Social phobia. From epidemiology to brain function* (Dissertation for the Degree of Doctor of Philosophy in Psychology). Retrieved from Acta Universitatis Upsaliensis database.

Furukawa, T. A. W., Chen, J., Watanabe, N., Nakano, Y., Ietsugu, T., Ogawa, S., . . . Noda, Y. (2009). Videotaped experiments to drop safety behaviors and self-focused attention for patients with social anxiety disorder: Do they change subjective and objective evaluations of anxiety and performance? *Journal of Behavior Therapy and Experimental Psychiatry, 40*(2), 202–210. doi:10.1016/j.jbtep.2008.08.003

Geary, D. C. (2007). An integrative model of human brain, cognitive, and behavioral evolution. *Acta Psychologica Sinica, 39*(3), 383–397.

Gee, B. A., Antony, M. M., Koerner, N., & Aiken, A. (2012). Appearing anxious leads to negative judgments by others. *Journal of Clinical Psychology, 68*(3), 304–318. doi:10.1002/jclp.20865

Gilbert, P. (2001). Evolution and social anxiety. The role of attraction, social competition, and social hierarchies. *The Psychiatric Clinics of North America, 24*(4), 723–751.

Glenberg, A. M., Havas, D., Becker, R., & Rinck, M. (2005). Grounding language in bodily states: The case for emotion. In D. Pecher & R. A. Zwaan (Eds.), *Grounding cognition* (pp. 115–128). New York, NY: Cambridge University Press.

Haxby, J. V., Hoffman, E. A., & Gobbini, M. I. (2000). The distributed human neural system for face perception. *Trends in Cognitive Sciences, 4*(6), 223–233.

Heerey, E. A., & Kring, A. M. (2007). Interpersonal consequences of social anxiety. *Journal of Abnormal Psychology, 116*(1), 125–134.

Heimberg, R. G. (2002). Cognitive-behavioral therapy for social anxiety disorder: Current status and future directions. *Biological Psychiatry, 51*(1), 101–108. doi:S0006322301011830 [pii]

Heimberg, R. G., Brozovich, F. A., & Rapee, R. M. (2010). A cognitive behavioral model of social anxiety disorder: Update and extension. In S. G. Hofmann & P. M. DiBartolo (Eds.), *Social anxiety: Clinical, developmental, and social perspectives* (2nd ed., pp. 395–422). New York, NY: Elsevier Academic Press.

Heinrichs, N., & Hofmann, S. G. (2001). Information processing in social phobia: A critical review. *Clinical Psychology Review, 21*(5), 751–770.

Herbert, J. D., Gaudiano, B. A., Rheingold, A. A., Myers, V. H., Dalrymple, K., & Nolan, E. M. (2005). Social skills training augments the effectiveness of cognitive behavioral group therapy for social anxiety disorder. *Behavior Therapy, 36*, 125–138.

Heuer, K., Rinck, M., & Becker, E. S. (2007). Avoidance of emotional facial expressions in social anxiety: The Approach-Avoidance Task. *Behaviour Research and Therapy, 45*(12), 2990–3001.

Hirsch, C. R., & Clark, D. M. (2004). Information-processing bias in social phobia. *Clinical Psychology Review, 24*(7), 799–825.

Hirsch, C. R., Clark, D. M. M., & Mathews, A. (2006). Imagery and interpretations in social phobia: Support for the combined cognitive biases hypothesis. *Behavior Therapy, 37*(3), 223–236.

Hjortsjö, C. H. (1970). *Man's face and mimic language*. Malmö, Sweden: Nordens Boktryckeri.

Hofmann, S. G. (2007). Cognitive factors that maintain social anxiety disorder: A comprehensive model and its treatment implications. *Cognitive Behaviour Therapy, 36*(4), 193–209.

Huppert, J. D., Pasupuleti, R. V., Foa, E. B., & Mathews, A. (2007). Interpretation biases in social anxiety: Response generation, response selection, and self-appraisals. *Behaviour Research and Therapy, 45*(7), 1505–1515.

Juth, P., Lundqvist, D., Karlsson, A., & Öhman, A. (2005). Looking for foes and friends: Perceptual and emotional factors when finding a face in the crowd. *Emotion, 5*(4), 379–395.

Kopp, S. (2010). Social resonance and embodied coordination in face-to-face conversation with artificial interlocutors. *Speech Communication, 52*(6), 587–597. doi:10.1016/j.specom.2010.02.007

Lakin, J. L., Jefferis, V. E., Cheng, C. M., & Chartrand, T. L. (2003). The chameleon effect as social glue: Evidence for the evolutionary significance of nonconscious mimicry. *Journal of Nonverbal Behavior, 27*(3), 145–162. doi:10.1023/A:1025389814290

Lang, P. J. (1985). The cognitive psychophysiology of emotion: Fear and anxiety. In A. H. Tuma & I. D. Mazer (Eds.), *Anxiety and the anxiety disorders* (pp. 131–170). Hillsdale, NJ: Erlbaum.

Lang, P. J. (1995). The emotion probe. Studies of motivation and attention. *American Psychologist, 50*(5), 372–385.

Lang, P. J., Davis, M., & Öhman, A. (2000). Fear and anxiety: Animal models and human cognitive psychophysiology. *Journal of Affective Disorders, 61*(3), 137–159.

Lange, W. G., & Becker, E. S. (in prep.). Looking back in anger: Social anxiety and ostracism.

Lange, W. G., Keijsers, G. P. J., Becker, E. S., & Rinck, M. (2008). Social anxiety and evaluation of social crowds: Explicit and implicit measures. *Behaviour Research and Therapy, 46*(8), 932–943.

Lange, W. G., Roelofs, K., & Becker, E. S. (in prep.). Don't stand so close to me: Personal space in social anxiety.

LeDoux, J. E. (1996). *The emotional brain: The mysterious underpinnings of emotional life*. New York, NY: Simon and Schuster.

Levitan, M. N., Crippa, J. A., Bruno, L. M., Pastore, D. L., Freire, R. C., Arrais, K. C., . . . Nardi, A. E. (2012). Postural balance in patients with social anxiety disorder. *Brazilian Journal of Medical and Biological Research, 45*(1), 38–42. doi:10.1590/S0100-879X2011007500155

Levitan, M. N., & Nardi, A. E. (2009). Social skill deficits in socially anxious subjects. *World Journal of Biological Psychiatry, 10*(6), 702–709. doi:10.3109/15622970802255919

Mallott, M. A., Maner, J. K., DeWall, N., & Schmidt, N. B. (2009). Compensatory deficits following rejection: The role of social anxiety in disrupting affiliative behavior. *Depression and Anxiety, 26*(5), 438–446. doi:10.1002/da.20555

Marzillier, J. S., Lambert, C., & Kellett, J. (1976). A controlled evaluation of systematic desensitization and social skills training for socially inadequate psychiatric patients. *Behaviour Research and Therapy, 14*(3), 225–238. doi: 10.1016/0005-7967(76)90015-2

Mathew, S. J., & Ho, S. (2006). Etiology and neurobiology of social anxiety disorder. *Journal of Clinical Psychiatry, 67*, 9–13.

Mauss, I. B., Wilhelm, F. H., & Gross, J. J. (2004). Is there less to social anxiety than meets the eye? Emotion experience, expression, and bodily responding. *Cognition and Emotion, 18*(5), 631–662.

McManus, F., Sacadura, C., & Clark, D. M. M. (2008). Why social anxiety persists: An experimental investigation of the role of safety behaviours as a maintaining factor. *Journal of Behavior Therapy and Experimental Psychiatry, 39*(2), 147–161.

Mogg, K., & Bradley, B. P. (1998). A cognitive-motivational analysis of anxiety. *Behaviour Research and Therapy, 36*(9), 809–848.

Mogg, K., & Bradley, B. P. (2007). *Attentional processing of emotional stimuli: Mechanisms and measurement*. Paper presented at the Opening meeting of the FWO Scientific Research Community "Automatic processes in psychopathology and health related behavior", Ghent, Belgium.

Neal, J. A., & Edelmann, R. J. (2003). The etiology of social phobia: Toward a developmental profile. *Clinical Psychology Review, 23*(6), 761–786.

Niedenthal, P. M., Barsalou, L. W., Winkielman, P., Krauth Gruber, S., & Ric, F. (2005). Embodiment in attitudes, social perception, and emotion. *Personality and Social Psychology Review, 9*(3), 184–211.

Oaten, M., Williams, K. D., Jones, A., & Zadro, L. (2008). The effects of ostracism on self-regulation in the socially anxious. *Journal of Social and Clinical Psychology, 27*(5), 471–504. doi:10.1521/jscp.2008.27.5.471

Öhman, A. (1993). Fear and anxiety as emotional phenomena: Clinical phenomenology, evolutionary perspectives, and information-processing mechanisms. In M. Lewis & J. M. Haviland (Eds.), *Handbook of emotions* (pp. 511–536). New York, NY: Guilford Press.

Öhman, A., Dimberg, U., & Öst, L.-G. (1985). Animal and social phobias: Biological constraints on learned fear responses. In S. Reiss & R. R. Bootzin (Eds.), *Theoretical issues in behavior therapy* (pp. 123–178). Orlando, FL: Academic Press.

Plasencia, M. L., Alden, L. E., & Taylor, C. T. (2011). Differential effects of safety behaviour subtypes in social anxiety disorder. *Behaviour Research and Therapy, 49*(10), 665–675. doi:10.1016/j.brat.2011.07.005

Rapee, R. M., & Heimberg, R. G. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy, 35*(8), 741–756.

Rinck, M., & Becker, E. S. (2007). Approach and avoidance in fear of spiders. *Journal of Behavior Therapy and Experimental Psychiatry, 38*, 105–120.

Rinck, M., Rörtgen, T., Lange, W.-G., Dotsch, R., Wigboldus, D. H. J., & Becker, E. S. (2010). Social anxiety predicts avoidance behaviour in virtual encounters. *Cognition & Emotion, 24*(7), 1269–1276.

Roelofs, K., Putman, P., Schouten, S., Lange, W.-G., Volman, I., & Rinck, M. (2010). Gaze direction differentially affects avoidance tendencies to happy and angry faces in socially anxious individuals. *Behaviour Research and Therapy, 48*(4), 290–294. doi:10.1016/j.brat.2009.11.008

Schofield, C. A., Coles, M. E., & Gibb, B. E. (2007). Social anxiety and interpretation biases for facial displays of emotion: Emotion detection and ratings of social cost. *Behaviour Research and Therapy, 45*(12), 2950–2963.

Seligman, M. E. (1971). Phobias and preparedness. *Behavior Therapy, 2*(3), 307–320.

Staugaard, S. R. (2010). Threatening faces and social anxiety: A literature review. *Clinical Psychology Review, 30*(6), 669–690. doi:10.1016/j.cpr.2010.05.001

Stein, M. B., Jang, K. L., & Livesley, W. J. (2002). Heritability of social anxiety-related concerns and personality characteristics: A twin study. *Journal of Nervous and Mental Disease, 190*(4), 219–224.

Voncken, M. J., Alden, L. E., Bögels, S. M., & Roelofs, J. (2008). Social rejection in social anxiety disorder: The role of performance deficits, evoked negative emotions and dissimilarity. *British Journal of Clinical Psychology, 47*(4), 439–450. doi:10.1348/014466508×334745

Voncken, M. J., & Bögels, S. M. (2008). Social performance deficits in social anxiety disorder: Reality during conversation and biased perception during speech. *Journal of Anxiety Disorders, 22*(8), 1384–1392.

Vrana, S. R., & Gross, D. (2004). Reactions to facial expressions: Effects of social context and speech anxiety on responses to neutral, anger, and joy expressions. *Biological Psychology, 66*(1), 63–78. doi:10.1016/j.biopsycho.2003.07.004

Vrijsen, J. N., Lange, W.-G., Becker, E. S., & Rinck, M. (2010). Socially anxious individuals lack unintentional mimicry. *Behaviour Research and Therapy, 48*(6), 561–564. doi:10.1016/j.brat.2010.02.004

Vrijsen, J. N., Lange, W.-G., Dotsch, R., Wigboldus, D. l.H. J., & Rinck, M. (2010). How do socially anxious women evaluate mimicry? A virtual reality study. *Cognition & Emotion, 24*(5), 840–847.

Vuilleumier, P. (2005). How brains beware: Neural mechanisms of emotional attention. *Trends in Cognitive Sciences, 9*(12), 585–594.

Weeks, J. W., Heimberg, R. G., & Heuer, R. (2011). Exploring the role of behavioral submissiveness in social anxiety. *Journal of Social and Clinical Psychology, 30*(3), 217–249.

Weeks, J. W., Heimberg, R. G., & Rodebaugh, T. L. (2008). The Fear of Positive Evaluation Scale: Assessing a proposed cognitive component of social anxiety. *Journal of Anxiety Disorders, 22*(1), 44–55.

Wieser, M. J., Pauli, P., Grosseibl, M., Molzow, I., & Mühlberger, A. (2010). Virtual social interactions in social anxiety-the impact of sex, gaze, and interpersonal distance. *Cyberpsychology Behavior and Social Networking, 13*(5), 547–554. doi:10.1089/cyber.2009.0432

Zadro, L., Boland, C., & Richardson, R. (2006). How long does it last? The persistence of the effects of ostracism in the socially anxious. *Journal of Experimental Social Psychology, 42*(5), 692–697.

17

Examining the Controversy Surrounding Social Skills in Social Anxiety Disorder

The State of the Literature

Brent W. Schneider and Cynthia L. Turk
Washburn University, USA

Social phobia, now referred to as social anxiety disorder (SAD), is characterized by fear of negative evaluation by others as a result of showing anxiety symptoms or acting in a way that will be humiliating (American Psychiatric Association [APA], 2000). In this way, concerns about social performance are embedded within the criteria for the diagnosis. Indeed, socially anxious individuals routinely express concerns about the adequacy of their social skills. Therefore, it is not surprising that clinicians and researchers have long been interested in the relationship between social skills deficits and social anxiety (Clark & Arkowitz, 1975), even before social phobia was first included as a diagnosis in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (APA, 1980). Understanding the relationship between social skills deficits and social anxiety is important in terms of how we conceptualize and treat the disorder.

Individuals with SAD routinely criticize their own social behavior. When asked to actually engage in a social interaction or performance—whether as part of a standardized behavioral assessment test or as part of an individualized exposure to a feared social situation—the objective quality of their behavior often seems better than would be expected from their self-report. Most socially anxious individuals underestimate the quality of their performance and overestimate the visibility of their anxiety (Cartwright-Hatton, Hodges, & Porter, 2003; Cartwright-Hatton, Tschernitz, & Gomersall, 2005; Clark & Arkowitz, 1975; Norton & Hope, 2001a; Rapee & Abbott, 2006; Rapee & Lim, 1992; Stopa & Clark, 1993; Voncken & Bögels, 2008).

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

Even though socially anxious individuals tend to underestimate the quality of their social performances, their social behavior may nevertheless be inadequate. Most clinicians who have worked with patients with SAD on a regular basis have observed patients who exhibit social behavior across the continuum of adequacy, from highly skilled to woefully inadequate. Whether the "typical" social behavior of the "typical" patient with SAD is adequate or not has been the subject of a fair amount of debate and research. The overall pattern of the data seems to suggest that patients with SAD, as a group, tend to perform more poorly than nonanxious individuals in laboratory situations in which they are asked to interact with novel persons or give a speech (Table 17.1). Some studies, however, have failed to find differences among individuals with and without significant social anxiety in performance ratings made by objective observers (Cartwright-Hatton et al., 2005; Clark & Arkowitz, 1975; Rapee & Lim, 1992; Strahan & Conger, 1998). Differences across studies in terms of statistical power, participant selection, tasks, and measures employed have undoubtedly influenced whether between group effects are found, suggesting perhaps that such differences exist but the magnitude of these differences is relatively small and, thus, inconsistently detected.

The degree to which the social behavior of socially anxious individuals deviates in a clinically meaningful way from that of nonanxious individuals has rarely been addressed. In one study, observer ratings of the social behavior of patients with generalized SAD fell within two standard deviations of the ratings given to a large group of nonanxious controls over 75% of the time across four different social tasks (Beidel, Rao, Scharfstein, Wong, & Alfano, 2010). Indeed, studies that include socially anxious and nonanxious groups typically find marked overlap in performance (Baker & Edelmann, 2002). Moreover, the literature suggests that many socially anxious individuals perform within the normal range, at least in terms of the social tasks that they have been called upon to engage in within the lab.

Even when social behavior seems objectively poor, it is usually unclear whether poor performance was a result of a lack of knowledge versus some aspect of anxiety inhibiting the expression of skillful behavior. The term "skill" generally suggests having the ability necessary for effective performance (Hopko, McNeil, Zvolensky, & Eifert, 2001). When poor performance is observed, alternative explanations besides a "social skills deficit" abound. Perhaps excessive self-focused attention or hypervigilance for environmental threat led the anxious individual to miss important environmental cues that would have otherwise elicited adaptive responses (Rapee & Heimberg, 1997). Perhaps the anxious individual is engaging in safety behaviors (e.g., not shaking someone's hand upon being introduced) in an attempt to circumvent what is perceived as a catastrophic outcome (e.g., the other person noticing one's sweaty palms and making a negative judgment), and these safety behaviors have the unintended effect of diminishing performance (Clark & Wells, 1995). As Hopko et al. (2001) point out, the only way that we can be confident that our patients possess adequate social skills is to observe them behaving in a skillful manner. When we observe them behaving in a deficient manner, we cannot speak definitively to the issue of whether there is a social skills deficit, whether anxiety is interfering with social performance, or whether the person has both a social skills deficit and is experiencing additional performance disruption due to anxiety (Hopko et al., 2001).

Table 17.1 Studies Examining the Quality of Observable Social Behavior Among Socially Anxious Individuals Identified Using Psychometrically Sound Self-report or Clinician-Administered Measures

Study	*Participants*	*Nature of social task(s)*	*Social skills assessment*	*Main findings*
Alden and Mellings (2004)	25 patients with generalized SAD; 26 community controls. Diagnosed with a structured clinical interview.	Conversation with a confederate	Confederates rated global skill.	SAD participants rated as less skillful than control participants.
Alden and Wallace (1995)	32 patients with generalized SAD and 32 nonclinical controls. Diagnosed with a structured clinical interview.	Conversation with opposite sex confederate	Experimenter rated positive verbal behavior, warmth/interest, and global likability.	SAD group conveyed less warmth and interest, emitted fewer positive verbal behaviors, and were less likable than control group.
Baker and Edelmann (2002)	18 patients with generalized SAD; 18 patients with other anxiety disorders; 18 normal controls. Diagnosed with a structured clinical interview.	Conversation with a confederate	Molecular behaviors (e.g., smiling) and global skill rated by a group of 21 minimally trained observers.	Controls > other anxiety disorder group > generalized SAD group for adequacy of overall skill, gestures, and speech fluency. Controls > both anxiety groups for adequacy of eye contact and speech clarity.

Beidel et al. (2007)	63 adolescents with SAD; 43 adolescents with no diagnosis. Diagnosed with structured clinical interviews.	Role-plays with a peer: conversation, offering to help, giving a compliment, receiving a compliment, and responding to inappropriate behavior. Participants also read aloud to an audience of an adult and peer.	Global skills rating	Observers rated SAD group as less skilled than the control group for the read aloud task and the role-play tasks
Beidel et al., 2010	101 individuals with generalized SAD without comorbid depression; 60 nongeneralized SAD; and 200 controls with no mental disorder. Diagnosed with a structured clinical interview.	Simulated social interaction test (SSIT; Curran, 1982); conversation task with opposite sex confederate; conversation task with same sex confederate; speech task.	Coders rated global skill in each of the tasks.	Global skill in SSIT and conversations: controls > nongeneralized SAD > generalized SAD. Global skill in the speech: controls and nongeneralized SAD > generalized SAD
Bögels, Rijsemus, and De Jong (2002)	36 high social anxiety participants; 36 low social anxiety participants. Classification based on a questionnaire.	Conversation	Observers utilized an adapted version of Rapee and Lim's (1992) social skills rating scale.	No differences between groups were observed for social skills.
Boone et al. (1999)	10 nongeneralized SAD; 15 generalized SAD without APD; generalized SAD with APD (for social skills analyses). Diagnosed with structured clinical interviews.	Speech; conversation	Coders rated global social skills and selected molecular behaviors (gaze, voice volume, intonation).	No significant omnibus group differences.

(*continued*)

Table 17.1 (*Continued*)

Study	*Participants*	*Nature of social task(s)*	*Social skills assessment*	*Main findings*
Cartwright-Hatton et al. (2003)	110 school children who completed a questionnaire measure of social anxiety.	Speech	Observers rated molecular behaviors (e.g., clear voice) and global impressions.	Correlation between level of social anxiety and observer ratings of social skills were not significant.
Cartwright-Hatton et al. (2005)	40 children classified as high or low social anxiety based on a questionnaire.	Conversation with an adult	Observers rated molecular behaviors (e.g., clear voice) and global impressions.	Observers rated high and low social anxiety groups similarly on molecular and global social skills.
Fydrich, Chambless, Perry, Buergener, and Beazley (1998)	34 patients with SAD; 14 patients with anxiety disorders other than SAD; 28 normal controls. Diagnosed with a structured clinical interview.	Two conversations	Observers rated behaviors using a modified version of Trower, Bryant, and Argyle's (1978). Ratings of molecular behaviors were totaled for an overall score.	SAD group performed more poorly than the other two groups, which did not differ from each other.
Heimberg, Hope, Dodge, and Becker (1990)	35 patients with generalized SAD; 22 patients with nongeneralized SAD. Diagnosed with a structured clinical interview.	Individualized behavioral assessment test (typically a conversation or a speech, but varied by participant based on their fear profile)	Research assistants rated global performance quality.	Generalized SAD group with poorer global performance than the nongeneralized SAD group.
Herbert, Hope, and Bellack (1992)	Nine patients with generalized SAD; 14 patients with generalized SAD with APD. Diagnosed with structured clinical interviews.	Two conversation tasks; speech	Rated for overall social skills; composite score derived from ratings of speech content, nonverbal behavior, and paralinguistic behavior.	No between groups differences emerged on any measure of social skills.

Hofmann, Gerlach, Wender, and Roth (1997)	14 patients with generalized SAD; 10 patients with nongeneralized SAD; 13 nonclinical controls with generalized social fears; 12 controls with nongeneralized social fears. Diagnosed with a structured clinical interview.	Speech	Filled pauses, speech errors, silent pauses, gaze behavior.	Combined SAD groups had more filled pauses, longer pauses, more frequent pauses, and spent more time pausing than controls. Generalized SAD group spent more time pausing than the other three groups.
Levitan et al. (2012)	18 patients with generalized SAD; 18 normal controls. Diagnosed with a structured clinical interview.	Speech	Observers rated behaviors using Trower et al.'s (1978) rating system, which measures molecular and molar behaviors.	Generalized SAD with poorer intonation, poorer fluency, and poorer overall performance.
Norton and Hope (2001a)	54 patients with SAD; 23 patients with dysthymia; 28 normal controls. Diagnosed with structured interviews	Speech; conversation, confederate responded naturally; conversation, confederate responded in a reserved manner.	Global ratings of performance quality by research assistants.	Observers rated the SAD group as performing more poorly than the other groups.
Rapee and Abbott (2006)	57 patients with SAD; 41 nonclinical controls. Diagnosed with a structured clinical interview.	Speech	Speech performance was rated by observers using Rapee and Lim's (1992) speech performance questionnaire	Observers rated the SAD group as performing more poorly than the control group.

(*continued*)

Table 17.1 (*Continued*)

Study	*Participants*	*Nature of social task(s)*	*Social skills assessment*	*Main findings*
Rapee and Lim (1992)	28 individuals with SAD and no comorbid mood disorder; 33 nonclinical participants. Patients diagnosed using a structured clinical interview.	Speech in which study participants served as audience members	Rating form for speech was developed by authors for this study. Ratings made by audience members.	No significant difference was found between SAD and nonclinical participants on observer ratings of global items
Scharfstein, Beidel, Sims, and Finnell (2011)	30 children with SAD; 30 children with Asperger's Disorder (AD); 30 typically developing peers as controls. Diagnosed with structured clinical interviews.	Role-plays with a peer: conversation, offering to help, giving a compliment, receiving a compliment, and responding to inappropriate behavior.	Observers coded global social skill and 12 specific social behaviors.	AD = control group but better than SAD for global skills, appropriateness of affect, and skill maintaining the conversation. Control group better than the SAD group, with the AD group not differing from controls or SAD children, for latency to talk, number of words spoken, response appropriateness, and vocal inflection.
Segrin and Kinney (1995)	31 high social anxiety participants; 33 low social anxiety participants. Classification based on a questionnaire.	Unstructured conversation while waiting in a room with a confederate (participant did not know the study had begun)	Observers rated global and molecular social skills; partner rated global social competence	High social anxiety participants, relative to nonanxious participants, received more negative ratings from conversation partners. Observer ratings did not differentiate the groups.

Spence, Donovan, and Brechman-Toussaint (1999)	27 children with SAD; 27 control children. Diagnosed with structured clinical interviews.	Naturalistic observation at school. Role-play task adapted from the Behavioral Assertiveness Test for Children (BAT-C; Bornstein, Bellack, & Hersen, 1977), which included six situations requiring positive assertion (e.g., giving a positive compliment) and six requiring a negative assertion (e.g., refusing an unreasonable request).	At school, observers rated the quality of the peer response (positive, negative, ignore) to each interaction with the target child. For the BAT-C, observers rated eye contact, length of response, latency to respond, and global assertiveness.	At school, children with SAD were less likely than controls to receive a positive response from peers; however, the groups did not differ in eliciting negative or ignore responses. For the BAT-C, no group differences were found other than the SAD group spoke fewer words than the control group.
Stopa and Clark (1993)	12 patients with SAD; 12 patients with anxiety disorders other than SAD; 12 normal controls. Diagnosed with a structured clinical interview.	Conversation	Observers rated positive (e.g., warm, pleasant) and negative (e.g., awkward, uncomfortable) social behaviors.	SAD group with more negative social behaviors than the two other groups. SAD group with fewer positive behaviors than the control group.
Strahan and Conger (1998)	27 high social anxiety men; 26 low social anxiety men. Classification based on questionnaire.	Simulated job interview	Observers rated content, fluency, nonverbals, and global competence.	Groups did not differ for global competence.

(*continued*)

Table 17.1 (*Continued*)

Study	*Participants*	*Nature of social task(s)*	*Social skills assessment*	*Main findings*
Thompson and Rapee (2002)	26 high social anxiety women; 22 low social anxiety women. Classification based on questionnaire.	Unstructured conversation while waiting in a room with a male confederate (participant did not know the study had begun); structured conversation same confederate with instructions to get to know each other as if at a party.	Observers rated behaviors using Trower et al.'s (1978) rating system, which measures molecular and molar behaviors.	Relative to the high anxiety group, the low social anxiety group exhibited better molar skills in both tasks. The low social anxiety group exhibited better molecular skills in the unstructured task. No group differences in molecular skills emerged for the structured task.
Tran and Chambless (1995)	16 nongeneralized SAD; 12 generalized SAD without APD, 16 generalized SAD with APD. Diagnosed with structured clinical interviews.	Speech; conversation with opposite sex confederate; conversation with same sex confederate	Observers rated global social skill.	For speech and conversation tasks, nongeneralized group rated as more skilled than the generalized SAD with APD group; the generalized SAD without APD group did not differ significantly from the other two groups.

Turner, Beidel, and Townsley (1992)	28 nongeneralized SAD; 46 generalized SAD without APD; 15 generalized SAD with APD. Diagnosed with structured clinical interviews.	Speech; conversation with opposite sex confederate; conversation with same sex confederate	Conversation task was rated for facial gaze, voice tone, number of verbal initiations, length of verbalization, and overall skill.	No significant differences on social skills among the three groups.
Voncken and Bögels (2008)	48 generalized SAD; 27 normal controls. Diagnosed with a structured clinical interview.	Speech; conversation with two confederates	Confederates rated 16 items (e.g., eye contact) on a social behavior subscale.	Confederates rated SAD participants as having less adequate social behavior than controls during the conversations; confederate ratings did not differentiate the two groups with regard to performance during the speech.
Wenzel, Graff-Dolezal, Macho, and Brendle (2005)	13 socially anxious undergraduates with romantic partners; 14 nonanxious undergraduates with romantic partners. Target participants categorized with questionnaires.	10-min discussion about events of their day; 10-min discussion about a problem area in their relationship; 10-min discussion about positive aspect of their relationship	Videotapes were coded using the Communication Skills Test (CST; Floyd & Markman, 1984); 10 molecular skills (e.g., nodding) rated; global impression of social skills rated	Across all conversations, SA participants displayed fewer positive behaviors. SA group displayed more very negative behaviors during the discussion of the problem. SA group performed more poorly on the global and most molecular ratings of social skill.

Problems with the Literature Examining the Social Skills of Socially Anxious Individuals

Defining Social Skills

There is not one widely accepted operational definition of the term "social skills" (Stravynski & Amado, 2001), yet most of us believe that we intuitively know what social skills are. Many proposed operational definitions fall into a "too broad" category, leaving out concrete and specific components of skillful behavior that would allow us to know exactly what to look for to determine skill level. Alternatively, many definitions fall into a "too specific" category, including an unwieldy array of social skills components without speaking to the larger picture of how these skills come together to comprise effective social behavior.

Regardless of the definition used, context is an important factor in judging the adequacy of social behavior. Individuals with good social skills are able to vary their social behavior around friends, family, strangers, authorities, in public, in private, and so on. McFall (1982, p. 7) notes that, "no particular behavior can be considered intrinsically skillful, independent of its context." Del Prette and Del Prette (2001) define social skills as "different classes of social behavior within the individual's repertoire to deal appropriately with demands of interpersonal situations" (p. 31). These definitions stress the idea that social situations are dynamic and that individuals must effectively adapt their behaviors to be successful. Therefore, social skills must be thought of as not only a specific set of behaviors but also as the ability to adapt behaviors to the larger social context. These definitions would also require that cultural context be taken into account in understanding the skills within an individual's repertoire. For an expanded discussion of multicultural diversity issues in SAD, see **Chapter 11**.

Sample Selection

The criteria used to identify socially anxious individuals for studies of social skill have evolved over the years. Early studies identified participants by asking questions about dating frequency (Arkowitz, Lichtenstein, McGovern, & Hines, 1975; Conger & Farrell, 1981; Glasgow & Arkowitz, 1975; Twentyman & McFall, 1975; Wessberg, Mariotto, Conger, Ferrell, & Conger, 1979). Although some socially anxious people do not date due to their anxiety, this approach to participant selection is problematic in that individuals may choose not to date for reasons other than social anxiety and some socially anxious individuals may date despite their anxiety (e.g., they were pursued by their partners). Furthermore, in older studies, participants were selected based on a rather narrow scope of problems, which was consistent with early conceptualizations of social phobia as being limited to one or two social situations and only resulting in a minimal disruption in role functioning (APA, 1980). As more was learned about SAD, researchers began to collect more representative samples of socially anxious individuals using more reliable and valid psychological measures.

A variety of self-report measures have been used to select participants for high and low levels of social anxiety in analog studies using undergraduate participants. For example, the Social Phobia and Anxiety Inventory (SPAI; Turner, Beidel, Dancu,

& Stanley, 1989) is a self-report measure that assesses the severity of social anxiety symptoms across a number of social and performance situations and has been used for participant selection in a number of analog studies of social skills (Baker & Edelmann, 2002; Strahan & Conger, 1998). The Fear of Negative Evaluation Scale (FNE; Watson & Friend, 1969) has also been used in analog studies of social skills (Thompson & Rapee, 2002; Wenzel et al., 2005). The Social Avoidance and Distress Scale (Watson & Friend, 1969) is another measure that researchers have chosen for this purpose (Meleshko & Alden, 1993; Papsdorf & Alden, 1998). Information about the psychometric properties of these/related and other self-report measures of social anxiety can be found in **Chapter 14**.

Most of the recent studies examining social skills among socially anxious individuals use clinical samples diagnosed with structured clinical interviews. Both the Anxiety Disorders Inventory Schedule for DSM-IV (ADIS-IV; Brown, DiNardo, & Barlow, 1994) and Structured Clinical Interview for DSM-IV (SCID; First, Gibbon, Spitzer, & Williams, 1997) are commonly used (Beidel et al., 2010; Voncken & Bögels, 2008). Information about the good psychometric properties of these interviews can be found in **Chapter 13**.

Structured interviews allow investigators to better report which subtypes of SAD are being examined, which is helpful, given the heterogeneity of individuals with the disorder. A few studies have examined the social performance of different subgroups of individuals with SAD: individuals with nongeneralized SAD; individuals with generalized SAD; and individuals with generalized SAD comorbid with avoidant personality disorder (APD) (Beidel et al., 2010; Boone et al., 1999; Herbert et al., 1992; Tran & Chambless, 1995; Turner et al., 1992). Although participants with SAD and APD report more anxiety than those with only SAD, studies generally have found that social performance does not differ between SAD groups and SAD with APD groups (Boone et al., 1999; Herbert et al., 1992; Tran & Chambless, 1995; Turner et al., 1992). Additionally, findings generally support the notion that those with generalized SAD display higher levels of social behavior impairment than those with nongeneralized SAD (Beidel et al., 2010; Boone et al., 1999; Heimberg et al., 1990).

Structured interviews have the added benefit of allowing investigators to identify and report any comorbid disorders. Individuals with SAD and comorbid disorders are typically retained in studies examining social skills to maximize external validity. However, the presence of comorbid conditions provides an alternative explanation when differences in social performance emerge between individuals with generalized SAD and other groups (i.e., normal controls, individuals with the nongeneralized subtype of SAD). For example, generalized SAD is commonly comorbid with depression (Mannuzza, Schneier, Chapman, & Liebowitz, 1995), which has also been associated with social skills deficits (Segrin, 2000), raising the possibility that performance differences are a function of group differences in depression rather than a feature of generalized SAD. Exceptions include studies by Beidel et al. (2010) and Rapee and Lim (1992) that did control for depression by excluding individuals with SAD and comorbid depression from analyses. Beidel et al. (2010) found performance deficits for individuals with SAD relative to controls, while Rapee and Lim (1992) did not.

With regard to comorbidity, although Axis I comorbidity is often reported, Axis II comorbidity typically has not been reported beyond the occasional study which reports

upon the presence or absence of APD (for an exception, see Beidel et al., 2010). As with depression, Axis II pathology could account for some of the differences between individuals with generalized SAD and other groups in terms of social performance, but this issue has not been examined.

Comparison Groups

As previously stated, the most common approach to examining social skills has been to compare individuals high versus low in social anxiety or individuals with SAD versus normal controls with no diagnosis. When studies compare a clinical group to a nonclinical group, they are unable to speak to the issue of whether group differences are attributable to social anxiety or something else (e.g., comorbidity, general impairment resulting from any psychopathology). Relatively few studies have included a clinical comparison group in addition to a group of individuals diagnosed with SAD. When these studies have been conducted, the most common clinical comparison group has consisted of a mixture of individuals with an anxiety disorder other than SAD. In a study by Baker and Edelmann (2002), a generalized SAD group, "other anxiety disorder" group, and a nonclinical group were assessed during a conversation. The nonclinical group displayed greater use of specific behavioral skills than either the "other anxiety group" or the SAD group. The "other anxiety" group was also rated as more globally adequate than the SAD group. Stopa and Clark (1993) compared individuals with SAD, individuals with an anxiety disorder other than SAD, and normal controls on a conversation task. The SAD group engaged in more negative social behaviors (e.g., left long gaps in the conversation) than the two other groups. The SAD group also engaged in fewer positive behaviors (e.g., asked interesting questions) than the control group. Last, using two conversation tasks, Fydrich et al. (1998) found that the participants with SAD performed more poorly than the participants with another anxiety disorder or no diagnosis; these latter two groups did not differ from each other with regard to total social skills.

Even fewer studies have included a clinical comparison group consisting of individuals with diagnoses other than anxiety disorders. Norton and Hope (2001a) included a group with a primary diagnosis of SAD, a group with a primary diagnosis of dysthymia, and a nonclinical control group. Observers rated the individuals with SAD as performing more poorly than the individuals in the dysthymia group, and the dysthymia group performed worse than the nonclinical group. Scharfstein, Beidel, Sims, and Finnell (2011) compared children with SAD, children with Asperger's Disorder (AD), and typically developing children on a series of five brief structured role-plays with peers. Observers rated the global social skills of the SAD group as poorer than those of either of the other two groups, which did not differ from each other. The same pattern of results was found for the more specific variables of appropriateness of affect and skill maintaining the conversation. The control group was also rated as performing better than the SAD group with regard to the specific behaviors of latency to talk, number of words spoken, response appropriateness, and vocal inflection; the AD group did not differ from either the SAD or control group on these variables. Overall, this small group of studies suggests that, in general, individuals with other

disorders display deficient social behaviors; however, these differences are not as great as those displayed by individuals with SAD.

Tasks used to Elicit Samples of Social Behavior in the Lab

To be effective, our behavior must vary depending upon our relationship to the people with whom we are interacting (e.g., strangers, parents, friends, children, bosses) and the nature of the social situation (e.g., speech, job interview, conversation with a romantic partner, meeting a new person, party). Studies examining the social behavior of socially anxious individuals have primarily employed variations on two different types of tasks: impromptu public speaking to a small audience and interacting with a novel person or persons during a conversation. These two tasks are thought to pose somewhat different challenges to socially anxious individuals. Specifically, speeches are primarily unidirectional while conversations are bidirectional (Thompson & Rapee, 2002). Speeches typically involve the goal of conveying information about a particular topic and do not require as much responsiveness to the reactions of others as conversations.

When asked to give a speech in the lab, participants are often provided with a topic or are asked to pick from a list of topics, such as "talk about your favorite vacation" or "talk about your favorite pet." They are then given a few minutes to prepare before talking to an audience composed of a small number of confederates. The composition of the audience (e.g., with regard to gender) and speech duration have been mixed across studies. For example, Norton and Hope (2001a) asked participants to give a 4-min speech in front of a three-person audience composed of men and women. As another example, Beidel et al. (2010) asked participants to give a 10-min impromptu speech to a three-person audience but did not specify the gender of the audience members.

Conversations typically involve having the socially anxious individual talk to a novel person. Instructions such as "get to know this person you just met at a party" or "you are meeting a new neighbor" are often given (Beidel et al., 2010). The burden of the conversation typically falls to the participant. Confederates are often trained to respond in a specific, neutral manner and to not take the lead in the conversation unless there is a specified amount of silence (Baker & Edelmann, 2002; Boone et al., 1999). In some studies, confederates are allowed to respond "naturally" (Norton & Hope, 2001a). The conversations typically last 3–5 min and rarely last more than 10 min. Studies vary in terms of matching the sex of the confederate to the sex of the participant, having separate interactions with male and female confederates, or interacting with more than one confederate at a time.

As previously stated, the overall pattern of the data seems to suggest that socially anxious individuals tend to perform more poorly on both global and specific measures of performance than nonanxious individuals on both of these types of tasks, irrespective of minor variations across studies (Table 17.1). Exceptions to this pattern are available, in which performance was equivalent between socially anxious and control groups (Cartwright-Hatton et al., 2005; Clark & Arkowitz, 1975; Rapee & Lim, 1992; Strahan & Conger, 1998).

Additional iterations on the themes of short impromptu speeches and conversations with *strangers* are certainly possible. However, there is probably a less pressing need

to explore variations on these common themes than to help us to understand the social behavior of socially anxious individuals in the context of *ongoing relationships*, such as with family members, friends, and romantic partners. An exception is a study by Wenzel et al. (2005). Socially anxious participants and their romantic partners were compared to nonanxious control participants and their romantic partners. The dyads were asked to discuss each of the following topics in separate 10-min intervals: the events of their day, a negative aspect of their relationship, and a positive aspect of their relationship. Overall, the socially anxious participants displayed fewer positive behaviors than the control group. The socially anxious participants also displayed more negative behaviors during the discussion about a negative aspect of their relationship than the control group. The implication is that socially anxious individuals may behave differently than others within the context of their ongoing relationships, but there is a dearth of research that examines this issue with methodologies other than self-report.

Nevertheless, these findings dovetail with some of the literature on close relationships and social anxiety, which describes socially anxious individuals as having problems within their intimate relationships. For example, socially anxious individuals report that they are less able to express strong emotion or assert themselves, are avoidant of conflict, and have an overreliance on others (Davila & Beck, 2002).

Moreover, individuals with SAD may elicit negative reactions from strangers and individuals within their social support system due to poor social behavior. In subtle ways, socially anxious individuals may "pull" negative reactions from other individuals (Alden & Taylor, 2004; Heerey & Kring, 2007; see also **Chapter 16**). In a "getting acquainted" role-play, individuals were less likely to want further interactions with socially anxious individuals than with nonanxious individuals (Meleshko & Alden, 1993; Papsdorf & Alden, 1998). Even in an unstructured social situation, with no instructions and with no knowledge by either party that they were being observed, socially anxious individuals were rated as less competent by their conversation partners than nonsocially anxious individuals (Segrin & Kinney, 1995). Interestingly, in the same study, objective observers were unable to differentiate the social skills of anxious and nonanxious participants, suggesting that the partners may have focused on subtle aspects of the target participant's behavior that were not assessed by the rating scales used by the observers.

Alden and Bieling (1998) demonstrated that socially anxious individuals did not differ from their nonanxious counterparts in terms of how much they were liked by their conversation partner when all participants were led to expect a positive social outcome from the conversation. However, socially anxious participants were less well liked than nonanxious participants when all participants expected a negative social outcome from the conversation. Unlike nonanxious participants, socially anxious participants engaged in safety behaviors (i.e., selecting nonrevealing conversation topics) under conditions of social threat, and these safety behaviors had the unintended effect of making them less likable. Such results suggest that socially anxious individuals are capable of appropriate social behavior but may instead engage in self-protective behaviors that undermine their performance when they appraise the situation to be threatening.

Once others' negative reactions are detected, the socially anxious individual may react with more negative social behaviors—creating an interpersonal cycle of negative

behaviors. Following an initial negative experience during a conversation with a socially anxious person, people may be less motivated to pursue additional conversations in the future (Alden & Bieling, 1998). Such disengagement would reinforce socially anxious individuals' negative views of themselves and expectations for negative outcomes from future social interactions. For more on an interpersonal perspective on social anxiety, see **Chapter 8**.

Assessment Measures Used to Determine the Quality of Social Behavior

For the behavioral assessment tests conducted to date, observers rate participants' performances using instruments that measure molecular (e.g., length of speech, eye contact, hand movements) and/or molar (i.e., the overall global impression) behaviors. In general, studies have employed a relatively equal mix of molar and molecular measures, although some studies may focus on one over the other depending on the research question. In a number of studies (Beidel et al., 2010; Norton & Hope, 2001b; Wenzel et al., 2005), raters used an existing assessment tool to rate participants' performance. For example, Rapee and Lim (1992) created a scale to measure specific and global behaviors that are important to public speaking; a self-rating version and observer-rating version exist and have been used in a number of studies (Rapee & Abbott, 2006; Rapee & Hayman, 1996; Rodebaugh & Chambless, 2002). Wenzel et al. (2005) used the CST (Floyd & Markman, 1984) to assess the extent to which interactions were characterized by positive and negative verbal and nonverbal behaviors. Researchers have also had raters assess a specific set of molecular behaviors (e.g., gestures, smiles, eye contact) of their own choosing (Baker & Edelmann, 2002; Wenzel et al., 2005). Researchers have also had raters provide global (i.e., molar) impression ratings of their own creation (Beidel et al., 2010; Wenzel et al., 2005). What seems clear is that there is no "gold standard" for rating the quality of performance during a social task.

Who serves as a rater and how that rater is trained also varies considerably among studies. At one extreme, raters are highly trained in an established coding system and training is ongoing to reduce bias and drift (Wenzel et al., 2005). At the other extreme, raters receive minimal training in order to maximize external validity (Baker & Edelmann, 2002). Most studies report reasonable inter-rater reliability and ensure that raters are blind to the group status of the participant.

Participants are often asked to provide ratings of their own performance after behavioral tasks. For example, Cartwright-Hatton et al. (2005) had children complete a questionnaire asking about their micro (i.e., molecular) behaviors (e.g., clear voice, etc.) and overall impression of their performance after completing a short conversation. Voncken and Bögels (2008) asked participants, after a conversation role-play, to complete a questionnaire measuring behavioral components (e.g., eye contact, fidgeting, listening to partner, showing interest, etc.), but to do so as the participant thought the conversation partner would complete it. These ratings are often compared to the ratings made by objective observers, and the discrepancy between these two sets of ratings is often examined.

The variety of tasks and assessment measures used creates problems reconciling discrepancies across studies. This problem is compounded by the fact that there is

no widely accepted agreement on a definition of social skills, making it unclear what exactly we should be looking at when we are trying to assess them.

This review has focused on observation of behavior to understand the adequacy of social behavior among socially anxious individuals. However, other forms of assessment for social skills exist, all of which possess advantages and disadvantages. Friends, roommates, or family members can be interviewed or asked to fill out assessment measures in reference to the participant to gain additional information about social skills that may not appear in the office of the clinician or the lab of the researcher. Clinical interviews and self-report measures are also available (Nangle, Hansen, Erdley, & Norton, 2010). Ideally, a multidimensional approach would provide the best overall clinical understanding of the adequacy of social behavior of a particular individual with SAD.

Treating Social Anxiety by Targeting Social Skills

Techniques commonly used in social skills training (SST) include education, therapist modeling, behavioral rehearsal, corrective feedback, social reinforcement, and homework assignments. Early studies of SST as a stand-alone treatment for social anxiety suggested its efficacy (Mersch, Emmelkamp, Bögels, & Van der Sleen, 1989; Stravynsky, Marks, & Yule, 1982; Wlazlo, Schroeder-Hartwig, Hand, Kaiser, & Münchau, 1990), although these studies have a number of methodological limitations that prevent strong conclusions from being drawn (see Heimberg & Juster, 1995, for a detailed critique). More recent studies have tended to include SST as part of a multicomponent cognitive behavioral treatment package rather than a stand-alone treatment. These combined treatments have demonstrated efficacy (Herbert et al., 2005; Turner, Beidel, & Cooley-Quille, 1995; Turner, Beidel, Cooley, Woody, & Messer, 1994; van Dam-Baggen, & Kraaimaat, 2000).

The rationale for SST in the treatment of SAD is typically grounded in the notion that the intervention corrects underlying social skills deficits. However, the mechanism of change in SST may be due to other psychological processes such as increased self-efficacy, anxiety reduction through inherent *exposure*, or correcting dysfunctional beliefs (e.g., about one's own social skills; about the likelihood of feared outcomes) (Bögels & Voncken, 2008; Herbert et al., 2005). Consequently, treatment effects as a result of SST cannot be assumed to reveal etiological significance. Furthermore, no study has yet examined mechanisms or mediators of change with regard to SST.

Conclusions and Future Directions

Although socially anxious individuals tend to perform more poorly than nonanxious individuals in laboratory situations involving speeches or interactions with strangers, many socially anxious individuals perform within the normal range on these tasks. There is likely heterogeneity with regard to social skills within the population of individuals with SAD, with some individuals having actual deficits but others with strong skills. This chapter encourages researchers to carefully consider issues related

to defining social skills, sample selection, comparison groups employed, the nature of the tasks used, and choice of assessment measures when designing studies. Studies that assess social behavior with familiar or intimate others are especially lacking. Existing treatment outcome studies that include SST do little to illuminate the role of social skills deficits in the etiology and maintenance of social anxiety. Future studies that examine mechanisms or mediators of change will make a greater contribution to the theoretical arguments behind the use of SST in the treatment of social anxiety.

References

Alden, L. E., & Bieling, P. (1998). Interpersonal consequences of the pursuit of safety. *Behaviour Research and Therapy, 36*(1), 53–64. doi:10.1016/S0005-7967(97)00072-7

Alden, L. E., & Mellings, T. M. B. (2004). Generalized social phobia and social judgments: The salience of self- and partner-information. *Journal of Anxiety Disorders, 18*(2), 143–157. doi:http://dx.doi.org/10.1016/S0887-6185(02)00244-X

Alden, L. E., & Taylor, C. T. (2004). Interpersonal processes in social phobia. *Clinical Psychology Review, 24*(7), 857–882. doi:http://dx.doi.org/10.1016/j.cpr.2004.07.006

Alden, L. E., & Wallace, S. T. (1995). Social phobia and social appraisal in successful and unsuccessful social interactions. *Behaviour Research and Therapy, 33*(5), 497–505. doi:http://dx.doi.org/10.1016/0005-7967(94)00088-2

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

Arkowitz, H., Lichtenstein, E., McGovern, K., & Hines, P. (1975). The behavioral assessment of social competence in males. *Behavior Therapy, 6*(1), 3–13. doi:http://dx.doi.org/10.1016/S0005-7894(75)80056-6

Baker, S. R., & Edelmann, R. J. (2002). Is social phobia related to lack of social skills? Duration of skill-related behaviours and ratings of behavioural adequacy. *British Journal of Clinical Psychology, 41*(3), 243–257. doi:http://dx.doi.org/10.1348/014466502760379118

Beidel, D. C., Rao, P. A., Scharfstein, L., Wong, N., & Alfano, C. A. (2010). Social skills and social phobia: An investigation of DSM-IV subtypes. *Behaviour Research and Therapy, 48*(10), 992–1001. doi:http://dx.doi.org/10.1016/j.brat.2010.06.005

Beidel, D. C., Turner, S. M., Young, B. J., Ammerman, R. T., Sallee, F. R., & Crosby, L. (2007). Psychopathology of adolescent social phobia. *Journal of Psychopathology and Behavioral Assessment, 29*(1), 47–54. doi:http://dx.doi.org/10.1007/s10862-006-9021-1

Bögels, S. M., Rijsemus, W., & De Jong, P. J. (2002). Self-focused attention and social anxiety: The effects of experimentally heightened self-awareness on fear, blushing, cognitions, and social skills. *Cognitive Therapy and Research, 26*(4), 461–472. doi:http://dx.doi.org/10.1023/A:1016275700203

Bögels, S. M., & Voncken, M. (2008). Social skills training versus cognitive therapy for social anxiety disorder characterized by fear of blushing, trembling, or sweating. *International Journal of Cognitive Therapy, 1*(2), 138–150. doi:http://dx.doi.org/10.1521/ijct.2008.1.2.138

Boone, M. L., McNeil, D. W., Masia, C. L., Turk, C. L., Carter, L. E., Ries, B. J., & Lewin, M. R. (1999). Multimodal comparisons of social phobia subtypes and avoidant personality disorder. *Journal of Anxiety Disorders, 13*(3), 271–292. doi:http://dx.doi.org/10.1016/S0887-6185(99)00004-3

Bornstein, M. R., Bellack, A. S., & Hersen, M. (1977). Social-skills training for unassertive children: A multiple-baseline analysis. *Journal of Applied Behavior Analysis, 10*(2), 183–195. doi:http://dx.doi.org/10.1901/jaba.1977.10-183

Brown, T. A., DiNardo, P. A., & Barlow, D. H. (1994). *Anxiety disorders interview schedule for DSM-IV (ADIS-IV)*. New York, NY: Guilford Press.

Cartwright-Hatton, S., Hodges, L., & Porter, J. (2003). Social anxiety in childhood: The relationship with self and observer rated social skills. *Journal of Child Psychology and Psychiatry, 44*(5), 737–742. doi:http://dx.doi.org/10.1111/1469-7610.00159

Cartwright-Hatton, S., Tschernitz, N., & Gomersall, H. (2005). Social anxiety in children: Social skills deficit, or cognitive distortion? *Behaviour Research and Therapy, 43*(1), 131–141. doi:http://dx.doi.org/10.1016/j.brat.2003.12.003

Clark, D. M., & Wells, A. (1995). A cognitive model of social phobia. In R. G. Heimberg, M. R. Liebowitz, D. A. Hope, & F. R. Schneier (Eds.), *Social phobia: Diagnosis, assessment, and treatment* (pp. 69–93). New York, NY: Guilford Press.

Clark, J. V., & Arkowitz, H. (1975). Social anxiety and self-evaluation of interpersonal performance. *Psychological Reports, 36*(1), 211–221.

Conger, J. C., & Farrell, A. D. (1981). Behavioral components of heterosocial skills. *Behavior Therapy, 12*(1), 41–55. doi:http://dx.doi.org/10.1016/S0005-7894(81)80105-0

Curran, J. P. (1982). A procedure for the assessment of social skills: The simulated social interaction test. In J. P. Curran & P. M. Monti (Eds.), *Social skills training: A practical handbook for assessment and treatment* (pp. 348–373). New York, NY: Guilford Press.

Davila, J., & Beck, J. G. (2002). Is social anxiety associated with impairment in close relationships? A preliminary investigation. *Behavior Therapy, 33*(3), 427–446. doi:http://dx.doi.org/10.1016/S0005-7894(02)80037-5

Del Prette, A., & Del Prette, Z. A. P. (2001). *Psicologia das relações interpessoais: Vivências para o trabalho em grupo*. Petrópolis, Brazil: Vozes.

First, M. W., Gibbon, M., Spitzer, R. L., & Williams, J. B. W. (1997). *Structured clinical interview for DSM-IV axis I disorders (SCID-I), clinical version*. Washington, DC: American Psychiatric Association.

Floyd, F. J., & Markman, H. (1984). An economic observational measure of couples' communication skill. *Journal of Consulting and Clinical Psychology, 52*, 97–103.

Fydrich, T., Chambless, D. L., Perry, K. J., Buergener, F., & Beazley, M. B. (1998). Behavioral assessment of social performance: A rating system for social phobia. *Behaviour Research and Therapy, 36*(10), 995–1010. doi:http://dx.doi.org/10.1016/S0005-7967(98)00069-2

Glasgow, R. E., & Arkowitz, H. (1975). The behavioral assessment of male and female social competence in dyadic heterosexual interactions. *Behavior Therapy, 6*(4), 488–498. doi:http://dx.doi.org/10.1016/S0005-7894(75)80005-0

Heerey, E. A., & Kring, A. M. (2007). Interpersonal consequences of social anxiety. *Journal of Abnormal Psychology, 116*(1), 125–134. doi:http://dx.doi.org/10.1037/0021-843X.116.1.125

Heimberg, R. G., Hope, D. A., Dodge, C. S., & Becker, R. E. (1990). DSM-III—R subtypes of social phobia: Comparison of generalized social phobics and public speaking phobics. *Journal of Nervous and Mental Disease, 178*(3), 172–179. doi:http://dx.doi.org/10.1097/00005053-199003000-00004

Heimberg, R. G., & Juster, H. R. (1995). Cognitive-behavioral treatments: Literature review. In R. G. Heimberg, M. R. Liebowitz, D. A. Hope, & F. R. Schneier (Eds.), *Social phobia: Diagnosis, assessment, and treatment*. (pp. 261–309). New York, NY: Guilford Press.

Herbert, J. D., Gaudiano, B. A., Rheingold, A. A., Myers, V. H., Dalrymple, K., & Nolan, E. M. (2005). Social skills training augments the effectiveness of cognitive behavioral group

therapy for social anxiety disorder. *Behavior Therapy, 36*(2), 125–138. doi:http://dx.doi.org/10.1016/S0005-7894(05)80061-9

Herbert, J. D., Hope, D. A., & Bellack, A. S. (1992). Validity of the distinction between generalized social phobia and avoidant personality disorder. *Journal of Abnormal Psychology, 101*(2), 332–339. doi:http://dx.doi.org/10.1037/0021-843X.101.2.332

Hofmann, S. G., Gerlach, A. L., Wender, A., & Roth, W. T. (1997). Speech disturbances and gaze behavior during public speaking in subtypes of social phobia. *Journal of Anxiety Disorders, 11*(6), 573–585. doi:http://dx.doi.org/10.1016/S0887-6185(97)00040-6

Hopko, D. R., McNeil, D. W., Zvolensky, M. J., & Eifert, G. H. (2001). The relation between anxiety and skill in performance-based anxiety disorders: A behavioral formulation of social phobia. *Behavior Therapy, 32*(1), 185–207. doi:http://dx.doi.org/10.1016/S0005-7894(01)80052-6

Levitan, M. N., Falcone, E. M., Placido, M., Krieger, S., Pinheiro, L., Crippa, J. A., …, & Nardi, A. E. (2012). Public speaking in social phobia: A pilot study of self-ratings and observers' ratings of social skills. *Journal of Clinical Psychology, 68*(4), 397–402. doi:http://dx.doi.org/10.1002/jclp.20868

Mannuzza, S., Schneier, F. R., Chapman, T. F., & Liebowitz, M. R. (1995). Generalized social phobia: Reliability and validity. *Archives of General Psychiatry, 52*(3), 230–237. doi:http://dx.doi.org/10.1001/archpsyc.1995.03950150062011

McFall, R. M. (1982). A review and reformulation of the concept of social skills. *Behavioral Assessment, 4*(1), 1–33. doi:http://dx.doi.org/10.1007/BF01321377

Meleshko, K. G., & Alden, L. E. (1993). Anxiety and self-disclosure: Toward a motivational model. *Journal of Personality and Social Psychology, 64*(6), 1000–1009. doi:http://dx.doi.org/10.1037/0022-3514.64.6.1000

Mersch, P. P., Emmelkamp, P. M., Bögels, S. M., & Van der Sleen, J. (1989). Social phobia: Individual response patterns and the effects of behavioral and cognitive interventions. *Behaviour Research and Therapy, 27*(4), 421–434. doi:http://dx.doi.org/10.1016/0005-7967(89)90013-2

Nangle, D. W., Hansen, D. J., Erdley, C. A., & Norton, P. J. (Eds.) (2010). *Practitioner's guide to empirically based measures of social skills.* New York, NY: Springer. doi:10.1007/978-1-4419-0609-0.

Norton, P. J., & Hope, D. A. (2001a). Kernels of truth or distorted perceptions: Self and observer ratings of social anxiety and performance. *Behavior Therapy, 32*(4), 765–786. doi:http://dx.doi.org/10.1016/S0005-7894(01)80020-4

Norton, P. J., & Hope, D. A. (2001b). Analogue observational methods in the assessment of social functioning in adults. *Psychological Assessment, 13*(1), 59–72. doi:http://dx.doi.org/10.1037/1040-3590.13.1.59

Papsdorf, M., & Alden, L. (1998). Mediators of social rejection in social anxiety: Similarity, self-disclosure, and overt signs of anxiety. *Journal of Research in Personality, 32*(3), 351–369. doi:http://dx.doi.org/10.1006/jrpe.1998.2219

Rapee, R. M., & Abbott, M. J. (2006). Mental representation of observable attributes in people with social phobia. *Journal of Behavior Therapy and Experimental Psychiatry, 37*(2), 113–126. doi:http://dx.doi.org/10.1016/j.jbtep.2005.01.001

Rapee, R. M., & Hayman, K. (1996). The effects of video feedback on the self-evaluation of performance in socially anxious subjects. *Behaviour Research and Therapy, 34*(4), 315–322. doi:http://dx.doi.org/10.1016/0005-7967(96)00003-4

Rapee, R. M., & Heimberg, R. G. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy, 35*(8), 741–756. doi:http://dx.doi.org/10.1016/S0005-7967(97)00022-3

Rapee, R. M., & Lim, L. (1992). Discrepancy between self- and observer ratings of performance in social phobics. *Journal of Abnormal Psychology, 101*(4), 728–731. doi:http://dx.doi.org/10.1037/0021-843X.101.4.728

Rodebaugh, T. L., & Chambless, D. L. (2002). The effects of video feedback on self-perception of performance: A replication and extension. *Cognitive Therapy and Research, 26*(5), 629–644. doi:http://dx.doi.org/10.1023/A:1020357210137

Scharfstein, L. A., Beidel, D. C., Sims, V. K., & Finnell, L. R. (2011). Social skills deficits and vocal characteristics of children with social phobia or Asperger's disorder: A comparative study. *Journal of Abnormal Child Psychology, 39*(6), 865–875. doi:http://dx.doi.org/10.1007/s10802-011-9498-2

Segrin, C. (2000). Social skills deficits associated with depression. *Clinical Psychology Review, 20*(3), 379–403. doi:http://dx.doi.org/10.1016/S0272-7358(98)00104-4

Segrin, C., & Kinney, T. (1995). Social skills deficits among the socially anxious: Rejection from others and loneliness. *Motivation and Emotion, 19*(1), 1–24. doi:http://dx.doi.org/10.1007/BF02260670

Spence, S. H., Donovan, C., & Brechman-Toussaint, M. (1999). Social skills, social outcomes, and cognitive features of childhood social phobia. *Journal of Abnormal Psychology, 108*(2), 211–221. doi:http://dx.doi.org/10.1037/0021-843X.108.2.211

Stopa, L., & Clark, D. M. (1993). Cognitive processes in social phobia. *Behaviour Research and Therapy, 31*(3), 255–267. doi:http://dx.doi.org/10.1016/0005-7967(93)90024-O

Strahan, E., & Conger, A. J. (1998). Social anxiety and its effects on performance and perception. *Journal of Anxiety Disorders, 12*(4), 293–305. doi:http://dx.doi.org/10.1016/S0887-6185(98)00016-4

Stravynski, A., & Amado, D. (2001). Social phobia as a deficit in social skills. In S. G. Hofmann & P. M. DiBartolo (Eds.), *From social anxiety to social phobia: Multiple perspectives* (pp. 107–129). Needham Heights, MA: Allyn & Bacon.

Stravynski, A., Marks, I., & Yule, W. (1982). Social skills problems in neurotic outpatients: Social skills training with and without cognitive modification. *Archives of General Psychiatry, 39*(12), 1378–1385. doi:http://dx.doi.org/10.1001/archpsyc.1982.04290120014003

Thompson, S., & Rapee, R. M. (2002). The effect of situational structure on the social performance of socially anxious and non-anxious participants. *Journal of Behavior Therapy and Experimental Psychiatry, 33*(2), 91–102. doi:http://dx.doi.org/10.1016/S0005-7916(02)00021-6

Tran, G. Q., & Chambless, D. L. (1995). Psychopathology of social phobia: Effects of subtype and avoidant personality disorder. *Journal of Anxiety Disorders, 9*(6), 489–501. doi:http://dx.doi.org/10.1016/0887-6185(95)00027-L

Trower, P., Bryant, B. M., & Argyle, M. (1978). *Social skills and mental health.* London, UK: Methuen.

Turner, S. M., Beidel, D. C., Cooley, M. R., Woody, S. R., & Messer, S. C. (1994). A multicomponent behavioral treatment for social phobia: Social effectiveness therapy. *Behaviour Research and Therapy, 32*(4), 381–390. doi:http://dx.doi.org/10.1016/0005-7967(94)90001-9

Turner, S. M., Beidel, D. C., & Cooley-Quille, M. (1995). Two-year follow-up of social phobics treated with social effectiveness therapy. *Behaviour Research and Therapy, 33*(5), 553–555. doi:http://dx.doi.org/10.1016/0005-7967(94)00086-Y

Turner, S. M., Beidel, D. C., Dancu, C. V., & Stanley, M. A. (1989). An empirically derived inventory to measure social fears and anxiety: The social phobia and anxiety inventory. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 1*(1), 35–40. doi:http://dx.doi.org/10.1037/1040-3590.1.1.35

Turner, S. M., Beidel, D. C., & Townsley, R. M. (1992). Social phobia: A comparison of specific and generalized subtypes and avoidant personality disorder. *Journal of Abnormal Psychology, 101*(2), 326–331. doi:http://dx.doi.org/10.1037/0021-843X.101.2.326

Twentyman, C. T., & McFall, R. M. (1975). Behavioral training of social skills in shy males. *Journal of Consulting and Clinical Psychology, 43*(3), 384–395. doi:http://dx.doi.org/10.1037/h0076743

van Dam-Baggen, R., & Kraaimaat, F. (2000). Group social skills training or cognitive group therapy as the clinical treatment of choice for generalized social phobia? *Journal of Anxiety Disorders, 14*(5), 437–451. doi:http://dx.doi.org/10.1016/S0887-6185(00)00038-4

Voncken, M. J., & Bögels, S. M. (2008). Social performance deficits in social anxiety disorder: Reality during conversation and biased perception during speech. *Journal of Anxiety Disorders, 22*(8), 1384–1392. doi:http://dx.doi.org/10.1016/j.janxdis.2008.02.001

Watson, D., & Friend, R. (1969). Measurement of social-evaluative anxiety. *Journal of Consulting and Clinical Psychology, 33*(4), 448–457. doi:http://dx.doi.org/10.1037/h0027806

Wenzel, A., Graff-Dolezal, J., Macho, M., & Brendle, J. R. (2005). Communication and social skills in socially anxious and nonanxious individuals in the context of romantic relationships. *Behaviour Research and Therapy, 43*(4), 505–519. doi:http://dx.doi.org/10.1016/j.brat.2004.03.010

Wessberg, H. W., Mariotto, M. J., Conger, A. J., Ferrell, A. D., & Conger, J. C. (1979). Ecological validity of role plays for assessing heterosocial anxiety and skill of male college students. *Journal of Consulting and Clinical Psychology, 47*(3), 525–535. doi:http://dx.doi.org/10.1037/0022-006X.47.3.525

Wlazlo, Z., Schroeder-Hartwig, K., Hand, I., Kaiser, G., & Münchau, N. (1990). Exposure in vivo vs. social skills training for social phobia: Long-term outcome and differential effects. *Behaviour Research and Therapy, 28*(3), 181–193. doi:http://dx.doi.org/10.1016/0005-7967(90)90001-Y

V

Broadening the Scope of Social Anxiety Disorder

Areas Warranting Enhanced Empirical Attention

18

Translational Research in Social Anxiety

Summary of Newest Developments and Future Directions

Angela Fang and Stefan G. Hofmann
Boston University, USA

Introduction

In their first request for applications announcement for the Clinical and Translational Research Award Program, the National Institutes of Health (NIH) defined translational research as the following:

> "Translational research includes two areas of translation. One is the process of applying discoveries generated during research in the laboratory, and in preclinical studies, to the development of trials and studies in humans. The second area of translation concerns research aimed at enhancing the adoption of best practices in the community. The term 'science' is meant to encompass the discovery of new knowledge about health and disease prevention, pre-emption, and treatment, as well as methodological research to develop or improve research tools" (NIH, 2005).

Therefore, according to the definition provided by the NIH, translational research involves bridging gaps between the laboratory and the community, or from the bench to the bedside, in order to effect meaningful improvements in health outcomes. This may be accomplished by conducting studies that not only inform existing treatment strategies or enhance treatment outcomes but also by examining novel approaches to diagnosis, prevention, and dissemination. The purpose of the current chapter is threefold: (1) to provide a broad overview of the state of translational research in social anxiety disorder (SAD); (2) to review the latest developments in research on SAD; and (3) to identify relatively neglected areas of research and suggest promising areas for further research. We will discuss findings in the areas of cognitive enhancers, neuroimaging, and behavioral genetics within the context of these three aims.

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

The Role of Cognitive Enhancers in Augmenting CBT With Pharmacotherapy

Recent attention has turned to a model of combination therapy, in which cognitive-behavioral therapy (CBT) is augmented by cognitive enhancers. Cognitive enhancers refer to a class of pharmacologic agents, which have been proposed to impact core learning processes involved in CBT such as extinction learning (Hofmann, 2007b). In contrast, traditional combination strategies for anxiety disorders have used anxiolytic medications (e.g., selective serotonin reuptake inhibitors, monoamine oxidase inhibitors, tricyclic antidepressants, and benzodiazepines) in conjunction with CBT. Studies have revealed that combining these treatment modalities for anxiety disorders does not necessarily offer additional advantages than either modality alone (Hofmann, 2012; Otto, McHugh, & Kantak, 2010). In particular, it appears that among the various anxiety disorders, combination treatment may only confer some benefits in the short term (but not long term) for panic disorder and generalized anxiety disorder, but not for SAD or obsessive–compulsive disorder (Hofmann, Sawyer, Korte, & Smits, 2009). The cognitive enhancers that have been the most extensively studied for enhancing treatment for anxiety disorders include D-cycloserine, cortisol, catecholamines, and yohimbine (Hofmann, 2012). For SAD in particular, D-cycloserine has been shown to have the most empirical support (Hofmann, Smits, Ansaani, Gutner, & Otto, 2011).

D-Cycloserine

The cognitive processes involved in CBT, exposure therapy, and extinction learning share common elements that can be targeted and enhanced with pharmacological agents (Hofmann, 2008). D-Cycloserine (DCS) is a partial agonist of the glycine recognition site of the *N*-methyl-D-aspartate receptor complex. Through its action on this receptor site in the amygdala, DCS has been shown to facilitate extinction learning through the consolidation of new learning during extinction (Davis, Ressler, Rothbaum, & Richardson, 2006). Studies have also shown that DCS has context-specific effects, such that only animals which receive adequate extinction trials benefit in terms of extinction learning from the memory enhancement effects of DCS (compared to animals which receive minimal extinction trials), and DCS appears to facilitate extinction only in the original context in which extinction was learned (Bouton, Vurbic, & Woods, 2008).

To date, three trials have been conducted examining the efficacy of DCS augmentation of CBT for SAD. In the first trial, Hofmann et al. (2006) randomized 27 participants with SAD to receive either 50 mg of DCS or placebo 1 hr before four sessions of a five-session exposure therapy protocol. Results demonstrated that following the acute phase of treatment and at the 1-month follow-up period, DCS-treated patients reported a significantly greater reduction in their social anxiety symptoms compared to placebo-treated patients (Hofmann et al., 2006). Guastella, Richardson, et al. (2008) replicated this finding in 56 participants with SAD using the same study design and treatment manual. Future research should examine dose–response effects of DCS in SAD in facilitating extinction learning. In addition, one area that deserves

further research attention is whether DCS can be administered after an exposure session. This would allow DCS to maximize therapeutic outcomes by being given only after successful extinction trials and avoid the risk of DCS enhancing learning after ineffective exposure experiences.

Cortisol

Cortisol, another cognitive enhancer, has effects on memory consolidation and memory retrieval. Cortisol is a glucocorticoid that is released by the adrenal cortex in response to stress and plays a particularly important role in the hypothalamic–pituitary–adrenal axis. See **Chapter 21** for expanded discussion on cortisol and SAD. Research has shown that cortisol not only facilitates the processes of memory acquisition and consolidation (Roozendaal, 2000) but also inhibits the retrieval of previously acquired information in both animals (de Quervain, Roozendaal, & McGaugh, 1998) and humans (de Quervain, Roozendaal, Nitsch, McGaugh, & Hock, 2000). Indeed, there is evidence that the impairing effects of cortisol on memory retrieval may only apply to individuals with SAD and not to healthy individuals, which suggests that cortisol acts specifically on fear-related memory circuits and may not have general anxiolytic effects (Soravia, de Quervain, & Heinrichs, 2009). These findings have led researchers to examine the potential augmentation effects of cortisol on psychosocial challenge tests as well as exposure therapy for SAD. One study investigated the effects of cortisone administration on subjective fear in response to a socio-evaluative threat task in individuals with SAD (Soravia et al., 2006). Twenty-one adult males with SAD were randomly assigned to receive either cortisone or placebo (25 mg) via oral administration 1 hr prior to exposure to the Trier Social Stress Test (Kirschbaum, Pinke, & Hellhammer, 1993). Results demonstrated that cortisone significantly reduced self-reported ratings of fear during the anticipation, exposure, and recovery phase of the social stressor, compared to individuals who received placebo. These results suggest that administration of low-dose cortisone may be an efficacious treatment for reducing phobic fear.

Future research on the efficacy of cortisol as an augmentation strategy to extinction-based therapies should explore the potential interfering effects of anxiolytic medications, as there is evidence suggesting that they may suppress cortisol or hinder cortisol reactivity (Fries, Hellhammer, & Hellhammer, 2006; Pomara, Willoughby, Sidtis, Cooper, & Greenblatt, 2005). Indeed, it may be that the attenuation of glucocorticoid activity by anxiolytic medications interferes with extinction-based therapies, which explains the lack of additional benefits conferred by combining treatment modalities between CBT and medications (Otto et al., 2010).

Oxytocin

An emerging body of literature has examined the effects of oxytocin on social cognition and behavior in animals and humans. See **Chapter 21** for expanded discussion on oxytocin and SAD. Oxytocin is a nine-amino-acid neuropeptide, which is produced in the magnocellular neurons in the paraventricular and supraoptic nuclei of the hypothalamus and released into the bloodstream via secretion from the posterior pituitary or into the brain via dendritic release from neurons in the hypothalamus.

Oxytocin also travels along axonal projections from the hypothalamus to other parts of the brain, such as the amygdala, hippocampus, striatum, and spinal cord. Recent research suggests that oxytocin's effects on maternal attachment, parturition, and lactation may be associated with peripheral release into the bloodstream, whereas its effects on facilitating social cognition and pro-social behavior may be associated with central release (Meyer-Lindenberg, Domes, Kirsch, & Heinrichs, 2011). The recent discovery that intranasal administration of oxytocin enables acute delivery to the brain (Born et al., 2002) has spawned an abundance of studies investigating the effects of intranasal oxytocin. Existing evidence indicates that intranasal oxytocin impacts core learning and memory processes, as it has been demonstrated that oxytocin enhances emotion recognition (Schulze et al., 2011), selectively impairs semantic implicit memory (Fehm-Wolfsdorf, Born, Voigt, & Fehm, 1984; Heinrichs, Meinlschmidt, Wippich, Ehlert, & Hellhammer, 2004) but improves positive social memories (Guastella, Mitchell, & Mathews, 2008), and attenuates reactivity to emotional stimuli in limbic structures (Domes et al., 2007). Furthermore, due to its implications for psychological disorders associated with social deficits, oxytocin has been studied in clinical populations, such as autism, Asperger's Syndrome, schizophrenia, and SAD (for a review, see MacDonald & MacDonald, 2010).

In patients with SAD, oxytocin appears to improve self-reported ratings of speech performance and speech appearance when administered as an adjunct to exposure therapy but does not reduce overall social anxiety symptom severity, compared to placebo-treated individuals (Guastella, Howard, Dadds, Mitchell, & Carson, 2009). In this study, 25 males with SAD were randomly assigned to receive 24 international units (IUs) of intranasal oxytocin or placebo 1 hr prior to four sessions of exposure therapy in a five-session protocol involving speech exposures. A variety of reasons may explain the lack of reduction in symptom severity among oxytocin-treated individuals. First, the optimal dosing, timing, and frequency of administrations of intranasal oxytocin have yet to be investigated. Second, previous studies suggest that although single-dose administrations of intranasal oxytocin are sensitive to neuroimaging techniques, they are rather insensitive to clinical and behavioral indices of mood and anxiety (Labuschagne et al., 2010). Future research is needed to address these limitations in the literature. In addition, greater research attention should be given to delineating the specificity of oxytocin's effects and how they are distinguished from pharmacologic agents with general anxiolytic properties.

In summary, the available literature on augmentation strategies with cognitive enhancers for SAD points to DCS as the most promising agent, as it facilitates extinction learning that takes place during exposure therapy and may enhance treatment outcome. Oxytocin also represents a relatively new area that deserves greater attention as far as its implications for normalizing dysfunctional social learning processes in SAD.

Neuroimaging Techniques as an Assessment and Treatment Tool

In recent years, neuroimaging studies in clinical and affective neuroscience have greatly contributed to our understanding of neurobiological mechanisms underlying

cognitive processes in anxiety disorders, including SAD (Hofmann, Ellard, & Siegle, 2012; see also **Chapter 4**). Whereas the traditional paradigm in translational medicine has been to use pharmacology to inform the pathophysiology of SAD, neuroscience research has enabled an alternative approach in exploring structural and functional brain activity to inform pathophysiology. In addition to investigating the neural bases of cognitive processes in SAD, researchers have examined neurobiological changes as a result of effective treatment for SAD. As we will describe further below, we recently also examined the potential validity of using fMRI data to predict treatment response in SAD (Doehrmann et al., 2013).

Hyperreactivity of the Amygdala

Previous research has consistently demonstrated hyperreactivity of the amygdala when processing social stimuli among patients with SAD compared to healthy controls (Birbaumer et al., 1998; Phan, Fitzgerald, Nathan, & Tancer, 2006; Stein, Goldin, Sareen, Zorrilla, & Brown, 2002; Straube, Mentzel, & Miltner, 2005; Yoon, Fitzgerald, Angstadt, McCarron, & Phan, 2007). The first study to examine the neural substrates of emotional face processing in SAD found greater activation of the left amygdala when viewing angry or contemptuous faces (Stein et al., 2002). In this study, 15 patients with generalized SAD were matched with 15 healthy control participants with no history of Axis I disorders on age, sex, handedness, and education. While undergoing fMRI, participants viewed a series of facial stimuli reflecting angry, fearful, contemptuous, happy, or nonexpressive facial expressions and were asked to identify the gender of each stimulus. Results showed that SAD patients produced significantly greater blood oxygen-level-dependent (BOLD) responses than healthy controls when viewing harsh (e.g., angry, fearful, contemptuous) versus accepting (e.g., happy) facial expressions. In a separate analysis, SAD patients had a greater BOLD signal change than healthy controls in the left anterior medial temporal lobe region (which includes the amygdala, uncus, and parahippocampal gyrus) for contemptuous and angry faces compared with happy faces.

Stein et al. (2002) demonstrated for the first time an emotion-specific effect of amygdala activation in SAD, which suggests that emotional processing of harsh facial expressions may be neurally distinct from processing accepting or happy facial expressions in patients with SAD. The findings also supported the role of the amygdala in processing emotionally salient cues. However, although the Stein et al. (2002) study demonstrated specificity of the amygdala's response to harsh rather than accepting faces, specificity of amygdala response to different negative facial expressions is less well understood.

Other studies examining the role of the amygdala in emotional processing in SAD have tested the specificity of the emotional stimulus (e.g., emotional valence) in eliciting amygdala hyperreactivity (Evans et al., 2008; Straube et al., 2005; Winston, Strange, O'Doherty, & Dolan, 2002; Yoon et al., 2007). The study by Straube et al. (2005) showed that the amygdala is hyperreactive to positive as well as negative expressions of facial affect. In this study, nine patients with SAD were compared to nine healthy control participants (five females per group) with no history of

psychological or neurologic disorders, and no history of psychotherapy or pharmacotherapy. Participants were asked to view a series of happy, angry, or neutral pictures while undergoing fMRI. Results showed that SAD patients had greater right amygdala activation in response to angry as well as happy faces when compared to controls, which suggested that the amygdala may not only be involved in the processing of emotional threat (e.g., harsh faces) but also the processing of safety signals (e.g., socially accepting information such as happy faces). However, it remains possible that the amygdala is hyperreactive to all emotional cues in SAD. A recent line of evidence suggests that happy faces may be conceived as another form of social threat by patients with SAD because they convey ridicule, and perhaps another opportunity for social rejection (Campbell et al., 2009). One study found that individuals with generalized SAD rated happy faces as less approachable than did healthy participants and that SAD severity negatively correlated with degree of approachability among SAD patients (Campbell et al., 2009). Further research is therefore needed to more closely examine whether happy faces are perceived as safety signals or another form of social threat in individuals with SAD. See **Chapter 20** for further discussion on fear of positive stimuli and positive evaluation in patients with SAD.

As far as emotionally neutral stimuli, evidence suggests that neutral face stimuli are associated with less robust amygdala activation (Campbell et al., 2007; Straube et al., 2005). Although an early finding demonstrated that exposure to neutral faces and aversive odor stimuli were associated with heightened amygdala responses (Birbaumer et al., 1998), later findings have more consistently found that neutral faces are associated with less emotionally arousing reactions and less robust amygdala activation among individuals with SAD, compared to activation with emotional faces (Campbell et al., 2007; Straube et al., 2005). Therefore, converging evidence from behavioral and neuroimaging data support this claim.

Several issues limit the comparability between these early studies of emotional processing in SAD. First, methodological differences make it difficult to draw meaningful comparisons. For example, the studies differed in terms of the nature of the task. Whereas the Stein et al. (2002) study involved making gender discriminations, the Straube et al. (2005) study involved simply viewing the stimuli. Differences in task demands may result in differences in the neural processing of emotional information, such as recruiting greater attentional resources or greater support from higher cortical regions (Freitas-Ferrari et al., 2010). Second, there is an overall lack of distinction between generalized and nongeneralized subtypes of SAD in study samples. Generalized SAD is characterized by having many social fears, usually involving both public performance and social interactional situations, whereas nongeneralized SAD can involve a single, specific, circumscribed, social fear (American Psychiatric Association, 2000). Research indicates that individuals with generalized SAD are a significantly more severe subgroup of SAD patients than nongeneralized SAD patients (Stein, Torgrud, & Walker, 2000). Since the Stein et al. (2002) study included patients with generalized SAD and the Straube et al. study did not distinguish their sample by subtype, it is unclear whether the results of Straube et al. apply more to patients with the generalized than the nongeneralized subtype. This lack of differentiation is also problematic because there is evidence that the magnitude of amygdala activation is positively correlated with SAD symptom severity (Phan et al., 2006), which suggests

that neural responses to emotional processing in SAD do vary as a function of SAD symptom severity.

Frontal Involvement in Emotion Regulation

One aspect of emotional processing that has received extensive empirical attention is emotion regulation, which refers to a set of cognitive control processes that modify the emotions we experience and express (Quirk & Beer, 2006). Emotion dysregulation among individuals with SAD has been identified as a major maintaining factor of the disorder (McClure, Pine, Cicchetti, & Cohen, 2006; Mennin, McLaughlin, & Flanagan, 2009). Therefore, psychological treatments for SAD often incorporate cognitive reappraisal, which is an emotion regulation strategy that aims to reframe and reinterpret an automatic thought to modify negative emotions associated with the thought, thereby reducing emotional distress.

Previous research on the neural correlates of emotion regulation in healthy individuals has found evidence for the involvement of frontal regions in suppression and reappraisal emotion regulation strategies (Banks, Eddy, Angstadt, Nathan, & Phan, 2007; Ochsner & Gross, 2005; Quirk & Beer, 2006). In particular, the orbitofrontal cortex (OFC), dorsolateral prefrontal cortex (DLPFC), dorsal medial prefrontal cortex (DMPFC), and ventrolateral prefrontal cortex (VLPFC) appear to be engaged during reappraisal of emotions compared to maintenance (Banks et al., 2007). Data also indicate that connectivity between these frontal areas and the amygdala is particularly important during emotion regulation (Banks et al., 2007).

Research on the neural mechanisms of emotion regulation in SAD is much more limited. However, available evidence suggests that during cognitive reappraisal of harsh facial expressions, patients with SAD were less likely than healthy participants to recruit frontal areas involved in cognitive reappraisal such as the DLPFC and dorsal ACC (Goldin, Manber, Hakimi, Canli, & Gross, 2009). This study involved 15 individuals with SAD and 17 healthy demographically matched individuals with nine females per group. Participants were presented with a randomized sequence of harsh faces (social threat), violent scenes (physical threat), and neutral scenes (control condition for nonphysical and nonsocial threat) while undergoing fMRI. For each stimulus, they were asked to either "look" at the stimulus without trying to control their emotional response or "regulate" their emotional response by thinking in a way that modified their interpretation of the stimulus and thereby reduce their negative reaction. Results from "look" trials demonstrated a greater neural response in emotion-related brain regions among patients when compared to healthy controls that was specific to social threat stimuli, and which was not observed for physical threat and neutral stimuli. Specifically, SAD patients showed greater BOLD responses in the medial OFC, subgenual ACC, and bilateral parahippocampal gyrus, relative to control participants. Results from "regulate" trials showed that healthy participants produced greater BOLD responses than SAD patients for harsh faces in brain regions involved in cognitive control (e.g., DLPFC, dorsal ACC). Neural activation patterns were similar between groups for physical threat stimuli. These findings suggest that individuals with SAD displayed exaggerated emotional responses and reduced neural

activation in regions implicated in cognitive control (or regulation), specifically for social threat stimuli.

Although this study addressed a major gap in the literature on the neural basis of emotion regulation in SAD, it had some methodological limitations. The most problematic concern is that the study did not exclude participants that received past CBT. The cognitive reappraisal strategy used in the study is a major component of CBT, and differential familiarity with the technique may be a confounding variable. Second, the sample consisted of participants with some additional comorbid psychiatric disorders including generalized anxiety disorder, agoraphobia, and specific phobia. As a result, the sample was likely more severe and more anxious than other studies with pure SAD samples. Psychiatric comorbidities in the sample limit the extent to which we can attribute emotional responses to disorder-specific stimuli and increase the possibility that broad constructs underlying emotional disorders, such as negative affect, may have accounted for the neural response to social threat stimuli rather than SAD itself.

Research on the role of frontal regions in emotion regulation has also found that amygdala activity appears to be inversely associated with DLPFC activation during cognitive reappraisal in both patients with SAD and healthy controls. This suggests that reappraisal involves interactions between prefrontal systems that implement emotion regulation processes and the amygdala, which evaluates the affective aspects of stimuli (Goldin, Manber-Ball, Werner, Heimberg, & Gross, 2009; Ochsner et al., 2004). However, there is evidence for more prefrontal cognitive control regions, including the DLPFC and VLPFC, that are inversely related to amygdala activation in healthy controls during cognitive reappraisal than in individuals with SAD (Goldin, Manber-Ball, et al., 2009). Taken together, the few studies examining the neural correlates of cognitive reappraisal in SAD indicate that patients with SAD may not be recruiting as much frontal support as healthy controls for reappraising negative thoughts and that SAD may be associated with disruptions in the interactions between frontal–amygdala systems that are important for successful emotion regulation.

Amygdala Response Before and After Treatment for SAD

It has been shown that amygdala responses decrease after successful treatment of SAD (Furmark et al., 2002). This study consisted of 18 patients with SAD, of whom 10 were males, who had no other current psychiatric or neurologic disorders and were not long-term users of prescribed medications. All participants were randomized to treatment with citalopram, cognitive-behavioral group therapy, or wait list control. There were three participants with generalized SAD and three participants with nongeneralized SAD in each treatment condition. Participants were matched as closely as possible for each condition by SAD severity, sex, and age. Pre- and posttreatment PET scans using regional cerebral blood flow responses were conducted during a provoked anxiety state (e.g., a public speaking task), in which the participants gave an impromptu speech to a silent audience of six to eight members standing around the scanner bed. Results revealed that improvement in treated groups was correlated with reduced cerebral blood flow responses during a public speaking task in the amygdala and hippocampus. Improvement, or responder status, was defined by reduced scores by

one standard deviation or more from the pretreatment mean value, on at least four out of nine self-report and/or physiological outcome measures of subjective anxiety. In addition, there were significant between group differences, with treated participants and responders showing significantly reduced cerebral blood flow in these regions relative to control participants and nonresponders, respectively. It is noteworthy that both treated groups showed equally reduced SAD symptom severity, as measured by self-report questionnaires. Observed changes in cerebral blood flow responses are therefore unlikely attributable to differences in efficacy between the citalopram and cognitive-behavioral group therapy treatments. Thus, the Furmark et al. (2002) study provided compelling evidence that reduction of SAD symptoms was associated with attenuated neural activity during public speaking in brain regions thought to play a major role in emotional processing in SAD.

Potential Use of Neuroimaging Assessment Data to Guide Treatment Selection

Although the available literature on neural mechanisms underlying SAD is still in its infancy, one promising area of future research is investigating the potential use and predictive validity of fMRI assessment data to guide treatment selection. For example, our research group recruited 39 medication-free patients with generalized SAD to undergo fMRI scans while viewing an emotional face processing paradigm before 12 sessions of group CBT (Doehrmann et al., 2013). The treatment protocol was described in detail elsewhere (Hofmann, 2007a; Hofmann & Otto, 2008). The fMRI paradigm involved viewing a sequence of emotional and nonemotional faces and scenes, which contrasted between angry versus neutral faces, as well as emotional versus neutral scenes. Results showed that there were significant associations between treatment response and pretreatment neural responses in regions of the higher-order visual cortex (e.g., dorsal and ventral areas of the occipital–temporal cortex). Combining the brain measures substantially exceeded predictions based on clinical measures at baseline and accounted for more than 50% of the variance in treatment response, supporting a more idiographic approach in using neuromarkers to improve predictions of success of cognitive-behavioral interventions for SAD. Future research should further examine the reliability and predictive validity of neuroimaging assessment data in predicting outcomes to different treatment modalities. Ultimately, these data may guide treatment selection by identifying individuals who may be more responsive to cognitive-behavioral interventions or pharmacotherapy.

To sum, these studies suggest that neuroimaging might be able to identify biomarkers for treatment response. However, a number of methodological limitations should be noted. Of primary concern is the vast difference in sample characteristics between studies. The systematic study of the effect of SAD subtype on neural response patterns in emotional processing has yet to be investigated, despite evidence from behavioral studies that the generalized subtype is associated with greater severity and functional impairment (Stein et al., 2000). Furthermore, studies are inconsistent in their exclusion criteria, specifically in terms of allowing additional psychiatric Axis I and II comorbidities, as well as previous and current pharmacotherapy or

psychotherapy. Another major caveat of reported findings is that gender interactions are rarely examined, which is often due to small sample sizes that are not adequately powered to explore gender effects. This is particularly problematic in light of existing evidence that there are sex differences in amygdala activation during facial processing (Killgore & Yurgelun-Todd, 2001). Neuroimaging studies on emotional processing in SAD have also largely consisted of young adult samples, and it is unclear the extent to which findings generalize to older adults. A recent meta-analysis found that differences between subjects in age and sex did not significantly alter results within studies because cases and controls were usually well matched (Freitas-Ferrari et al., 2010). However, age or gender differences across studies may affect the sensitivity for detecting differences between SAD patients and control participants (Freitas-Ferrari et al., 2010).

Behavioral Genetics and Endophenotypes of SAD

Although any of the neurobiological mechanisms discussed above with regard to cognitive enhancers and specific neural abnormalities in SAD may represent endophenotypes of SAD, one area deserving of greater research attention is the investigation of potential endophenotypes that link disease-promoting sequence variations in genes (e.g., alleles or single nucleotide polymorphisms) to the phenotypes and symptoms associated with SAD.

Serotonin Transporter (5-HTT) Gene

Examination of variations of the serotonin transporter (5-HTT) gene appears to be a promising area of future research, as serotonergic neurotransmission has been implicated in SAD (Hood et al., 2011; Stein & Vythilingum, 2007). One study examined the effect of a serotonin transporter polymorphism on amygdala activity and symptom severity in individuals with SAD (Furmark et al., 2004). In this study, regional cerebral blood flow in the amygdala was measured in 18 patients (10 females) with SAD during an anxiety-provoking public speaking task. Individuals who had one or two copies of the short allele in the promoter region of 5-HTT showed significantly greater levels of anxiety, and greater right-amygdala activation during the anxiety-provoking task, as compared to individuals who were homozygous for the long allele. This suggests that the extent of amygdala activation in individuals with SAD is dependent on variations of the serotonin transporter gene. Furthermore, research shows that this genetic variant predicts treatment response to SSRIs (Stein, Seedat, & Gelernter, 2006). This study consisted of 32 patients with generalized SAD (23 males) who were randomly assigned to receive paroxetine or fluvoxamine at a maximally tolerated dose (Stein et al., 2006). Results demonstrated that genetic variation in the serotonin transporter gene promoter was associated with response to SSRIs as measured by responder status and symptom severity, from pretreatment to posttreatment. Taken together, these studies suggest that the serotonin system plays an important role in mediating the neural circuitry underlying SAD and provides

a foundation for further exploration in determining whether assessment of 5-HTT genotype could be used to successfully tailor pharmacologic treatments for patients with SAD.

Oxytocin Receptor (OXTR) Gene

Another potentially beneficial area of future research is examining genetic variations of the oxytocin receptor (OXTR) gene. One study showed that a single nucleotide polymorphism (rs53576) in the OXTR gene interacted with social support to reduce stress in humans (Chen et al., 2011). In this study, genetic samples were taken from 194 healthy male participants. Salivary cortisol samples and subjective stress ratings were taken before, during, and after a standardized psychosocial laboratory stress procedure. Participants were randomized to prepare for the stressful task either alone or with social support (e.g., a female friend or partner). Chen and colleagues (2011) found that stress responses differed between the two genotypes and depended on the presence or absence of social support, such that only those with one or two copies of the G allele of the OXTR polymorphism had reduced cortisol responses to stress with social support, compared to those with the same genotype without social support. Furthermore, another study found that a different single nucleotide polymorphism, rs2254298, within the OXTR gene interacted with high levels of early adversity in the parental environment (e.g., maternal history of recurrent major depression) to predict levels of depression, physical anxiety, and social anxiety (Thompson, Parker, Hallmayer, Waugh, & Gotlib, 2011). These results indicated that specific polymorphisms in the OXTR gene may be candidate genes that could serve as endophenotypes of SAD and thereby help to identify individuals at risk for developing SAD.

Brain-Derived Neurotrophic Factor

Brain-derived neurotrophic factor (BDNF) is part of the neurotrophin family of growth factors, which plays a role in the synaptic plasticity of neurons involved in learning and memory in the adult central nervous system (Egan et al., 2003; Hariri et al., 2003). Specifically, animal studies have shown that BDNF impacts fear learning, as genetic and pharmacological inhibition of BDNF signaling significantly reduces long-term potentiation and ultimately leads to diffuse memory impairments (Yu et al., 2009). Evidence also suggests that BDNF mediates the consolidation of extinction memory within the infralimbic medial prefrontal cortex (IL mPFC). Furthermore, research supports the hypothesis that increasing hippocampal BDNF may enhance the efficacy of exposure-based treatments for anxiety (Kobayashi et al., 2005). In this study, 42 outpatients with panic disorder received CBT, and treatment response was found to be correlated with serum BDNF levels, as patients with a poor response to CBT showed significantly lower serum BDNF levels (25.9 ng/ml [SD 8.7]), compared to patients with a favorable response to CBT (33.7 ng/ml [SD 7.5]). Serum BDNF levels have been suggested to be reflective of BDNF levels in the brain (Karege, Schwald, & Cisse, 2002).

Recent research on BDNF has focused on a human-specific single nucleotide polymorphism (Val66Met) in the BDNF gene, which has been identified as a potential endophenotype of psychological disorders such as anxiety and depression (Momose et al., 2002; Sen et al., 2003; Sklar et al., 2002; Ventriglia et al., 2002). This gene variant ($BDNF_{Met}$) also appears to impact hippocampal volume and hippocampal dependent memory (Bueller et al., 2006; Egan et al., 2003; Hariri et al., 2003) and is associated with impaired extinction learning in both mice and humans (Soliman et al., 2010). It is possible that the BDNF Val66Met polymorphism may be associated with greater risk for anxiety disorders such as SAD, as $BDNF_{Met}$ allele carriers in both mice and humans demonstrate impaired extinction learning in a classical fear conditioning and extinction paradigm, compared to non-$BDNF_{Met}$ allele carriers (Soliman et al., 2010). Further exploration is warranted to determine whether BDNF polymorphisms are associated specifically with SAD and whether they may serve as biological predictors of treatment response.

Taken together, the BDNF Val66Met polymorphism appears to impact extinction learning in anxiety disorders, and specific BDNF genotypes may modulate treatment. More research is needed to examine the association between BDNF genotypes, serum BDNF levels, and treatment response in anxiety disorders, as well as elucidate whether the $BDNF_{Met}$ allele serves as a general risk factor for multiple forms of psychopathology, or is specific to mood or anxiety disorders, or SAD in particular.

Conclusions

Translational research aims to garner new knowledge in basic science to inform clinical applications and improve health outcomes. In terms of SAD, translational research in the areas of cognitive enhancers, neuroscience, and behavioral genetics has already begun to clarify the neurobiological underpinnings of SAD and provide therapeutic implications for enhancing treatment outcomes for patients with SAD.

Research on the augmentation of CBT with cognitive enhancers for anxiety disorders has pointed to the efficacy of D-cycloserine in facilitating core learning processes involved in exposure therapy for SAD. Cognitive enhancers offer an advantage over alternative pharmacologic options (e.g., anxiolytic medications) for treating SAD because they target specific cognitive mechanisms known to be involved in the pathophysiology of SAD and represent a neuroscience-informed approach to the treatment of SAD.

Emerging evidence from neuroimaging studies has contributed to a better understanding of the pathophysiology of SAD (see **Chapter 4** for additional review). The basic paradigm used in these studies is a comparison between SAD patient groups and healthy individuals to identify abnormal patterns of functional brain activation. Perhaps the most well-established finding is hyperreactivity of the amygdala during emotional processing among individuals with SAD (Birbaumer et al., 1998; Phan et al., 2006; Stein et al., 2002; Straube et al., 2005). Even more compelling evidence for the role of the amygdala in emotional processing in SAD is that amygdala responses are attenuated in SAD patients during a public speaking task after successful medication treatment or psychotherapy (Furmark et al., 2002). There is also an emerging

body of research implicating the importance of functional connectivity between the amygdala and PFC in the successful downregulation of emotion (Banks et al., 2007). Recent studies also provide preliminary support for the potential use of neuroimaging data to predict treatment response and guide treatment selection in SAD. Thus, neuroimaging findings have advanced our understanding of the nature of emotion dysregulation in SAD and its neural underpinnings, which may be directly translatable to improvements in treatments for SAD by identifying and targeting abnormal processes and neural pathways.

Studies in behavioral genetics have pointed to the role of serotonergic, dopaminergic, and oxytocinergic gene variants in social anxiety vulnerability, as well as SSRI treatment response. Further exploration of these genetic variants, as well as the $BDNF_{Met}$ allele, may have directly translatable clinical applications by informing prevention, diagnosis, and treatment.

Finally, cross-cultural differences in neural correlates of emotional processing in SAD have been relatively neglected in the literature. Race and ethnicity data are seldom reported as demographic characteristics, even though standardized stimulus sets sometimes contain non-Caucasian facial stimuli. Research on the cultural manifestations of SAD has identified *taijinkyofusho* (TKS) as the cultural variant of SAD, which is commonly found in Eastern cultures (Lewis-Fernandez et al., 2010). Recent evidence shows that a majority of US patients with SAD endorsed one of the symptoms of the offensive subtype of TKS, which suggests that TKS may not be as culturally specific as previously thought (Choy, Schneier, Heimberg, Oh, & Liebowitz, 2008). Further research is needed to clarify the effect of culture on emotional processing, and whether neural processes reflect such effects.

References

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

Banks, S. J., Eddy, K. T., Angstadt, M., Nathan, P. J., & Phan, K. L. (2007). Amygdala-frontal connectivity during emotion regulation. *Social Cognitive and Affective Neuroscience, 2*, 303–312. doi:10.1093/scan/nsm029

Birbaumer, N., Grodd, W., Diedrich, O., Klose, U., Erb, M., Lotze, M., . . . Flor, H. (1998). fMRI reveals amygdala activation to human faces in social phobics. *Neuroreport: An International Journal for the Rapid Communication of Research in Neuroscience, 9*, 1223–1226. (Accession No. 00001756-199804200-00048).

Born, J., Lange, T., Kern, W., McGregor, G. P., Bickel, U., & Fehm, H. L. (2002). Sniffing neuropeptides: A transnasal approach to the human brain. *Nature Neuroscience, 5*, 514–516. doi:10.1038/nn0602-849

Bouton, M. E., Vurbic, D., & Woods, A. M. (2008). D-cycloserine facilitates context-specific fear extinction learning. *Neurobiology of Learning and Memory, 90*, 504–510. doi:10.1016/j.nlm.2008.07.003

Bueller, J. A., Aftab, M., Sen, S., Gomez-Hassan, D., Burmeister, M., & Zubieta, J. K. (2006). BDNF Val66Met allele is associated with reduced hippocampal volume in healthy subjects. *Biological Psychiatry, 599*, 812–815. doi:10.1016/j.biopsych.2005.09.022

Campbell, D. W., Sareen, J., Paulus, M. P., Goldin, P. R., Stein, M. B., & Reiss, J. P. (2007). Time-varying amygdala response to emotional faces in generalized social phobia. *Biological Psychiatry, 62*, 455–463. doi:10.1016/j.biopsych.2006.09.017

Campbell, D. W., Sareen, J., Stein, M. B., Kravetsky, L. B., Paulus, M. P., Hassard, S. T., & Reiss, J. P. (2009). Happy but not so approachable: The social judgments of individuals with generalized social phobia. *Depression and Anxiety, 26*, 419–424. doi:10.1002/da.20474

Chen, F. S., Kumsta, R., von Dawans, B., Monakhov, M., Ebstein, R. P., & Heinrichs, M. (2011). Common oxytocin receptor gene (OXTR) polymorphism and social support interact to reduce stress in humans. *Proceedings of the National Academy of Sciences of the United States of America, 108*, 19937–19942. doi:10.1073/pnas.1113079108

Choy, Y., Schneier, F. R., Heimberg, R. G., Oh, K.-S., & Liebowitz, M. R. (2008). Features of the offensive subtype of Taijin-Kyofu-Sho in US and Korean patients with DSM-IV social anxiety disorder. *Depression and Anxiety, 25*, 230–240. doi:10.1002/da.20295

Davis, M., Ressler, K., Rothbaum, B. O., & Richardson, R. (2006). Effects of D-cycloserine on extinction: Translation from preclinical to clinical work. *Biological Psychiatry, 60*, 369–375. doi:10.1016/j.biopsych.2006.03.084

de Quervain, D. J., Roozendaal, B., & McGaugh, J. L. (1998). Stress and glucocorticoids impair retrieval of long-term spatial memory. *Nature, 394*, 787–790. doi:10.1038/29542

de Quervain, D. J., Roozendaal, B., Nitsch, R. M., McGaugh, J. L., & Hock, C. (2000). Acute cortisone administration impairs retrieval of long-term declarative memory in humans. *Nature Neuroscience, 3*, 313–314. doi:10.1038/73873

Doehrmann, O., Ghosh, S. S., Pollo, F. E., Reynolds, G. O., Whitfield-Gabrieli, S., Hofmann, S. G., . . . , Gabrieli, J. D. (2013). Predicting treatment response in social anxiety disorder from functional magnetic resonance imaging. *Archives of General Psychiatry, 70*(1), 87–97. doi:10.1001/2013.jamapsychiatry.5

Domes, G., Heinrichs, M., Glascher, J., Buchel, C., Braus, D. F., & Herpertz, S. C. (2007). Oxytocin attenuates amygdala responses to emotional faces regardless of valence. *Biological Psychiatry, 62*, 1187–1190. doi:10.1016/j.biopsych.2007.03.025

Egan, M. F., Kojima, M., Callicott, J. H., Goldberg, T. E., Kolachana, B. S., Bertolino, A., . . . Weinberger, D. R. (2003). The BDNF val66met polymorphism affects activity-dependent secretion of BDNF and human memory and hippocampal function. *Cell, 112*, 257–269. doi:10.1016/S0092-8674(03)00035-7

Evans, K. C., Wright, C. I., Wedig, M. M., Gold, A. L., Pollack, M. H., & Rauch, S. L. (2008). A functional MRI study of amygdala responses to angry schematic faces in social anxiety disorder. *Depression and Anxiety, 25*, 496–505. doi:10.1002/da.20347

Fehm-Wolfsdorf, G., Born, J., Voigt, K. H., & Fehm, H. L. (1984). Human memory and neurohypophyseal hormones: Opposite effects of vasopressin and oxytocin. *Psychoneuroendocrinology, 9*, 285–292. doi:10.1016/0306-4530(84)90007-6

Freitas-Ferrari, M. C., Hallak, J. E. C., Trzesniak, C., Filho, A. S., Machado-de-Sousa, J. P., Chagas, M. H. N., . . . Crippa, J. A. (2010). Neuroimaging in social anxiety disorder: A systematic review of the literature. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 34*, 565–580. doi:10.1016/j.pnpbp.2010.02.028

Fries, E., Hellhammer, D. H., & Hellhammer, J. (2006). Attenuation of the hypothalamic-pituitary-adrenal axis responsivity to the Trier Social Stress Test by the benzodiazepine alprazolam. *Psychoneuroendocrinology, 31*, 1278–1288. doi:10.1016/j.psyneuen.2006.09.009

Furmark, T., Tillfors, M., Garpenstrand, H., Marteinsdottir, I., Langstrom, B., Oreland, L., & Fredrikson, M. (2004). Serotonin transporter polymorphism related to amygdala excitability and symptom severity in patients with social phobia. *Neuroscience Letters, 362*, 189–192. doi:10.1016/j.neulet.2004.02.070

Furmark, T., Tillfors, M., Marteinsdottir, I., Fischer, H., Pissiota, A., Langstrom, B., & Fredrikson, M. (2002). Common changes in cerebral blood flow in patients with social phobia treated with citalopram or cognitive-behavioral therapy. *Archives of General Psychiatry, 59*, 425–433. doi:10.1001/archpsyc.59.5.425

Goldin, P. R., Manber, T., Hakimi, S., Canli, T., & Gross, J. J. (2009). Neural bases of social anxiety disorder: Emotional reactivity and cognitive regulation during social and physical threat. *Archives of General Psychiatry, 66*, 170–180. doi:10.1001/archgenpsychiatry.2008.525

Goldin, P. R., Manber-Ball, T., Werner, K., Heimberg, R., & Gross, J. J. (2009). Neural mechanisms of cognitive reappraisal of negative self-beliefs in social anxiety disorder. *Biological Psychiatry, 66*, 1091–1099. doi:10.1016/j.biopsych.2009.07.014

Guastella, A. J., Howard, A. L., Dadds, M. R., Mitchell, P., & Carson, D. S. (2009). A randomized controlled trial of intranasal oxytocin as an adjunct to exposure therapy for social anxiety disorder. *Psychoneuroendocrinology, 34*, 917–923. doi:10.1016/j.psyneuen.2009.01.005

Guastella, A. J., Mitchell, P. B., & Mathews, F. (2008). Oxytocin enhances the encoding of positive social memories in humans. *Biological Psychiatry, 64*, 256–258. doi:10.1016/j.biopsych.2008.02.008

Guastella, A. J., Richardson, R., Lovibond, P. F., Rapee, R. M., Gaston, J. E., Mitchell, P., & Dadds, M. R. (2008). A randomized controlled trial of d-cycloserine enhancement of exposure therapy for social anxiety disorder. *Biological Psychiatry, 63*, 544–549. doi:10.1016/j.biopsych.2007.11.011

Hariri, A. R., Goldberg, T. E., Mattay, V. S., Kolachana, B. S., Callicott, J. H., Egan, M. F., & Weinberger, D. R. (2003). Brain-derived neurotrophic factor val66met polymorphism affects human memory-related hippocampal activity and predicts memory performance. *Journal of Neuroscience, 23*, 6690–6694. Retrieved from http://www.jneurosci.org/content/23/17/6690.long

Heinrichs, M., Meinlschmidt, G., Wippich, W., Ehlert, U., & Hellhammer, D. H. (2004). Selective amnesic effects of oxytocin on human memory. *Physiology and Behavior, 83*, 31–38. doi:10.1016/j.physbeh.2004.07.020

Hofmann, S. G. (2007a). Cognitive factors that maintain social anxiety disorder: A comprehensive model and its treatment implications. *Cognitive Behaviour Therapy, 36*, 195–209. doi:10.1080/16506070701421313

Hofmann, S. G. (2007b). Enhancing exposure-based therapy from a translational research perspective. *Behaviour Research and Therapy, 45*, 1987–2001. doi:10.1016/j.brat.2007.06.006

Hofmann, S. G. (2008). Cognitive processes during fear acquisition and extinction in animals and humans: Implications for exposure therapy of anxiety disorders. *Clinical Psychology Review, 28*, 199–210. doi:10.1016/j.cpr.2007.04.009

Hofmann, S. G. (2012). *Psychobiological approaches for anxiety disorders: Treatment combination strategies.* Oxford, UK: John Wiley & Sons.

Hofmann, S. G., Ellard, K. K., & Siegle, G. J. (2012). Neurobiological correlates of cognitions in fear and anxiety: A cognitive-neurobiological information processing model. *Cognition and Emotion, 26*, 282–299. doi:10.1080/02699931.2011.579414

Hofmann, S. G., Meuret, A. E., Smits, J. A. J., Simon, N. M., Pollack, M. H., Eisenmenger, K., & Otto, M. W. (2006). Augmentation of exposure therapy with d-cycloserine for social anxiety disorder. *Archives of General Psychiatry, 63*, 298–304. doi:10.1001/archpsyc.63.3.298

Hofmann, S. G., & Otto, M. W. (2008). *Cognitive-behavior therapy of social anxiety disorder: Evidence-based and disorder specific treatment techniques.* New York, NY: Routledge.

Hofmann, S. G., Sawyer, A. T., Korte, K. J., & Smits, J. A. (2009). Is it beneficial to add pharmacotherapy to cognitive-behavioral therapy when treating anxiety disorders? A meta-analytic review. *International Journal of Cognitive Therapy, 2*, 160–175. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2732196/

Hofmann, S. G., Smits, J. A. J., Asnaani, A., Gutner, C. A., & Otto, M. W. (2011). Cognitive enhancers for anxiety disorders. *Pharmacology, Biochemistry, and Behavior, 99*, 275–284. doi:10.1016/j.pbb.2010.11.020

Hood, S. D., Potokar, J. P., Davies, S. J., Hince, D. A., Morris, K., Seddon, K. M., . . . Argyropoulos, S. V. (2011). Dopaminergic challenges in social anxiety disorder: Evidence for dopamine D3 desensitisation following successful treatment with serotonergic antidepressants. *Journal of Psychopharmacology, 24*, 709–716. doi:10.1177/0269881108098144

Karege, F., Schwald, M., & Cisse, M. (2002). Postnatal developmental profile of brain-derived neurotrophic factor in the rat brain and platelets. *Neuroscience Letters, 328*, 261–264. doi:10.1016/S0304-3940(02)00529-3

Killgore, W. D., & Yurgelun-Todd, D. A. (2001). Sex differences in amygdala activation during the perception of facial affect. *NeuroReport, 12*, 2543–2547. (Accession No. 00001756-200108080-00050)

Kirschbaum, C., Pinke, D. H., & Hellhammer, D. H. (1993). The "Trier Social Stress Test" – A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology, 28*, 76–81. doi:10.1159/000119004

Kobayashi, K., Shimizu, E., Hashimoto, K., Mitsumori, M., Koike, K., Okamura, N., . . . , Iyo, M. (2005). Serum brain-derived neurotrophic factor (BDNF) levels in patients with panic disorder: As a biological predictor of response to group cognitive behavioral therapy. *Progress in Neuro-psychopharmacology and Biological Psychiatry, 29*, 658–663. doi:10.1016/j.pnpbp.2005.04.010

Labuschagne, I., Phan, K. L., Wood, A., Angstadt, M., Chua, P., Heinrichs, M., . . . Nathan, P. J. (2010). Oxytocin attenuates amygdala reactivity to fear in generalized social anxiety disorder. *Neuropsychopharmacology, 35*, 2403–2413. doi:10.1038/npp.2010.123

Lewis-Fernandez, R., Hinton, D. E., Laria, A. J., Patterson, E. H., Hofmann, S. G., Craske, M. G., . . . Liao, B. (2010). Culture and the anxiety disorders: Recommendations for DSM-V. *Depression and Anxiety, 27*, 212–229. doi:10.1002/da.20647

MacDonald, K., & MacDonald, T. M. (2010). The peptide that binds: A systematic review of oxytocin and its prosocial effects in humans. *Harvard Review of Psychiatry, 18*, 1–21. doi:10.3109/10673220903523615

McClure, E. B., Pine, D. S., Cicchetti, D., & Cohen, D. J. (2006). Social anxiety and emotion regulation: A model for developmental psychopathology perspectives on anxiety disorders. In D. Cicchetti, & D. J. Cohen (Eds.), *Developmental psychopathology, Vol 3: Risk, disorder, and adaptation* (2nd ed., pp. 470–502). Hoboken, NJ: John Wiley & Sons.

Mennin, D. S., McLaughlin, K. A., & Flanagan, T. J. (2009). Emotion regulation deficits in generalized anxiety disorder, social anxiety disorder, and their co-occurrence. *Journal of Anxiety Disorders, 23*, 866–871. doi:10.1016/j.janxdis.2009.04.006

Meyer-Lindenberg, A., Domes, G., Kirsch, P., & Heinrichs, M. (2011). Oxytocin and vasopressin in the human brain: Social neuropeptides for translational medicine. *Nature Reviews Neuroscience, 12*, 524–538. doi:10.1038/nrn3044

Momose, Y., Murata, M., Kobayashi, K., Tachikawa, M., Nakabayashi, Y., Kanazawa, I., & Toda, T. (2002). Association studies of multiple candidate genes for Parkinson's disease using single nucleotide polymorphisms. *Annals of Neurology, 51*, 133–136. doi:10.1002/ana.10079

National Institutes of Health. (2005). Definitions under Subsection 1 (Research Objectives), Section I (Funding Opportunity Description), Part II (Full Text of Announcement), of

RFA-RF-06-002: Institutional Clinical and Translational Science Award. Retrieved from http://grants.nih.gov/grants/guide/rfa-files/RFA-RM-06-002.html, accessed on May 16, 2012.

Ochsner, K. N., & Gross, J. J. (2005). The cognitive control of emotion. *Trends in Cognitive Sciences, 9*, 242–249. doi:10.1016/j.tics.2005.03.010

Ochsner, K. N., Ray, R. D., Cooper, J. C., Robertson, E. R., Chopra, S., Gabrieli, J. D., & Gross, J. J. (2004). For better or for worse: Neural systems supporting the cognitive down- and up-regulation of negative emotion. *Neuroimage, 23*, 483–499. doi:10.1016/j.neuroimage.2004.06.030

Otto, M. W., McHugh, R. K., & Kantak, K. M. (2010). Combined pharmacotherapy and cognitive-behavioral therapy for anxiety disorders: Medication effects, glucocorticoids, and attenuated treatment outcomes. *Clinical Psychology: Science and Practice, 17*, 91–103. doi:10.1097/YCO.0b013e3280115e52

Phan, K. L., Fitzgerald, D. A., Nathan, P. J., & Tancer, M. E. (2006). Association between amygdala hyperactivity to harsh faces and severity of social anxiety in generalized social phobia. *Biological Psychiatry, 59*, 424–429. doi:10.1016/j.biopsych.2005.08.012

Pomara, N., Willoughby, L. M., Sidtis, J. J., Cooper, T. B., & Greenblatt, D. J. (2005). Cortisol response to diazepam: Its relationship to age, dose, duration of treatment, and presence of generalized anxiety disorder. *Psychopharmacology, 178*, 1–8. doi:10.1007/s00213-004-1974-8

Quirk, G. J., & Beer, J. S. (2006). Prefrontal involvement in the regulation of emotion: Convergence of rat and human studies. *Current Opinion in Neurobiology, 16*, 723–727. doi:10.1016/j.conb.2006.07.004

Roozendaal, B. (2000). 1999 Curt P. Richter Award. Glucocorticoids and the regulation of memory consolidation. *Psychoneuroendocrinology, 25*, 213–238. doi:10.1016/S0306-4530(99)00058-X

Schulze, L., Lischke, A., Greif, J., Herpertz, S. C., Heinrichs, M., & Domes, G. (2011). Oxytocin increases recognition of masked emotional faces. *Psychoneuroendocrinology, 36*, 1378–1382. doi:0.1016/j.psyneuen.2011.03.011

Sen, S., Nesse, R. M., Stoltenberg, S. F., Li, S., Gleiberman, L., Chakravarti, A., . . . Burmeister, M. (2003). A BDNF coding variant is associated with the NEO personality inventory domain neuroticism, a risk factor for depression. *Neuropsychopharmacology, 28*, 397–401. doi:10.1038/sj.npp.1300053

Sklar, P., Gabriel, S. B., McInnis, M. G., Bennett, P., Lim, Y. M., Tsan, G., . . . Lander, S. (2002). Family-based association study of 76 candidate genes in bipolar disorder: BDNF is a potential risk locus. Brain-derived neurotrophic factor. *Molecular Psychiatry, 76*, 579–593. doi:10.1038/sj.mp.4001058

Soliman, F., Glatt, C. E., Bath, K. G., Levita, L., Jones, R. M., Pattwell, S. S., . . . Casey, B. J. (2010). A genetic variant BDNF polymorphism alters extinction learning in both mouse and human. *Science, 327*, 863–866. doi:10.1126/science.1181886

Soravia, L. M., de Quervain, D. J. F., & Heinrichs, M. (2009). Glucocorticoids do not reduce subjective fear in healthy subjects exposed to social stress. *Biological Psychology, 81*, 184–188. doi:10.1016/j.biopsycho.2009.04.001

Soravia, L. M., Heinrichs, M., Aerni, A., Maroni, C., Schelling, G., Ehlert, U., . . . de Quervain, D.J. (2006). Glucocorticoids reduce phobic fear in humans. *Proceedings of the National Academy of Sciences of the United States of America, 103*, 5585–5590. doi:10.1073/pnas.0509184103

Stein, D. J., & Vythilingum, B. (2007). Social anxiety disorder: Psychobiological and evolutionary underpinnings. *CNS Spectrums, 12*, 806–809. doi:10.1016/j.pscychresns.2009.05.004

Stein, M. B., Goldin, P. R., Sareen, J., Zorrilla, L. T. E., & Brown, G. G. (2002). Increased amygdala activation to angry and contemptuous faces in generalized social phobia. *Archives of General Psychiatry, 59*, 1027–1034. doi:10.1001/archpsyc.59.11.1027

Stein, M. B., Seedat, S., & Gelernter, J. (2006). Serotonin transporter gene promoter polymorphism predicts SSRI response in generalized social anxiety disorder. *Psychopharmacology (Berl), 187*, 68–72. doi:10.1007/s00213-006-0349-8

Stein, M. B., Torgrud, L. J., & Walker, J. R. (2000). Social phobia symptoms, subtypes, and severity: Findings from a community survey. *Archives of General Psychiatry, 57*, 1046–1052. doi:10.1001/archpsyc.57.11.1046

Straube, T., Mentzel, H.-J., & Miltner, W. H. R. (2005). Common and distinct brain activation to threat and safety signals in social phobia. *Neuropsychobiology, 52*, 163–168. doi:10.1159/000087987

Thompson, R. J., Parker, K. J., Hallmayer, J. F., Waugh, C. E., & Gotlib, I. H. (2011). Oxytocin receptor gene polymorphism (rs2254298) interacts with familial risk for psychopathology to predict symptoms of depression and anxiety in adolescent girls. *Psychoneuroendocrinology, 36*, 144–147. doi:10.1016/j.psyneuen.2010.07.003

Ventriglia, M., Bocchio Chiavetto, L., Benussi, L., Binetti, G., Zanetti, O., Riva, M. A., & Gennarelli, M. (2002). Association between the BDNF 196 A/G polymorphism and sporadic Alzheimer's disease. *Molecular Psychiatry, 7*, 136–137. doi:10.1038/sj/mp/4000952

Winston, J. S., Strange, B. A., O'Doherty, J., & Dolan, R. J. (2002). Automatic and intentional brain responses during evaluation of trustworthiness of faces. *Nature Neuroscience, 5*, 277–283. doi:10.1038/nn816

Yoon, K. L., Fitzgerald, D. A., Angstadt, M., McCarron, R. A., & Phan, K. L. (2007). Amygdala reactivity to emotional faces at high and low intensity in generalized social phobia: A 4-Tesla functional MRI study. *Psychiatry Research: Neuroimaging, 154*, 93–98. doi:10.1016/j.pscychresns.2006.05.004

Yu, H., Wang, Y., Pattwell, S., Jing, D., Liu, T., Zhang, Y., . . . Chen, Z. Y. (2009). Variant BDNF Val66Met polymorphism affects extinction of conditioned aversive memory. *Journal of Neuroscience, 29*, 4056–4064. doi:10.1523/JNEUROSCI.5539-08.2009

19

Positivity Impairment as a Broad-Based Feature of Social Anxiety

Eva Gilboa-Schechtman, Iris Shachar, and Yair Sahar

Bar-Ilan University, Israel

The hallmark feature of social anxiety disorder (SAD) is a painful and persistent preoccupation with the evaluation of others. This concern prevents socially anxious individuals from actively participating in events they find interesting or inspiring, halts them as they are about to contribute to a conversation, keeps them from taking jobs that are commensurate with their abilities, and stops them from approaching a person with whom they could achieve intimacy. Given these anxieties and avoidances, it is not surprising that socially anxious individuals report high levels of negative affect (NA), and functional impairment in several life areas (Aderka et al., 2012). Unsurprisingly also, SAD is associated with lower well-being (Sherbourne et al., 2010).

The understanding of the full impact of social anxiety (SA) on well-being has been enriched in the past decade by the emerging interest in the study of positivity (Fredrickson, 2001; Seligman, Alex, Joseph, & Boniwell, 2003). Recent developments in the study of affect conceptualize positive and negative affectivity as two distinct psychological constructs (or dimensions) rather than as two ends of an affective continuum (Rafaeli & Revelle, 2006). Above and beyond the influence of NA, positive affect (PA) has been shown to affect psychological well-being (Catalino & Fredrickson, 2011; Lyubomirsky, King, & Diener, 2005; Tugade & Fredrickson, 2004). Specifically, PA was found to strengthen one's psychological resilience (Fredrickson, Tugade, Waugh, & Larkin, 2003), to promote physical health (Boehm & Kubzansky, 2012; Davidson, Mostofsky, & Whang, 2010), to increase longevity (Xu & Roberts, 2010), and to enhance creativity (Rego, Sousa, Marques, & Cunha, 2012).

The tripartite model of anxiety and depression (Clark & Watson, 1991) builds upon the independence of PA and NA in an attempt to explain the pattern of comorbidity between anxiety and depression. According to this model, while anxiety is largely characterized by increased NA and increased physiological arousal, depression is characterized by increased NA and decreased PA. Initially, Watson and Clark (1995) postulated that low PA is unique to depression, but later studies found that SAD, like depression and unlike other anxiety disorders, is also associated with low PA (Brown, Chorpita, &

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

Barlow, 1998; Kotov, Watson, Robles, & Schmidt, 2007; Watson, Clark, & Tellegen, 1988). In fact, low PA has begun to emerge as one of the central features of SAD (Weeks, Heimberg, Rodebaugh, Goldin, & Gross, 2012; Weeks & Howell, 2012).

Given the importance of PA to overall well-being on the one hand, and the strong association between SAD and low PA on the other, the present chapter seeks to clarify and delineate the prevalent, yet ill-defined, concept of "positive impairment" in SA. We propose to address this topic adopting an evolutionary–interpersonal perspective, which provides a theoretical framework to examine the structure of positivity as well as the nature of SA within the context of a functional analysis of human sociality (Bugental, 2000).

From such a perspective, each individual participates in several evolutionarily shaped social structures. Among the most prominent are affiliative relationships (friendship, companionship, intimacy) and hierarchical relationships (authority, social rank, social power). Indeed, the need to affiliate with or belong to a social group is considered one of the central social motives across species, and basic psychological systems are hypothesized to constantly monitor for inclusionary status (Baumeister & Leary, 1995). Similarly, a need to advance in the social hierarchy and to be sensitive to threat to one's status within a group appears to be inherited from our primate ancestors (Sapolsky, 2005). Social exclusion (i.e., ostracism or social rejection) and social submission (e.g., being defeated) threatened one's standing in and belonging to a social group. Such events also decreased one's chances of future social effectiveness and collaboration. In contrast, social acceptance and social ascendance increase one's chances of social flourishing. In the following, we consider cues or events denoting the possibility of social acceptance or social ascendance as "socially positive."

Trower and Gilbert's (1989) evolutionary model suggests that, whereas all individuals utilize both the social rank and the affiliation systems, individuals with SAD tend to over-utilize the social rank system and under-utilize the affiliation system (Trower & Gilbert, 1989; see also **Chapter 2**). In other words, individuals with SAD may tend to view interpersonal situations from a competitive, rather than an affiliative, perspective. Consistent with this perspective, studies have demonstrated that socially anxious individuals perceive themselves as submissive and rate their social status as low and unsatisfactory (Aderka, Weisman, Shahar, & Gilboa-Schechtman, 2009; Oakman, Gifford, & Chlebowsky, 2003; Trower & Gilbert, 1989).

To understand the nature of positive impairment in SA, four questions will be examined. *First*, we consider the scope of positive impairment. As mentioned above, high SA individuals report low levels of PA, particularly following social interactions (Watson, Clark, McIntyre, & Hamaker, 1992). Does decreased PA in SAD occur in other situations geared to elicit PA (e.g., when someone achieves a long-sought-for goal, wins an honor, or admires a beautiful painting)? Although the empirical literature on this question is rather sparse, we consider the implications therein. *Second*, we examine the question of reactivity. Do individuals suffering from SAD tend to under- or over-react to socially positive stimuli or events? In order to address this issue we review how socially anxious individuals attend to, interpret, evaluate, imagine and respond to positive stimuli and events. *Third*, we examine the question of regulation. When a socially positive event occurs, what are the strategies employed by individuals high in SA in an attempt to regulate (prolong, savor, dampen, or suppress) this affective

state? *Fourth*, we explore the question of causation. Does positivity impairment play a causal role in the development or maintenance of SA? Specifically, we ask whether biases in the processing of positive stimuli or events are causally related to the etiology and maintenance of SA.

Scope of Positivity Impairment

An accumulating body of research has identified a negative relationship between SA and global PA, even when severity of depression is statistically controlled (Brown, Silvia, Myin-Germeys, & Kwapil, 2007; Brown et al., 1998; Kashdan & Steger, 2006). A comprehensive meta-analysis conducted by Kashdan (2007) reported a robust and sizable negative correlation between SA and PA. These findings examined positive affectivity as a global and unified construct, commonly measured by the 20-item Positive and Negative Affect Schedule (PANAS; Watson et al., 1988). The PA subscale of the PANAS includes the following varied adjectives: *interested, alert, attentive, excited, enthusiastic, inspired, proud, determined, strong* and *active*. Importantly, although the PANAS is extensively used as a measure of general PA, it has been found to sample primarily high activation positive emotions (Russell & Carroll, 1999), rather than low activation emotions such as *content*, or *satisfied*. In fact, the emotion words used in the PANAS appear to be more closely associated with feelings of agency and dominance than with feelings of affiliation (see Mehrabian, 1997 for a similar argument).

Stimulated by a functional perspective, much of contemporary theorizing highlights the utility of examining *distinct* positive affective states, rather than global positive affectivity (Catalino & Fredrickson, 2011; Shiota, Neufeld, Yeung, Moser, & Perea, 2011). Indeed, there is growing interest in creating a more nuanced understanding of specific positive emotions (Shiota, Campos, Keltner, & Hertenstein, 2004). Recent empirical evidence supports a differentiation between multiple positive emotional states such as happiness/joy, amusement, contentment, pride, nurturant love, and attachment love (Fredrickson & Branigan, 2005; Herring, Burleson, Roberts, & Devine, 2011; Juslin & Laukka, 2003; Shiota et al., 2011). Taken as a whole, these findings support the notion that positive emotions, like negative emotions, involve distinct response signatures.

Perhaps the most intriguing, as well as the most informative, investigation of the SA–PA relationship concerns the emotions of pride, attachment love, and nurturant love/compassion. These positive emotions map onto two previously mentioned systems postulated to govern interpersonal relatedness: one of dominance, social rank or power; and the other of affiliation, reciprocity and intimacy (Alden, Wiggins, & Pincus, 1990; Gilbert, 2000; Trower & Gilbert, 1989).

Despite its theoretical significance, an examination of pride in SA has yet to be undertaken. Indeed, SA has been linked to the more intense and frequent experience of shame (Gilbert, 2000). Pride, considered by some to be a mirror image of shame (Weisfeld & Dillon, 2012), has been conceptualized as a self-conscious emotion that signals the accomplishment of a valued task to members of the group, enabling an individual the possibility to better their social standing within a given social hierarchy (Gilbert & Trower, 2001; Tiedens, Ellsworth, & Mesquita, 2000; Tracy & Robins,

2007a). Pride is cross-culturally recognized and easily distinguished from other similar emotions (e.g., happiness) using nonverbal cues such as facial expressions and bodily postures (Tracy & Robins, 2007b). Given the theoretical postulations regarding the importance of the dominance system for SA on the one hand, and the recent findings regarding the link between pride and other psychopathologies, such as depression, on the other (Gruber, Oveis, Keltner, & Johnson, 2011), it seems particularly important to examine the experience of pride in SA.

There is a similar gap in the literature on SA and the experience of affiliative emotions. Affiliative emotions are known under a variety of names, including the already mentioned affiliative and nurturing love, compassion, empathy, and gratitude. *Attachment love* is hypothesized to address our species' need for nurturance and protection, and has been depicted as the surge of trust and warmth experienced in response to an attachment figure (Griskevicius, Shiota, & Neufeld, 2010; Shaver, Morgan, & Wu, 1996). This emotion is thought to facilitate acceptance of help from others (Shiota, Keltner, & John, 2006). In contrast, *nurturant love* has been postulated to be related to the need to care for the young (Griskevicius et al., 2010; Shiota et al., 2006).

So far, no study has experimentally examined attachment love or nurturant love (or other affiliative emotions such as gratitude, compassion, or empathy) in socially anxious individuals. Existing research does suggest that SA is associated with decreased satisfaction with close relationships. For example, high levels of SA are associated with low perceived intimacy (Weisman, Aderka, Marom, Hermesh, & Gilboa-Schechtman, 2011) and reduced perceived quality of intimate relations (Rodebaugh, 2009; Sparrevohn & Rapee, 2009; Weeks et al., 2012). Understanding the nature of affiliative difficulties may assist in improving socially anxious individuals' satisfaction with, and maintenance of, close relationships.

Clearly, not all positive emotions concern interpersonal relationships. Some arise from humans' intrinsic enjoyment of cognitive complexity (Keltner & Haidt, 1999; Shiota, Keltner, & Mossman, 2007). Surprisingly little research has examined SA individuals' (or individuals suffering from other emotional disorders for that matter) impairment in amusement, awe, or contentment. Research inquiring into the experience of these emotions will help us to understand and delineate the boundaries of positive impairment in SA.

Existing evidence pointing to the relevance of discrete positive emotions in bipolar disorder (Gruber & Johnson, 2009) and depression (Gruber et al., 2011) has already begun to reshape our conceptions of these conditions. Discrete positive emotions may reveal valuable information regarding the underlying mechanisms of SAD. Specifically, a broad decrease in multiple areas of PA may indicate impairment of a general reward-sensitive mechanism, whereas a more focal impairment in several distinct emotions may be indicative of the impact of the (mal)functioning of specific systems (Shiota et al., 2006).

Reactivity to Positive Signals

Heightened reactivity to, and impaired regulation in response to, social threat is widely considered to be at the epicenter of SAD (Clark & Wells, 1995; Gilbert & Trower,

2001; Hofmann, Heinrichs, & Moscovitch, 2004; Rapee & Heimberg, 1997) and many experimental studies have indeed supported this position (Ly & Roelofs, 2009; Oaten, Williams, Jones, & Zadro, 2008; Rapee & Lim, 1992). The term *emotional reactivity* has been variously defined, but it is mostly used according to two meanings: as a response to an emotional perceptual stimulus (henceforth *stimulus reactivity*), or as a response to an emotional event (henceforth *event reactivity*). The former deals with the effect of stimuli such as facial expressions, whereas the latter deals with affective changes experienced, predicted, or remembered, as a result of an event such as social rejection (Nelson & Shankman, 2011). In the following, we first review the literature on reactivity as stimulus reactivity and then review the literature on event reactivity.

Stimulus Reactivity

Theoretical accounts converge in suggesting that misinterpretations of affiliative social signals as threatening are likely to deepen one's distress and contribute to the maintenance of SAD. Yet, facial expressions connoting acceptance and affiliation (such as smiles) are typically used only as control stimuli. Understanding the factors that influence biased or inaccurate interpretations of affiliative signals is needed to form a more complete and accurate model of SAD. Such understanding may also advance a more nuanced conceptualization of the interpersonal and situational factors that influence the accurate perceptions of emotional states of others. Since smiles are the most prevalent stimuli used in stimulus-reactivity research, and since they also occur naturally outside the laboratory, we focus our review on the processing of these expressions.

Attention

Studies using various attentional tasks document differential processing of smiling facial expressions in SA, compared to nonanxious individuals. Using the dot probe task with emotional facial expressions, SA was found to be associated with a tendency to direct attention *away* from smiling faces (Chen, Ehlers, Clark, & Mansell, 2002; Pishyar, Harris, & Menzies, 2004). Using the visual search paradigm with arrays of smiling, disgusted, and angry faces (face-in-the-crowd paradigm) Gilboa-Schechtman, Foa, and Amir (1999) found that individuals with SAD were more distracted than nonanxious controls by both angry and smiling crowds. Using eye-tracking methodology, Gamble and Rapee (2010) examined attention to pairs of emotional (smiling or angry) and neutral faces in individuals with SAD and in controls. They found that, while controls exhibited higher vigilance for smiling than for angry expressions, SAD individuals were equally vigilant toward emotional (i.e., both smiling and angry) faces than to neutral faces. Recently, Chen, Clarke, MacLeod, and Guastella (2012) also used eye-tracking to examine engagement and disengagement from smiling and angry facial expressions. They found that, as compared to controls, SAD individuals were faster to disengage from smiling faces. SAD was not found to be associated with a bias in attentional engagement for smiling expressions.

Interpretation and Evaluation

Tasks that focused on the ability of SAs to accurately label facial expressions have not typically identified an impact of SA on accuracy, especially when participants had unlimited time to complete the task (Campbell et al., 2009; Heuer, Lange, Isaac, Rinck, & Becker, 2010; Joormann & Gotlib, 2006). Similarly, many rating studies did not find differences between high and low SA individuals in evaluating single emotional expressions (Stein, Goldin, Sareen, Zorrilla, & Brown, 2002) or mixed displays of smiling, neutral, and angry expressions (Gilboa-Schechtman, Presburger, Marom, & Hermesh, 2005; Lange et al., 2011). Other studies have examined response latencies to morphed or degraded presentations of emotional expressions. Such studies have typically found that depression, but not SA, was associated with a higher threshold for the identification of happy expressions (Gilboa-Schechtman, Foa, Vaknin, Marom, & Hermesh, 2008; Joormann & Gotlib, 2006). Recently, women (but not men) with SA were found to require less emotional information to identify smiling, sad, and fearful expressions, compared to nonanxious women (Arrais et al., 2010).

However, when a time constraint was introduced in labeling studies, or when rating studies required participants to rate more subtle (especially interpersonal) attributes of facial expressions, a different pattern of findings emerged. The introduction of a time constraint in labeling studies revealed that, while low SAs demonstrated a positive "misattribution" bias, interpreting disgust as happy, high SAs lacked this bias, confusing disgust with contempt (Heuer et al., 2010). On an untimed rating task, SADs evaluated happy faces as less "approachable" than nonanxious controls (Campbell et al., 2009).

Evidence regarding socially anxious individuals' reduced tendencies to evaluate smiling facial expressions as positive can also be inferred from socially anxious individuals' approach and avoidance tendencies. The approach avoidance task (AAT) is used to examine automatic aspects of behavioral approach and avoidance (Heuer, Rinck, & Becker, 2007). In the AAT, positive stimuli are expected to be associated with faster arm flexion than arm extension whereas negative stimuli are associated with the opposite pattern (Cacioppo, Priester, & Berntson, 1993). High SA participants were found to display greater avoidance (lower approach) tendencies for both smiling and angry faces relative to participants low in SA (Heuer et al., 2007; Roelofs et al., 2010). SA was not found to affect avoidance of neutral faces or explicit judgments of emotional expressions. In addition, Lange, Keijsers, Becker, and Rinck (2008) have found that, as compared to individuals low in SA, individuals high in SA tended to push (i.e., *avoid*), rather than pull (i.e., *approach*) facial crowds consisting of smiling and angry faces (see also **Chapter 16**).

Memory

The literature on memory biases for experimentally presented stimuli in SA presents a mixed picture, with some studies documenting enhanced processing of threatening stimuli (Foa, Gilboa-Schechtman, Amir, & Freshman, 2000), while others do not (Coles & Heimberg, 2002; Rapee, McCallum, Melville, Ravenscroft, & Rodney, 1994; Rinck & Becker, 2005 for reviews). Still other studies find support for the

erosion of positive memory biases in SAD (Liang, Hsu, Hung, Wang, & Lin, 2011). In general, however, the support for negative memory bias, or for diminished positive bias, in SAD using emotional faces or words is rather modest.

To summarize, data from attention and interpretation (but not memory) studies suggest that, in no-stress conditions, individuals high in SA do not appear to associate a smiling face with unambiguously positive outcomes. Moreover, under some circumstances, SAD individuals, but not controls, process angry and smiling faces in a balanced (similar) manner. Importantly, such even-handed processing of angry and smiling expressions is also observed in imaging studies (Ball et al., 2012). However, these findings are obtained mostly with indirect (implicit) rather with direct (explicit) assessment measures.

Cognitive Biases in the Face of Social Stress

Cognitive theories of SA (Clark & Wells, 1995) emphasize that the threat of a negative social outcome (e.g., social rejection) is likely to exacerbate attention, interpretation, and memory biases in SAD. Being rejected is found to make people more sensitive to interpersonal cues. Indeed, in nonselected populations, individuals who have been rejected (as compared to those who are accepted) are more sensitive to the emotional tone of other people's voices; more accurate in detecting emotions from others' facial expressions (Pickett, Gardner, & Knowles, 2004), more accurate in distinguishing between real and fake smiles (Bernstein, Young, Brown, Sacco, & Claypool, 2008), and more likely to orient attention in accordance with another person's eye-gaze (Wilkowski, Robinson, & Friesen, 2009). Thus, signals indicating potential sources of renewed affiliation would be expected to receive preferential processing following rejection (Maner, DeWall, Baumeister, & Schaller, 2007).

Indeed, using a variety of attentional indices, DeWall, Maner, and Rouby (2009) found that, compared with nonexcluded participants, participants who experienced the threat of social exclusion attended selectively and preferentially to smiling faces. Buckner, DeWall, Schmidt, and Maner (2010) also found that, as compared to nonsocial threat, threat of social exclusion was related to enhanced attention to positive faces among high SA individuals (commonly interpreted as related to enhanced affiliative tendency). In contrast, Mallott, Maner, DeWall, and Schmidt (2009) found that high SA individuals failed to react to rejection in a positive or pro-social manner and exhibited some evidence of negative social responses.

Additional studies examining attention to smiles under conditions of social stress (before an anticipated performance challenge, or following a change in social relatedness) are necessary to understand whether, and how, SA affects attunement to signals of social affiliation in the face of changing social fortunes. Moreover, it is yet unclear how socially anxious individuals attend to and interpret socially affiliative signals following the experience of unambiguous social acceptance or social ascendance.

What's in a smile?

As mentioned above, smiles have been extensively used in the study of SA. Indeed, smiles are the most common visual expressions of affiliative intent, but they may

connote an internal state of enjoyment as well as a state of dominance or condescension (Niedenthal, Mermillod, Maringer, & Hess, 2010). Thus, smiles are also intrinsically ambiguous. Why might socially anxious individuals fail to associate smiles with positive outcomes (acceptance) and even perceive smiling faces as threatening?

A data-driven study, which used principal components analysis on ratings of faces along various dimensions, found that the evaluation of faces can be represented within a two-dimensional space defined by *trustworthiness* and *dominance* (Todorov, Said, Engel, & Oosterhof, 2008). These two dimensions can be thought of as isomorphic to the dimensions proposed by the interpersonal circumplex models (Wiggins, 1979), and with the recently advanced two-dimensional representations in social judgments (Abele, Cuddy, Judd, & Yzerbyt, 2008; Cuddy, Fiske, & Glick, 2008). These models echo the affiliation and dominance systems discussed earlier (Trower & Gilbert, 1989). Importantly, in terms of their positions along these two dimensions, smiling faces were rated as high in both friendliness (or affiliation) and dominance (Knutson, 1996). Although speculative, it seems likely that, if individuals preferentially attend to signals of dominance versus signals of affiliation, they might interpret smiling faces as threatening; whereas if they preferentially attend to signals of affiliation, they might tend to interpret it as positive. A more thorough examination of smiles would involve the systematic examination of smiles' functions (Niedenthal et al., 2010).

Event Reactivity

Using positive and negative vignettes, Gilboa-Schechtman et al. have found that individuals with SAD tend to underestimate the likelihood of socially positive events while concurrently over-estimating both the positive and the negative impact of such events (Gilboa-Schechtman, Franklin, & Foa, 2000). Thus, positive events failed to elicit an unambiguously positive reaction from SAD individuals.

Using experimental manipulation, Alden et al. found that, upon receiving positive feedback following a social interaction, individuals with high levels of SA expected to experience higher levels of anxiety regarding a future social interaction (Alden, Mellings, & Laposa, 2004). In addition, after receiving positive feedback, people with SAD predicted that their partner would expect more from them in the next interaction and that they would fall short of those expectations (Alden & Wallace, 1995; Wallace & Alden, 1997). Finally, Alden, Taylor, Mellings, and Laposa (2008) found that the tendency to interpret positive social events as indicative of negative future outcomes partially mediated the relationship between SA and decreased PA (Alden et al., 2008).

Socially anxious individuals appear to be impaired in their memory for positive self-relevant information, especially when faced with or following a social interaction. For example, Cody and Teachman (2010) conducted a study in which participants received positive and negative feedback regarding their own performance, and regarding the performance of a confederate. Memory for this information was assessed immediately after, as well as two days after the experimental manipulation. High SA participants showed more positively biased recognition for the confederate's feedback compared to their own. High SAs also demonstrated negatively biased recognition (relative to the low SAs) for positive feedback regarding their own performance over time.

Combined, these findings indicate that SA individuals predict that events connoting social approval or advance in social standing raise NA. SA individuals focus on the possible negative implications of successful social interactions, and this focus may dampen (or even prevent) their experience of satisfaction, pride, or enjoyment from these encounters. Finally, SA appears to be associated with diminished memories for signals of appreciation.

Regulation of Positive Emotions

Emotion regulation refers to the "processes by which individuals influence which emotions they have, when they have them, and how they experience and express these emotions" (Gross, 1998, p. 5). Gross and Thompson (2007) presented five classes of emotion regulation strategies varying in the point in time in which they "enter" the regulatory process.

So far, research on emotion regulation has centered on the regulation of *negative* emotions. Less is known about positive emotion regulation in general, and regulation of positive emotions in psychopathology in particular. However, it has been suggested that attention to positive information promotes emotion regulation (Wadlinger & Isaacowitz, 2011). In addition, regulation strategies may differ in effectiveness when applied to the regulation of negative versus positive emotions. For instance, emotional suppression reduces positive but not negative affect (Gross, 1998; Gross & Levenson, 1997). Studies in SA suggest that SAD individuals are less likely to engage in emotion regulation in general, due to negative beliefs regarding the effectiveness of emotion regulation techniques (Sung et al., 2012). We now examine the available evidence concerning socially anxious individuals' propensity to engage in regulatory strategies which may shorten or dampen positive experiences.

Situation selection refers to a regulatory strategy taken at a preliminary stage, before the emotion-evoking event has taken place. For example, an individual may choose one situation over another (e.g., staying at home over going to a party). In other words, situation selection may be understood as choosing to engage in or avoid a situation (Werner, Goldin, Ball, Heimberg, & Gross, 2011). In situation selection, individuals rely on expectations and past experiences from similar situations to inform their affective forecasting. Because SAD individuals tend to underestimate the likelihood of positive social events and overestimate their potential costs (Gilboa-schechtman et al., 2000), they may avoid situations in which there is a potential for a positive evaluation by others (Weeks & Howell, 2012).

Situation modification refers to actions taken by an individual designed to alter various aspects of the situation in order to influence its emotional impact. Safety behaviors may be considered as an example of this strategy (e.g., speaking in a soft voice in order to make a favorable expression). Clearly, not all situation modification strategies are maladaptive (e.g., one may use humor to ward off criticism). Recent findings have demonstrated that different safety behaviors (i.e., avoidance versus impression management) lead to distinct expectations for future interactions (Plasencia, Alden, & Taylor, 2011). Given that socially anxious individuals are concerned with being the center of attention, and are wary of positive feedback, they may use situation modification strategies to prevent the unfolding of such situations.

Attentional deployment refers to the alteration of one's attention in an attempt to affect emotional intensity. These strategies may include changes in attentional focus away from (distraction) or to the (concentration) stimuli in question. Gross and Thompson (2007) mention the act of "rumination" as a faulty concentration strategy in emotion regulation. One may consider "savoring" as an attentional strategy, which serves to enhance the impact of positive experiences (Miyamoto & Ma, 2011). Importantly, socially anxious individuals reported engaging in less savoring of positive emotions (Eisner, Johnson, & Carver, 2009). Using self-report (i.e., explicit) measures on the frequency of attentional deployment, no differences were found between SADs and nonanxious control individuals (Werner et al., 2011). Yet, as we already mentioned, several recent studies have found differences between high and low SA individuals while assessing attentional deployment implicitly (Mallott et al., 2009). Additional studies in this vein, assessing attentional deployment and attunement in the anticipation of, or following the experiences of, social acceptance and social ascendance are needed.

Cognitive change, in the form of cognitive reappraisal, refers to the meaning given to a situation in order to alter its emotional charge. Weeks (2010) proposed one such mechanism of cognitive reappraisal, the disqualification of positive social outcomes (DPSO, Weeks, 2010). This mechanism refers to a thinking pattern, in which an individual eliminates what others might consider positive (Beck, 1976). Individuals with SAD obtained significantly higher scores on DPSO compared to nonsocially anxious control participants (Weeks, 2010). Similar results have been obtained by Vassilopoulos and Banerjee (2010), who found that the propensity to discount positive events mediated the relationship between SA and PA. Re-evaluating a positive experience as unimportant, stressful, or inauthentic, deflates its potential emotional impact.

Response modulation occurs when the emotional affect has already unfolded, and an attempt to modulate the physiological, experiential, and behavioral aspects of the emotional response is under way. Because an emotional reaction may lead SA individuals to be more exposed and conspicuous, a strategy of emotion suppression may dominate their emotional regulation mechanisms (Werner et al., 2011). As mentioned earlier, decreasing emotionally expressive behavior decreases positive but not negative emotions (Gross, 1998). Emotion suppression is prevalent among SA individuals, and this relationship is mediated by beliefs that overt emotional expression is negative (Spokas, Luterek, & Heimberg, 2009). Suppressing positive emotions may prevent SAs from experiencing these fully (Kashdan & Breen, 2008); and reduces both the intensity, and the likelihood, of experiencing positive events on the day following the suppression efforts (Farmer & Kashdan, 2012). Indeed, the negative correlation between SA and PA was mediated by the tendency to suppress emotions (see Kashdan, Weeks, & Savostyanova, 2011 for a review). SAD individuals reported being less emotionally expressive and paying less attention to their positive emotions than were individuals with generalized anxiety disorder or controls (Turk, Heimberg, Luterek, Mennin, & Fresco, 2005; see also Farmer & Kashdan, 2012 for nonclinical replication). In sum, emotion suppression appears to play a significant role in positivity impairment in SA.

Emotion regulation strategies appear to play a causal role in the development and maintenance of psychopathology (Gross & Thompson, 2007). Yet, regulatory efforts for positive emotions are only partially explored, and even less so within the context

of SA. Different mechanisms of emotion suppression may exist for specific positive emotions. For example, individuals may believe that they should suppress pride but express exhilaration (endorsing both "I shouldn't show my enthusiasm about my successful performance" and "expressing my awe of a musical piece is appropriate as it signals that I am a spiritual person"). Understanding the frequency, range, and flexibility of emotion regulation strategies in SA is important, as these strategies may be causally related to the maintenance of SA.

The Causal Status of Positive Affect and Positive Events in Social Anxiety

Evidence directly supporting the causal role of PA in SA can be derived from two main lines of research: longitudinal studies examining the impact of positive events on SA, and cognitive bias modification training (attention and interpretation) studies examining the effects of changing cognitive patterns on mood and emotional reactivity.

Longitudinal Studies

Several longitudinal studies combine in suggesting that close positive relationships and experiences of mastery/belonging serve as protective factors against SA. For instance, positive class climate was found as protective for sensitive children who suffer from "anxious-solitude" tendencies (Avant, Gazelle, & Faldowski, 2011). Relatedly, in a study on first graders' adjustment, a positive child–teacher relationship moderated the relationship between shyness and adjustment, such that closer relationships predicted better adjustment (Arbeau, Coplan, & Weeks, 2010). A prospective study of SA among adolescents found that SA decreases when friendship qualities (i.e., companionship and intimacy) increase (Vernberg, Abwender, Ewell, & Beery, 1992). Finally, sports participation in childhood was associated with better psychological adjustment a year later. Specifically, shy children who participated in sports over time reported a significant decrease in anxiety (Findlay & Coplan, 2008). It is likely that the association between close positive relationships and SA is reciprocal, as SA is likely to influence the probability of forming close relationships (Alden & Taylor, 2004). This reciprocal relationship may create an "upward spiral" as affiliative social behaviors tend to evoke corresponding responses in terms of affiliation (Tiedens & Fragale, 2003; Tiedens & Jimenez, 2003).

An additional line of research suggests that warm and supporting family relationships serve as protective factors against the *maintenance* of SA. Longitudinal studies aimed to predict the onset of SA based on family functioning yielded a mixed pattern of results, with some studies supporting such an association while others found only limited support for it (Knappe et al., 2009). Importantly, using a prospective design, Knappe et al. (2009) found that lack of emotional warmth was independently associated with the *persistence* of SA in adolescence, above and beyond the influence of dysfunctional family functioning and parental psychopathology. Moreover, a recent study by Knappe, Beesdo-Baum, Fehm, Lieb, and Wittchen (2012) found that a pattern of higher maternal overprotection, higher paternal rejection, and

lower paternal emotional warmth was uniquely associated with increased levels of SA in offsprings.

Interestingly, so far, most studies examined the impact of affiliative social relationships on the onset and maintenance of SA. However, given the centrality of concerns with social standing and social rank in SA (Aderka et al., 2009), it is also important to examine the causal effects of the influence of experiences of increased social rank on SA.

Cognitive Modification Training Studies

In the past decade, a growing body of research has focused on the alleviation of anxiety symptoms via the modification of basic cognitive processes such as attention and interpretation. A causal role of positive information processing in the maintenance of SA may be inferred from the modifications of cognitive biases. Specifically, modifying attention for/interpretation of positive information, and observing a later decline in anxiety symptoms and an increase in PA may argue for such causal proposition.

Attentional Bias Modification Programs

Attentional bias modification (ABM) programs have been used to implicitly teach individuals to shift their attention from threat location towards nonthreatening locations. Such interventions have been found to have a beneficial effect on emotional experience in several anxiety disorders, including SAD (for reviews, see Eldar & Bar-Haim, 2010; MacLeod & Mathews, 2012).

Recently, researchers have begun to use attentional training paradigms in a new manner, by teaching individuals to attend to positive, rather than neutral, information. For example, Li, Tan, Qian, and Liu (2008) found that attentional training toward positive faces in SA was associated with reductions in attentional bias for negative faces and self-reported fear of social interaction. Similarly, Heeren, Reese, McNally, and Philippot (2012) found reductions in self-reported SA following training to attend to positive stimuli. In the latter study, participants also improved in verbal, behavioral, and physiological measures of SA. However, none of the aforementioned studies assessed positive mood following their "positively" oriented ABM programs.

Such an attempt was recently undertaken by Grafton, Ang, and Macleod (2012), who used ABM for manipulating (unselected) participants' selective attentional response to positive information. Participants in the "attend positive" condition were faster to respond to cues replacing positive words, and also reported higher levels of PA following positive feedback, compared to participants in the "avoid positive" condition. Therefore, beyond demonstrating the ability to differentially modify attentional response to positive information, the findings of Grafton et al. point to a causal role of selective attention to positive information in experiencing PA. Taken together, these findings suggest that the experimental induction of a positive attentional bias may not only attenuate anxious response to a subsequent stressor (Johnson, 2009; Taylor, Bomyea, & Amir, 2010), but also directly contribute to enhanced PA.

Interpretation Bias Modification Programs

Interpretation bias modification (IBM) programs have been used to implicitly teach individuals to interpret ambiguous events in a less threatening or a more benign manner. For example, high SA individuals receiving a single-session positive IBM reported experiencing less anticipatory anxiety about a future social situation relative to a control group (Murphy, Hirsch, Mathews, Smith, & Clark, 2007). Beard and Amir found that individuals with elevated SA levels reported a reduction in their symptoms following an eight-session IBM using a modified word–sentence association paradigm (WSAP; Beard & Amir, 2009). These results were recently replicated in a study using a 12-session adaptation of the same procedure (Amir & Taylor, 2012). An integrated program that included both interpretation (WSAP) and attentional training methods was effective in reducing self-reported and behavioral measures of SA (Beard, Weisberg, & Amir, 2011).

Recent findings suggest that approach-related motivations and behavior can be influenced by applying an adapted approach/avoidance task paradigm that manipulates the tendency to approach a specific cue, as opposed to avoid it (Wiers, Eberl, Rinck, Becker, & Lindenmeyer, 2011; Wiers, Rinck, Kordts, Houben, & Strack, 2010). An AAT-based CBM program aiming to modify SA individuals' automatic approach tendencies for smiling facial expressions was conducted (Taylor & Amir, 2012). Participants in the approach-positive condition displayed greater social approach behaviors and also elicited more positive post-interaction reactions from their conversation partner, relative to those in the control condition. Participants' subjective level of anxiety was not significantly affected during the task, and therefore the effects cannot be explained as a result of reduced anxiety levels. Combined, research from longitudinal and experimental paradigms suggests that PA may play a role in the maintenance of SA.

Implications and Future Directions

Core Features of Social Anxiety Revisited

Our review suggests that individuals suffering from SAD and individuals with high SA show a complex pattern of positivity erosion, although the scope of this erosion is still incompletely understood. Increased emphasis on the differential processing of signals of positive regard versus events connoting social acceptance or social ascendance may enhance the conceptualization of SAD. First, our conceptualization may extend beyond sensitivity to social threat to include biased processing of signals of affiliation. This development is consistent with evolutionary, interpersonal, and cognitive accounts of SAD (Alden & Taylor, 2004; Gilbert, 2001; Weeks & Howell, 2012). Moreover, examination of reactivity to socially affiliative as well as socially threatening cues may broaden and refine our current conceptualizations of this disorder, and contribute to the understanding of the comorbidity between SA and depression (Gilboa-schechtman et al., 2000; Kashdan et al., 2011). Second, with respect to the assessment of SAD, the present framework may help shift efforts to elucidate the nature and severity of distress from looking solely at functioning in nonstressful

situations, to the examination of reactivity to challenges and changes in belongingness and dominance domains (Clark & Wells, 1995; Weeks, Jakatdar, & Heimberg, 2010). Exploring SA within the context of the sensitivity of basic psychological systems represents a shift toward a theory-based, rather than a symptom-based, approach to emotional disorders.

Positive Impairment and Social Anxiety Revisited

Dominance and affiliation have been linked to both PA and well-being. A great number of studies have established strong associations between affiliative interactions on the one hand, and PA and well-being (WLB) on the other (Brown et al., 2007; Watson, 2000; Watson et al., 1992). Perceived intimacy has also been linked to PA and WLB (Busch & Hofer, 2012). Other studies have documented the relationship of dominance to both PA and WLB. Hammer and Good (2010) found that self-perception of dominance in men was related to psychological resilience. Having higher socio-economic status in comparison to neighbors contributes to WLB (Firebaugh & Schroeder, 2009). Finally, a report by the World Health Organization places social–economical positioning as a major contributor to variations in communities' mental health (Friedli, 2009). SA, as understood from both evolutionary and interpersonal perspectives, is associated with decreased experiences of both affiliative bonds and of interpersonal dominance. Deficiencies and difficulties in both of these domains are likely to independently contribute to decreased PA and WLB.

Treatment Implications

The preceding review suggests the need to specifically target positive emotions in the treatment of SA. Treatment may benefit from explicitly focusing on the enhancement of positive experience either by the use of cognitive modification procedures, or by instructing individuals to practice more adaptive regulatory strategies (e.g., enhancing overt signals of PA; focusing on subsequent cognitive and behavioral manifestations [savoring]). Indeed, the enhancement of positive experiences was found to be associated with changes in basic cognitive, physiological, and behavioral functioning, creating an "upward spiral" process (Kok & Fredrickson, 2010). Interventions geared to enhance positivity (such as the positive affect stimulation and sustainment [PASS]) have recently been developed for the treatment of depression. The PASS module utilizes behavioral activation to increase the frequency of positive events and then seeks to capitalize on these positive events to enhance and sustain PA (McMakin, Siegle, & Shirk, 2011). An intervention that directly targets positive emotion may also be effective for SAD.

Symptom reduction is not the only desired outcome of therapeutic interventions. The ultimate goal is human flourishing: the capacity to inspire and to be inspired; the capacity to form stable, secure, and satisfying bonds; the capacity to be creative in multiple life domains. Understanding the nature of positivity impairment may bring us closer to this goal.

References

Abele, A. E., Cuddy, A. J. C., Judd, C. M., & Yzerbyt, V. Y. (2008). Fundamental dimensions of social judgment. *European Journal of Social Psychology, 38*(7), 1063–1065. doi:10.1002/ejsp.574

Aderka, I. M., Hofmann, S. G., Nickerson, A., Hermesh, H., Gilboa-Schechtman, E., & Marom, S. (2012). Functional impairment in social anxiety disorder. *Journal of Anxiety Disorders, 26*(3), 393–400. doi:10.1016/j.janxdis.2012.01.003

Aderka, I. M., Weisman, O., Shahar, G., & Gilboa-Schechtman, E. (2009). The roles of the social rank and attachment systems in social anxiety. *Personality and Individual Differences, 47*(4), 284–288. doi:10.1016/j.paid.2009.03.014

Alden, L. E., Mellings, T. M. B., & Laposa, J. M. (2004). Framing social information and generalized social phobia. *Behaviour Research and Therapy, 42*(5), 585–600. doi:10.1016/S0005-7967(03)00163-3

Alden, L. E., & Taylor, C. T. (2004). Interpersonal processes in social phobia. *Clinical Psychology Review, 24*(7), 857–882.

Alden, L. E., Taylor, C. T., Mellings, T. M. J. B., & Laposa, J. M. (2008). Social anxiety and the interpretation of positive social events. *Journal of Anxiety Disorders, 22*(4), 577–90. doi:10.1016/j.janxdis.2007.05.007

Alden, L. E., & Wallace, T. (1995). Social phobia and social appraisal in successful and unsuccessful social interactions. *Behaviour Research and Therapy, 33*(5), 497–505.

Alden, L. E., Wiggins, J. S., & Pincus, A. L. (1990). Construction of circumplex scales for the Inventory of Interpersonal Problems. *Journal of Personality Assessment, 55*(3–4), 521–536. doi:10.1080/00223891.1990.9674088

Amir, N., & Taylor, C. T. (2012). Interpretation training in individuals with generalized social anxiety disorder: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 80*(3), 497–511. doi:10.1037/a0026928

Arbeau, K. A., Coplan, R. J., & Weeks, M. (2010). Shyness, teacher-child relationships, and socio-emotional adjustment in grade 1. *International Journal of Behavioral Development, 34*(3), 259–269. doi:10.1177/0165025409350959

Arrais, K. C., Machado-de-Sousa, J. P., Trzesniak, C., Filho, A. S., Ferrari, M. C. F., Osório, F. L., . . . Crippa, J. A. (2010). Social anxiety disorder women easily recognize fearful, sad and happy faces: The influence of gender. *Journal of Psychiatric Research, 44*(8), 535–540. doi:10.1016/j.jpsychires.2009.11.003

Avant, T. S., Gazelle, H., & Faldowski, R. (2011). Classroom emotional climate as a moderator of anxious solitary children's longitudinal risk for peer exclusion: A child × environment model. *Developmental Psychology, 47*(6), 1711–1727. doi:10.1037/a0024021

Ball, T. M., Sullivan, S., Flagan, T., Hitchcock, C. A., Simmons, A., Paulus, M. P., & Stein, M. B. (2012). Selective effects of social anxiety, anxiety sensitivity, and negative affectivity on the neural bases of emotional face processing. *NeuroImage, 59*(2), 1879–1887. doi:10.1016/j.neuroimage.2011.08.074

Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychological Bulletin, 117*(3), 497–529. doi:10.1037/0033-2909.117.3.497

Beard, C., & Amir, N. (2009). Interpretation in social anxiety: When meaning precedes ambiguity. *Cognitive Therapy and Research, 33*(4), 406–415. doi:10.1007/s10608-009-9235-0

Beard, C., Weisberg, R. B., & Amir, N. (2011). Combined cognitive bias modification treatment for social anxiety disorder: A pilot trial. *Depression and Anxiety, 28*(11), 981–988. doi:10.1002/da.20873

Beck, A. T. (1976). *Cognitive therapy and the emotional disorders* (p. 356). Oxford, UK: International Universities Press.

Bernstein, M. J., Young, S. G., Brown, C. M., Sacco, D. F., & Claypool, H. M. (2008). Adaptive responses to social exclusion: Social rejection improves detection of real and fake smiles. *Psychological Science, 19*(10), 981–983. doi:10.1111/j.1467-9280.2008.02187.x

Boehm, J. K., & Kubzansky, L. D. (2012). The heart's content: The association between positive psychological well-being and cardiovascular health. *Psychological Bulletin, 138*(4), 655–691. doi:10.1037/a0027448

Brown, L. H., Silvia, P. J., Myin-Germeys, I., & Kwapil, T. R. (2007). When the need to belong goes wrong: The expression of social anhedonia and social anxiety in daily life. *Psychological Science, 18*(9), 778–782. doi:10.1111/j.1467-9280.2007.01978.x

Brown, T. A., Chorpita, B. F., & Barlow, D. H. (1998). Structural relationships among dimensions of the DSM-IV anxiety and mood disorders and dimensions of negative affect, positive affect, and autonomic arousal. *Journal of Abnormal Psychology, 107*(2), 179–192. doi:10.1037/0021-843X.107.2.179

Buckner, J. D., Dewall, C. N., Schmidt, N. B., & Maner, J. K. (2010). A Tale of Two Threats: Social anxiety and attention to social threat as a function of social exclusion and non-exclusion threats. *Cognitive Therapy and Research, 34*(5), 449–455. doi:10.1007/s10608-009-9254-x

Bugental, D. B. (2000). Acquisition of the algorithms of social life: A domain-based approach. *Psychological Bulletin, 126*(2), 187–219. doi:10.1037//0033-2909.126.2.187

Busch, H., & Hofer, J. (2012). Self-regulation and milestones of adult development: Intimacy and generativity. *Developmental Psychology, 48*(1), 282–293. doi:10.1037/a0025521

Cacioppo, J. T., Priester, J. R., & Berntson, G. G. (1993). Rudimentary determinants of attitudes: II. Arm flexion and extension have differential effects on attitudes. *Journal of Personality and Social Psychology, 65*(1), 5–17. doi:10.1037/0022-3514.65.1.5

Campbell, D. W., Sareen, J., Stein, M. B., Kravetsky, L. B., Paulus, M. P., Hassard, S. T., & Reiss, J. P. (2009). Happy but not so approachable: The social judgments of individuals with generalized social phobia. *Depression and Anxiety, 26*(5), 419–424. doi:10.1002/da.20474

Catalino, L. I., & Fredrickson, B. L. (2011). A Tuesday in the life of a flourisher: The role of positive emotional reactivity in optimal mental health. *Emotion (Washington, D.C.), 11*(4), 938–950. doi:10.1037/a0024889

Chen, N. T. M., Clarke, P. J. F., MacLeod, C., & Guastella, A. J. (2012). Biased attentional processing of positive stimuli in social anxiety disorder: An eye movement study. *Cognitive Behaviour Therapy, 41*(2), 96–107. doi:10.1080/16506073.2012.666562

Chen, Y., Ehlers, A., Clark, D. M., & Mansell, W. (2002). Patients with generalized social phobia direct their attention away from faces. *Behaviour Research and Therapy, 40*(6), 677–687. doi:10.1016/S0005-7967(01)00086-9

Clark, D. M., & Wells, A. (1995). A cognitive model of social phobia. In R. G. Heimberg, M. R. Liebowitz, D. A. Hope, & F. R. Schneier (Eds.), *Social phobia: Diagnosis, assessment, and treatment* (pp. 69–93). New York, NY: Guilford Press.

Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology, 100*(3), 316–336. doi:10.1037/0021-843X.100.3.316

Cody, M. W., & Teachman, B. A. (2010). Post-event processing and memory bias for performance feedback in social anxiety. *Journal of Anxiety Disorders, 24*(5), 468–479. doi:10.1016/j.janxdis.2010.03.003

Coles, M. E., & Heimberg, R. G. (2002). Memory biases in the anxiety disorders. *Clinical Psychology Review, 22*(4), 587–627. doi:10.1016/S0272-7358(01)00113-1

Cuddy, A. J., Fiske, S. T., & Glick, P. (2008). Warmth and competence as universal dimensions of social perception: The Stereotype Content Model and the BIAS Map. In M. P. Zanna (Ed.), *Advances in experimental social psychology* (Vol. *40*, pp. 61–149). New York, NY: Academic Press. doi:10.1016/S0065-2601(07)00002-0

Davidson, K. W., Mostofsky, E., & Whang, W. (2010). Don't worry, be happy: Positive affect and reduced 10-year incident coronary heart disease: The Canadian Nova Scotia Health Survey. *European Heart Journal, 31*(9), 1065–1070. doi:10.1093/eurheartj/ehp603

DeWall, C. N., Baumeister, R. F., & Vohs, K. D. (2008). Satiated with belongingness? Effects of acceptance, rejection, and task framing on self-regulatory performance. *Journal of Personality and Social Psychology, 95*(6), 1367–1382. doi:10.1037/a0012632

DeWall, C. N., Maner, J. K., & Rouby, D. A. (2009). Social exclusion and early-stage interpersonal perception: Selective attention to signs of acceptance. *Journal of Personality and Social Psychology, 96*, 729–741.

Eisner, L. R., Johnson, S. L., & Carver, C. S. (2009). Positive affect regulation in anxiety disorders. *Journal of Anxiety Disorders, 23*(5), 645–649. doi:10.1016/j.janxdis.2009.02.001

Eldar, S., & Bar-Haim, Y. (2010). Neural plasticity in response to attention training in anxiety. *Psychological Medicine, 40*(4), 667–677. doi:10.1017/S0033291709990766

Farmer, A. S., & Kashdan, T. B. (2012). Social anxiety and emotion regulation in daily life: Spillover effects on positive and negative social events. *Cognitive Behaviour Therapy, 41*(2), 152–162. doi:10.1080/16506073.2012.666561

Findlay, L. C., & Coplan, R. J. (2008). Come out and play: Shyness in childhood and the benefits of organized sports participation. *Canadian Journal of Behavioural Science/Revue Canadienne des Sciences du Comportement, 40*(3), 153–161. doi:10.1037/0008-400X.40.3.153

Firebaugh, G., & Schroeder, M. B. (2009). Does your neighbor's income affect your happiness? *American Journal of Sociology, 115*(3), 805–831. doi:10.1086/603534

Foa, E. B., Gilboa-Schechtman, E., Amir, N., & Freshman, M. (2000). Memory bias in generalized social phobia: Remembering negative emotional expressions. *Journal of Anxiety Disorders, 14*(5), 501–519. doi:10.1016/S0887-6185(00)00036-0

Fredrickson, B. L. (2001). The role of positive emotions in positive psychology: The broaden-and-build theory of positive emotions. *American Psychologist, 56*(3), 218–226. doi:10.1037/0003-066X.56.3.218

Fredrickson, B. L., & Branigan, C. (2005). Positive emotions broaden the scope of attention and thought-action repertoires. *Cognition & Emotion, 19*(3), 313–332. doi:10.1080/02699930441000238

Fredrickson, B. L., Tugade, M. M., Waugh, C. E., & Larkin, G. R. (2003). What good are positive emotions in crisis? A prospective study of resilience and emotions following the terrorist attacks on the United States on September 11th, 2001. *Journal of Personality and Social Psychology, 11*(2), 365–376. doi:10.1037/0022-3514.84.2.365

Friedli, L. (2009). *Mental health, resilience and inequalities* – a report for WHO Europe and the Mental Health Foundation London/Copenhagen: Mental Health Foundation and WHO Europe.

Gamble, A. L., & Rapee, R. M. (2010). The time-course of attention to emotional faces in social phobia. *Journal of Behavior Therapy and Experimental Psychiatry, 41*(1), 39–44. doi:10.1016/j.jbtep.2009.08.008

Gilbert, P. (2000). The relationship of shame, social anxiety and depression: The role of the evaluation of social rank. *Clinical Psychology & Psychotherapy, 7*(3), 174–189. doi:10.1002/1099-0879(200007)7:3<174::AID-CPP236>3.0.CO;2-U

Gilbert, P. (2001). Evolution and social anxiety: The role of attraction, social competition, and social hierarchies. *Psychiatric Clinics of North America, 24*(4), 723–751. doi:10.1016/S0193-953X(05)70260-4

Gilbert, P., & Trower, P. (2001). Evolution and process in social anxiety. In W. R. Crozier & L. E. Alden (Eds.), *International handbook of social anxiety: Concepts, research and interventions relating to the self and shyness* (pp. 259–279). New York, NY: John Wiley & Sons, Ltd.

Gilboa-Schechtman, E., Foa, E. B., & Amir, N. (1999). Attentional biases for facial expressions in social phobia: The face-in-the-crowd paradigm. *Cognition & Emotion, 13*(3), 305–318. doi:10.1080/026999399379294

Gilboa-Schechtman, E., Foa, E., Vaknin, Y., Marom, S., & Hermesh, H. (2008). Interpersonal sensitivity and response bias in social phobia and depression: Labeling emotional expressions. *Cognitive Therapy and Research, 32*(5), 605–618. doi:10.1007/s10608-008-9208-8

Gilboa-schechtman, E., Franklin, M. E., & Foa, E. B. (2000). Anticipated reactions to social events: Differences among individuals with generalized social phobia, obsessive compulsive disorder, and nonanxious controls. *Cognitive Therapy and Research, 24*(6), 731–746.

Gilboa-Schechtman, E., Presburger, G., Marom, S., & Hermesh, H. (2005). The effects of social anxiety and depression on the evaluation of facial crowds. *Behaviour Research and Therapy, 43*(4), 467–474. doi:10.1016/j.brat.2004.03.001

Grafton, B., Ang, C., & MacLeod, C. (2012). Always look on the bright side of life: The attentional basis of positive affectivity. *European Journal of Personality, 26*(2), 133–144. doi:10.1002/per.1842

Griskevicius, V., Shiota, M. N., & Neufeld, S. L. (2010). Influence of different positive emotions on persuasion processing: A functional evolutionary approach. *Emotion (Washington, D.C.), 10*(2), 190–206. doi:10.1037/a0018421

Gross, J. J. (1998). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology, 74*(1), 224–237. doi:10.1037/0022-3514.74.1.224

Gross, J. J., & Levenson, R. W. (1997). Hiding feelings: The acute effects of inhibiting negative and positive emotion. *Journal of Abnormal Psychology, 106*(1), 95–103. doi:10.1037/0021-843X.106.1.95

Gross, J. J., & Thompson, R. A. (2007). Emotion regulation: Conceptual foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 3–24). New York, NY: Guilford Press.

Gruber, J., & Johnson, S. L. (2009). Positive emotional traits and ambitious goals among people at risk for mania: The need for specificity. *International Journal of Cognitive Therapy, 2*(2), 176–187. doi:10.1521/ijct.2009.2.2.176

Gruber, J., Oveis, C., Keltner, D., & Johnson, S. L. (2011). A discrete emotions approach to positive emotion disturbance in depression. *Cognition & Emotion, 25*(1), 40–52. doi:10.1080/02699931003615984

Hammer, J. H., & Good, G. E. (2010). Positive psychology: An empirical examination of beneficial aspects of endorsement of masculine norms. *Psychology of Men & Masculinity, 11*(4), 303–318. doi:10.1037/a0019056

Heeren, A., Reese, H. E., McNally, R. J., & Philippot, P. (2012). Attention training toward and away from threat in social phobia: Effects on subjective, behavioral, and physiological measures of anxiety. *Behaviour Research and Therapy, 50*(1), 30–39. doi:10.1016/j.brat.2011.10.005

Herring, D. R., Burleson, M. H., Roberts, N. A., & Devine, M. J. (2011). Coherent with laughter: Subjective experience, behavior, and physiological responses during amusement and joy. *International Journal of Psychophysiology, 79*(2), 211–218. doi:10.1016/j.ijpsycho.2010.10.007

Heuer, K., Lange, W.-G., Isaac, L., Rinck, M., & Becker, E. S. (2010). Morphed emotional faces: Emotion detection and misinterpretation in social anxiety. *Journal of Behavior Therapy and Experimental Psychiatry, 41*(4), 418–425. doi:10.1016/j.jbtep.2010.04.005

Heuer, K., Rinck, M., & Becker, E. S. (2007). Avoidance of emotional facial expressions in social anxiety: The Approach-Avoidance Task. *Behaviour Research and Therapy, 45*(12), 2990–3001. doi:10.1016/j.brat.2007.08.010

Hofmann, S. G., Heinrichs, N., & Moscovitch, D. A. (2004). The nature and expression of social phobia: Toward a new classification. *Clinical Psychology Review, 24*(7), 769–797. doi:10.1016/j.cpr.2004.07.004

Johnson, D. R. (2009). Goal-directed attentional deployment to emotional faces and individual differences in emotional regulation. *Journal of Research in Personality, 43*(1), 8–13. doi:10.1016/j.jrp.2008.09.006

Joormann, J., & Gotlib, I. H. (2006). Is this happiness I see? Biases in the identification of emotional facial expressions in depression and social phobia. *Journal of Abnormal Psychology, 115*(4), 705–714. doi:10.1037/0021-843X.115.4.705

Juslin, P. N., & Laukka, P. (2003). Communication of emotions in vocal expression and music performance: Different channels, same code? *Psychological Bulletin, 129*(5), 770–814. doi:10.1037/0033-2909.129.5.770

Kashdan, T. B. (2007). Social anxiety spectrum and diminished positive experiences: Theoretical synthesis and meta-analysis. *Clinical Psychology Review, 27*(3), 348–365. doi:10.1016/j.cpr.2006.12.003

Kashdan, T. B., & Breen, W. E. (2008). Social anxiety and positive emotions: A prospective examination of a self-regulatory model with tendencies to suppress or express emotions as a moderating variable. *Behavior Therapy, 39*(1), 1–12. doi:10.1016/j.beth.2007.02.003

Kashdan, T. B., & Steger, M. F. (2006). Expanding the topography of social anxiety. An experience-sampling assessment of positive emotions, positive events, and emotion suppression. *Psychological Science, 17*(2), 120–128. doi:10.1111/j.1467-9280.2006.01674.x

Kashdan, T. B., Weeks, J. W., & Savostyanova, A. A. (2011). Whether, how, and when social anxiety shapes positive experiences and events: A self-regulatory framework and treatment implications. *Clinical Psychology Review, 31*(5), 786–799. doi:10.1016/j.cpr.2011.03.012

Keltner, D., & Haidt, J. (1999). Social functions of emotions at four levels of analysis. *Cognition & Emotion, 13*(5), 505–522.

Knappe, S., Beesdo, K., Fehm, L., Höfler, M., Lieb, R., & Wittchen, H.-U. (2009). Do parental psychopathology and unfavorable family environment predict the persistence of social phobia? *Journal of Anxiety Disorders, 23*(7), 986–994. doi:10.1016/j.janxdis.2009.06.010

Knappe, S., Beesdo-Baum, K., Fehm, L., Lieb, R., & Wittchen, H.-U. (2012). Characterizing the association between parenting and adolescent social phobia. *Journal of Anxiety Disorders, 26*(5), 608–616. doi:10.1016/j.janxdis.2012.02.014

Knutson, B. (1996). Facial expressions of emotion influence interpersonal trait inferences. *Journal of Nonverbal Behavior, 20*(3), 165–182. doi:10.1007/BF02281954

Kok, B. E., & Fredrickson, B. L. (2010). Upward spirals of the heart: Autonomic flexibility, as indexed by vagal tone, reciprocally and prospectively predicts positive emotions and social connectedness. *Biological Psychology, 85*(3), 432–436. doi:10.1016/j.biopsycho.2010.09.005

Kotov, R., Watson, D., Robles, J. P., & Schmidt, N. B. (2007). Personality traits and anxiety symptoms: The multilevel trait predictor model. *Behaviour Research and Therapy, 45*(7), 1485–1503. doi:10.1016/j.brat.2006.11.011

Lange, W.-G., Heuer, K., Langner, O., Keijsers, G. P. J., Becker, E. S., & Rinck, M. (2011). Face value: Eye movements and the evaluation of facial crowds in social anxiety. *Journal of Behavior Therapy and Experimental Psychiatry, 42*(3), 355–363. doi:10.1016/j.jbtep.2011.02.007

Lange, W.-G., Keijsers, G., Becker, E. S., & Rinck, M. (2008). Social anxiety and evaluation of social crowds: Explicit and implicit measures. *Behaviour Research and Therapy, 46*(8), 932–943. doi:10.1016/j.brat.2008.04.008

Li, S., Tan, J., Qian, M., & Liu, X. (2008). Continual training of attentional bias in social anxiety. *Behaviour Research and Therapy, 46*(8), 905–912. doi:10.1016/j.brat.2008.04.005

Liang, C.-W., Hsu, W.-Y., Hung, F.-C., Wang, W.-T., & Lin, C.-H. (2011). Absence of a positive bias in social anxiety: The application of a directed forgetting paradigm. *Journal of Behavior Therapy and Experimental Psychiatry, 42*(2), 204–210. doi:10.1016/j.jbtep.2010.12.002

Ly, V., & Roelofs, K. (2009). Social anxiety and cognitive expectancy of aversive outcome in avoidance conditioning. *Behaviour Research and Therapy, 47*(10), 840–847. doi:10.1016/j.brat.2009.06.015

Lyubomirsky, S., King, L., & Diener, E. (2005). The benefits of frequent positive affect: Does happiness lead to success? *Psychological Bulletin, 131*(6), 803–855. doi:10.1037/0033-2909.131.6.803

MacLeod, C., & Mathews, A. (2012). Cognitive bias modification approaches to anxiety. *Annual Review of Clinical Psychology, 8*, 189–217. doi:10.1146/annurev-clinpsy-032511-143052

Mallott, M. A., Maner, J. K., DeWall, N., & Schmidt, N. B. (2009). Compensatory deficits following rejection: The role of social anxiety in disrupting affiliative behavior. *Depression and Anxiety, 26*(5), 438–446. doi:10.1002/da.20555

Maner, J. K., DeWall, C. N., Baumeister, R. F., & Schaller, M. (2007). Does social exclusion motivate interpersonal reconnection? Resolving the "porcupine problem." *Journal of Personality and Social Psychology, 92*(1), 42–55. doi:10.1037/0022-3514.92.1.42

McMakin, D. L., Siegle, G. J., & Shirk, S. R. (2011). Positive Affect Stimulation and Sustainment (PASS) module for depressed mood: A preliminary investigation of treatment-related effects. *Cognitive Therapy and Research, 35*(3), 217–226. doi:10.1007/s10608-010-9311-5

Mehrabian, A. (1997). Comparison of the PAD and PANAS as models for describing emotions and for differentiating anxiety from depression. *Journal of Psychopathology and Behavioral Assessment, 19*(4), 331–357. doi:10.1007/BF02229025

Miyamoto, Y., & Ma, X. (2011). Dampening or savoring positive emotions: A dialectical cultural script guides emotion regulation. *Emotion (Washington, D.C.), 11*(6), 1346–1357. doi:10.1037/a0025135

Murphy, R., Hirsch, C. R., Mathews, A., Smith, K., & Clark, D. M. (2007). Facilitating a benign interpretation bias in a high socially anxious population. *Behaviour Research and Therapy, 45*(7), 1517–1529. doi:10.1016/j.brat.2007.01.007

Nelson, B. D., & Shankman, S. A. (2011). Does intolerance of uncertainty predict anticipatory startle responses to uncertain threat? *International Journal of Psychophysiology, 81*(2), 107–115. doi:10.1016/j.ijpsycho.2011.05.003

Niedenthal, P. M., Mermillod, M., Maringer, M., & Hess, U. (2010). The Simulation of Smiles (SIMS) model: Embodied simulation and the meaning of facial expression. *Behavioral and Brain Sciences, 33*(6), 417–433. doi:10.1017/S0140525×10000865

Oakman, J., Gifford, S., & Chlebowsky, N. (2003). A multilevel analysis of the interpersonal behavior of socially anxious people. *Journal of Personality, 71*(3), 397–434. doi:10.1111/1467-6494.7103006

Oaten, M., Williams, K. D., Jones, A., & Zadro, L. (2008). The effects of ostracism on self-regulation in the socially anxious. *Journal of Social and Clinical Psychology, 27*(5), 471–504. doi:10.1521/jscp.2008.27.5.471

Pickett, C. L., Gardner, W. L., & Knowles, M. (2004). Getting a cue: The need to belong and enhanced sensitivity to social cues. *Personality & Social Psychology Bulletin, 30*(9), 1095–1107. doi:10.1177/0146167203262085

Pishyar, R., Harris, L. M., & Menzies, R. G. (2004). Attentional bias for words and faces in social anxiety. *Anxiety, Stress & Coping, 17*(1), 23–36. doi:10.1080/10615800310001601458

Plasencia, M. L., Alden, L. E., & Taylor, C. T. (2011). Differential effects of safety behaviour subtypes in social anxiety disorder. *Behaviour Research and Therapy, 49*(10), 665–675. doi:10.1016/j.brat.2011.07.005

Rafaeli, E., & Revelle, W. (2006). A premature consensus: Are happiness and sadness truly opposite affects? *Motivation and Emotion, 30*(1), 1–12. doi:10.1007/s11031-006-9004-2

Rapee, R. M., & Heimberg, R. G. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy, 35*(8), 741–756. doi:10.1016/S0005-7967(97)00022-3

Rapee, R. M., & Lim, L. (1992). Discrepancy between self- and observer ratings of performance in social phobics. *Journal of Abnormal Psychology, 101*(4), 728–731. doi:10.1037/0021-843X.101.4.728

Rapee, R. M., McCallum, S. L., Melville, L. F., Ravenscroft, H., & Rodney, J. M. (1994). Memory bias in social phobia. *Behaviour Research and Therapy, 32*(1), 89–99. doi:10.1016/0005-7967(94)90087-6

Rego, A., Sousa, F., Marques, C., & Cunha, M. P. E. (2012). Authentic leadership promoting employees' psychological capital and creativity. *Journal of Business Research, 65*(3), 429–437. doi:10.1016/j.jbusres.2011.10.003

Rinck, M., & Becker, E. S. (2005). A comparison of attentional biases and memory biases in women with social phobia and major depression. *Journal of Abnormal Psychology, 114*(1), 62–74. doi:10.1037/0021-843X.114.1.62

Rodebaugh, T. L. (2009). Social phobia and perceived friendship quality. *Journal of Anxiety Disorders, 23*(7), 872–878. doi:10.1016/j.janxdis.2009.05.001

Roelofs, K., Putman, P., Schouten, S., Lange, W.-G., Volman, I., & Rinck, M. (2010). Gaze direction differentially affects avoidance tendencies to happy and angry faces in socially anxious individuals. *Behaviour Research and Therapy, 48*(4), 290–294. doi:10.1016/j.brat.2009.11.008

Russell, J. A., & Carroll, J. M. (1999). On the bipolarity of positive and negative affect. *Psychological Bulletin, 125*(1), 3–30. doi:10.1037/0033-2909.125.1.3

Sapolsky, R. M. (2005). The influence of social hierarchy on primate health. *Science, 308*(5722), 648–652. doi:10.1126/science.1106477

Seligman, M. E. P., Alex, P., Joseph, S., & Boniwell, I. (2003). Positive psychology: Fundamental assumptions. *The Psychologist, 16*(3), 126–127.

Shaver, P. R., Morgan, H. J., & Wu, S. (1996). Is love a "basic" emotion? *Personal Relationships, 3*(1), 81–96. doi:10.1111/j.1475-6811.1996.tb00105.x

Sherbourne, C. D., Sullivan, G., Craske, M. G., Roy-Byrne, P., Golinelli, D., Rose, R. D., . . . Stein, M. B. (2010). Functioning and disability levels in primary care outpatients with one or more anxiety disorders. *Psychological Medicine, 40*(12), 2059–2068. doi:10.1017/S0033291710000176

Shiota, M. N., Campos, B., Keltner, D., & Hertenstein, M. J. (2004). Positive emotion and the regulation of interpersonal relationships. In P. Philippot & R. S. Feldman (Eds.), *The regulation of emotion* (pp. 127–155). Mahwah, NJ: Lawrence Erlbaum Associates.

Shiota, M. N., Keltner, D., & John, O. P. (2006). Positive emotion dispositions differentially associated with Big Five personality and attachment style. *The Journal of Positive Psychology, 1*(2), 61–71. doi:10.1080/17439760500510833

Shiota, M. N., Keltner, D., & Mossman, A. (2007). The nature of awe: Elicitors, appraisals, and effects on self-concept. *Cognition & Emotion, 21*(5), 944–963. doi:10.1080/02699930600923668

Shiota, M. N., Neufeld, S. L., Yeung, W. H., Moser, S. E., & Perea, E. F. (2011). Feeling good: Autonomic nervous system responding in five positive emotions. *Emotion (Washington, D.C.), 11*(6), 1368–1378. doi:10.1037/a0024278

Sparrevohn, R. M., & Rapee, R. M. (2009). Self-disclosure, emotional expression and intimacy within romantic relationships of people with social phobia. *Behaviour Research and Therapy, 47*(12), 1074–1078. doi:10.1016/j.brat.2009.07.016

Spokas, M., Luterek, J. A., & Heimberg, R. G. (2009). Social anxiety and emotional suppression: The mediating role of beliefs. *Journal of Behavior Therapy and Experimental Psychiatry, 40*(2), 283–291.

Stein, M. B., Goldin, P. R., Sareen, J., Zorrilla, L. T. E., & Brown, G. G. (2002). Increased amygdala activation to angry and contemptuous faces in generalized social phobia. *Archives of General Psychiatry, 59*(11), 1027–1034. doi:10.1001/archpsyc.59.11.1027

Sung, S. C., Porter, E., Robinaugh, D. J., Marks, E. H., Marques, L. M., Otto, M. W., . . . , Simon, N. M. (2012). Mood regulation and quality of life in social anxiety disorder: An examination of generalized expectancies for negative mood regulation. *Journal of Anxiety Disorders, 26*(3), 435–441. doi:10.1016/j.janxdis.2012.01.004

Taylor, C. T., & Amir, N. (2012). Modifying automatic approach action tendencies in individuals with elevated social anxiety symptoms. *Behaviour Research and Therapy, 50*(9), 529–536. doi:10.1016/j.brat.2012.05.004

Taylor, C. T., Bomyea, J., & Amir, N. (2010). Attentional bias away from positive social information mediates the link between social anxiety and anxiety vulnerability to a social stressor. *Journal of Anxiety Disorders, 24*(4), 403–408. doi:10.1016/j.janxdis.2010.02.004

Tiedens, L. Z., Ellsworth, P. C., & Mesquita, B. (2000). Stereotypes about sentiments and status: Emotional expectations for high- and low-status group members. *Personality and Social Psychology Bulletin, 26*(5), 560–574. doi:10.1177/0146167200267004

Tiedens, L. Z., & Fragale, A. R. (2003). Power moves: Complementarity in dominant and submissive nonverbal behavior. *Journal of Personality and Social Psychology, 84*(3), 558–568. doi:10.1037/0022-3514.84.3.558

Tiedens, L. Z., & Jimenez, M. C. (2003). Assimilation for affiliation and contrast for control: Complementary self-construals. *Journal of Personality and Social Psychology, 85*(6), 1049–1061. doi:10.1037/0022-3514.85.6.1049

Todorov, A., Said, C. P., Engel, A. D., & Oosterhof, N. N. (2008). Understanding evaluation of faces on social dimensions. *Trends in Cognitive Sciences, 12*(12), 455–460. doi:10.1016/j.tics.2008.10.001

Tracy, J. L., & Robins, R. W. (2007a). The psychological structure of pride: A tale of two facets. *Journal of Personality and Social Psychology, 92*(3), 506–525. doi:10.1037/0022-3514.92.3.506

Tracy, J. L., & Robins, R. W. (2007b). The prototypical pride expression: Development of a nonverbal behavior coding system. *Emotion (Washington, D.C.), 7*(4), 789–801. doi:10.1037/1528-3542.7.4.789

Trower, P., & Gilbert, P. (1989). New theoretical conceptions of social anxiety and social phobia. *Clinical Psychology Review, 9*(1), 19–35. doi:10.1016/0272-7358(89)90044-5

Tugade, M. M., & Fredrickson, B. L. (2004). Resilient individuals use positive emotions to bounce back from negative emotional experiences. *Journal of Personality and Social Psychology, 86*(2), 320–333. doi:10.1037/0022-3514.86.2.320

Turk, C. L., Heimberg, R. G., Luterek, J. A., Mennin, D. S., & Fresco, D. M. (2005). Emotion dysregulation in generalized anxiety disorder: A comparison with social anxiety disorder. *Cognitive Therapy and Research, 29*(1), 89–106. doi:10.1007/s10608-005-1651-1

Vassilopoulos, S. P., & Banerjee, R. (2010). Social interaction anxiety and the discounting of positive interpersonal events. *Behavioural and Cognitive Psychotherapy, 38*, 597–609.

Vernberg, E. M., Abwender, D. A., Ewell, K. K., & Beery, S. H. (1992). Social anxiety and peer relationships in early adolescence: A prospective analysis. *Journal of Clinical Child Psychology, 21*(2), 189–196. doi:10.1207/s15374424jccp2102_11

Wadlinger, H. A., & Isaacowitz, D. M. (2011). Fixing our focus: Training attention to regulate emotion. *Personality and Social Psychology Review, 15*(1), 75–102. doi:10.1177/1088868310365565

Wallace, S. T., & Alden, L. E. (1997). Social phobia and positive social events: The price of success. *Journal of Abnormal Psychology, 106*(3), 416–424. doi:10.1037/0021-843X.106.3.416

Watson, D. (2000). Basic problems in positive mood regulation. *Psychological Inquiry, 11*(3), 205–209.

Watson, D., & Clark, L. A. (1995). Depression and the melancholic temperament. *European Journal of Personality, 9*(5), 351–366. doi:10.1002/per.2410090505

Watson, D., Clark, L. A., McIntyre, C. W., & Hamaker, S. (1992). Affect, personality, and social activity. *Journal of Personality and Social Psychology, 63*(6), 1011–1025. doi:10.1037/0022-3514.63.6.1011

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*(6), 1063–1070. doi:10.1037/0022-3514.54.6.1063

Weeks, J. W. (2010). The Disqualification of Positive Social Outcomes Scale: A novel assessment of a long-recognized cognitive tendency in social anxiety disorder. *Journal of Anxiety Disorders, 24*(8), 856–865. doi:10.1016/j.janxdis.2010.06.008

Weeks, J. W., Heimberg, R. G., Rodebaugh, T. L., Goldin, P. R., & Gross, J. J. (2012). Psychometric evaluation of the Fear of Positive Evaluation Scale in patients with social anxiety disorder. *Psychological Assessment, 24*(2), 301–312. doi:10.1037/a0025723

Weeks, J. W., & Howell, A. N. (2012). The bivalent fear of evaluation model of social anxiety: Further integrating findings on fears of positive and negative evaluation. *Cognitive Behaviour Therapy,* 37–41. doi:10.1080/16506073.2012.661452

Weeks, J. W., Jakatdar, T. A., & Heimberg, R. G. (2010). Comparing and contrasting fears of positive and negative evaluation as facets of social anxiety. *Journal of Social and Clinical Psychology, 29*(1), 68–94.

Weisfeld, G. E., & Dillon, L. M. (2012). Applying the dominance hierarchy model to pride and shame, and related behaviors. *Journal of Evolutionary Psychology, 10*(1), 15–41. doi:10.1556/JEP.10.2012.1.2

Weisman, O., Aderka, I. M., Marom, S., Hermesh, H., & Gilboa-Schechtman, E. (2011). Social rank and affiliation in social anxiety disorder. *Behaviour Research and Therapy, 49*(6–7), 399–405. doi:10.1016/j.brat.2011.03.010

Werner, K. H., Goldin, P. R., Ball, T. M., Heimberg, R. G., & Gross, J. J. (2011). Assessing emotion regulation in social anxiety disorder: The Emotion Regulation Interview. *Journal of Psychopathology and Behavioral Assessment, 33*(3), 346–354. doi:10.1007/s10862-011-9225-x

Wiers, R. W., Eberl, C., Rinck, M., Becker, E. S., & Lindenmeyer, J. (2011). Retraining automatic action tendencies changes alcoholic patients' approach bias for alcohol and improves treatment outcome. *Psychological Science, 22*(4), 490–497. doi:10.1177/0956797611400615

Wiers, R. W., Rinck, M., Kordts, R., Houben, K., & Strack, F. (2010). Retraining automatic action-tendencies to approach alcohol in hazardous drinkers. *Addiction, 105*(2), 279–287. doi:10.1111/j.1360-0443.2009.02775.x

Wiggins, J. S. (1979). A psychological taxonomy of trait-descriptive terms: The interpersonal domain. *Journal of Personality and Social Psychology, 37*(3), 395–412. doi:10.1037//0022-3514.37.3.395

Wilkowski, B. M., Robinson, M. D., & Friesen, C. K. (2009). Gaze-triggered orienting as a tool of the belongingness self-regulation system. *Psychological Science, 20*(4), 495–501. doi:10.1111/j.1467-9280.2009.02321.x

Xu, J., & Roberts, R. E. (2010). The power of positive emotions: It's a matter of life or death—Subjective well-being and longevity over 28 years in a general population. *Health Psychology, 29*(1), 9–19. doi:10.1037/a0016767

20

Fear of Positive Evaluation

The Neglected Fear Domain in Social Anxiety

Justin W. Weeks and Ashley N. Howell

Ohio University, USA

In a seminal review, social anxiety disorder (SAD, i.e., social phobia) was once dubbed "the neglected anxiety disorder" (Liebowitz, Gorman, Fyer, & Klein, 1985, p. 729) due to a relative dearth of research on this disorder in comparison to the other anxiety disorders. Subsequently, a massive body of data has accumulated on the etiology, symptomology, and treatment of SAD. A hallmark feature of this disorder that has long been featured in cognitive-behavioral models (Clark & Wells, 1995; Rapee & Heimberg, 1997), and which has been well substantiated by empirical studies (Coles, Turk, Heimberg, & Fresco, 2001; Horley, Williams, Gonsalvez, & Gordon, 2004; Mansell & Clark, 1999), is the fear of negative evaluation (FNE). FNE consists of feelings of apprehension about others' negative evaluations, distress over these evaluations, and the expectation that others will evaluate one negatively (Watson & Friend, 1969). Yet, despite the enhanced empirical attention toward, and innumerable advances in research on, SAD since Liebowitz et al. called for greater attention to this disorder nearly three decades ago, there is currently a neglected fear *domain* within SAD: the fear of positive evaluation (FPE; Weeks, Heimberg, & Rodebaugh, 2008).

Weeks, Heimberg, and Rodebaugh (2008) have posited that fear of evaluation *in general* may mark the core of social anxiety, including both FNE and FPE. FPE consists of feelings of apprehension about others' positive evaluations of oneself and distress over these evaluations.

Continually accumulating evidence supports FPE as a core cognitive component of social anxiety. This evidence has led to both updated and novel cognitive-behavioral models of SAD (Heimberg, Brozovich, & Rapee, 2010; Weeks & Howell, 2012, respectively). The purposes of the present review are to: (1) present the theoretical overview of FPE; (2) discuss the assessment of FPE; (3) review evidence pertaining to the overall construct validity of FPE; (4) break down extant findings on FPE with regard to the three basic experiences emphasized within cognitive-behavioral models (i.e., cognitions, emotions/arousal, and behaviors); (5) discuss the clinical implications of FPE; and (6) provide directions for future research.

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

Theoretical Overview

Psycho-Evolutionary Considerations

Consistent with psycho-evolutionary models of social anxiety (Gilbert, 2001; see also **Chapter 2**), FPE and FNE are purported to serve distinct, adaptive goals; individuals who perceive themselves as ranking socially lower than others are motivated to: (a) avoid giving such a positive impression that they would be viewed as a threat by other members of the group (i.e., to avoid an upward shift in a social hierarchy [FPE]), while also motivated to (b) not appear so socially undesirable as to be ostracized from the group (i.e., to avoid a downward shift in a social hierarchy [FNE]) (Weeks, Rodebaugh, Heimberg, Norton, & Jakatdar, 2009). In support of this position, FPE is indicated to stem in part from concerns of social reprisal due to making positive social impressions on others (i.e., the threat of social conflict with individuals perceived to rank higher on a social hierarchy) (Weeks & Howell, 2012). In other words, socially anxious individuals feel compelled to be as inconspicuous as possible in all feared social situations, as attention of any kind (positive or negative) is perceived to threaten their social standing—if they appear "too good," others will retaliate by way of "putting them back in their place," and to maintain their own, superior social positions; appear "not good enough," and others may well oust them from the group altogether. Figure 20.1 illustrates the competing demands of FPE and FNE upon maintaining one's social status, as well as the consequences of social status change as perceived by nonsocially anxious versus socially anxious individuals. Given that positive and negative social feedback represent two distinct valences of social evaluation, we dubbed our novel conceptualization of social anxiety-related fear the *bivalent fear of evaluation* model (Weeks & Howell, 2012).

Positivity Impairment and SAD

In addition to an emerging focus on FPE as an important feature of social anxiety, other constructs related to positivity have recently received empirical attention in this area. For example, low trait levels of positive affect characterize SAD (Brown, Chorpita, & Barlow, 1998; Kashdan, 2007), and SAD is associated with low quality of life (Hambrick, Turk, Heimberg, Schneier, & Liebowitz, 2003). Furthermore, social anxiety is associated with decreased positive automatic thoughts (i.e., facilitative- or coping-oriented thoughts), as well as increased negative automatic thoughts, while engaging in and anticipating social situations (Weeks, 2010). Collectively, elements such as these constitute what has been termed *positivity impairment*, which is increasingly highlighted to be a pervasive and impairing feature of SAD (Kashdan, Weeks, & Savostyanova, 2011; Weeks & Heimberg, 2012). While a thorough review of positivity impairment as a broad-based feature of social anxiety is outside the scope of this review, these issues have been summarized elsewhere (Kashdan et al., 2011; **Chapter 19**). FPE appears to be one of many facets of positivity impairment; moreover, preliminary evidence suggests that FPE may be a possible causal factor for at least some other positivity impairments (see **Emotions and Arousal**; *Positive and negative affect: State level*).

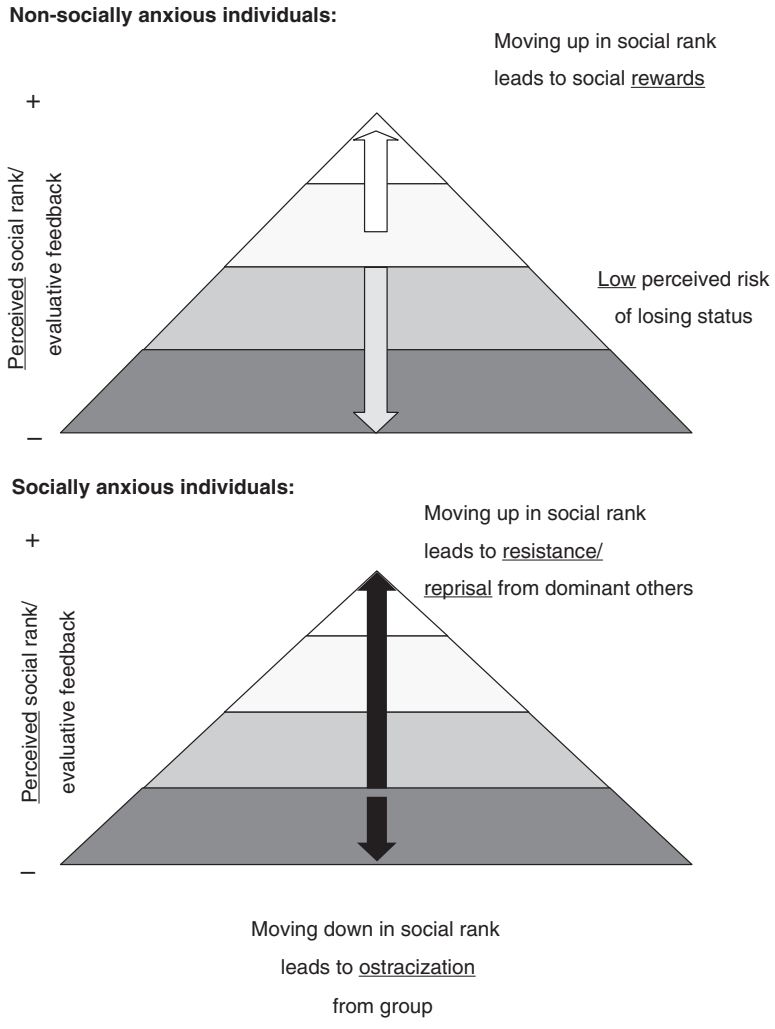

Figure 20.1 Social hierarchy dynamics model underlying fears of positive and negative evaluation within nonsocially anxious and socially anxious individuals.

Basic Properties of FPE

FPE and FNE are both evidenced to have a dimensional (as opposed to taxonic) latent structure (Weeks, Norton, & Heimberg, 2009). This suggests that FPE is not relevant for only a subset of the population/SAD patients—rather, similar to FNE, levels of FPE are continuously distributed throughout the population.

In line with this general conception, FPE shows stability across time, gender, and ethnic background. Specifically, levels of FPE remained stable for as long as 4.5 months in a waitlist control group of SAD patients (Weeks, Heimberg, Rodebaugh, Goldin, & Gross, 2012), and over a 5-week period in undergraduate samples (Weeks, Heimberg, Rodebaugh, & Norton, 2008). Furthermore, when measured across multiple weeks, FPE and FNE maintain their distinctiveness, with no evidence that one construct

prospectively causes the other over the short term (Rodebaugh, Weeks, Gordon, Langer, & Heimberg, 2012). Moreover, given that the vast majority of studies on FPE to date have been conducted in adult samples, there is also some preliminary evidence that FPE may be stable across age, in that FPE was a strong positive predictor of social anxiety symptoms in a sample of adolescents (Hirsch et al., 2011); however, the examination of FPE across varying age groups requires extensive additional research attention. With regard to gender, no significant gender differences in FPE have yet been reported (Weeks, Heimberg, and Rodebaugh, 2008; Weeks, Heimberg, et al., 2012). With regard to ethnic/racial background, a two-factor fear of evaluation model with separate factors for FPE and FNE was factorially invariant across the four major ethnic groups of the United States (Norton & Weeks, 2009). Taken together, the available evidence suggests that FPE is not only continuously distributed throughout the overall population, but that it relates stably to social anxiety across developmental and socio-cultural contexts.

Hence, consistent with our notion that social anxiety is characterized by simultaneous concerns of appearing "too good" (i.e., FPE) and "not good enough" (i.e., FNE) (Figure 20.1), we view FPE and FNE to be hand-in-hand, co-core features of social anxiety. In other words, feelings of social inferiority, combined with the natural desire to be socially included, will inherently lead to the development of both of these concerns in tandem; if not, those who feel socially inferior would (perceivedly) face either persistent retaliation/ridicule within a group, or imminent social exile (Gilbert, 2001; **Chapter 2**).

Assessment of FPE

In order to assess FPE, Weeks, Heimberg, and Rodebaugh (2008) developed the Fear of Positive Evaluation Scale (FPES); a 10-item self-report questionnaire. Given its brevity, the FPES is easily incorporable into assessment batteries, and we hope that our review will inspire greater use of this measure. The FPES has shown good internal consistency (all αs > .80) and factorial validity across undergraduate (Weeks, Heimberg, & Rodebaugh, 2008; Weeks, Jakatdar, & Heimberg, 2010) and clinical (Fergus et al., 2009; Weeks, Heimberg, et al., 2012) samples (see also **Chapter 14** for some additional psychometric details on the FPES). The construct validity of the FPES will be reviewed within the subsequent sections of our review.

Overall Construct Validity

FPE relates strongly to FNE and social anxiety across undergraduate (Weeks, Heimberg, & Rodebaugh, 2008, Weeks, Heimberg, Rodebaugh, & Norton, 2008) and clinical (Fergus et al., 2009; Weeks, Heimberg, et al., 2012) samples, providing strong support for convergent validity. Furthermore, FPE relates more strongly overall to social anxiety than to depression, generalized anxiety symptoms/worry (Fergus et al., 2009; Weeks, Heimberg, & Rodebaugh, 2008; Weeks, Heimberg, et al., 2012), panic disorder symptoms, obsessive–compulsive disorder symptoms, and anxiety sensitivity (Weeks, Heimberg, Rodebaugh, & Norton, 2008), indicating strong discriminant validity. Moreover, patients with SAD report greater FPE than nonanxious controls (Werner, Goldin, Ball, Heimberg, & Gross, 2011; Weeks, Heimberg, et al., 2012) as

well as greater FPE than patients with other anxiety disorders (Fergus et al., 2009); FPE also lowers in response to cognitive-behavioral therapy for SAD, although treatment effects are greater for FNE (Fergus et al., 2009; Weeks, Heimberg, et al., 2012).

Distinctions Between FPE and Theoretically Overlapping Constructs

Fear of negative evaluation Importantly, findings to date show that FPE is distinct from FNE. Confirmatory factor analyses have consistently supported a two-factor fear of evaluation model with separate factors for FPE and FNE in both undergraduate (Weeks, Heimberg, & Rodebaugh, 2008, Weeks et al., 2010) and clinical (Fergus et al., 2009) samples. Moreover, FPE accounts for unique variance in social anxiety above and beyond that accounted for by FNE in both undergraduate (Weeks, Heimberg, & Rodebaugh, 2008; Weeks, Heimberg, Rodebaugh, & Norton, 2008) and clinical (Fergus et al., 2009; Weeks, Heimberg, et al., 2012) samples.

An alternative viewpoint on these two socio-evaluative fears is that FPE may simply *appear* distinct from FNE, but that the effects of FPE may in fact be rooted in FNE. In other words, despite apparent distinguishing features between FPE and FNE, the relation between social anxiety and FPE could be entirely due to feared eventual negative evaluation that is anticipated as a consequence of initial positive evaluation. To illustrate, a socially anxious individual who is told that he/she made some intriguing points during a conversation might fear the possibility that the next conversation will not meet the same standard, thereby resulting in disappointment for the conversation partner, and ultimately, greater negative evaluation than might have been experienced if the first conversation had not gone well to begin with. This is in contrast to fearing positive evaluation *per se*, which would beg a direct social comparison of the self to others, thereby causing the socially anxious individual to feel conspicuous and "in the spotlight" (Weeks, Heimberg, & Rodebaugh, 2008)—this, in turn, would prompt concerns that others would perceive him/her as stealing attention during the conversation (or future conversations) that would more appropriately be garnered by those of higher social rank (Figure 20.1).

For illustrative purposes, however, assuming the *former* account, FPE would simply reflect a desire to avoid future negative evaluation. Consistent with this alternative perspective, findings by Wallace and Alden (1995, 1997) suggested that, when confronted with apparent social success, individuals with high social anxiety are likely to believe that others will expect more from them in the future. Although Wallace and Alden did not demonstrate specifically that perceived changes in social standards led to any changes in FNE, the possibility that FPE could merely represent *fear of delayed negative evaluation* was an important alternative account to our bivalent fear of evaluation model (Weeks & Howell, 2012) which required testing.

Rodebaugh et al. (2012) compared these two accounts using a longitudinal design, by examining the relations between FPE and FNE across three timepoints (each assessed once per week) in a sample of undergraduates. Competing models which were consistent with (1) our bivalent fear of evaluation model and (2) fear of delayed negative evaluation were assessed. The best-fitting model was an autoregressive latent-trajectory model in which each type of fear had *distinct* trait-like components, in line with our bivalent model; importantly, the correlation between these trait-like components appeared to fully account for the relations between FPE and FNE over time.

Furthermore, in testing the prospective relationships between FPE and FNE and social anxiety, FPE and FNE were both strongly and equally correlated with social anxiety (rs = .75 and .78, respectively) (Rodebaugh et al., 2012). These findings provided strong additional support for our bivalent fear of evaluation model, and directly countered the alternative theoretical account of *fear of delayed negative evaluation*.

This is of course not to say that fear of delayed negative evaluation may not be an important component of responses to positive feedback for socially anxious individuals. However, recalling the theoretical underpinnings of FPE (see **Theoretical Overview**), FPE is more strongly associated than FNE with concerns of social reprisal due to making positive social impressions on others (Weeks & Howell, 2012; see **Cognitions** section for additional details), and FPE is associated with distinct cognitive and emotional responses to social threat in comparison to FNE (see **Breaking Down Extant Findings: Cognitive-Behavioral Components**). Taken together, FPE and FNE are best characterized as strongly related, albeit clearly distinct, cognitive components of social anxiety.

Disqualification of positive social outcomes Disqualifying the positive (Beck, 1976) involves the rejection of positive experiences. It has been previously noted that individuals with social anxiety frequently tend to disqualify positive social experiences by minimally attributing their social success to their own ability or effort, and instead attributing their social success to external factors (Heimberg & Becker, 2002). For example, if a socially anxious individual delivers a joke that is apparently well received, he/she would likely attribute the positive social feedback to the agreeableness or politeness of those who laughed at the joke, rather than to his/her own ability to be funny.

Weeks (2010) developed a self-report questionnaire to assess disqualification of positive social outcomes (DPSO), the Disqualification of Positive Social Outcomes Scale (DPSOS; see also Vassilopoulos & Banerjee, 2010, for an alternative, recently developed measure of discounting positive interpersonal events). The DPSOS was designed to assess disqualification tendencies with regard to two distinct facets of DPSO: (a) other-oriented attributions (i.e., attributions of positive social experiences to the characteristics/experiences of others rather than to one's own abilities or effort; e.g., "People will laugh at my jokes even if they aren't funny, simply because that is the polite thing to do"); and (b) self-oriented attributions (i.e., self-targeted statements which directly reflect DPSO; e.g., "I frequently dismiss my own social successes and accomplishments").

FPE and FNE both relate strongly and positively to DPSO tendencies (both self- and other-oriented) at the latent level (Weeks, 2010), with FPE shown to relate significantly more strongly than FNE to DPSO at the level of the self (Weeks, 2010; Weeks & Howell, 2012). Importantly, this suggests that FPE may be particularly tied to such disqualifying self-statements. In fact, DPSO at the level of the self has been found to mediate the relationship between FPE and negative automatic thoughts in an analogue clinical sample (Weeks & Howell, 2012). Hence, the above findings, as well as previous cognitive-behavioral models of social anxiety, suggest that SAD may be associated with a mental representation of the self (Rapee & Heimberg, 1997) that is characterized simultaneously by *positivity-minimizing* as well as negativity-magnifying cognitive biases. And taken together, the above findings suggest that FPE may play an etiological role in these biases.

Depression As mentioned previously, FPE relates more strongly overall to social anxiety than to depression (Fergus et al., 2009; Weeks, Heimberg, & Rodebaugh, 2008, Weeks, Heimberg, et al., 2012). Moving beyond correlational evidence, in a review of various factors that hold the potential to clarify the dispositional basis of social anxiety, Naragon-Gainey and Watson (2011) recently called for multivariate analyses to examine the uniqueness of FPE (among other factors) to social anxiety with regard to depression. In response to this call, Wang, Hsu, Chiu, and Liang (2012) recently tested an intriguing hierarchical model of social interaction anxiety and depression, with the goal of illuminating both the overlap and the distinctions between these two conditions. Given previous findings involving affect and social anxiety (Brown et al., 1998), Wang et al. specified: positive and negative affect as higher-order *general* (i.e., vulnerability) factors for both social interaction anxiety and depression; FNE as a lower-order *specific* factor that would account for more variance in social interaction anxiety than depression (but which would account for significant variance in depression nevertheless); and FPE as a lower-order *unique* factor, in that Wang et al. expected that FPE would emerge as the key feature that would differentiate social interaction anxiety from depression (and thus, FPE was not expected to account for significant variance in depression).

Utilizing structural equation modeling and hierarchical regression analyses in a large Taiwanese undergraduate sample, Wang et al. (2012) obtained support for their hypothesized model. Specifically, positive and negative affect emerged as general factors, FNE was found to be a specific factor in that it had a larger effect on social interaction anxiety than on depression, and FPE emerged as a unique factor linked only to social interaction anxiety. Moreover, an alternative, competing model which specified FPE as a lower-order *specific* factor that would account for more variance in social interaction anxiety than depression, but which would account for significant variance in depression nevertheless, was ruled out, thus providing further evidence that FPE is a key differentiating factor between social anxiety and depression. Wang et al. interpreted their findings thusly: "The present findings especially highlight the importance of FPE as a unique factor in [social interaction anxiety]," and "... depressed individuals do not show FPE" (p. 321).

An important point to consider is that cultural factors may have influenced the results obtained by Wang et al. (2012), and that replication of their model in samples of varying geographical and ethnic/racial backgrounds remains essential for establishing the generalizability of their findings. On this note, although these findings are not yet published, we have recently successfully replicated the structural model of Wang et al. in a large U.S. sample of undergraduates, and utilizing responses to the same measures (Weeks, unpublished data); thus, there is preliminary evidence that their findings generalize to American (at least, to American Midwestern) samples.

Fear of success More than 40 years ago, Horner (1969) proposed the construct *fear of success,* the disposition to become anxious about achieving success due to expectations of negative consequences of succeeding, as a means of explaining putative gender differences in achievement motivation. Horner (1969) asserted that women are motivated to avoid success when they expect negative consequences (e.g., rejection by others, social isolation, feelings of being unfeminine) as a result of adopting stereotypic masculine gender roles which can facilitate occupational success

(e.g., being competitive or assertive) yet which traditionally conflict with stereotypic feminine gender roles. Fear of success has received equivocal support over the years (see Tresemer, 1976, for a review). However, given the thematic overlap between fear of success and FPE, it remained important to examine the inter-relationships between these constructs and social anxiety, in order to confirm their distinctiveness.

First, Horner's (1969) hypothesis that significant gender differences exist with respect to fear of success marks a critical distinction between the constructs of fear of success and FPE. No *a priori* reason existed to suggest that FPE would vary by gender and, as noted previously, no gender differences in FPE have been reported to date. More importantly, Weeks et al. (2010) found that FPE related significantly more strongly to social anxiety than to fear of success, and this was a very large effect. Thus, FPE is indicated to indeed be distinct from fear of success.

Preference for negative feedback Valentiner, Skowronski, McGrath, Smith, and Renner (2011) have obtained some interesting findings demonstrating inter-relationships between social anxiety, low social self-esteem, and a *preference for negative feedback* about one's social worth. Valentiner et al. have interpreted their findings as being consistent with *self-verification theory* (Swann, 1983), which asserts that individuals tend to seek out, prefer, and elicit information that is congruent with their self-image and tend to reject and fail to process information that is inconsistent with that image. This preference for congruent information purportedly exists even when one's self-image is negative, as is generally the case with social anxiety (Leary & Kowalski, 1995). Could FPE simply reflect a tendency to reject positive social feedback/prefer negative social feedback?

There is now evidence from multiple studies and across independent research teams that FPE is distinct from the tendency to prefer negative feedback. Weeks et al. (2010) found that FPE (and FNE) emerged as distinct at the latent level from preferences for either negative or positive feedback in a large undergraduate sample. Furthermore, latent correlations among these factors revealed that FPE related negatively to preferences for either negative or positive feedback, and that FNE related significantly and negatively to a preference for negative feedback—taken together, these findings suggest that those with high fear of evaluation in general prefer not to be evaluated at all, be it negatively or positively. It bears noting that this finding is entirely consistent with our position that socially anxious individuals essentially prefer to be as inconspicuous as possible at all times (see **Basic Properties of FPE**). Furthermore, FPE related more than twice as strongly to FNE than to either preference for feedback, providing further support for the distinction between FPE and negative feedback preference.

Valentiner et al. (2011) found that, in a clinical sample treated with cognitive-behavioral group therapy (comprised of general outpatients with anxiety disorders, approximately 70% of whom were diagnosed with SAD), preference for negative feedback related positively to FPE and FNE at both pre- and post-treatment. It is worth noting here that the difference in the directions of relationships between FPE, FNE, and preference for negative feedback reported by Weeks et al. (2010) and Valentiner et al. is likely due to the use of a forced-choice response measure in the latter study; according to the findings of Valentiner et al., it appears that if socially anxious individuals feel that they *must* choose between receiving either positive or negative

feedback, they tend to prefer negative feedback. More importantly for the purposes of our review, however, Valentiner et al. found that both FPE and FNE improved by post-treatment in this sample, whereas preference for negative feedback did not improve; Valentiner et al. interpreted this finding as highlighting the specificity between FPE (and FNE) from negative feedback preference, in that cognitive-behavioral therapy effectively lowered FPE and FNE (Weeks, Heimberg, et al., 2012), but does not appear to effectively target preference for negative social feedback.

Breaking Down Extant Findings: Cognitive-Behavioral Components

In order to facilitate the integration of findings on FPE to date with other cognitive-behavioral models of social anxiety (Clark & Wells, 1995; Heimberg et al., 2010; Hofmann, 2007), we will now review these extant findings with regard to the three basic experiences emphasized within these models: cognitions, emotions/arousal, and behaviors.

Cognitions

Disqualification of positive social outcomes As reviewed above, FPE has been shown to be distinct from, albeit strongly related to, disqualification of positive social outcomes (see **Distinctions Between FPE and Theoretically Overlapping Constructs**). Indeed, FPE was found to lead to increased disqualification of positive social outcomes at the level of the self which, in turn, led to increased negative automatic thoughts in typical social situations (Weeks & Howell, 2012). A key principle within our bivalent fear of evaluation model of social anxiety is that FPE may *cause* disqualification of positive social outcomes, which may in turn serve as a mental safety behavior (Wells et al., 1995) in the face of the threat of positive evaluation. Providing some support for the premise that DPSO is a mental safety behavior for FPE-related state anxiety, Weeks, Heimberg, Rodebaugh, and Norton (2008) had an unselected undergraduate sample take part in a bogus personality test. Upon completing the "test," all participants were provided with 10 (identical) positive statements (e.g., "People assume that you have many talents"), as though each of these statements was tailored feedback to the participants on the basis of the personality test. Participants were informed that the purpose of this study was to improve the alleged personality test; as part of this pretense, participants were asked to provide state ratings of the perceived accuracy of each statement, and (as an alleged independent index of the "accuracy" of the personality profile) discomfort experienced in response to each statement.

In support of the DPSO-mental safety behavior hypothesis, trait FPE led to increased state discomfort/anxiety in response to receipt of the positive social feedback, which in turn led to decreased perceived accuracy of the positive social feedback—in other words, it appeared that participants with higher levels of FPE were significantly discomforted by positive feedback, and accordingly, interpreted those positive statements as less true of them. These mediational findings are in strong accord with the notion that disqualification of the positive is an active effort on the

part of socially anxious individuals to manage threat in the form of positive evaluation (Weeks, Heimberg, Rodebaugh, & Norton, 2008). Essentially, if one is concerned about appearing "too good," one may convince oneself that he/she is not very good as a means of allaying this fear.

Social comparison and concerns of social reprisal As discussed above, FPE is posited to be rooted in the tendency to perceive social interactions within the context of a dominance hierarchy, as well as in concerns of social reprisal due to making positive social impressions (Figure 20.1). In support of these premises, FPE (and FNE) relate negatively to social self-rankings (Weeks et al., 2010). Moreover, confirmatory factor analyses in a large undergraduate sample revealed that FPE related significantly more strongly at the latent level to concerns of social reprisal due to positive impressions on others than did FNE. Furthermore, FPE related uniquely to such concerns of social reprisal even upon accounting for shared variance from FNE and social anxiety in general; in fact, social anxiety did not even relate significantly to concerns of social reprisal due to positive impressions upon accounting for shared variance from FPE and FNE. It is worth noting that FNE also related uniquely (with regard to FPE and social anxiety) to concerns of social reprisal due to positive social impressions in this study, albeit less than half as strongly as did FPE.

Positive and negative automatic thoughts An intriguing positivity deficit (Kashdan et al., 2011; Weeks & Heimberg, 2012) associated with social anxiety, in light of the near-exclusive focus within contemporary models and studies of social anxiety (Clark & Wells, 1995; Rapee & Heimberg, 1997) upon elevated negative automatic thoughts, is that high levels of social anxiety are also associated with decreased positive automatic thoughts (i.e., facilitative-, or coping-oriented thoughts) (Weeks, 2010). Even more interesting is that, whereas FPE, FNE, and social anxiety all related uniquely and positively to negative automatic thoughts in typical social situations, FPE and FNE alone related uniquely and negatively to the experience of positive automatic thoughts. This suggests that fears of evaluation may account for this particular positivity deficit. Perhaps it is the case that FPE leads to decreased positive automatic thoughts due to the mental safety behavior of disqualification of positive social outcomes; in contrast, FNE may lead to decreased positive thoughts due to an over-emphasis on negative thinking (i.e., direct anticipation of negative outcomes), which could cancel out the counter-experience of facilitative, coping thinking. This is an interesting area for future research.

Emotions and Arousal

Positive and negative affect: Trait level As reviewed above, SAD is characterized by low trait positive affect as well as high trait negative affect (Brown et al., 1998; Kashdan, 2007). Consistent with our position that FPE is a co-core cognitive component of SAD, this same pattern holds for FPE and FNE. Weeks et al. (2010) modified the instructions of the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) to assess affect specifically tied to typical social interactions, and we have subsequently used this modified version of the PANAS to directly test our bivalent fear of evaluation model (Weeks & Howell, 2012). In both of these studies (Weeks

et al., 2010; Weeks & Howell, 2012), both FPE and FNE related uniquely (regarding the opposing valence of fear of evaluation) and negatively to social interaction-specific positive affect, and uniquely and positively to social interaction-specific negative affect. Interestingly, these relations for FPE remained robust even when accounting for the effects of social anxiety; in contrast, upon controlling for the effects of social anxiety, FNE maintained a significant relationship only with negative, and not with positive, affect (Weeks & Howell, 2012). Taken together with the findings reviewed in the previous section involving positive and negative automatic thoughts, we posited that, at least at the trait level, FPE may be more specific to SAD-related positivity deficits (e.g., reduced positive thoughts and feelings experienced in typical social situations) than FNE or social anxiety are to SAD-related negativity surfeits (e.g., excessive negative thoughts and feelings). Our contention here is consistent with the findings of Wang et al. (2012) highlighting FPE as a unique factor of social interaction anxiety, whereas FNE and positive/negative affect appear to cut across social anxiety and depression. But, does this pattern hold for state levels of positive and negative affect as well?

Positive and negative affect: State level In a very recent study, Weeks and Zoccola (2013) examined FPE- and FNE-associated state responses to an impromptu speech task in a sample of undergraduates (see *Physiological arousal* for additional findings by Weeks and Zoccola [2013]). Participants delivered a 5-min speech on why they would be an ideal candidate for a hypothetical job. State positive and negative affect (as well as state anxiety) during the speech were compared to state responses during a 3-min pre-speech relaxation period, in order to assess changes in response to social threat. Consistent with previous findings (Brown et al., 1998; Kashdan, 2007), FPE and FNE both related positively to negative affect, and negatively to positive affect at the bivariate level. However, multivariate repeated measures analyses revealed that FPE alone related uniquely to changes in positive affect in response to the speech (but not to negative affect [or state anxiety]), whereas FNE alone related uniquely to changes in negative affect (and state anxiety), but not to positive affect.

Interestingly, positive affect varied as a function of the interaction between FPE level and speech phase, such that high levels of FPE were associated with low (maintained) positive affect both prior to and in response to the speech, whereas low and moderate levels of FPE were associated with significant *increases* in positive affect. Furthermore, pre-speech state anxiety fully mediated the relationship between FPE and diminished positive affect during the speech. In other words, higher FPE appeared to cause higher anticipatory state anxiety, which subsequently caused the experience of positive affect during the speech to be dampened. Taken together, these findings suggest that higher FPE may prevent a *normative increase* in positive affect that would otherwise be experienced while socially engaged (Weeks & Zoccola, 2013). Put another way, high FPE may diminish the positive feelings/rewarding nature of social connectedness that seems intrinsic for those without this fear. Hence, FPE may not only be one of many facets of positivity impairment within SAD (Kashdan et al., 2011; Weeks & Heimberg, 2012), but there is preliminary evidence that FPE may also be a causal factor for other SAD-related positivity impairments (e.g., dampened positive emotional experiences in social situations).

State anxiety FPE has been linked to elevated state anxiety in response to positive social feedback in a number of studies to date. For example, as noted previously, Weeks, Heimberg, Rodebaugh, and Norton (2008) reported that FPE was associated with increased state anxiety in response to receiving a positive (bogus) personality profile.

Furthermore, regarding the study by Weeks and Zoccola (2013; see *Positive and negative affect*), FPE and FNE both related positively to state anxiety, both in anticipation of and during the speech. In fact, FPE related significantly more strongly ($z = 2.02$, $p = .04$) to state anxiety prior to the speech ($r = .42$) than did FNE ($r = .25$), whereas FPE and FNE related equivalently to state anxiety during the speech (FPE: $r = .39$; FNE: $r = .43$). Although FPE was not found to relate to *changes* in state anxiety in response to the speech, it is interesting to note that the above findings suggest that this was due to a ceiling effect for the impact of FPE upon state anxiety in response to social threat in this particular study, in that FPE related equivalently and moderately to state anxiety both in anticipation of and during the speech. Indeed, recalling that disqualification of positive social outcomes may serve as a mental safety behavior for FPE (Weeks, 2010), in interpreting their findings, Weeks and Zoccola noted that their participants could not overtly disqualify positive social outcomes while holding to the instructions of their speech task, as doing so would have run counter to making a case for their being an "ideal" job candidate. In line with this, mediational results suggested that FPE caused an especially strong increase in anticipatory state anxiety, presumably because participants high in FPE could not rely on typical safety behaviors for managing their response to the threat of appearing ideal, and this increase in anticipatory anxiety caused positive affect to be dampened while socially engaged.

We recently conducted a study examining the relationship between gaze avoidance and SAD symptoms in response to a computerized social interaction simulation (Weeks, Howell, & Goldin, 2013). Consistent with our bivalent fear of evaluation model, it was expected that patients with SAD, in comparison to nonsocially anxious control participants, would exhibit increased state anxiety in response to videos simulating *both* positive and negative social feedback (see also **Behavior**, regarding gaze avoidance effects). The simulation task consisted of viewing 26 dynamic videos (13 positive and 13 negative), each 12 s in duration, with statements delivered by actors on the screen as if "to the participant" (e.g., "You seem very smart"). Indeed, SAD patients experienced nearly twice the amount of state anxiety in response to the positive videos in comparison to controls, a large effect. Moreover, we have since replicated this effect in an ongoing extension study using the same paradigm, but with patients who specifically qualified for principal SAD of the generalized subtype and demographically matched healthy controls (Weeks, Howell, Srivastav, & Goldin, 2012); in our extension study, we have obtained an even larger effect, in that state anxiety in response to positive social stimuli was nearly three times higher for SAD patients than for healthy controls.

Carter, Sbrocco, Riley, and Mitchell (2012) conducted an intriguing study in an undergraduate sample involving experimental manipulation, wherein all participants delivered a 3-min, videotaped speech that they were led to believe would be rated by faculty judges. On the basis of random assignment, participants then received either (a) positive or (b) negative bogus feedback on the performance of their speech, or

(c) no feedback, and were subsequently asked to re-deliver their speech directly to the panel of judges. For the first speech, FNE alone related uniquely and positively to state anxiety during the speech task. However, for the second speech, regardless of feedback type (positive, negative, or neutral), FPE alone related uniquely and positively to state anxiety during the speech task. Carter et al. concluded that FPE and FNE are distinct predictors of state anxiety in response to social threat.

Although Carter et al. (2012) did not find FPE to be uniquely associated with state anxiety during the first speech in their study, it is worth noting that all participants delivered a speech on a perceived *negative* aspect of their own body. Recalling that Weeks and Zoccola (2013) found that higher FPE was consistently associated with higher state anxiety during a speech focusing on the self as *ideal*, and posited that presenting oneself as ideal inherently prevented participants from engaging in the mental safety behavior of disqualifying the positive, the speech topic utilized by Carter et al. appears to hold important implications for their findings. It seems probable that focusing on a negative aspect of oneself during a speech would naturally enhance disqualification of positive social outcomes, which we would expect to attenuate the impact of FPE upon state anxiety experiences (Weeks, 2010; Weeks, Heimberg, Rodebaugh, & Norton, 2008). Unfortunately, Carter et al. did not report the relationship between FPE and state anxiety during the first speech; thus, the strength of this relationship was not available for interpretation. Carter et al. went on to note, however, that participants may have felt better able to deliver their speech the second time due to rehearsal effects, and that this could have resulted in participants fearing positive evaluation due to improved public speaking; this is an interesting interpretation, and could explain why FPE alone accounted for anxiety during the second speech (see *Physiological arousal* for additional findings by Carter et al.). We find Carter and colleagues' findings to be an intriguing extension of this literature, and we recommend that researchers take disqualification of positive social outcomes into consideration when conducting future studies on FPE; doing so can only further inform this area of research.

Physiological arousal In the study by Carter et al. (2012), higher FPE and FNE were both uniquely associated with greater subjective somatic responses (e.g., pounding heart, dizziness, feeling hot or flushed, feeling like passing out) during the public speaking task. It is interesting that higher FPE was associated with subjective physiological arousal in this study, given that the assigned topic of the speech was focused on a perceived *negative* aspect of the participants' body. If FPE is associated with greater (perceived) physiological arousal when expectations of evaluation were likely to be negative, these effects would be expected to be even greater when expectations of evaluation were likely to be positive.

Indeed, in the recent study by Weeks and Zoccola (2013), FPE uniquely predicted (relative to FNE and social anxiety) increases in mean heart rate during their speech task relative to the pre-speech relaxation condition; in contrast, neither FNE nor social anxiety overall related to changes in heart rate. Moreover, heart rate increases were greatest for those participants who endorsed higher FPE, suggesting that FPE may be associated with pathophysiological responses to social threat, particularly in situations in which socially anxious individuals are unable to disqualify the positive in order to temper the effects of FPE.

Behavior

Avoidance of social interactions and performance situations One of the gold standard measures for social avoidance is the avoidance subscale of the Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987; see **Chapter 13** for details). The LSAS was designed to assess avoidance (as well as fear) in social interaction and performance situations. Weeks, Heimberg, et al. (2012) reported findings on LSAS total scores in a multisite clinical sample; all of the participants in this study were patients who were assigned a principal diagnosis of SAD (see also **Clinical Implications of Fear of Positive Evaluation** for additional findings from this study). However, for the purposes of this section of our review, we re-analyzed the clinician-administered LSAS responses from participants in this study, focusing exclusively on the social avoidance subscales.

In this clinical sample, FPE related strongly to both the social interaction ($r = .62$) and performance ($r = .59$) avoidance subscales of the LSAS, indicating that FPE is indeed related to clinically-severe social avoidance. Moreover, FPE and FNE related equivalently to both LSAS avoidance subscales (both zs < 0.74, both ps $> .45$), indicating that FPE and FNE may play equally important roles in socially avoidant behavior (Heimberg, unpublished data).

Self-reported submissive behaviors Recalling the theoretical underpinnings of FPE, socially anxious individuals tend to perceive social situations within the context of a dominance hierarchy (Gilbert, 2001; **Chapter 2**; Figure 20.1). As a corollary, FPE should logically correspond with submissive displays, as social anxiety-related submissive gestures are proposed to de-escalate competition between individuals and facilitate group cohesion across social ranks (Gilbert, 2001; Weeks, Heimberg, & Heuer, 2011). Indeed, FPE relates strongly and positively to self-reported submissive behaviors (Weeks et al., 2010; Weeks, Heimberg, Rodebaugh, & Norton, 2008). In fact, this effect remains quite robust even upon controlling for FNE (Weeks et al., 2010). These findings uphold our contention that FPE and FNE are tied to perceptions of inferiority, which then leads to social submissiveness.

Objectively assessed submissive behavior: Gaze avoidance As noted previously, we have recently (Weeks et al., 2013) tested our bivalent fear of evaluation model with regard to what is perhaps the most prominent of social anxiety-related submissive gestures: eye gaze avoidance (Horley et al., 2004). We examined the relationship between gaze avoidance and SAD symptoms, via computerized eye tracking of gaze tendencies in response to a dynamic social interaction simulation (see *State anxiety* section above for details on our social simulation). Consistent with our bivalent model, SAD patients exhibited global gaze avoidance (with regard to "eye contact" with the actors in the simulation videos, as indexed by total time holding eye gaze, number of fixation durations upon the eyes of the actors, and the durations of these fixations) in response to both the positive and negative video clips in comparison to the controls. Moreover, the SAD group exhibited equivalent gaze avoidance in response to stimuli of both emotional valences (Weeks et al., 2013), indicating that SAD patients tend to avoid eye contact with others regardless of the valence of emotional cues being received

from those others. Moreover, we have since replicated this effect in an ongoing extension study, using the same paradigm, but with patients who specifically qualified for diagnoses of principal SAD of the generalized subtype and demographically-matched healthy controls (Weeks, Heimberg, et al., 2012); in this extension study, we have obtained even larger effects. Our findings suggest that high FPE leads to gaze avoidance with pleasant others, whereas high FNE leads to gaze avoidance with unpleasant others, presumably as a means of remaining as inconspicuous as possible, and maintaining one's social standing through the act of submissiveness (see Gilbert, 2001; **Chapter 2**).

Summary

In sum, increasingly accumulating evidence highlights relations between FPE and experiences on the cognitive, emotional/physiological arousal, and behavioral levels which largely parallel those found for social anxiety and FNE. Importantly, however, FPE accounts for unique variance in the majority of these experiences above and beyond that accounted for by FNE (and indeed, in some cases, even above and beyond that accounted for by social anxiety itself [Weeks & Zoccola, 2013]). Furthermore, on the whole, FPE appears to relate more uniquely to positivity deficits than FNE and social anxiety relate to negativity surfeits (Weeks & Howell, 2012; Weeks et al., 2010), although this rather broad statement appears to hold more consistently at the trait level than at the state level (see *State anxiety* effects; Weeks & Zoccola, 2013).

Clinical Implications of Fear of Positive Evaluation

By this point in our review, we expect that the clinical implications of FPE have begun to become clear. Given continually-accumulating evidence that FPE is a core cognitive component of SAD, and that existing treatments for SAD do not systematically target FPE, this raises the question of whether doing so would result in enhanced treatment gains for SAD patients. Indeed, according to the findings reviewed above, it seems possible – and perhaps even probable – that therapists are currently only systematically targeting half of the domain of fear that is experienced by this patient population in focusing only on FNE.

Patients with SAD report greater FPE than nonanxious controls (Weeks, Heimberg, et al., 2012; Werner et al., 2011) and patients with other anxiety disorders (Fergus et al., 2009). Furthermore, FPE related positively to SAD-related disability, and negatively to quality of life, in a large sample of SAD patients (Weeks, Heimberg, et al., 2012). The latter finding suggests that cognitive-behavioral treatment protocols which systematically target FPE could result not only in reduced social anxiety symptoms overall, but could also enhance patients' ability to experience enhanced quality of life, thereby further augmenting clinically significant treatment outcomes.

Given the implications of systematically targeting FPE, an important preliminary question is posed: do existing treatments for SAD demonstrate efficacy in lowering FPE? Indeed, FPE lowers in response to exposure therapy (Fergus et al., 2009) and cognitive-behavioral therapy for SAD (Weeks, Heimberg, et al., 2012), and both of

these effect sizes were large (see also Valentiner et al., 2011, for findings in a general outpatient sample consisting of patients diagnosed with various anxiety disorders). However, a crucial point here is that effect sizes pertaining to FPE reductions in SAD samples are smaller than those pertaining to FNE (see Fergus et al., 2009; Weeks, Heimberg, et al., 2012), highlighting room for improvement in existing treatments for reducing clinically-severe FPE (see below for details). It is further worth noting that Fergus et al. and Valentiner et al. did report having utilized in-session exposures which focused on receipt of positive (e.g., compliments, applause, admiration) as well as negative (e.g., jeering, booing) feedback, indicating that their treatment approach did directly target FPE; however, the typical frequency with which the positive-evaluative exposures were assigned per patient was not reported, and thus, was likely to have been administered in a non-systematic fashion across patients. No data is yet available regarding the effects of pharmacotherapy for FPE.

Taken together, the findings we have reviewed throughout our chapter highlight several possible refinements to existing treatment protocols for SAD patients. First, the findings of Fergus et al. (2009) suggest that positive feedback-oriented exposures may be an important component for systematic incorporation into standard treatment for SAD patients. Second, given that: (a) disqualification of positive social outcomes is frequently reported by patients receiving therapy for SAD and has been identified by clinicians as an obstacle to symptom improvement in psychotherapy (Heimberg & Becker, 2002); (b) that DPSO is evidenced to serve as a mental safety behavior in response to FPE (Weeks, Heimberg, Rodebaugh, & Norton, 2008; Weeks & Howell, 2012); (c) and that use of safety behaviors has been linked to maintained social anxiety symptoms and negative beliefs about social situations (Wells et al., 1995), this suggests that more enhanced targeting of DPSO is called for in SAD treatment. For example, rather than focusing on it as simply one of many thinking errors to which SAD patients are prone to experiencing (e.g., in cognitive-behavioral therapy; see Hope, Heimberg, Juster, & Turk, 2000), perhaps the incorporation of a concentrated module focusing expressly on psychoeducation and cognitive work which targets DPSO would better prepare patients to engage in exposures that yield outcomes that are perceived positively by patients (Weeks, 2010). This seems particularly likely given that existing treatments for SAD do not include a targeted approach to FPE.

Third, in addition to the possibility that the failure to systematically address FPE may leave half of the domain of social fear untreated for SAD patients, it is also likely the case that failure to do so serves to undercut the overarching treatment of FNE and social anxiety in general. To illustrate, without psychoeducation on FPE and targeted cognitive restructuring to address FPE-related thoughts, SAD patients may in fact experience clinically significant distress during the debriefing of successful exposures, due to their therapists' positive feedback pertaining to their very success in treatment! This rather paradoxical result would seem to at best be confusing to patients, given that they may not understand why "doing well" in the treatment would lead to greater anxiety; and at worst, perceived to be a barrier to continued treatment which could ultimately lead to patient dropout. Last, we recommend routine assessment of FPE (Weeks, Heimberg, & Rodebaugh, 2008) and possibly DPSO (Weeks, 2010) at intake and prior to termination, as results from these assessments may meaningfully inform sessions, and provide a means of evaluating whether FNE and FPE are both

satisfactorily reduced prior to termination—ideally, both core domains of fear would be in the nonclinical range in order for treatment to be deemed successful.

One final intriguing clinical implication involving FPE pertains to recent findings involving self-compassion. Self-compassion has been defined as having a warm and accepting stance toward the aspects of oneself that are disliked (Neff, 2003), and is closely related to mindfulness (Werner et al., 2011). In light of the increased focus on self-compassion and mindfulness within mindfulness- and acceptance-based interventions for SAD (see **Chapter 27** for a thorough review), Werner et al. (2011) recently reported that greater FPE (and FNE) related to lower overall self-compassion. More specifically, regarding distinct facets of self-compassion (Neff, 2003), both FPE and FNE were associated with increased *self-judgment*, perceived *isolation*, and *over-identification* (i.e., when one becomes overwhelmed with negative emotion and identifies with it). Interestingly, however, FPE alone related inversely to *self-kindness* and *mindfulness*; in contrast, social anxiety and FNE exhibited no significant relations with these latter two constructs. The findings of Werner et al. highlight an additional possible positivity deficit associated with FPE, in that self-compassion is evidenced to be a potentially adaptive coping response to social anxiety (Leary, Tate, Adams, Allen, & Hancock, 2007). Given that FPE appears to relate more strongly to self-compassion as a whole than either FNE or social anxiety in general, the findings of Werner et al. may highlight a novel utility of mindfulness- and acceptance-based interventions for SAD—namely, their specific potential for treating clinically severe FPE. This is an interesting area for future research.

Directions for Future Research

Our review highlights a number of important areas for future research on FPE. First, although a good number of studies have been conducted on this socio-evaluative fear to date, the vast majority of these studies, as is evident from our review, have been conducted in adult samples from the United States. It remains essential to examine whether the findings reviewed generalize to children, adolescents, and older adults. In addition, although researchers have recently begun testing hypotheses pertaining to FPE in non-American samples (Weeks, Heimberg et al., 2012), given that FPE is evidenced to be an important feature of social anxiety, cross-cultural research in this area appears warranted. This seems particularly important given that the prevalence and expression of social anxiety/SAD symptoms appears to depend upon cultural context (Hofmann, Asnaani, & Hinton, 2010).

Second, continued examination of FPE via multimodal assessment is necessary to better understand this construct and its role in social anxiety—additional studies assessming state-based and behavioral outcomes associated with FPE would be particularly informative. Furthermore, additional experimental designs, in which social feedback of varying valence to participants (i.e., positive, negative, and neutral) is manipulated (Carter et al., 2012) are essential, as such manipulations will allow for the causal effects underlying FPE to be more clearly delineated. In addition, further studies assessing FPE (along with related variables; e.g., FNE, social anxiety) within a longitudinal framework, possibly through the utilization of ecological momentary

assessment, would serve to further inform whether FPE indeed remains relatively stable over the course of time (as our bivalent fear of evaluation model and current data [Rodebaugh et al., 2012] would suggest), or if it may fluctuate over time; for example, in response to the valence of immediate social outcomes.

Finally, as highlighted in the last section, a number of important clinical avenues will be important to pave through translational research. Most notably, clinical trials evaluating whether the systematic incorporation of FPE-based exposures into SAD treatment will significantly augment treatment outcomes appear particularly called for. In addition, studies examining FPE as a potential barrier to seeking services and/or risk factor for premature treatment termination would be informative.

Overall Summary

In conclusion, continually accumulating evidence supports FPE as a core cognitive component of SAD. Fear of evaluation *in general* appears to lie at the heart of social anxiety, and spans both extremes, from positive to negative social evaluations. Despite that FPE and FNE are indicated to play equally important roles in this highly prevalent and debilitating disorder, research addressing FPE is relatively lacking. We hope that our review generates additional interest and efforts on the part of clinical scientists and practitioners in better addressing this neglected fear domain within SAD.

References

Beck, A. T. (1976). *Cognitive therapy and the emotional disorders.* New York, NY: International Universities Press.

Brown, T. A., Chorpita, B. F., & Barlow, D. H. (1998). Structural relationships among dimensions of the DSM-IV anxiety and mood disorders and dimensions of negative affect, positive affect, and autonomic arousal. *Journal of Abnormal Psychology, 107*, 179–192. doi:10.1037/0021-843X.107.2.179

Carter, M. M., Sbrocco, T., Riley, S., & Mitchell, F. E. (2012). Comparing fear of positive evaluation to fear of negative evaluation in predicting anxiety from a social challenge. *Journal of Experimental Psychopathology, 3*(5), 782–793. doi:10.5127/jep.022211

Clark, D. M., & Wells, A. (1995). A cognitive model of social phobia. In R. G. Heimberg, M. R. Liebowitz, D. A. Hope, & F. R. Schneier (Eds.), *Social phobia: Diagnosis, assessment, and treatment* (pp. 69–93). New York, NY: Guilford Press.

Coles, M. E., Turk, C. L., Heimberg, R. G., & Fresco, D. M. (2001). Effects of varying levels of anxiety within social situations: Relationship to memory perspective and attributions in social phobia. *Behaviour Research and Therapy, 39*, 651–665. doi:10.1016/S0005-7967(00)00035-8

Fergus, T. A., Valentiner, D. P., McGrath, P. B., Stephenson, K., Gier, S., & Jencius, S. (2009). The Fear of Positive Evaluation Scale: Psychometric properties in a clinical sample. *Journal of Anxiety Disorders, 23*, 1177–1183. doi:10.1016/j.janxdis.2009.07.024

Gilbert, P. (2001). Evolution and social anxiety: The role of attraction, social competition, and social hierarchies. *The Psychiatric Clinics of North America, 24*, 723–751. doi:10.1016/S0193-953X(05)70260-4

Hambrick, J. P., Turk, C. L., Heimberg, R. G., Schneier, F. R., & Liebowitz, M. R. (2003). The experience of disability and quality of life in social anxiety disorder. *Depression and Anxiety, 18*, 6–50. doi:10.1002/da.10110

Heimberg, R. G., & Becker, R. E. (2002). *Cognitive-behavioral group therapy for social phobia: Basic mechanisms and clinical strategies* (pp. 176–198). New York, NY: Guilford Press.

Heimberg, R. G., Brozovich, F. A., & Rapee, R. M. (2010). A cognitive-behavioral model of social anxiety disorder: Update and extension. In S.G. Hofmann & P. M. DiBartolo (Eds.), *Social anxiety: Clinical, developmental, and social perspectives* (2nd ed., pp. 395–422). New York, NY: Elsevier. doi:10.1016/B978-0-12-375096-9.00028-6

Hirsch, E., Stewart, C. E., Lackovic, S., Fox, J. K., Colognori, D., Ryan, J. L., . . . Warner, C. M. (2011, November). *The Fear of Positive Evaluation Scale in a community sample of adolescents: Psychometric properties and relationship to social anxiety.* Poster presented at the annual meeting of the Association for Behavioral and Cognitive Therapies, Toronto, Canada.

Hofmann, S. G. (2007). Cognitive factors that maintain social anxiety disorder: A comprehensive model and its treatment implications. *Cognitive Behaviour Therapy, 36*, 193–209. doi:10.1080/16506070701421313

Hofmann, S. G., Asnaani, A., & Hinton, D. E. (2010). Cultural aspects in social anxiety and social anxiety disorder. *Depression and Anxiety, 27*, 1117–1127. doi:10.1002/da.20759

Hope, D. A., Heimberg, R. G., Juster, H. R., & Turk, C. L. (2000). *Managing social anxiety: A cognitive-behavioral therapy approach.* New York, NY: Oxford University Press.

Horley, K., Williams, L. M., Gonsalvez, C., & Gordon, E. (2004). Face to face: Visual scanpath evidence for abnormal processing of facial expressions in social phobia. *Psychiatry Research, 127*, 43–53. doi:10.1016/j.psychres.2004.02.016

Horner, M. (1969). Fail: Bright women. *Psychology Today, 3*, 36–38.

Kashdan, T. B. (2007). Social anxiety spectrum and diminished positive experiences: Theoretical synthesis and meta-analysis. *Clinical Psychology Review, 27*, 348–365. doi:10.1016/j.cpr.2006.12.003

Kashdan, T. B., Weeks, J. W., & Savostyanova, A. (2011). Whether, how, and when social anxiety shapes positive experiences and events: A self-regulatory framework and treatment implications. *Clinical Psychology Review, 31*(5), 786–799. doi:10.1016/j.cpr.2011.03.012

Leary, M. R., & Kowalski, R. M. (1995). *Social anxiety.* New York, NY: Guilford.

Leary, M. R., Tate, E. G., Adams, C. E., Allen, A. B., & Hancock, J. (2007). Self-compassion and reactions to unpleasant self-relevant events: The implications of treating oneself kindly. *Journal of Personality and Social Psychology, 92*, 887–904. doi:10.1037/0022-3514.92.5.887

Liebowitz, M. R. (1987). Social phobia. *Modern Problems in Pharmacopsychiatry, 22*, 141–173.

Liebowitz, M. R., Gorman, J. M., Fyer, A. J., & Klein, D. F. (1985). Social phobia: Review of a neglected anxiety disorder. *Archives of General Psychiatry, 42*, 729–736. doi:10.1001/archpsyc.1985.01790300097013

Mansell, W., & Clark, D. M. (1999). How do I appear to others? Social anxiety and processing of the observable self. *Behaviour Research and Therapy, 37*, 419–443. doi:10.1016/S0005-7967(98)00148-X

Naragon-Gainey, K., & Watson, D. (2011). Clarifying the dispositional basis of social anxiety: A hierarchical perspective. *Personality and Individual Differences, 50*, 926–934. doi:10.1016/j.paid.2010.07.012

Neff, K. D. (2003). Self-compassion: An alternative conceptualization of a healthy attitude toward oneself. *Self and Identity, 2*, 85–102. doi:10.1080/15298860309032

Norton, P. J., & Weeks, J. W. (2009). A multi-ethnic examination of socio-evaluative fears. *Journal of Anxiety Disorders, 23*, 904–908. doi:10.1016/j.janxdis.2009.05.008

Rapee, R. M., & Heimberg, R. G. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy, 35*, 741–756. doi:10.1016/S0005-7967(97)00022-3

Rodebaugh, T. L., Weeks, J. W., Gordon, E. A., Langer, J. K., & Heimberg, R. G. (2012). The longitudinal relationship between fears of positive and negative evaluation. *Anxiety, Stress, and Coping, 25*, 167–182. doi:10.1080/10615806.2011.569709

Swann, W. B. (1983). Self-verification: Bringing social reality into harmony with the self. *Psychological Perspectives on the Self, 2*, 33–66.

Tresemer, D. (1976). The cumulative record of research on "fear of success." *Sex Roles, 2*, 217–236. doi:10.1007/BF00287650

Valentiner, D. P., Skowronski, J. J., McGrath, P. B., Smith, S. A., & Renner, K. A. (2011). Self-verification and social anxiety: Preference for negative social feedback and low social self-esteem. *Behavioural and Cognitive Psychotherapy, 39*, 601–617. doi:10.1017/S1352465811000300

Vassilopoulos, S. P., & Banerjee, R. (2010). Social interaction anxiety and the discounting of positive interpersonal events. *Behavioural and Cognitive Psychotherapy, 38*, 597–609. doi:10.1017/S1352465810000433

Wallace, S. T., & Alden, L. E. (1995). Social anxiety and standard setting following social success or failure. *Cognitive Therapy and Research, 19*, 613–631. doi:10.1007/BF02227857

Wallace, S. T., & Alden, L. E. (1997). Social phobia and positive social events: The price of success. *Journal of Abnormal Psychology, 106*, 416–424. doi:10.1037//0021-843X.106.3.416

Wang, W. T., Hsu, W. Y., Chiu, Y. C., & Liang, C. W. (2012). The hierarchical model of social interaction anxiety and depression: The critical roles of fears of evaluation. *Journal of Anxiety Disorders, 26*, 215–224. doi:10.1016/j.janxdis.2011.11.004

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*, 1063-1070. doi:10.1037//0022-3514.54.6.1063

Watson, D., & Friend, R. (1969). Measurement of social-evaluative anxiety. *Journal of Consulting and Clinical Psychology, 33*(4), 448–457. doi:10.1037/h0027806

Weeks, J. W. (2010). The Disqualification of Positive Social Outcomes Scale: A novel assessment of a long-recognized cognitive tendency in social anxiety disorder. *Journal of Anxiety Disorders, 24*, 856–865. doi:10.1016/j.janxdis.2010.06.008

Weeks, J. W., & Heimberg, R. G. (2012). Positivity impairments: Pervasive and impairing (yet nonprominent?) features of social anxiety disorder. *Cognitive Behaviour Therapy, 41*, 79–82. doi:10.1080/16506073.2012.680782

Weeks, J. W., Heimberg, R. G., & Heuer, R. (2011). Exploring the role of submissiveness in social anxiety: Testing an evolutionary model of social anxiety disorder. *Journal of Social and Clinical Psychology, 30*, 217–249. doi:10.1521/jscp.2011.30.3.217

Weeks, J. W., Heimberg, R. G., & Rodebaugh, T. L. (2008). The Fear of Positive Evaluation Scale: Assessing a proposed cognitive component of social anxiety disorder. *Journal of Anxiety Disorders, 22*, 44–55. doi:10.1016/j.janxdis.2007.08.002

Weeks, J. W., Heimberg, R. G., Rodebaugh, T. L., Goldin, P., & Gross, J. (2012). Psychometric evaluation of the Fear of Positive Evaluation Scale in patients with social anxiety disorder. *Psychological Assessment, 24*, 301–312. doi:10.1037/a0025723

Weeks, J. W., Heimberg, R. G., Rodebaugh, T. L., & Norton, P. J. (2008). Exploring the relationship between fear of positive evaluation and social anxiety. *Journal of Anxiety Disorders, 22*(3), 386–400. doi:10.1016/j.janxdis.2007.04.009

Weeks, J. W., & Howell, A. N. (2012). The bivalent fear of evaluation model of social anxiety: Further integrating findings on fears of positive and negative evaluation [Special issue.]. *Cognitive Behaviour Therapy, 41*, 83–95. doi:10.1080/16506073.2012.661452

Weeks, J. W., Howell, A. N., & Goldin, P. R. (2013). Gaze avoidance in social anxiety disorder. *Depression and Anxiety, 30*, 749–756.

Weeks, J. W., Howell, A. N., Srivastav, A., & Goldin, P. R. (2012, November). Assessing social anxiety-related gaze avoidance via eye-tracking: Further validating the bivalent fear of evaluation model of social anxiety. In J. Richey (Chair), *The role of positive emotions in SAD: Behavioral and neurobiological evidence for a new treatment approach.* Paper presented at the annual meeting of the Association for Behavioral and Cognitive Therapies, National Harbor, MD.

Weeks, J. W., Jakatdar, T. A., & Heimberg, R. G. (2010). Comparing and contrasting fears of positive and negative evaluation as facets of social anxiety. *Journal of Social and Clinical Psychology, 29*, 68–94. doi:10.1521/jscp.2010.29.1.68

Weeks, J. W., Norton, P. J., & Heimberg, R. J. (2009). Exploring the latent structure of two cognitive components of social anxiety: Taxometric analyses of fears or negative and positive evaluation. *Depression and Anxiety, 26*, E40–E48. doi:10.1002/da.20414

Weeks, J. W., Rodebaugh, T. L., Heimberg, R. G., Norton, P. J., & Jakatdar, T. A. (2009). "To avoid evaluation, withdraw": Fears of evaluation and depressive cognitions lead to social anxiety and submissive withdrawal. *Cognitive Therapy and Research, 33*(4), 375–389. doi:10.1007/s10608-008-9203-0

Weeks, J. W., & Zoccola, P. (2013). *"Having the heart to be evaluated": The differential effects of Fears of Positive and Negative Evaluation on emotional and cardiovascular responses to social threat*. Manuscript submitted for publication.

Wells, A., Clark, D. M., Salkovskis, P., Ludgate, J., Hackmann, A., & Gelder, M. (1995). Social phobia: The role of in-situation safety behaviors in maintaining anxiety and negative beliefs. *Behavior Therapy, 26*, 153–161. doi:10.1016/S0005-7894(05)80088-7

Werner, K. H., Goldin, P. R., Ball, T. M., Heimberg, R. G., & Gross, J. J. (2011). Assessing emotion regulation in social anxiety disorder: The Emotion Regulation Interview. *Journal of Psychopathology and Behavioral Assessment, 33*(3), 346–354. doi:10.1007/s10862-011-9225-x

21

The Neuroendocrinology of Social Anxiety Disorder

Gail A. Alvares and Adam J. Guastella
University of Sydney, Australia

Social anxiety disorder is characterized by excessive fear and subsequent avoidance of social situations. Individuals with social anxiety exhibit persistent negative beliefs about their role in social situations, including how others will view their own appearance or performance. Not only do these beliefs modify behavior in future events, but they also influence how past social events are remembered, negatively biasing memories. Social anxiety is also associated with excessive autonomic responses to feared situations which often results in excessive avoidance and the use of safety behaviors. Such behaviors also represent a maintenance factor in limiting the individual's capacity to learn that they can effectively cope in social situations.

The aim of this chapter is to explore the role that hormones play in the regulation of social behavior with a focus on how this relates to these core features of social anxiety. This chapter will focus on stress hormones, particularly glucocorticoids, as well as the neuropeptides oxytocin and arginine vasopressin, and the gonadal hormones testosterone and estradiol. These hormones naturally interact to modulate physiological, cognitive, and behavioral responses to social situations. Notably, these hormones are essential for social information processing, how this information is then stored in memory, and what aspects of these memories are likely to be retrieved. Additionally, these hormones regulate a wide range of social behaviors, from social approach to avoidance and even aggression. Taken together, the evidence reviewed below implicates differences in endogenous levels of these hormones in anxiety pathophysiology, but also a role for exogenous administration as treatment adjuncts for social anxiety.

The Stress Response

Socially stressful situations trigger a cascade of automatic and adaptive behavioral and physiological responses, including activation of the hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic–adrenal system. In the latter system, the

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

hypothalamus activates the sympathetic division of the autonomic nervous system to influence the adrenal medulla to produce and release epinephrine (i.e., adrenalin) and norepinephrine. Stress also causes the activation of the HPA axis, the central regulatory system that connects the central nervous system with the endocrine system (see Tsigos & Chrousos, 2002 for review). Briefly, the paraventricular nucleus of the hypothalamus produces corticotrophin-releasing hormone which promotes the secretion of adrenocorticotropic hormone (ACTH) by the pituitary (Lamberts, Verleun, Oosterom, De Jong, & Hackeng, 1984). ACTH then stimulates the adrenal cortex to synthesize and release glucocorticoid hormones (i.e., cortisol in humans and corticosterone in most animals). Glucocorticoids function to increase blood glucose levels and break down protein and fat to make these nutrients available for tissues when necessary. They also increase inflammatory responses and later inhibit the release of further ACTH when blood glucose levels eventually decline to inhibit HPA axis activity (De Kloet, 1991). Glucocorticoids, as the end product of HPA axis activation, are the major component of an adaptive stress response. Importantly, due to high densities of glucocorticoid receptors found in the hippocampus, amygdala, and frontal lobes (De Kloet, 1991), glucocorticoids are critical for learning and memory processes, particularly for emotional stimuli.

Endogenous Glucocorticoids

An adaptive stress response prepares individuals for perceived threat and, subsequently, returns the body to a homeostatic condition. It additionally functions to induce longer-term adaptive behavioral and cognitive responses (McEwen, 2006), including the activation of a range of hormonal and neurotransmitter systems to enhance memory for emotionally arousing information. Evidence for this comes from examining overall levels of glucocorticoids, glucocorticoid reactivity to social stress, and glucocorticoid effects on anxiety and affect.

In humans, studying the reactivity of endogenous cortisol activity to social stress situations allows an examination of potential endocrine involvement in the pathophysiology of social anxiety. A review of standardized acute laboratory stressors in humans found that, of all tasks reviewed, public speaking tasks with a socially evaluative threat component exhibited the strongest effect sizes on physiological and cognitive measures (Dickerson & Kemeny, 2004). The Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993), for example, involves completion of a free speech and mental arithmetic task in front of an audience and demonstrates consistent and replicable increases in cortisol reactivity. Cold pressor tasks, in which individuals immerse their hand into cold water, have also been associated with comparable increases in blood pressure, cortisol reactivity, and subjective distress relative to the TSST (Schwabe, Haddad, & Schachinger, 2008).

Exacerbation of HPA axis activity in response to such laboratory social stressors influences subsequent memory, notably memory consolidation and retrieval. Emotionally arousing information is better remembered, even after a long-time period, and elevated glucocorticoid levels enhance this process (McGaugh & Roozendaal, 2002). Degree of arousal at the time of encoding plays a critical role in this process, with cold pressor stress enhancing memory for neutral information (Andreano

& Cahill, 2006) and enhancing memory consolidation for emotionally arousing stimuli (Cahill, Gorski, & Le, 2003). Glucocorticoids also influence the way that memories are retrieved. Initial preclinical studies demonstrated that higher levels of corticosterone in rats, induced by foot shock, impaired memory retrieval for previously acquired spatial knowledge (de Quervain, Roozendaal, & McGaugh, 1998). Homologous evidence in humans confirms this association, with decreased recall of emotionally arousing words in individuals after exposure to laboratory stressors (Domes, Heinrichs, Rimmele, Reichwald, & Hautzinger, 2004; Kuhlmann, Piel, & Wolf, 2005).

In healthy humans, higher HPA axis activity in response to stress is associated with increased bias toward threat (van Honk et al., 2000), social avoidance, and freezing behavior (Roelofs, Bakvis, Hermans, van Pelt, & van Honk, 2007). In individuals with social anxiety disorder, increased cortisol responses to psychological stressors have been observed over a number of studies (Condren, O'Neill, Ryan, Barrett, & Thakore, 2002; Furlan, DeMartinis, Schweizer, Rickels, & Lucki, 2001; Levin et al., 1993; Martel et al., 1999). Further, hyperactivity of HPA axis in response to stress is also associated with increased behavioral avoidance in social anxiety (Roelofs et al., 2009). This evidence implies that enhanced sensitivity or hyper-reactivity of this system may represent an etiological factor in social anxiety, particularly in response to social stress.

Exogenous Glucocorticoid Administration

Given the role of glucocorticoids in modulating emotional memory, and the role of emotional fear associations in the etiology of anxiety, it may be assumed that enhanced glucocorticoids under stress may contribute to the formation of an aversive fear memory. However, a critical aspect of behavioral treatments for social anxiety relies on facing fears about social situations through exposure to learned fear associations (Centonze, Siracusano, Calabresi, & Bernardi, 2005), based on Pavlovian conditioning models (Pavlov, 1927). In brief, fear associations are formed by repeated pairings of an initially neutral stimulus (a conditioned stimulus; CS) with an aversive stimulus (the unconditioned stimulus; US) that elicits a conditioned response (CR; e.g., startle) to the corresponding CS; thus, a corresponding fear memory is formed. As a consequence, presentation of the CS alone will elicit a fear response. Extinction of this learned fear involves repeated presentations of the CS in the absence of the US, which gradually results in a decrease of fear responses. Theories generally agree that this is not due to forgetting the previously learned association but to a formation of a new association that competes with the original memory (Myers & Davis, 2007). Similarly, in exposure-based therapies for social anxiety, patients are encouraged to expose themselves to a feared social situation (either in real life, or imagined) to learn that the expected outcome will not occur, or will not be as bad as expected. This procedure is considered analogous to extinction training.

Notably, glucocorticoids enhance memory consolidation in extinction (Wolf, 2008). For example, corticosterone inhibitors impair (Barrett & Gonzalez-Lima, 2004), while glucocorticoid agonists facilitate (Yang, Chao, & Lu, 2005), extinction in animal models. These effects appear to be mediated by noradrenergic activation in the basolateral amygdala (de Quervain, Aerni, Schelling, & Roozendaal, 2009).

Glucocorticoid administration also impairs memory retrieval for aversive memories in rats, an effect which appears to be mediated by the medial temporal lobe (de Quervain et al., 2009). Homologous findings in healthy humans indicate that high cortisol levels reduce cerebral blood flow in the medial temporal lobe during memory retrieval (de Quervain et al., 2003; Oei et al., 2007). Thus, glucocorticoids facilitate extinction of learned fear by both enhancing consolidation of an extinction memory and weakening aversive memory traces by inhibiting memory retrieval. That is, the aversive cue is no longer followed by the usual aversive memory retrieval but instead becomes associated with a less aversive experience, which is stored as an extinction memory.

A number of studies confirm that glucocorticoid administration enhances extinction of clinical fear. In one study, acutely administered cortisone to social anxiety disorder patients prior to a TSST resulted in reduced self-reported fear across the task (Soravia et al., 2006). Further, in participants given placebo, individuals with higher endogenous cortisol exhibited less subjective fear. The authors concluded that, although cortisol may buffer fear in stressful situations, a dysregulation of the endogenous stress system in clinical anxiety might also act to reduce subjective stress when activated by an exposure treatment. In the same paper, the authors also reported that acute hydrocortisone administration progressively reduced fear in specific spider phobia. This effect was maintained 2 days after the last administration, implying a longer-term memory consolidation effect (Soravia et al., 2006). More recently, cortisol was also shown to enhance exposure therapy for specific phobia (de Quervain et al., 2011). Patients treated with cortisol prior to three sessions of exposure-based therapy exhibited significantly less fear symptoms in comparison to a placebo group at both posttreatment and 1-month follow-up. The results from these clinical studies suggest that both acute and repeated doses of cortisol may reduce self-reported anxiety symptoms and potentially enhance consolidation of these newly learned associations (i.e., extinction) in individuals with phobic fears. Further, the evidence favors combinatorial treatment with exposure-based therapy, implying that augmentative treatment approach may be warranted for future studies (see **Chapter 18** for additional discussion on this issue).

Social Neuropeptides

The roles of oxytocin and vasopressin in mammalian social cognition and behavior have been well reviewed recently (McCall & Singer, 2012; Meyer-Lindenberg, Domes, Kirsch, & Heinrichs, 2011). Currently, our understanding of these neuropeptides comes from correlational studies of endogenous levels (measured in plasma, saliva, urine, or cerebrospinal fluid) and experimental studies using intranasal or intravenous administration methods. Another line of research, outside the scope of this chapter, examines effects of oxytocin and vasopressin receptor polymorphisms (see **Chapter 4** for a review). These studies provide evidence that oxytocin facilitates the beneficial effects of positive and supportive social interactions, as well as buffers against the negative impact of social stress. Studies in vasopressin, however, exhibit mixed evidence, with roles in social memory, cognition, and pair bonding, but also anxiogenic effects. There is limited evidence as to whether measures of peripheral levels of these neuropeptides accurately reflect central neuropeptidergic functioning,

although neuropeptides administered as a nasal spray may cross the blood brain barrier to exert central effects (Born et al., 2002). Recent neuroimaging research, however, has allowed for further insights into potential underlying mechanisms, with applications for social anxiety treatment.

Synthesis, Storage, and Sites of Action

Oxytocin and vasopressin (also known as antidiuretic hormone) are evolutionarily highly conserved neurohypophyseal hormones. Oxytocin promotes uterine contractions and lactation during childbirth, while vasopressin plays a key role in the regulation of water retention by the kidneys (Brownstein, Russell, & Gainer, 1980). Synthesis of oxytocin and vasopressin occurs mainly via the supraoptic and paraventricular nuclei of the hypothalamus. These nuclei project to the posterior pituitary, or neurohypophysis, which stores and releases oxytocin peripherally into the bloodstream (Gimpl & Fahrenholz, 2001). Parvocellular, and some magnocellular, neurons in the paraventricular nuclei synthesize oxytocin and vasopressin and project to various regions within the central nervous system. A single oxytocin receptor and two vasopressin receptor subtypes (V1a and V1b) are centrally expressed and distributed widely throughout the brain (two other vasopressin receptors are found peripherally). These central regions particularly underlie control of many social behaviors, such as the nucleus accumbens, ventral tegmental area, amygdala, and hippocampus (Landgraf & Neumann, 2004).

Endogenous Release

Oxytocin and vasopressin are well characterized for roles in pair bonding, parental care, and formation of social memories (Lim & Young, 2006). Although vasopressin expression is associated with more male-typical social behaviors, such as scent marking, paternal care, and aggression (Wang, Young, De Vries, & Insel, 1998), endogenous vasopressin is also necessary for normal social recognition and anxiety-related behaviors (Bielsky & Young, 2004). Importantly, vasopressin interacts with the HPA axis, in particular with ACTH, to regulate a normal stress response (Lolait, Stewart, Jessop, Young, & O'Carroll, 2007). Further, both peripheral and central levels of oxytocin reduce the response to physical and social stressors (Neumann, 2002). Endogenous oxytocin naturally functions as an anxiolytic, increasing release of the inhibitory neurotransmitter γ-aminobutyric acid (GABA) in the central amygdala (Viviani & Stoop, 2008) and attenuating HPA axis activity (Neumann, 2002) in response to fearful stimuli.

Increased endogenous oxytocin levels in human plasma are associated with positive social interactions. For example, elevations in peripheral oxytocin are associated with increased trust (Zak, Kurzban, & Matzner, 2005), and peripheral oxytocin levels increase after positive contact with a partner (Grewen, Girdler, Amico, & Light, 2005). Lactating women, in whom endogenous oxytocin is increased, show attenuated ACTH and cortisol responses to psychosocial stressors (Altemus, Deuster, Galliven, Carter, & Gold, 1995; Heinrichs et al., 2001). However, higher plasma oxytocin has also been associated with relationship distress and elevated HPA axis activity in

both young women and older postmenopausal women (Taylor et al., 2006; Turner, Altemus, Enos, Cooper, & McGuinness, 1999).

Atypical neuropeptide levels have been associated with a number of clinical disorders such as autism, depression, and schizophrenia (see Meyer-Lindenberg et al., 2011 for review). Of note, higher levels of social anxiety symptoms, in patients diagnosed with social anxiety disorder, were associated with increased plasma oxytocin levels (Hoge, Pollack, Kaufman, Zak, & Simon, 2008). Greater plasma levels of vasopressin have also been associated with a lifetime history of general aggression and aggression toward others in individuals with antisocial personality disorder (Coccaro, Kavoussi, Hauger, Cooper, & Ferris, 1998). These studies implicate dysregulated neuropeptide functioning or expression in individuals with greater anxiety or stress responsiveness.

Exogenous Manipulation and Administration

Oxytocin and vasopressin modulate approach and withdrawal behaviors across a number of species that are essential for normal social behavior. First, both oxytocin- and vasopressin-knockout mice exhibit deficits in social recognition, despite displaying normal overall memory (Bielsky & Young, 2004; Ferguson et al., 2000). Second, oxytocin and vasopressin are necessary for pair bonding; oxytocin is, however, specifically necessary for the motivational changes that transition social avoidance behavior to maternal care (Pedersen, Vadlamudi, Boccia, & Amico, 2006). Onset of maternal behavior is mediated by oxytocin receptor distribution in the nucleus accumbens and ventral tegmental area, regions critical for reward processing (Landgraf & Neumann, 2004). Last, partner preferences in vole models are driven by specific oxytocin and vasopressin receptor distributions between species (Young & Wang, 2004). While successful mating experiences facilitate partner preference, oxytocin infusions into the cerebral ventricles can induce partner preference in the absence of any mating behavior (Williams, Insel, Harbaugh, & Carter, 1994). In naturally promiscuous voles, V1a receptor antagonists injected into the ventral pallidum block pair bonding, while agonists induce partner preference (Lim, Murphy, & Young, 2004). These findings suggest a role for these neuropeptides in the acquisition or appetitive phase of social learning and memory by facilitating motivational and behavioral transitions to enhance approach and reduce withdrawal.

Oxytocin Administration in Humans

Oxytocin administered as a nasal spray facilitates the perception and interpretation of social stimuli (Guastella & MacLeod, 2012). Since Kosfeld, Heinrichs, Zak, Fischbacher, & Fehr (2005) first found that acute oxytocin increases trusting behavior, a wealth of single-dose administration studies have emerged arguing that oxytocin modulates aspects of social cognition, behavior, and emotion (Meyer-Lindenberg et al., 2011). These effects may be mediated through increasing attention to socially relevant areas of human faces, such as the eyes (Guastella, Mitchell, & Dadds, 2008). This is of particular interest given that social anxiety is associated with abnormal processing of faces, with hypervigilance toward social stimuli but avoidance of eye

regions (Horley, Williams, Gonsalvez, & Gordon, 2004). Oxytocin also seems to influence accurate detection and appraisal of emotional information at both automatic and strategic levels of processing, with a stronger bias toward recall of positive social information (Guastella & MacLeod, 2012). This suggests that oxytocin facilitates the early and rapid detection of emotional information, but may also enhance cognitive appraisals at elaborative stages of information processing, with a particular bias toward positive social information.

In terms of anxiety, acute and chronic oxytocin administration in rats reduces stress responsivity (Slattery & Neumann, 2010; Windle et al., 2004). In humans, intranasal oxytocin attenuates cortisol levels and subjective anxiety reports after psychosocial stress, an effect which was augmented by social support (Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003). Oxytocin also reduces anticipatory anxiety (de Oliveira, Zuardi, Graeff, Queiroz, & Crippa, 2012) and attenuates cortisol responses to public speaking tasks in individuals with impaired emotional regulation abilities (Quirin, Kuhl, & Düsing, 2011). Recently, we administered oxytocin to healthy male individuals prior to a socially evaluative public speech task and measured changes in eye gaze to a virtual audience and self-reported appraisals of speech performance (Alvares, Chen, Balleine, Hickie, & Guastella, 2012). We found that oxytocin reduced negative cognitive self-appraisals of speech performance specifically for those participants with higher trait anxiety, implying a selective moderation effect for individuals with increased anxiety. Oxytocin also appears to abolish fear conditioning in response to fearful or threatening stimuli (Kirsch et al., 2005; Petrovic, Kalisch, Singer, & Dolan, 2008).

These effects may be due to reducing the perceived potential threat associated with ambiguous or fearful social interactions (Kirsch et al., 2005). Oxytocin modulates amygdala activation and associated cortical and subcortical areas (Baumgartner, Heinrichs, Vonlanthen, Fischbacher, & Fehr, 2008; Domes et al., 2007; Kirsch et al., 2005). Critically, oxytocin reduces amygdala hyperactivity to threatening social stimuli in healthy (Kirsch et al., 2005) and socially anxious (Labuschagne et al., 2010) individuals. As amygdala hyperactivity in response to social threat has been previously observed in social anxiety disorder (Phan, Fitzgerald, Nathan, & Tancer, 2006), this moderating role of oxytocin on amygdala hyperactivity implies a potential neural mechanism to remediate social fear.

The effects of neuropeptide administration are currently being explored across a number of clinical disorders, including autism, schizophrenia, borderline personality, and anxiety (see Meyer-Lindenberg et al., 2011 for review). Of particular relevance, patients with social anxiety disorder given oxytocin as an adjunct to five sessions of cognitive behavioral therapy exhibited reduced negative beliefs about their perceived performance after public speaking over time compared to those who received placebo (Guastella, Howard, Dadds, Mitchell, & Carson, 2009). Acute oxytocin administration may therefore have therapeutic potential by reducing negatively biased social information processing. Additionally, social anxiety is associated with biased avoidance of positive social information (Weeks, Jakatdar, & Heimberg, 2010). As oxytocin acts to enhance the reward value associated with positive information and reduce the threat associated with negative social information, this may represent an underlying mechanism to treat these cognitive biases.

Vasopressin Administration in Humans

The evidence for the effects of vasopressin on human social cognition and behavior is more limited. Whereas oxytocin enhances memory for positive social stimuli (Guastella, Mitchell, & Mathews, 2008), vasopressin increases familiarity for both positive and negative faces (Guastella, Kenyon, Alvares, Carson, & Hickie, 2010). Further, vasopressin enhances recognition of sexual words (Guastella, Kenyon, Unkelbach, Alvares, & Hickie, 2011), comparable to previous findings that oxytocin enhances recognition of positive sex and relationship words (Unkelbach, Guastella, & Forgas, 2008). Vasopressin also appears to modulate physiological responses to social information, enhancing negative unconscious responses to ambiguous social cues (Thompson, Gupta, Miller, Mills, & Orr, 2004). Sexually dimorphic effects have also been reported, with vasopressin decreasing perceptions of friendliness and increasing agonistic facial responses in males, but increasing affiliative motor responses and friendliness perceptions in females (Thompson, George, Walton, Orr, & Benson, 2006).

Although there is an overwhelming amount of evidence for a role of oxytocin to enhance social information encoding and reduce social threat, vasopressin appears to regulate anxiogenic-related behaviors in response to stress. Vasopressin is associated with neural transmission in the amygdala and promotes secretion of ACTH (Axelrod & Reisine, 1984; Huber, Veinante, & Stoop, 2005), actually increasing the endocrine response to stress. For example, intranasal vasopressin significantly increases cortisol and heart rate during a psychosocial stressor (Ebstein et al., 2009), an effect that is much more pronounced when being socially evaluated by others (Shalev et al., 2011). Because vasopressin studies in humans are relatively limited at this stage, any conclusions about applications to clinical anxiety are still preliminary. However, given a potential role in increased social stress responsivity, the development of vasopressin receptor antagonists may warrant further exploration.

Gonadal Hormones

Although gonadal hormones are not centrally produced, both androgens and estrogens influence central brain regions to exert control over social behavior in both genders (van Wingen, Ossewaarde, Bäckström, Hermans, & Fernández, 2011). In particular, sex differences in learning and memory may be due to sexually dimorphic expression of these hormones (McLaughlin, Baran, & Conrad, 2009). Regions involved in fear and emotion regulation, such as the amygdala and ventromedial prefrontal cortex (vmPFC), are also sexually dimorphic structures (Goldstein et al., 2001). Further, rates of anxiety disorders are more prevalent in females compared to males, and stress evokes a number of sex differences across behavioral and cognitive tasks (Andreano & Cahill, 2009). Although no clinical studies in anxiety disorders have been conducted using administration of gonadal hormones, the evidence presented below suggests a potential role for these hormones to modulate emotion and social information processing. In this section we specifically refer to the androgen testosterone and the estrogen estradiol.

Synthesis and Sites of Action

In addition to their traditionally viewed functions in reproductive and sexual behavior, androgens and estrogen regulate social behavior in a number of species (van Wingen et al., 2011). Unlike oxytocin and vasopressin, testosterone and estradiol are synthesized in the gonads (testes in males and ovaries in females, respectively) and reach the central nervous system through systemic circulation. Both hormones are the end products of the hypothalamic–pituitary–gonadal (HPG) axis, whereby the hypothalamus produces gonadotropin-releasing hormone to act on the anterior pituitary to produce luteinizing and follicle-stimulating hormones. These hormones then act on the gonads to produce estradiol and testosterone, although small amounts of testosterone are secreted from the adrenal cortex in both males and females. Central effects of estrogen are mediated by estrogen receptors α (ERα) and β (ERβ) (Östlund, Keller, & Hurd, 2003). Testosterone acts on androgen receptors found mainly in the hippocampus, prefrontal cortex, and amygdala regions (Sarkey, Azcoitia, Garcia-Segura, Garcia-Ovejero, & DonCarlos, 2008).

Endogenous Release

Testosterone The link between testosterone and aggression is well established across a number of species. While the evidence is stronger in nonhuman animal models, increases in endogenous testosterone in humans is positively associated with increased aggressive and antisocial behavior (Book, Starzyk, & Quinsey, 2001). However, testosterone also modulates social information processing, cognitive performance, motivation, and the mediation of anxious states. In animals, such evidence is examined through the removal of an animal's testes through gonadectomy, resulting in primary androgen deprivation; for example, gonadectomy in rodents impairs memory in hippocampal-dependent tasks, which can be restored through testosterone replacement (Edinger & Frye, 2004).

Gonadectomy also increases anxiety behavior (Ceccarelli, Scaramuzzino, & Aloisi, 2001), while replacement of androgens reverses anxiety-induced behaviors and restores cognition and memory (Edinger & Frye, 2004). Increased testosterone in rats is associated with anxious responses to threat, such as behavioral freezing, fear-induced analgesia, increased startle response, and inhibited exploratory behavior (Edinger & Frye, 2005; Toufexis, Myers, & Davis, 2006). In humans, low basal salivary testosterone has been associated with anxiety in male adolescents (Granger et al., 2003), while low prenatal testosterone *in utero* may be associated with anxiety in adulthood (de Bruin, Verheij, Wiegman, & Ferdinand, 2006).

In terms of affective behavior, higher endogenous levels of testosterone are associated with enhanced recognition and recall of emotional and socially relevant information. Recent evidence shows that greater endogenous testosterone levels in men were associated with increased arousal ratings and better free recall of previously encoded neutral pictures, which related to increased amygdala activation (Ackermann et al., 2012). While testosterone levels in healthy males are positively associated with amygdala activation to emotionally threatening faces (Derntl et al., 2009; see also Stanton, Wirth, Waugh, & Schultheiss, 2009), females exhibit additional associations between

testosterone levels and activation in the amygdala, hypothalamus, temporal cortex, and orbitofrontal cortex (OFC) during presentation of angry versus happy faces (Hermans, Ramsey, & van Honk, 2008). Together, these studies suggest that endogenous testosterone has a role in anxious states, emotional memory, and processing of social stimuli via modulation of amygdala-dependent networks.

Estradiol Estrogens are reported to have both anxiogenic and anxiolytic properties, with differences attributed to two distinct receptor systems mediating effects on mood and anxiety (Lund, Rovis, Chung, & Handa, 2005). While ERα is critical for reproductive functions, ERβ appears to play a stronger role in synaptic plasticity in the amygdala and in mediating emotional behavior (Östlund et al., 2003). For example, ERβ knockout mice display increased anxiety but intact reproductive abilities (Krężel, Dupont, Krust, Chambon, & Chapman, 2001). ERβ is particularly expressed in regions of the brain associated with fear and anxiety responses, and administration of ERβ agonists into ovariectomized rats results in reductions of anxiolytic behaviors (Lund et al., 2005). Further, ovariectomized female rats show increased anxiety- and depression-related behaviors that can be reversed by subcutaneous injection of estrogens (Walf & Frye, 2006).

Depression and anxiety symptoms, as well as stress reactivity, are modulated by the menstrual cycle, particularly when estradiol levels are low (Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999; Toufexis et al., 2006). Although interactions with other relevant hormones such as progesterone should not be discounted, it seems as though estradiol in particular specifically influences the neural circuitry and morphological structures that underpin emotional memory and fear extinction learning (Goldstein et al., 2001; Shansky et al., 2010) and may play a key role in modulating state levels of anxiety through specific effects on ERβ (Lund et al., 2005). Functional imaging studies reveal that activation of the vmPFC and amygdala in response to emotions varies across the menstrual cycle (Goldstein et al., 2005). Further, sex differences in stress reactivity and fear acquisition (in rats and humans) are mediated by differing levels of estradiol in females in comparison to males, with higher levels of estradiol thought to attenuate stress-induced arousal (Dalla & Shors, 2009; Goldstein, Jerram, Abbs, Whitfield-Gabrieli, & Makris, 2010). In terms of extinction, higher levels of estradiol facilitate fear recall extinction in both rats (Milad, Igoe, Lebron-Milad, & Novales, 2009) and women (Milad et al., 2010), an effect which is thought to be driven by medial PFC and amygdala activity in humans or homologous rodent regions (Zeidan et al., 2011).

Exogenous Administration

Testosterone Exogenous administration studies have argued for a more direct link between testosterone and the processing of emotional social stimuli (van Wingen et al., 2011), with cumulative evidence suggestive that testosterone influences the processing of social information and modulates automatic responses to fear or threat. In placebo-controlled designs, a single dose of sublingual testosterone in females has been shown to decrease trust of unfamiliar others (Bos, Terburg, & van Honk, 2010) and increase fairness behavior in a bargaining social interaction

(Eisenegger, Naef, Snozzi, Heinrichs, & Fehr, 2010). However, this effect was reversed for those individuals who believed they had been given testosterone, regardless of whether they had actually received it or not, implying that beliefs in hormone efficacy may moderate actual effects on social interaction (Eisenegger et al., 2010). These effects may be in part related to a potential reduction in the ability to detect relevant social cues, with testosterone administration delaying detection of threatening faces compared to placebo (van Honk & Schutter, 2007). Testosterone also plays a role in hippocampal-dependent processing of opposite sex face memorization in females (van Wingen, Mattern, Verkes, Buitelaar, & Fernández, 2008). In terms of fear or threat detection, testosterone has been shown to reduce startle responses (Hermans, Putman, Baas, Koppeschaar, & van Honk, 2006) and central stress reactivity to aversive pictures (Hermans et al., 2007). Testosterone also reduces attentional biases toward fearful faces (van Honk et al., 2001) and vigilance toward threatening faces, without affecting self-reported anxiety (van Honk, Peper, & Schutter, 2005).

Testosterone appears to enhance amygdala and OFC activation to threatening stimuli (Hermans et al., 2008) compared to nonemotional stimuli van Wingen, Zylicz, et al. (2008) and enhances nucleus accumbens activity to reward anticipation (Hermans et al., 2010). This increase in amygdala activation in response to threat may be argued to indicate enhanced aggressive approach or increased threat responsivity tendencies (van Honk et al., 2001). However, testosterone also acts to reduce the functional connectivity between the amygdala and OFC (van Wingen, Mattern, Verkes, Buitelaar, & Fernández, 2010), suggesting that testosterone may actually act to regulate communication between the amygdala and OFC to reduce processing of threatening faces as negative (Bos, Hermans, Ramsey, & van Honk, 2012; van Wingen et al., 2011). By reducing activation of the amygdala, and connectivity to other regions involved in social threat processing, acute testosterone may facilitate reductions in emotional processing during socially threatening encounters. This evidence implies that testosterone exerts multiple motivational properties, increasing attention to cues of threat, reducing fear and punishment sensitivity, and reducing emotional processing under threat or stress (van Honk et al., 2005). These may provide a number of mechanisms by which acute increases in testosterone may reduce characteristic avoidance of negative or threatening information in social anxiety.

Estradiol Limited studies have been conducted examining estradiol administration in humans. However, interesting findings have been obtained from a recent novel translational study investigating the role of estradiol on fear extinction in female rats and humans (Zeidan et al., 2011). The authors showed that an ERβ agonist in rats facilitated extinction recall, while estradiol administration facilitated memory consolidation postextinction training. The authors then demonstrated homologous evidence in humans in which higher natural estradiol levels in women increased vmPFC and amygdala activation to facilitate extinction recall. This evidence suggests that increased estradiol, whether endogenous or exogenously promoted, may facilitate memory processes underlying learning and potentially enhance extinction of learned fear. Indeed, in female rats, chronic administration of estrogens acting on ERβ improves consolidation of fear extinction (Chang et al., 2009; Milad et al., 2009).

In humans, postmenopausal women given 4 weeks of combined estrogen and progesterone treatment revealed significant increases in OFC activity to negative pictures, with decreases in medial prefrontal activity to negative and positive pictures (Love, Smith, Persad, Tkaczyk, & Zubieta, 2010). Although this study may suggest a role for short-term estrogen treatment to potentially influence emotional processing, the treatment was combined with progesterone so it is unclear what the specific effects of estradiol may be and how such treatment may affect males or nonmenopausal women. However, preliminary evidence from single-dose studies suggests a role for estrogens to influence emotional circuitry processing (van Wingen et al., 2011). In particular, the potential for estradiol agonists, specifically acting on ERβ, to influence extinction in humans has not yet been examined as far as we are aware; however, estradiol manipulations may potentially reduce state anxiety and facilitate extinction of learned fear.

Conclusions and Future Directions

This chapter has reviewed evidence to suggest that stress and centrally acting peptidergic and gonadal hormones modulate the processing of social information. While there may be divergent effects in various species and differences noted in potential underlying mechanisms, increasing evidence suggests that these hormones individually and synergistically target etiological and maintenance factors that are at the core of social anxiety. Glucocorticoids index stress responsivity but also play key roles in facilitating fear extinction and inhibiting retrieval of fear memories. Oxytocin and vasopressin act in concert to regulate social cognition and memory to bias encoding and recall of emotionally salient information. Of particular relevance to social anxiety, oxytocin attenuates the effects of acute stress by reducing negative mental self-representations and physiological stress responsivity. Testosterone enhances sensitivity for rewarding and motivated behavior, with reductions in threat sensitivity and fear. Last, estradiol modulates mood and anxiety, with some limited evidence for acute administration to exert effects on neural circuitry underlying emotion processing. Cumulative evidence suggests that these hormones may act together as part of an emotional regulatory circuit.

A number of reviews have emerged recently discussing how these hormones do not act individually but synergistically (Bos, Panksepp, Bluthé, & van Honk, 2012; Joels & Baram, 2009; McCall & Singer, 2012). For example, both testosterone and estradiol interact with several limbic regions to influence the expression of oxytocin and vasopressin (Choleris, Devidze, Kavaliers, & Pfaff, 2008), while oxytocin acts to reduce HPA axis activity in response to social stress (Heinrichs et al., 2003). Testosterone and cortisol also exert mutually antagonistic properties to respectively suppress HPA and HPG axis activity. While activation of the stress response in socially threatening interactions promotes a cascade of hormonal reactions, individual differences in trait anxiety, or clinical levels of anxiety, may modulate how these hormones interact naturally. In particular, social anxiety may be characterized by hyperactivation or increased sensitivity of the endocrine response to stress, promoting enhanced physiological response, increased negative cognitions, and behavioral avoidance. Initial evidence from administration studies suggests that exogenous applications of some of

these hormones in a social context may act to remediate some of these changes to assist with treatment.

The most successful psychological intervention for social anxiety by far has been cognitive-behavioral therapy, which aims to redress maladaptive cognitions and behaviors (see **Chapter 22** for a review). However, such treatments are not universally efficacious, with considerable rates of partial responders or nonadherence to treatment. Thus, there is a pressing need for novel interventions that augment existing best-practice interventions. The strongest evidence for a facilitatory role in the behavioral components of social anxiety is cortisol, with clear enhancing effects on the extinction of learned fear and inhibition of fear memory retrieval. Further, oxytocin appears to modulate cognition, reducing biased negative mental self-representations and attenuating self-reported fear and anxiety. While the evidence for testosterone, estradiol, and vasopressin are more mixed, it is clear that these hormones exert effects on emotional memory and social information processing that are characteristically biased in social anxiety disorder.

A number of limitations in this field of research need to be considered when interpreting this evidence. Acute and chronic administration studies are largely gender-biased; for example, the majority of oxytocin and testosterone studies are conducted in males and females, respectively. Interestingly, when studies do employ designs including both genders, sexually dimorphic effects are often observed. This implies that research is still yet to fully determine the full spectrum of potential effects of hormonal administration on social behavior and emotion. Further, use of radioactive labeling of these hormones and positron emission tomography scanning within human administration studies will provide valuable insights into mechanisms as well as differential effects of various manipulations (i.e., intranasal, intravenous, and sublingual).

In conclusion, this chapter has reviewed evidence that the endocrinology underpinning stress and anxiety may inform a better understanding of factors that play a role in the etiology, maintenance, and treatment of social anxiety symptoms. Further, novel treatments, including oxytocin, cortisol, and possibly testosterone and estradiol agonists, may provide a new avenue for future research into the treatment of social anxiety disorder. In particular, the evidence reviewed favors use of these hormones as an adjunct to existing exposure-based treatments. This suggests that these hormones may have the potential to treat some of the core cognitive, behavioral, and emotional components of social anxiety, with augmentative approaches warranted for future investigation.

References

Ackermann, S., Spalek, K., Rasch, B., Gschwind, L., Coynel, D., Fastenrath, M.,… de Quervain, D. J.-F. (2012). Testosterone levels in healthy men are related to amygdala reactivity and memory performance. *Psychoneuroendocrinology, 37*(9), 1417–1424. doi:10.1016/j.psyneuen.2012.01.008

Altemus, M., Deuster, P. A., Galliven, E., Carter, C. S., & Gold, P. W. (1995). Suppression of hypothalamic-pituitary-adrenal axis responses to stress in lactating women. *The Journal of Clinical Endocrinology & Metabolism, 80*(10), 2954–2959. doi:10.1210/jc.80.10.2954

Alvares, G. A., Chen, N. T. M., Balleine, B. W., Hickie, I. B., & Guastella, A. J. (2012). Oxytocin selectively moderates negative cognitive appraisals in high trait anxious males. *Psychoneuroendocrinology, 27*(12), 2022–2031. doi:10.1016/j.psyneuen.2012.04.018

Andreano, J. M., & Cahill, L. (2006). Glucocorticoid release and memory consolidation in men and women. *Psychological Science, 17*(6), 466–470. doi:0.1111/j.1467-9280.2006.01729.x

Andreano, J. M., & Cahill, L. (2009). Sex influences on the neurobiology of learning and memory. *Learning & Memory, 16*(4), 248–266. doi:10.1101/lm.918309

Axelrod, J., & Reisine, T. D. (1984). Stress hormones: Their interaction and regulation. *Science, 224*, 452–459. doi:10.1126/science.6143403

Barrett, D., & Gonzalez-Lima, F. (2004). Behavioral effects of metyrapone on Pavlovian extinction. *Neuroscience Letters, 371*(2–3), 91–96. doi:10.1016/j.neulet.2004.08.046

Baumgartner, T., Heinrichs, M., Vonlanthen, A., Fischbacher, U., & Fehr, E. (2008). Oxytocin shapes the neural circuitry of trust and trust adaptation in humans. *Neuron, 58*, 639–650. doi:10.1016/j.neuron.2008.04.009

Bielsky, I. F., & Young, L. J. (2004). Oxytocin, vasopressin, and social recognition in mammals. *Peptides, 25*(9), 1565–1574. doi:10.1016/j.peptides.2004.05.019

Book, A. S., Starzyk, K. B., & Quinsey, V. L. (2001). The relationship between testosterone and aggression: A meta-analysis. *Aggression and Violent Behavior, 6*(6), 579–599. doi:10.1016/S1359-1789(00)00032-X

Born, J., Lange, T., Kern, W., McGregor, G. P., Bickel, U., & Fehm, H. L. (2002). Sniffing neuropeptides: A transnasal approach to the human brain. *Nature Neuroscience, 5*, 514–516. doi:10.1038/nn0602-849

Bos, P. A., Hermans, E. J., Ramsey, N. F., & van Honk, J. (2012). The neural mechanisms by which testosterone acts on interpersonal trust. *Neuroimage, 61*(3), 730–737. doi:10.1016/j.neuroimage.2012.04.002

Bos, P. A., Panksepp, J., Bluthé, R.-M., & van Honk, J. (2012). Acute effects of steroid hormones and neuropeptides on human social–emotional behavior: A review of single administration studies. *Frontiers in Neuroendocrinology, 33*, 17–35. doi:10.1016/j.yfrne.2011.01.002

Bos, P. A., Terburg, D., & van Honk, J. (2010). Testosterone decreases trust in socially naïve humans. *Proceedings of the National Academy of Sciences, 107*(22), 9991. doi:10.1073/pnas.0911700107

Brownstein, M. J., Russell, J. T., & Gainer, H. (1980). Synthesis, transport, and release of posterior pituitary hormones. *Science, 207*(4429), 373–378. doi:10.1126/science.6153132

Cahill, L., Gorski, L., & Le, K. (2003). Enhanced human memory consolidation with post-learning stress: Interaction with the degree of arousal at encoding. *Learning & Memory, 10*(4), 270–274. doi:10.1101/lm.62403

Ceccarelli, I., Scaramuzzino, A., & Aloisi, A. M. (2001). Effects of gonadal hormones and persistent pain on non-spatial working memory in male and female rats. *Behavioural Brain Research, 123*(1), 65–76. doi:10.1016/S0166-4328(01)00195-4

Centonze, D., Siracusano, A., Calabresi, P., & Bernardi, G. (2005). Removing pathogenic memories. *Molecular Neurobiology, 32*(2), 123–132. doi:10.1385/MN:32:2:123

Chang, Y.-J., Yang, C.-H., Liang, Y.-C., Yeh, C.-M., Huang, C.-C., & Hsu, K.-S. (2009). Estrogen modulates sexually dimorphic contextual fear extinction in rats through estrogen receptor β. *Hippocampus, 19*(11), 1142–1150. doi:10.1002/hipo.20581

Choleris, E., Devidze, N., Kavaliers, M., & Pfaff, D. W. (2008). Steroidal/neuropeptide interactions in hypothalamus and amygdala related to social anxiety. *Progress in Brain Research, 170*, 291–303. doi:10.1016/S0079-6123(08)00424-X

Coccaro, E. F., Kavoussi, R. J., Hauger, R. L., Cooper, T. B., & Ferris, C. F. (1998). Cerebrospinal fluid vasopressin levels: Correlates with aggression and serotonin function in personality-disordered subjects. *Archives of General Psychiatry, 55*(8), 708–714. doi:10.1001/archpsyc.55.8.708

Condren, R., O'Neill, A., Ryan, M., Barrett, P., & Thakore, J. (2002). HPA axis response to a psychological stressor in generalised social phobia. *Psychoneuroendocrinology, 27*(6), 693–703. doi:10.1016/S0306-4530(01)00070-1

Dalla, C., & Shors, T. J. (2009). Sex differences in learning processes of classical and operant conditioning. *Physiology & Behavior, 97*(2), 229–238. doi:10.1016/j.physbeh.2009.02.035

de Bruin, E. I., Verheij, F., Wiegman, T., & Ferdinand, R. F. (2006). Differences in finger length ratio between males with autism, pervasive developmental disorder–not otherwise specified, ADHD, and anxiety disorders. *Developmental Medicine & Child Neurology, 48*(12), 962–965. doi:10.1017/S0012162206002118

De Kloet, E. (1991). Brain corticosteroid receptor balance and homeostatic control. *Frontiers in Neuroendocrinology, 12*(2), 95–164.

de Oliveira, D. C. G., Zuardi, A. W., Graeff, F. G., Queiroz, R. H. C., & Crippa, J. A. S. (2012). Anxiolytic-like effect of oxytocin in the simulated public speaking test. *Journal of Psychopharmacology, 26*(4), 497–504. doi:10.1177/0269881111400642

de Quervain, D. J.-F., Aerni, A., Schelling, G., & Roozendaal, B. (2009). Glucocorticoids and the regulation of memory in health and disease. *Frontiers in Neuroendocrinology, 30*(3), 358–370. doi:10.1016/j.yfrne.2009.03.002

de Quervain, D. J.-F., Bentz, D., Michael, T., Bolt, O. C., Wiederhold, B. K., Margraf, J., & Wilhelm, F. H. (2011). Glucocorticoids enhance extinction-based psychotherapy. *Proceedings of the National Academy of Sciences, 108*(16), 6621. doi:10.1073/pnas.1018214108

de Quervain, D. J.-F., Henke, K., Aerni, A., Treyer, V., McGaugh, J. L., Berthold, T., … Hock, C. (2003). Glucocorticoid-induced impairment of declarative memory retrieval is associated with reduced blood flow in the medial temporal lobe. *European Journal of Neuroscience, 17*(6), 1296–1302. doi:10.1046/j.1460-9568.2003.02542.x

de Quervain, D. J.-F., Roozendaal, B., & McGaugh, J. L. (1998). Stress and glucocorticoids impair retrieval of long-term spatial memory. *Nature, 394*, 787–790. doi:10.1038/29542

Derntl, B., Windischberger, C., Robinson, S., Kryspin-Exner, I., Gur, R. C., Moser, E., & Habel, U. (2009). Amygdala activity to fear and anger in healthy young males is associated with testosterone. *Psychoneuroendocrinology, 34*(5), 687–693. doi:10.1016/j.psyneuen.2008.11.007

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130*(3), 355. doi:10.1037/0033-2909.130.3.355

Domes, G., Heinrichs, M., Glascher, J., Buchel, C., Braus, D. F., & Herpertz, S. C. (2007). Oxytocin attenuates amygdala responses to emotional faces regardless of valence. *Biological Psychiatry, 62*(10), 1187–1190. doi: 10.1016/j.biopsych.2007.03.025

Domes, G., Heinrichs, M., Rimmele, U., Reichwald, U., & Hautzinger, M. (2004). Acute stress impairs recognition for positive words—Association with stress-induced cortisol secretion. *Stress, 7*(3), 173–182. doi: 10.1080/10253890412331273213

Ebstein, R. P., Israel, S., Lerer, E., Uzefovsky, F., Shalev, I., Gritsenko, I., … Yirmiya, N. (2009). Arginine vasopressin and oxytocin modulate human social behavior. *Annals of the New York Academy of Sciences, 1167*(1), 87–102. doi:10.1111/j.1749-6632.2009.04541.x

Edinger, K. L., & Frye, C. A. (2004). Testosterone's analgesic, anxiolytic, and cognitive-enhancing effects may be due in part to actions of its 5 α-reduced metabolites in the

hippocampus. *Behavioral Neuroscience, 118*(6), 1352–1364. doi:10.1037/0735-7044.118.6.1352

Edinger, K. L., & Frye, C. A. (2005). Testosterone's anti-anxiety and analgesic effects may be due in part to actions of its 5 α-reduced metabolites in the hippocampus. *Psychoneuroendocrinology, 30*(5), 418–430. doi:10.1016/j.psyneuen.2004.11.001

Eisenegger, C., Naef, M., Snozzi, R., Heinrichs, M., & Fehr, E. (2010). Prejudice and truth about the effect of testosterone on human bargaining behaviour. *Nature, 463*(7279), 356–359. doi:10.1038/nature08711

Ferguson, J. N., Young, L. J., Hearn, E. F., Matzuk, M. M., Insel, T. R., & Winslow, J. T. (2000). Social amnesia in mice lacking the oxytocin gene. *Nature Genetics, 25*(3), 284–288. doi:10.1038/77040

Furlan, P. M., DeMartinis, N., Schweizer, E., Rickels, K., & Lucki, I. (2001). Abnormal salivary cortisol levels in social phobic patients in response to acute psychological but not physical stress. *Biological Psychiatry, 50*(4), 254–259. doi:10.1016/S0006-3223(00)01126-4

Gimpl, G., & Fahrenholz, F. (2001). The oxytocin receptor system: Structure, function, and regulation. *Physiological Reviews, 81*, 629–683.

Goldstein, J. M., Jerram, M., Abbs, B., Whitfield-Gabrieli, S., & Makris, N. (2010). Sex differences in stress response circuitry activation dependent on female hormonal cycle. *The Journal of Neuroscience, 30*(2), 431–438. doi:10.1523/JNEUROSCI.3021-09.2010

Goldstein, J. M., Jerram, M., Poldrack, R., Ahern, T., Kennedy, D. N., Seidman, L. a. J., & Makris, N. (2005). Hormonal cycle modulates arousal circuitry in women using functional magnetic resonance imaging. *The Journal of Neuroscience, 25*(40), 9309–9316.

Goldstein, J. M., Seidman, L. J., Horton, N. J., Makris, N., Kennedy, D. N., CavinessJr, V. S., . . . Tsuang, M. T. (2001). Normal sexual dimorphism of the adult human brain assessed by in vivo magnetic resonance imaging. *Cerebral Cortex, 11*(6), 490–497. doi:10.1093/cercor/11.6.490

Granger, D. A., Shirtcliff, E. A., Zahn-Waxler, C., Usher, B., Klimes-Dougan, B., & Hastings, P. (2003). Salivary testosterone diurnal variation and psychopathology in adolescent males and females: Individual differences and developmental effects. *Development and Psychopathology, 15*(2), 431–449. doi:10.1017/S0954579403000233

Grewen, K. M., Girdler, S. S., Amico, J., & Light, K. C. (2005). Effects of partner support on resting oxytocin, cortisol, norepinephrine, and blood pressure before and after warm partner contact. *Psychosomatic Medicine, 67*(4), 531–538. doi:10.1097/01.psy.0000170341.88395.47

Guastella, A. J., Howard, A. L., Dadds, M. R., Mitchell, P., & Carson, D. S. (2009). A randomized controlled trial of intranasal oxytocin as an adjunct to exposure therapy for social anxiety disorder. *Psychoneuroendocrinology, 34*, 917–923. doi:10.1016/j.psyneuen.2009.01.005

Guastella, A. J., Kenyon, A. R., Alvares, G. A., Carson, D. S., & Hickie, I. B. (2010). Intranasal arginine vasopressin enhances the encoding of happy and angry faces in humans. *Biological Psychiatry, 67*(12), 1220–1222. doi:10.1016/j.biopsych.2010.03.014

Guastella, A. J., Kenyon, A. R., Unkelbach, C., Alvares, G. A., & Hickie, I. B. (2011). Arginine vasopressin selectively enhances recognition of sexual cues in male humans. *Psychoneuroendocrinology, 36*(2), 294–297. doi:10.1016/j.psyneuen.2010.07.023

Guastella, A. J., & MacLeod, C. (2012). A critical review of the influence of oxytocin nasal spray on social cognition in humans: Evidence and future directions. *Hormones and Behavior, 61*(3), 410–418. doi:10.1016/j.yhbeh.2012.01.002

Guastella, A. J., Mitchell, P. B., & Dadds, M. R. (2008). Oxytocin increases gaze to the eye region of human faces. *Biological Psychiatry, 63*, 3–5. doi:10.1016/j.biopsych.2007.06.026

Guastella, A. J., Mitchell, P. B., & Mathews, F. (2008). Oxytocin enhances the encoding of positive social memories in humans. *Biological Psychiatry, 64*(3), 256–258. doi:10.1016/j.biopsych.2008.02.008

Heinrichs, M., Baumgartner, T., Kirschbaum, C., & Ehlert, U. (2003). Social support and oxytocin interact to suppress cortisol and subjective responses to psychosocial stress. *Biological Psychiatry, 54*, 1389–1398. doi:10.1016/S0006-3223(03)00465-7

Heinrichs, M., Meinlschmidt, G., Neumann, I., Wagner, S., Kirschbaum, C., Ehlert, U., & Hellhammer, D. H. (2001). Effects of suckling on hypothalamic-pituitary-adrenal axis responses to psychosocial stress in postpartum lactating women. *The Journal of Clinical Endocrinology & Metabolism, 86*(10), 4798–4804. doi:10.1210/jc.86.10.4798

Hermans, E. J., Bos, P. A., Ossewaarde, L., Ramsey, N. F., Fernández, G., & van Honk, J. (2010). Effects of exogenous testosterone on the ventral striatal bold response during reward anticipation in healthy women. *Neuroimage, 52*(1), 277–283. doi:10.1016/j.neuroimage.2010.04.019

Hermans, E. J., Putman, P., Baas, J. M., Gecks, N. M., Kenemans, J. L., & van Honk, J. (2007). Exogenous testosterone attenuates the integrated central stress response in healthy young women. *Psychoneuroendocrinology, 32*(8–10), 1052–1061. doi:10.1016/j.psyneuen.2007.08.006

Hermans, E. J., Putman, P., Baas, J. M., Koppeschaar, H. P., & van Honk, J. (2006). A single administration of testosterone reduces fear-potentiated startle in humans. *Biological Psychiatry, 59*(9), 872–874. doi:10.1016/j.biopsych.2005.11.015

Hermans, E. J., Ramsey, N. F., & van Honk, J. (2008). Exogenous testosterone enhances responsiveness to social threat in the neural circuitry of social aggression in humans. *Biological Psychiatry, 63*(3), 263–270. doi:10.1016/j.biopsych.2007.05.013

Hoge, E. A., Pollack, M. H., Kaufman, R. E., Zak, P. J., & Simon, N. M. (2008). Oxytocin levels in social anxiety disorder. *CNS Neuroscience & Therapeutics, 14*(3), 165–170. doi:10.1111/j.1755-5949.2008.00051.x

Horley, K., Williams, L. M., Gonsalvez, C., & Gordon, E. (2004). Face to face: Visual scanpath evidence for abnormal processing of facial expressions in social phobia. *Psychiatry Research, 127*, 43–53. doi:10.1016/j.psychres.2004.02.016

Huber, D., Veinante, P., & Stoop, R. (2005). Vasopressin and oxytocin excite distinct neuronal populations in the central amygdala. *Science, 308*, 245–248. doi:10.1126/science.1105636

Joels, M., & Baram, T. Z. (2009). The neuro-symphony of stress. *Nature Reviews Neuroscience, 10*(6), 459–466. doi:10.1038/nrn2632

Kirsch, P., Esslinger, C., Chen, Q., Mier, D., Lis, S., Siddhanti, S., . . . Meyer-Lindenberg, A. (2005). Oxytocin modulates neural circuitry for social cognition and fear in humans. *The Journal of Neuroscience, 25*, 11489–11493. doi:10.1523/JNEUROSCI.3984-05.2005

Kirschbaum, C., Kudielka, B. M., Gaab, J., Schommer, N. C., & Hellhammer, D. H. (1999). Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus-pituitary-adrenal axis. *Psychosomatic Medicine, 61*(2), 154–162.

Kirschbaum, C., Pirke, K.-M., & Hellhammer, D. H. (1993). The "trier social stress test"—A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology, 28*, 76–81. doi:10.1159/000119004

Kosfeld, M., Heinrichs, M., Zak, P. J., Fischbacher, U., & Fehr, E. (2005). Oxytocin increases trust in humans. *Nature, 435*, 673–676. doi:10.1038/nature03701

Krężel, W., Dupont, S., Krust, A., Chambon, P., & Chapman, P. F. (2001). Increased anxiety and synaptic plasticity in estrogen receptor β-deficient mice. *Proceedings of the National Academy of Sciences, 98*(21), 12278–12282. doi:10.1073/pnas.221451898

Kuhlmann, S., Piel, M., & Wolf, O. T. (2005). Impaired memory retrieval after psychosocial stress in healthy young men. *The Journal of Neuroscience, 25*(11), 2977–2982. doi:10.1523/JNEUROSCI.5139-04.2005

Labuschagne, I., Phan, K. L., Wood, A., Angstadt, M., Chua, P., Heinrichs, M., . . . Nathan, P. J. (2010). Oxytocin attenuates amygdala reactivity to fear in generalized social anxiety disorder. *Neuropsychopharmacology, 35*, 2403–2413. doi:10.1038/npp.2010.123

Lamberts, S. W. J., Verleun, T., Oosterom, R., De Jong, F., & Hackeng, W. H. L. (1984). Corticotropin-releasing factor (ovine) and vasopressin exert a synergistic effect on adrenocorticotropin release in man. *The Journal of Clinical Endocrinology & Metabolism, 58*(2), 298–303. doi:10.1210/jcem-58-2-298

Landgraf, R., & Neumann, I. D. (2004). Vasopressin and oxytocin release within the brain: A dynamic concept of multiple and variable modes of neuropeptide communication. *Frontiers in Neuroendocrinology, 25*, 150–176. doi:10.1016/j.yfrne.2004.05.001

Levin, A. P., Saoud, J. B., Strauman, T., Gorman, J. M., Fyer, A. J., Crawford, R., & Liebowitz, M. R. (1993). Responses of "generalized" and "discrete" social phobics during public speaking. *Journal of Anxiety Disorders, 7*(3), 207–221. doi:10.1016/0887-6185(93)90003-4

Lim, M. M., Murphy, A. Z., & Young, L. J. (2004). Ventral striatopallidal oxytocin and vasopressin v1a receptors in the monogamous prairie vole (*Microtus ochrogaster*). *Journal of Comparative Neurology, 468*, 555–570. doi:10.1002/cne.10973

Lim, M. M., & Young, L. J. (2006). Neuropeptidergic regulation of affiliative behavior and social bonding in animals. *Hormones and Behavior, 50*, 506–517. doi:10.1016/j.yhbeh.2006.06.028

Lolait, S. J., Stewart, L. Q., Jessop, D. S., Young, W. S., & O'Carroll, A.-M. (2007). The hypothalamic-pituitary-adrenal axis response to stress in mice lacking functional vasopressin v1b receptors. *Endocrinology, 148*(2), 849–856. doi:10.1210/en.2006-1309

Love, T., Smith, Y. R., Persad, C. C., Tkaczyk, A., & Zubieta, J.-K. (2010). Short-term hormone treatment modulates emotion response circuitry in postmenopausal women. *Fertility and Sterility, 93*(6), 1929–1937. doi:10.1016/j.fertnstert.2008.12.056

Lund, T. D., Rovis, T., Chung, W. C. J., & Handa, R. J. (2005). Novel actions of estrogen receptor-β on anxiety-related behaviors. *Endocrinology, 146*(2), 797–807. doi:10.1210/en.2004-1158

Martel, F. L., Hayward, C., Lyons, D. M., Sanborn, K., Varady, S., & Schatzberg, A. F. (1999). Salivary cortisol levels in socially phobic adolescent girls. *Depression and Anxiety, 10*(1), 25–27. doi:10.1002/(SICI)1520-6394(1999)10:1<25::AID-DA4>3.0.CO;2-O

McCall, C., & Singer, T. (2012). The animal and human neuroendocrinology of social cognition, motivation and behavior. *Nature Neuroscience, 15*(5), 681–688. doi:10.1038/nn.3084

McEwen, B. S. (2006). Stress, adaptation, and disease: Allostasis and allostatic load. *Annals of the New York Academy of Sciences, 840*(1), 33–44. doi:10.1111/j.1749-6632.1998.tb09546.x

McGaugh, J. L., & Roozendaal, B. (2002). Role of adrenal stress hormones in forming lasting memories in the brain. *Current Opinion in Neurobiology, 12*(2), 205–210. doi:10.1016/S0959-4388(02)00306-9

McLaughlin, K., Baran, S., & Conrad, C. (2009). Chronic stress- and sex-specific neuromorphological and functional changes in limbic structures. *Molecular Neurobiology, 40*(2), 166–182. doi:10.1007/s12035-009-8079-7

Meyer-Lindenberg, A., Domes, G., Kirsch, P., & Heinrichs, M. (2011). Oxytocin and vasopressin in the human brain: Social neuropeptides for translational medicine. *Nature Reviews Neuroscience, 12*, 524–538. doi:10.1007/s12035-009-8079-7

Milad, M. R., Igoe, S. A., Lebron-Milad, K., & Novales, J. E. (2009). Estrous cycle phase and gonadal hormones influence conditioned fear extinction. *Neuroscience, 164*(3), 887–895. doi:10.1016/j.neuroscience.2009.09.011

Milad, M. R., Zeidan, M. A., Contero, A., Pitman, R. K., Klibanski, A., Rauch, S. L., & Goldstein, J. M. (2010). The influence of gonadal hormones on conditioned fear extinction in healthy humans. *Neuroscience, 168*(3), 652–658. doi:10.1016/j.neuroscience.2010.04.030

Myers, K. M., & Davis, M. (2007). Mechanisms of fear extinction. *Molecular Psychiatry, 12*, 120–150. doi:10.1038/sj.mp.4001939

Neumann, I. D. (2002). Involvement of the brain oxytocin system in stress coping: Interactions with the hypothalamo-pituitary-adrenal axis. *Progress in Brain Research, 139*, 147–162. doi:10.1016/S0079-6123(02)39014-9

Oei, N. Y. L., Elzinga, B. M., Wolf, O. T., De Ruiter, M. B., Damoiseaux, J. S., Kuijer, J. P. A., . . . Rombouts, S. A. R. B. (2007). Glucocorticoids decrease hippocampal and prefrontal activation during declarative memory retrieval in young men. *Brain Imaging and Behavior, 1*(1), 31–41. doi:10.1007/s11682-007-9003-2

Östlund, H., Keller, E., & Hurd, Y. L. (2003). Estrogen receptor gene expression in relation to neuropsychiatric disorders. *Annals of the New York Academy of Sciences, 1007*(1), 54–63. doi:10.1196/annals.1286.006

Pavlov, I. P. (1927). *Conditioned reflexes: An investigation of the physiological activity of the cerebral cortex*. London, UK: Oxford University Press.

Pedersen, C. A., Vadlamudi, S. V., Boccia, M. L., & Amico, J. A. (2006). Maternal behavior deficits in nulliparous oxytocin knockout mice. *Genes Brain and Behavior, 5*, 274–281. doi:10.1111/j.1601-183X.2005.00162.x

Petrovic, P., Kalisch, R., Singer, T., & Dolan, R. J. (2008). Oxytocin attenuates affective evaluations of conditioned faces and amygdala activity. *The Journal of Neuroscience, 28*(26), 6607–6615. doi:10.1523/JNEUROSCI.4572-07.2008

Phan, K. L., Fitzgerald, D. A., Nathan, P. J., & Tancer, M. E. (2006). Association between amygdala hyperactivity to harsh faces and severity of social anxiety in generalized social phobia. *Biological Psychiatry, 59*(5), 424–429. doi:10.1016/j.biopsych.2005.08.012

Quirin, M., Kuhl, J., & Düsing, R. (2011). Oxytocin buffers cortisol responses to stress in individuals with impaired emotion regulation abilities. *Psychoneuroendocrinology, 36*, 898–904. doi:10.1016/j.psyneuen.2010.12.005

Roelofs, K., Bakvis, P., Hermans, E. J., van Pelt, J., & van Honk, J. (2007). The effects of social stress and cortisol responses on the preconscious selective attention to social threat. *Biological Psychology, 75*(1), 1–7. doi:10.1016/j.biopsycho.2006.09.002

Roelofs, K., van Peer, J., Berretty, E., de Jong, P., Spinhoven, P., & Elzinga, B. M. (2009). Hypothalamus–pituitary–adrenal axis hyperresponsiveness is associated with increased social avoidance behavior in social phobia. *Biological Psychiatry, 65*(4), 336–343. doi:10.1016/j.biopsych.2008.08.022

Sarkey, S., Azcoitia, I., Garcia-Segura, L. M., Garcia-Ovejero, D., & DonCarlos, L. L. (2008). Classical androgen receptors in non-classical sites in the brain. *Hormones and Behavior, 53*(5), 753–764. doi:10.1016/j.yhbeh.2008.02.015

Schwabe, L., Haddad, L., & Schachinger, H. (2008). HPA axis activation by a socially evaluated cold-pressor test. *Psychoneuroendocrinology, 33*(6), 890–895. doi:10.1016/j.psyneuen.2008.03.001

Shalev, I., Israel, S., Uzefovsky, F., Gritsenko, I., Kaitz, M., & Ebstein, R. P. (2011). Vasopressin needs an audience: Neuropeptide elicited stress responses are contingent upon perceived social evaluative threats. *Hormones and Behavior, 60*(1), 121–127. doi:10.1016/j.yhbeh.2011.04.005

Shansky, R. M., Hamo, C., Hof, P. R., Lou, W., McEwen, B. S., & Morrison, J. H. (2010). Estrogen promotes stress sensitivity in a prefrontal cortex–amygdala pathway. *Cerebral Cortex, 20*(11), 2560–2567. doi:10.1093/cercor/bhq003

Slattery, D. A., & Neumann, I. D. (2010). Chronic ICV oxytocin attenuates the pathological high anxiety state of selectively bred wistar rats. *Neuropharmacology, 58*, 56–61. doi:10.1016/j.neuropharm.2009.06.038

Soravia, L. M., Heinrichs, M., Aerni, A., Maroni, C., Schelling, G., Ehlert, U., . . . de Quervain, D. J.-F. (2006). Glucocorticoids reduce phobic fear in humans. *Proceedings of the National Academy of Sciences, 103*(14), 5585–5590. doi:10.1073/pnas.0509184103

Stanton, S. J., Wirth, M. M., Waugh, C. E., & Schultheiss, O. C. (2009). Endogenous testosterone levels are associated with amygdala and ventromedial prefrontal cortex responses to anger faces in men but not women. *Biological Psychology, 81*(2), 118–122. doi:10.1016/j.biopsycho.2009.03.004

Taylor, S., Gonzaga, G., Klein, L., Hu, P., Greendale, G., & Seeman, T. (2006). Relation of oxytocin to psychological stress responses and hypothalamic-pituitary-adrenocortical axis activity in older women. *Psychosomatic Medicine, 68*(2), 238–245.

Thompson, R. R., George, K., Walton, J. C., Orr, S. P., & Benson, J. (2006). Sex-specific influences of vasopressin on human social communication. *Proceedings of the National Academy of Sciences, 103*(20), 7889–7894. doi:10.1073/pnas.0600406103

Thompson, R. R., Gupta, S., Miller, K., Mills, S., & Orr, S. (2004). The effects of vasopressin on human facial responses related to social communication. *Psychoneuroendocrinology, 29*(1), 35–48. doi:10.1016/S0306-4530(02)00133-6

Toufexis, D. J., Myers, K. M., & Davis, M. (2006). The effect of gonadal hormones and gender on anxiety and emotional learning. *Hormones and Behavior, 50*(4), 539–549. doi:10.1016/j.yhbeh.2006.06.020

Tsigos, C., & Chrousos, G. P. (2002). Hypothalamic–pituitary–adrenal axis, neuroendocrine factors and stress. *Journal of Psychosomatic Research, 53*(4), 865–871. doi:10.1016/S0022-3999(02)00429-4

Turner, R. A., Altemus, M., Enos, T., Cooper, B., & McGuinness, T. (1999). Preliminary research on plasma oxytocin in normal cycling women: Investigating emotion and interpersonal distress. *Psychiatry, 62*(2), 97–113.

Unkelbach, C., Guastella, A. J., & Forgas, J. P. (2008). Oxytocin selectively facilitates recognition of positive sex and relationship words. *Psychological Science, 19*, 1092–1094. doi:10.1111/j.1467-9280.2008.02206.x

van Honk, J., Peper, J. S., & Schutter, D. J. (2005). Testosterone reduces unconscious fear but not consciously experienced anxiety: Implications for the disorders of fear and anxiety. *Biological Psychiatry, 58*(3), 218–225. doi:10.1016/j.biopsych.2005.04.003

van Honk, J., & Schutter, D. J. (2007). Testosterone reduces conscious detection of signals serving social correction implications for antisocial behavior. *Psychological Science, 18*(8), 663–667. doi:10.1111/j.1467-9280.2007.01955.x

van Honk, J., Tuiten, A., Hermans, E. J., Putman, P., Koppeschaar, H., Thijssen, J., . . . Van Doornen, L. (2001). A single administration of testosterone induces cardiac accelerative responses to angry faces in healthy young women. *Behavioral Neuroscience, 115*(1), 238–242. doi:10.1037/0735-7044.115.1.238

van Honk, J., Tuiten, A., van Den Hout, M., Koppeschaar, H., Thijssen, J., de Haan, E., & Verbaten, R. (2000). Conscious and preconscious selective attention to social threat: Different neuroendocrine response patterns. *Psychoneuroendocrinology, 25*(6), 577–591. doi:10.1016/S0306-4530(00)00011-1

van Wingen, G. A., Mattern, C., Verkes, R. J., Buitelaar, J., & Fernández, G. (2008). Testosterone biases automatic memory processes in women towards potential mates. *Neuroimage, 43*(1), 114–120. doi:10.1016/j.neuroimage.2008.07.002

van Wingen, G. A., Mattern, C., Verkes, R. J., Buitelaar, J., & Fernández, G. (2010). Testosterone reduces amygdala–orbitofrontal cortex coupling. *Psychoneuroendocrinology, 35*(1), 105–113. doi:10.1016/j.psyneuen.2009.09.007

van Wingen, G. A., Ossewaarde, L., Bäckström, T., Hermans, E. J., & Fernández, G. (2011). Gonadal hormone regulation of the emotion circuitry in humans. *Neuroscience, 191*, 38–45. doi:10.1016/j.neuroscience.2011.04.042

van Wingen, G. A., Zylicz, S. A., Pieters, S., Mattern, C., Verkes, R. J., Buitelaar, J. K., & Fernández, G. (2008). Testosterone increases amygdala reactivity in middle-aged women to a young adulthood level. *Neuropsychopharmacology, 34*(3), 539–547. doi:10.1038/npp.2008.2

Viviani, D., & Stoop, R. (2008). Opposite effects of oxytocin and vasopressin on the emotional expression of the fear response. *Progress in Brain Research, 170*, 207–218. doi:10.1016/S0079-6123(08)00418-4

Walf, A. A., & Frye, C. A. (2006). A review and update of mechanisms of estrogen in the hippocampus and amygdala for anxiety and depression behavior. *Neuropsychopharmacology, 31*(6), 1097–1111. doi:10.1038/sj.npp.1301067

Wang, Z., Young, L. J., De Vries, G. J., & Insel, T. R. (1998). Voles and vasopressin: A review of molecular, cellular, and behavioral studies of pair bonding and paternal behaviors. *Progress in Brain Research, 119*, 483–499. doi:10.1016/S0079-6123(08)61589-7

Weeks, J. W., Jakatdar, T. A., & Heimberg, R. G. (2010). Comparing and contrasting fears of positive and negative evaluation as facets of social anxiety. *Journal of Social and Clinical Psychology, 29*(1), 68–94. doi:10.1521/jscp.2010.29.1.68

Williams, J. R., Insel, T. R., Harbaugh, C. R., & Carter, C. S. (1994). Oxytocin administered centrally facilitates formation of a partner preference in female prairie voles (*Microtus ochrogaster*). *Journal of Neuroendocrinology, 6*, 247–250. doi:10.1111/j.1365-2826.1994.tb00579.x

Windle, R. J., Kershaw, Y. M., Shanks, N., Wood, S. A., Lightman, S. L., & Ingram, C. D. (2004). Oxytocin attenuates stress-induced c-fos mRNA expression in specific forebrain regions associated with modulation of hypothalamo–pituitary–adrenal activity. *The Journal of Neuroscience, 24*(12), 2974–2982. doi:10.1523/JNEUROSCI.3432-03.2004

Wolf, O. T. (2008). The influence of stress hormones on emotional memory: Relevance for psychopathology. *Acta Psychologica, 127*(3), 513–531. doi:10.1016/j.actpsy.2007.08.002

Yang, Y.-L., Chao, P.-K., & Lu, K.-T. (2005). Systemic and intra-amygdala administration of glucocorticoid agonist and antagonist modulate extinction of conditioned fear. *Neuropsychopharmacology, 31*(5), 912–924. doi:10.1038/sj.npp.1300899

Young, L. J., & Wang, Z. (2004). The neurobiology of pair bonding. *Nature Neuroscience, 7*, 1048–1054. doi:10.1038/nn1327

Zak, P. J., Kurzban, R., & Matzner, W. T. (2005). Oxytocin is associated with human trustworthiness. *Hormones and Behavior, 48*, 522–527. doi:10.1016/j.yhbeh.2005.07.009

Zeidan, M. A., Igoe, S. A., Linnman, C., Vitalo, A., Levine, J. B., Klibanski, A., . . . Milad, M. R. (2011). Estradiol modulates medial prefrontal cortex and amygdala activity during fear extinction in women and female rats. *Biological Psychiatry, 70*(10), 920–927. doi:10.1016/j.biopsych.2011.05.016

VI
Treatment

22

Cognitive-Behavioral Therapy for Social Anxiety Disorder

The State of the Science

Dina Gordon, Judy Wong, and Richard G. Heimberg

Adult Anxiety Clinic of Temple University, USA

Cognitive-behavioral models of social anxiety disorder (SAD; Clark & Wells, 1995; Heimberg, Brozovich, & Rapee, 2010; see **Chapter 1** of this volume) suggest that a reduction in social anxiety may be achieved by creating opportunities to reframe maladaptive beliefs related to the self and others in social situations and by targeting patterns of avoidance. Consistent with these models, cognitive-behavioral therapy (CBT) aims to modify distorted cognitions and avoidant behavior with cognitive restructuring and exposure to feared situations. This chapter provides a review of empirically examined techniques, including exposure administered alone and in combination with cognitive restructuring, social skills training (SST), and applied relaxation (AR).

Exposure

Exposure treatment for social anxiety involves direct and indirect engagement with the feared stimulus, such as initiating a conversation or speaking in front of an audience (McNeil, Lejuez, & Sorrel, 2010; see **Chapter 23** of this volume). Whereas avoidance maintains distorted beliefs about the danger of a social situation or the inevitability of unceasing anxiety, exposure creates a context in which a socially anxious individual may receive feedback that provides important disconfirmatory information that modifies irrational beliefs. Exposure is most effective when an individual is fully engaged with the physiological and emotional arousal associated with the feared situation (Foa & Kozak, 1986); this optimally occurs when the client is instructed not to use safety behaviors and to focus attention externally on the targeted situation rather than internally on the self (Wells et al., 1995; Wells & Papageorgiou, 1998).

Exposure techniques have resulted in greater reductions in social anxiety than waitlist (Butler, Cullington, Munby, Amies, & Gelder, 1984), pill placebo (Turner, Beidel, & Jacob, 1994), and relaxation training (Alström, Nordlund, Persson, Hårding,

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

& Ljungqvist, 1984) conditions. Overall, exposure as a stand-alone treatment is efficacious in the treatment of SAD, and most meta-analyses of treatment outcome for SAD (Acarturk, Cuijpers, van Straten, & de Graaf, 2009; Powers, Sigmarsson, & Emmelkamp, 2008) show little difference in outcome between exposure alone and exposure combined with other techniques such as cognitive restructuring. Nevertheless, Heimberg and Juster (1995) have questioned the maintenance of treatment gains from exposure, citing preliminary evidence that there may be greater likelihood of loss of gains over the long term with exposure alone (Alström et al., 1984; Mattick, Peters, & Clarke, 1989) as well as a higher frequency of treatment seeking during the follow-up period (Butler et al., 1984). Subsequently, the combination of exposure and cognitive restructuring has been the focus of much research.

Combination of Exposure and Cognitive Restructuring

According to cognitive-behavioral models, dysfunctional beliefs and biased information processing are largely responsible for the maintenance of anxiety. It is not the social situation itself, but rather an individual's subjective interpretation of that situation and the perceived likelihood and cost of negative outcomes, that evokes anxiety (Beck & Emery, 1985). This assertion provides the initial rationale for adding cognitive restructuring to exposure. Additional rationale arises from the very nature of social situations as unpredictable, variable, and ever-changing, as compared to many phobic stimuli of a nonsocial type, thus complicating the process of habituation (Butler, 1985) and increasing the potential value of disconfirmatory information about the meaning of the outcomes of social events. Cognitive restructuring can be extremely useful in advance of feared situations, helping to reduce anticipatory anxiety and thus avoidance; to the extent that avoidance persists, exposure does not occur and cannot be helpful.

Consistent with this reasoning, most cognitive-behavioral treatments for SAD incorporate cognitive restructuring. One of the most widely used CBT protocols for SAD (Hope, Heimberg, & Turk, 2010) integrates cognitive restructuring into exposure exercises by instructing individuals with SAD to identify and challenge maladaptive thoughts and to use the exposure as a test of the accuracy of those beliefs. Repeated graduated exposure and cognitive restructuring allow individuals with SAD the opportunity to test and reframe their fears into more logical, helpful thoughts. More adaptive interpretations allow closer attentiveness to social performance and interaction, make way for a greater openness to fully engage in anxiety-evoking social situations, and reduce postevent processing (see **Chapter 15** of this volume).

Research investigating CBT for SAD has been quite productive in recent years, and there are now a number of empirically supported protocols for both individual (Clark et al., 2003; Hope et al., 2010) and group (Heimberg & Becker, 2002) treatment. In line with the cognitive model of SAD proposed by Clark and Wells (1995), individual cognitive therapy (CT; Clark et al., 2003, 2006) comprises exposure and cognitive restructuring, with a strong emphasis on elimination of safety behaviors. Another key element of CT is the use of *video feedback* (see **Chapter 23** of this volume) to compare predicted and actual performance in the service of reframing distorted mental

self-representations. Compared to applied relaxation (AR) which combines exposure and progressive muscle relaxation (PMR) (described in more detail later), patients in the CT condition were twice as likely to be classified as responders (Clark et al., 2006). Moreover, gains made during individual CT have been sustained at 5-year follow-up (Mörtberg, Clark, & Bejerot, 2011).

Cognitive-behavioral group therapy (CBGT) is comprised of psychoeducation, *in vitro* and *in vivo* exposure, cognitive restructuring, and homework assignments (Heimberg & Becker, 2002). Compared to an educational-supportive group therapy, CBGT resulted in greater reductions in anxiety posttreatment (Heimberg et al., 1990) and better maintenance of gains over a 5-year period (Heimberg, Salzman, Holt, & Blendell, 1993). CBGT has compared favorably to medication (reviewed later in this chapter). It has also been adapted to an individual format (Hope et al., 2010) that demonstrated effect sizes similar to those of the group protocol (Goldin et al., 2012; Ledley et al., 2009). In comparing individual CBT to a wait-list condition, Ledley et al. (2009) found high protocol adherence, little attrition, and significantly greater improvements on both self-report and clinician-administered measures of social anxiety.

Efficacy studies have produced substantial evidence in favor of CBT for SAD; however, the generalizability of such studies is limited by the environment in which they are conducted (e.g., research settings in universities or medical schools), the structure of and monitored adherence to manualized treatment protocols, and strict exclusion criteria which often eliminate individuals of certain ages or who have comorbid mood or substance use disorders (Lincoln et al., 2003). Several benchmarking studies have now been conducted to address the generalizability and transportability of empirically supported treatments for SAD to private practice and community settings. These studies show encouraging results in both group (Gaston, Abbot, Rapee, & Neary, 2006; McEvoy, 2007; McEvoy, Nathan, Rapee, & Campbell, 2012) and individual (Lincoln et al., 2003) formats, as effect sizes in community settings were comparable to those reported in the laboratory and published in meta-analyses.

Social Skills Training

Some research points to deficient interpersonal skills in socially anxious individuals; however, this proposition has mixed support (Rapee & Lim, 1992; Stopa & Clark, 1993). Examples of such deficiencies include poor eye contact and difficulty maintaining a conversation. Rapee and Lim (1992) point out that an objective observer's judgment of a socially anxious individual's performance during exposures is often at odds with the individual's self-reported lack of interpersonal skill, in that the performance is often deemed to be appropriate by objective observers. Consistent with a negative interpretation bias in evaluating their own performance, socially anxious individuals typically underestimate their social competence (Rapee & Lim, 1992; Taylor & Alden, 2005), although there is certainly room for improvement in the performance of many clients when rated by objective observers (Stopa & Clark, 1993). With regard to circumstances in which objective impairments in social behavior are observed, their origin may be attributable to various factors including a true deficit in social skills,

a physical and cognitive preoccupation with anxiety that inhibits the application of social skills, or a combination of these or other factors. For a more detailed review of the controversy regarding social skills deficiencies versus performance deficits, see **Chapter 17** of this volume.

Social skills training (SST) is a behavioral intervention designed to provide an opportunity for individuals to improve upon verbal and nonverbal interpersonal skills by rehearsing social behaviors with the instruction, modeling, corrective feedback, and reinforcement of a therapist. Numerous studies support the efficacy of SST in treating social anxiety (Mersch, Emmelkamp, Bogels, & Van der Sleen, 1989; Trower, Yardley, Bryant & Shaw, 1978; Wlazlo, Schroeder-Hartwig, Hand, & Kaiser, 1990). However, Ponniah and Hollon (2008) argue that, because these trials did not include adequate control conditions, it cannot be stated unequivocally that SST is sufficient in producing successful outcomes on its own. The one controlled study of SST showed that this treatment failed to produce significantly more improvement in social skills or social anxiety than a wait-list control condition (Marzillier, Lambert, & Kellett, 1976). However, a more recent study comparing CBGT alone to CBGT coupled with SST found greater gains for the combination condition (Herbert et al., 2005).

It is possible that these mixed findings have to do with alternate explanations for the mechanism of action in SST. For example, the benefits associated with SST may be explained by the repeated exposure involved in rehearsing social situations or the cognitive reappraisal that may stem from corrective feedback about the adequacy of one's interpersonal skills. Another possibility is that some socially anxious individuals, such as those whose primary fear is the visibility of physical symptoms associated with anxiety (e.g., blushing, sweating), are more likely to benefit from SST; Bögels and Voncken (2008) found evidence that such individuals benefited equally from SST and CT. Overall, the literature examining the efficacy of SST is mixed, with some evidence demonstrating that SST in combination with other approaches is efficacious; however, without dismantling the various treatment components, it is difficult to conclude whether SST specifically accounted for such success.

Relaxation Techniques

Relaxation techniques have been utilized to help individuals with SAD cope with the somatic symptoms of anxiety, based on the premise that excessive physiological arousal impedes performance in social situations. Relaxation training, administered alone or in combination with exposure to anxiety-evoking stimuli, has mixed support in the empirical literature. PMR, a technique in which various muscle groups are sequentially tensed and relaxed, shows little efficacy in reducing social anxiety when administered alone (Alström et al., 1984). Systematic desensitization, a technique combining PMR with imaginal exposure to a hierarchy of anxiety-evoking social situations, failed to demonstrate superiority to wait-list controls (Kanter & Goldfried, 1979; Marzillier et al., 1976).

Applied relaxation combines relaxation and exposure techniques via the application of PMR while facing feared situations. Individuals initially learn to recognize early signs of anxiety and cope with them by practicing relaxation until they reach a relaxed

state. These skills are then transferred to anxiety-evoking situations (Öst, 1987). Early studies of AR (Jerremalm, Jansson, & Öst, 1986; Öst, Jerremalm, & Johansson, 1981) produced encouraging findings in the treatment of SAD. In a more recent comparison of Clark's CT, AR, and a wait-list control, the two active conditions were superior to wait-list on most measures of psychopathology and general distress, yet CT was more efficacious than AR in reducing social anxiety (Clark et al., 2006). Further research is needed to determine whether AR has the potential to augment established CBT treatments.

Comparison of CBT to Other Forms of Psychotherapy

Mindfulness-Based Therapies

Recently, there has been a growing interest in mindfulness-based psychotherapies. Mindfulness refers to a state of nonjudgmental awareness of the present moment, and mindfulness practices involve focusing attention on one's internal experiences in a nonjudgmental manner. Mindfulness approaches are believed to promote emotion regulation, which may improve psychological functioning and symptom reduction in psychiatric disorders, including SAD (Goldin & Gross, 2010). For more in-depth discussion of mindfulness-based approaches to SAD, please see **Chapter 27** of this volume.

Two studies have compared a mindfulness-based treatment to a CBT for SAD. Koszycki, Benger, Shlik, and Bradwejn (2007) randomly assigned 53 individuals with generalized SAD to either mindfulness-based stress reduction (MBSR; Kabat-Zinn, 1990) or CBGT (Heimberg & Becker, 2002). Patients in the MBSR condition received eight weekly 2.5-hr sessions and an all-day meditation retreat (27.5 hr of total treatment), and CBGT patients received 12 weekly 2.5-hr group sessions (30 hr of total treatment). Both groups demonstrated improvement, but CBGT patients showed greater improvement on clinician- and self-rated measures of social anxiety and greater treatment response and remission rates. The MBSR group showed comparable improvement in mood, general functioning, and quality of life, which is notable given that the MBSR intervention was not developed to target SAD.

Piet, Hougaard, Hecksher, and Rosenberg (2010) compared mindfulness-based cognitive therapy (MBCT; Segal, Teasdale, & Williams, 2002) to group CBT in a pilot study conducted in Denmark. Twenty-six young adults with SAD were randomized to one of the two treatment groups: one group received MBCT first, followed by group CBT, and the other received the two treatments in the reversed order. MBCT consisted of eight weekly 2-hr group sessions, along with daily mindfulness homework. Group CBT combined elements of the group treatment developed by Heimberg and Becker (2002) and individual treatment based on the Clark and Wells' (1995) model and consisted of 12 weekly 2-hr group sessions, with the addition of two weekly, 2-hr individual therapy sessions. Both groups showed improvement and were not significantly different after the first intervention period (i.e., MBCT compared to group CBT), after the second intervention period (i.e., MBCT followed by group CBT compared to group CBT followed by MBCT), or at 6- or 12-month follow-up. However, there was a trend toward larger effect sizes for group CBT. The researchers

were also interested in the effect of adding a mindfulness treatment program to CBT, but contrary to study hypotheses, augmenting CBT with MBCT did not result in larger effect sizes compared to CBT alone. Interpretation of the results of this study is limited by the small size of the groups (*n*s = 13).

Thus, the emerging research on mindfulness-based treatments suggests that they are viable alternatives to CBT, though likely less efficacious in reducing social anxiety. Future research should examine whether fully integrating mindfulness-based techniques into CBT protocols would improve efficacy.

Interpersonal Psychotherapy

Interpersonal psychotherapy (IPT; Klerman, Weissman, Rounsaville, & Chevron, 1984) was first developed to treat depression and aims to reduce distress and impairment by targeting interpersonal difficulties. Likewise, IPT approaches to SAD conceptualize the disorder as a dysfunction in interpersonal processes and social relationships. Alden and Taylor (2004) describe the core dysfunction as a self-perpetuating interpersonal cycle in which individuals with SAD, who anticipate negative social responses, engage in self-protective social behaviors that elicit negative responses, thereby maintaining their negative expectations. This self-perpetuating cycle parallels the negative belief feedback loops found in cognitive-behavioral models of SAD. In addition, treatments based on either model emphasize exposure to social situations and practice of new adaptive behaviors. However, a main difference in treatment is that IPT aims to promote prosocial behaviors and better communication of a person's wants and feelings in relationships, rather than targeting his/her dysfunctional beliefs and avoidance behaviors.

Preliminary support for IPT as a treatment for SAD is mixed. Lipsitz, Markowitz, Cherry, and Fyer (1999) conducted an open trial of 14-week, individual IPT. Nine ethnically diverse patients showed a significant decrease in social anxiety distress, and an independent assessor rated seven of these patients as treatment responders. This study was followed by a randomized controlled trial comparing IPT to a supportive therapy (ST; Lipsitz et al., 2008). Contrary to study hypotheses, IPT was not superior to ST, and the proportion of IPT treatment responders (42%) was lower than in major CBT trials, where the rates of response for intent-to-treat samples range from 52% to 76% (Clark et al., 2006; Davidson et al., 2004; Heimberg et al., 1998).

Two studies to date have compared IPT for SAD to a cognitive treatment. Borge et al. (2008) compared IPT to CT in a randomized controlled trial with 80 patients with primary SAD at a Norwegian inpatient clinic. The treatments were modified for use in a tertiary care inpatient treatment setting and included a mix of group and individual sessions over 10 weeks. The number of dropouts did not differ significantly between treatments, and residential IPT (RIPT) and residential CT (RCT) were equally efficacious, with patients showing significant improvement across a number of indicators at posttreatment and at 1-year follow-up.

In the largest and best-conducted controlled trial in this area to date, Stangier, Schramm, Heidenreich, Berger, and Clark (2011) compared IPT and CT to a wait-list control group at two sites in Germany. The sample was comprised of 117 individuals

with SAD randomized to one of the three groups, and the active treatments consisted of 16 individual sessions over 20 weeks. IPT and CT were both superior to wait-list, and ratings of treatment credibility and therapeutic alliance were high for both treatments. However, a significantly greater percentage of people who received CT (65.8%) were classified as treatment responders compared to the IPT group (42.1%), and this difference was maintained at 1-year follow-up. In addition, more people who received IPT sought additional treatment following the study.

In summary, there is evidence for the efficacy of IPT for SAD, although it does not seem to be as efficacious as CBT. Further research comparing the two approaches is warranted, particularly for the purpose of identifying differential moderators of treatment outcome (Borge, Hoffart, & Sexton, 2010). This is important as this could inform clinicians as to when IPT may be the best fit for patients and whether it is a useful option for CBT nonresponders.

Comparisons and Combinations with Pharmacotherapy

Medication treatments for SAD are described in **Chapter 24**. Here, we look briefly at CBT in comparison to and in combination with pharmacotherapy for SAD. Early trials showed cognitive-behavioral treatments to be superior to medications, but the medications evaluated in those studies had not themselves demonstrated superiority to pill placebo.

CBGT has been compared to several pharmacotherapies with previously established efficacy for the treatment of SAD. Gelernter et al. (1991) demonstrated essentially equivalent outcomes for CBGT, phenelzine (a monoamine oxidase inhibitor), and alprazolam (a high-potency benzodiazepine), and Otto et al. (2000) demonstrated CBGT to be roughly equivalent to the benzodiazepine clonazepam. In a multi-site study comparing CBGT to phenelzine, an educational-supportive group therapy, and pill placebo in 133 patients with SAD, a similarly high percentage of patients completing CBGT (75%) and phenelzine (77%) treatments were classified as responders by independent assessors (Heimberg et al., 1998). However, after responders completed six additional months of maintenance treatment and a 6-month follow-up period, half of the original responders to phenelzine relapsed compared to just 17% of responders to CBGT (Liebowitz et al., 1999), suggesting that the group therapy is superior to medication in retaining treatment gains.

Clark et al. (2003) compared individual CT to the selective serotonin reuptake inhibitor (SSRI) fluoxetine plus self-exposure to placebo plus self-exposure in 60 patients with SAD. After 16 weeks, there were significant improvements in all three conditions, but CT was superior to both the medication and placebo conditions at midtreatment, posttreatment, and 12-month follow-up. It is unclear whether differences in session length or the inclusion of self-exposure instructions in the administration of medication and placebo confounded the interpretation of these results, although the difference between CT and the other conditions was substantial.

In search of more effective treatments for SAD, CBT–medication combinations have been examined, but the results do not provide a clear message. Davidson et al. (2004) examined the efficacy of combining group CBT with fluoxetine. Group

CBT was similar to Heimberg's CBGT but also included SST. All active treatments (fluoxetine alone, CBT alone, fluoxetine plus CBT, CBT plus placebo) were superior to placebo, but they were equally efficacious. Combining fluoxetine with group CBT, therefore, did not provide increased benefit. In another study, Blanco et al. (2010) compared CBGT, phenelzine, CBGT plus phenelzine, and pill placebo. The combined treatment was significantly more efficacious than placebo, but surprisingly this was not the case for phenelzine or CBGT alone.

Blomhoff et al. (2001) examined the efficacy of exposure and the SSRI sertraline. All treatment conditions (sertraline alone, exposure alone, sertraline plus exposure, and pill placebo with no exposure) lasted 24 weeks, with patients in the exposure group completing 12 weeks of primary care physician-facilitated exposure and 12 weeks of self-exposure. After 12 weeks, all active treatment groups were superior to placebo. At posttreatment, only the sertraline group was superior to placebo. However, by the 1-year follow-up, participants in the exposure group had demonstrated further improvement, whereas those receiving sertraline or the combination treatment deteriorated on some measures (Haug et al., 2003). The results suggest that combined treatment does not enhance efficacy and may even detract from it; however, the study examined exposure administered by physicians with minimal CBT training, the pattern of results was unexpected, and questions have been raised about the interpretations of the results (Bandelow, 2004). In summary, research findings are mixed. To date, it is unclear whether combining traditional pharmacotherapy with CBT confers any benefit above the use of these treatments alone.

Combination of Nontraditional Pharmacotherapy and Exposure

Recently, basic research on the neural substrates of fear extinction has prompted interest in the use of nontraditional pharmacotherapies that may enhance the effectiveness of exposure (see also **Chapter 18** for expanded discussion on this issue). One such example is D-cycloserine (DCS), a partial NMDA receptor agonist, which facilitates extinction of learned fear in animals (Davis, Ressler, Rothbaum, & Richardson, 2006) and in humans (Ressler et al., 2004). A controlled trial investigated whether administering DCS prior to exposure enhanced its efficacy (Hofmann et al., 2006), comparing exposure alone (exposure plus pill placebo) to exposure plus DCS in a sample of patients with SAD. The abbreviated exposure-based protocol consisted of one psychoeducation session and four exposure sessions. Participants were required to have a fear of public speaking, as exposures focused solely on this activity. At posttreatment, individuals in the DCS group showed greater reductions on self-reported symptoms of anxiety. Although clinician-administered ratings of global severity did not differ between the two groups at posttreatment or follow-up, results at both time points displayed trends in the same direction as the self-report measures. The second randomized trial, conducted by Guastella et al. (2008), essentially replicated the results of Hofmann et al. (2006). Furthermore, benefits were maintained at 1-month follow-up. Together, results from these studies suggest the efficacy of DCS as an

adjunct to exposure for SAD. A trial of DCS-facilitated group CBT has recently been conducted, but results were less encouraging than the trials reported above (Hofmann et al., 2012).

Neuroimaging Findings

Research incorporating neuroimaging techniques such as functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) has begun to identify the brain regions active in SAD (see also **Chapter 4**) and the changes in activation of various brain regions following various forms for treatment. Individuals with SAD show greater activity in the amygdala, uncus, and parahippocampal gyrus than healthy controls in response to contemptuous and angry faces (Stein, Goldin, Sareen, Eyler Zorrilla, & Brown, 2002). An examination of the neural mechanisms of emotion regulation in SAD using fMRI showed that individuals with SAD were less successful than controls in recruiting cognitive and emotional regulation brain networks in response to threatening social stimuli (i.e., harsh facial expressions) but not to images of physical threat (Goldin, Manber, Hakimi, Canli, & Gross, 2009).

Treatment studies indicate that medication, CBGT, individual CBT, and MBSR for SAD are associated with changes in the activation of brain regions (Furmark et al., 2002; Goldin, Ramel, & Gross, 2009; Goldin et al., 2013). Furmark et al. (2002) conducted a randomized controlled trial to examine the neural sites of action upon treatment with the SSRI citalopram, CBGT, or wait-list control. Both active treatment conditions led to significant and approximately equal improvements, whereas the wait-list remained unchanged. PET imaging of regional cerebral blood flow before and after treatment identified sites in the right hemisphere, including the amygdala, hippocampus, and other adjacent regions involved in defensive responses to threatening stimuli. Blood flow was significantly reduced to these brain regions, both in the active treatment groups compared to controls and among individuals classified as responders compared to nonresponders in the active treatment groups. Changes in the activation of brain regions associated with response to threat may be elicited by both citalopram and CBGT.

As discussed above, several open trials have supported the efficacy of MBSR for SAD. In a recent fMRI study, individuals who underwent MBSR showed increased self-endorsement of positive traits and decreased self-endorsement of negative traits (Goldin, Ramel, et al., 2009). These improvements were associated with increased activity in the brain networks related to attention regulation and reduced activity in regions related to self-referential processing and language. Further, Goldin and Gross (2010) found increased activation in brain regions related to attentional deployment and decreased activation in the amygdala. More recently, we reported that individual CBT led to greater and quicker recruitment of dorsolateral and dorsomedial prefrontal cortex neural responses associated with reduced amygdala reactivity to negative self-beliefs and reduced severity of social anxiety symptoms (Goldin et al., 2013). For a broader review of the application of social neuroscience to SAD, see **Chapter 4** of this volume.

Factors Affecting Treatment Outcome

Subtype of SAD

In diagnosing SAD, an individual may be categorized as either having generalized or nongeneralized subtypes of the disorder (American Psychiatric Association, 2000). A diagnosis of generalized SAD indicates that *most* social situations are feared, commonly encompassing both social and performance situations. Nongeneralized SAD refers to a relatively more circumscribed fear of social or performance situations, such as fear of speaking in public or fear of eating in public, although this is a heterogeneous category including those with single and multiple (but not most) feared situations. Not surprisingly, individuals with generalized SAD tend to suffer from more severe social anxiety because their anxiety and avoidance extend to most areas of their lives and result in broader impairment. Consequently, those with generalized SAD, entering treatment with more severe levels of symptomatology than individuals with nongeneralized SAD, are less likely to achieve high end-state functioning following CBT (Brown, Heimberg, & Juster, 1995; Hope, Herbert, & White, 1995).

Comorbidity

SAD is highly comorbid with other anxiety and mood disorders (Kessler, Stang, Wittchen, Stein, & Walters, 1999); therefore, it is important to consider the effects of comorbidity on the course and outcome of treatment for SAD. In a comparison of individuals with uncomplicated SAD, SAD with a comorbid anxiety disorder, and SAD with a comorbid mood disorder, those with comorbid mood disorder reported greater symptom severity before and after CBGT (Erwin, Heimberg, Juster, & Mindlin, 2002). Individuals with comorbid anxiety disorders did not differ from those with uncomplicated SAD.

With regard to the presence of self-reported and clinician-rated symptoms of depression, a study which excluded individuals who had major depressive disorder in the past 6 months but who endorsed elevated pretreatment depressive symptoms found that elevated symptoms of depression were related to greater severity of SAD overall, reduced change in social anxiety symptoms throughout treatment, and higher treatment dropout rates (Ledley et al., 2005). Consistent with this finding, individuals who entered CBGT for SAD with a higher pretreatment level of self-reported depressive symptoms experienced diminished posttreatment reduction in anticipatory social anxiety (Chambless, Tran, & Glass, 1997). However, this association was not found for clinician ratings of depression.

Concerning the reciprocal effect of depression and social anxiety on each other during treatment, Moscovitch, Hofmann, Suvak, and In-Albon (2005) found that changes in depression were fully mediated by changes in social anxiety among individuals with SAD receiving CBGT. Whereas changes in social anxiety accounted for 91% of the variance in depression, changes in depression only accounted for 6% of the variance in social anxiety scores. It seems that improvements in depressive symptoms follow improvement in social anxiety among patients with primary SAD. For a

more detailed discussion of the comorbidity of social anxiety with mood disorders, see **Chapter 10** of this volume.

Outcome Expectancy

Individuals seeking treatment for SAD may express mixed expectations about their ability to improve. Several studies indicate that socially anxious individuals partaking in CBGT who expected treatment gains and found the protocol credible experienced greater improvements than those who were less optimistic about treatment (Chambless et al., 1997; Safren, Heimberg, & Juster, 1997; Westra, Dozois, & Marcus, 2007). This remained the case when pretreatment severity of SAD was controlled (Safren et al., 1997). More recently, Price and Anderson (2012) controlled for group-specific variance in order to account for the shared experience of the group therapy and found that treatment outcome expectancy accounted for 16–33% of the variance in decreased public-speaking anxiety. These results suggest that clinicians should emphasize the importance of a patient's role in the ultimate success of treatment early on in therapy.

Treatment Modality

Individual and group formats of CBT for SAD have been shown to produce similar outcomes in most meta-analyses (Acarturk et al., 2009; Powers et al., 2008) and in one Iranian study that directly compared them (Dogaheh, Mohammadkhani, & Dolatsheh, 2011). However, there is some evidence of a slight advantage for individual therapy (Aderka, 2009; Stangier, Heidenreich, Peitz, Lauterbach, & Clark, 2003). Each format has benefits and drawbacks. Group therapy provides a context in which individuals with SAD may access additional social support and facilitate the normalization of anxiety. Logistically, the group modality facilitates the execution of exposures by increasing access to role-players for protocols which utilize in-session exposures. Moreover, a patient's therapeutic relationship with the therapist may interfere with anxiety evocation (Hayes, Hope, VanDyke, & Heimberg, 2007), and the therapist may not always be the appropriate role-player for all scenarios (e.g., dating situations). Group members may also assist each other in cognitive restructuring and by providing feedback following exposures. Group therapy may not always be the best option, however. For example, individuals whose high symptom severity interferes with their ability to fully engage in the group process would likely benefit most from individual therapy. The individual format is also better suited to address idiosyncratic concerns and allows for more flexibility in the duration and pace of treatment. The role of such moderating factors requires further research.

Inclusion of Supplementary Treatment Components

Motivational interviewing. Motivation for therapy varies among individuals, which is why some treatment protocols incorporate motivational interviewing exercises (see also **Chapter 13**). Westra and Dozois (2006) examined whether preceding group

CBT with three sessions of motivational interviewing would lead to an improvement in outcome among patients with SAD, panic disorder, and generalized anxiety disorder. Compared to individuals who did not receive the pretreatment intervention, those who participated in the motivational exercises exhibited greater positive expectancy for change prior to treatment, were more adherent with homework during treatment, and had a greater likelihood of being classified as responders to CBT. Motivational exercises have been included in Hope et al. (2010).

Video feedback. The rationale for using video feedback as part of a CBT protocol for SAD is that viewing oneself performing or interacting in social situations provides a more objective context for correcting distorted images of the self. Video feedback, first reported by Rapee and Hayman (1996) and later amended to include cognitive preparation that enhanced the effects of video feedback (Harvey, Clark, Ehlers, & Rapee, 2000), instructs a socially anxious individual to predict in detail how he or she would look in a social situation, vividly imagine how he or she looked, and then watch the video of him or herself from a stranger's perspective. Several studies have demonstrated beneficial effects of incorporating video feedback into treatment (Harvey et al., 2000; Kim, Lundh, & Harvey, 2002; Rapee & Hayman, 1996; Rodebaugh, Heimberg, Schultz, & Blackmore, 2010).

Video feedback was found to improve one's self-perception of speech task performance, especially among those individuals with the greatest self-observer discrepancy (i.e., most unrealistically negative impressions of their performance), indicating that self-observer discrepancy is a predictor of response to video feedback (Rodebaugh & Rapee, 2005). A recent meta-analysis suggested that the use of video feedback did not moderate SAD treatment efficacy despite the presence of seemingly larger effect sizes in studies using video feedback than in those omitting it (Aderka, 2009); however, the meta-analysis was limited by the inclusion of only five studies in the moderation analyses. Video feedback may be a useful treatment addition for some individuals, particularly those with highly distorted self-perception.

Homework Compliance

Finally, homework compliance has been associated with treatment gains. Adherent individuals completing cognitive behavioral treatment for SAD experienced better outcomes than those with poorer compliance at various stages of treatment: Leung and Heimberg (1996) found this association immediately following treatment and Edelman and Chambless (1995) did so at 6-month follow-up. One study failed to replicate those findings (Woody & Adessky, 2002).

Issues for Further Study

Treatment Among Understudied Populations

There has been very little research to date looking at the efficacy or effectiveness of interventions for SAD in members of cultural groups other than people of European

descent in Western cultures. In general, there is little research on CBT across cultural groups, and there is a question of whether the theories and concepts underlying cognitive-behavioral treatments developed from the Western perspective are appropriate across cultural groups (Rathod & Kingdon, 2009). A handful of case studies have been written about CBT for the treatment of SAD in Japan (Toyokawa & Nedate, 2006), with an African American client (Fink, Turner, & Beidel, 1996), and in immigrants to the United States (Weiss, Singh, & Hope, 2011). In addition, Walsh and Hope (2010) describe a case in which they adapted a CBT protocol to be more lesbian, gay, bisexual (LGB)-affirmative for a self-identified gay client with SAD. All case studies cited above describe successful CBT treatment, implemented only with slight modifications to treatment protocols.

Chen et al. (2007) examined group CBT for 57 Japanese patients with SAD. Seven patients dropped out (12.3%); the drop-out rate was smaller in comparison to studies of group CBT in Western countries, which tend to range from 20% to 25% (Hofmann & Suvak, 2006), suggesting that the acceptability of the treatment among Japanese patients is at least comparable to that of their Western counterparts. For the intent-to-treat sample at posttreatment, there was small to moderate symptom reduction across the several self-report measures administered in the study. The authors compared their results to other group CBT studies and found their effect sizes to be significantly larger, though they caution over-interpretation of this finding by noting that their treatment groups were small (three to four people) and that the more individualized attention may have accounted for the increased effectiveness of their treatment. The authors also note that limitations to the study include lack of a comparison group, lack of an independent assessor (i.e., assessments were administered by the therapist), failure to control for psychotropic medication use, and a small sample size.

The case studies and the study by Chen et al. provide preliminary evidence that psychotherapy treatments for SAD developed in Western countries can be effective across cultural groups. For more in-depth discussion of culture and SAD, please refer to **Chapter 11** of this volume.

Nonresponders to Treatment

Although the evidence clearly demonstrates the efficacy of CBT for SAD, some socially anxious individuals are not adequately responsive to treatment. Little research exists that addresses the question of why some individuals do not improve significantly following CBT. In an examination of possible factors that predict response to group CBT for SAD, Chambless et al. (1997) found that higher pretreatment depression negatively predicted short-term and long-term outcome. Lower treatment expectancies and avoidant personality traits also predicted poorer outcome at posttreatment, but the pattern was less robust at 6-month follow-up. A replication of the Chambless et al. study by Scholing and Emmelkamp (1999) supported the finding that pretreatment depression was a significant predictor of outcome; however, the effect of depression was smaller and appeared only at posttest but not at 18-month follow-up. Contrary to Chambless et al., they did not find personality traits to be predictive; they did not measure treatment expectancies, which they acknowledge could be a clinically significant

predictive factor. However, given the generally mixed findings in this area of research, Scholing and Emmelkamp recommended that future research focus away from pretreatment client characteristics and more on how therapeutic process factors—such as homework compliance and therapeutic alliance—influence treatment outcome. Ultimately the goal is to understand how we can better improve our treatments to serve nonresponders.

A subset of treatment nonresponders includes those who terminate treatment early, and it has been repeatedly found that a relatively high rate of socially anxious participants in CBT studies drop out of treatment. The percentage of dropouts ranges widely among studies, though a number of studies show attrition rates that hover in the 16–22% range (Couttraux et al., 2000; Davidson et al., 2004; Dogaheh et al., 2011; Goldin et al., 2012; Heimberg et al., 1990, 1998; Stangier et al., 2011). In an attempt to identify factors that distinguish treatment completers from dropouts, Hofmann and Suvak (2006) looked at 133 individuals who received either CBGT or a behavioral group treatment for SAD in an outpatient anxiety clinic. No differences were found in demographic characteristics, social anxiety symptomatology, levels of depression, additional Axis I diagnoses, personality disorder symptomatology, or self-rated attitudes toward treatment. Following dropout, participants were contacted to obtain reasons for early treatment termination. Some of the respondents (totaling 17 of the 34 dropouts) indicated that they terminated treatment due to treatment-related factors, including discomfort with the group format ($n = 3$) and feelings that treatment was ineffective ($n = 4$). The authors note that a limitation of their study, however, was that they were unable to measure other variables that may have better differentiated dropouts from completers, such as therapist style or therapist–client match. Further research is needed to identify ways to decrease early treatment termination, as well as to identify patients for whom CBT or group treatment is not a good fit.

Summary

We have examined the literature on the efficacy of CBT for SAD, focusing on the specific combination of exposure to feared social situations and cognitive restructuring techniques. Although there is not strong evidence that cognitive techniques are an essential part of this mix, the combination has demonstrated positive outcomes in a number of studies, roughly equivalent to medication treatments (although this literature is sparse), and superior to other cognitive-behavioral techniques (e.g., AR) and other systems of psychotherapy (e.g., IPT). CBT also appears to retain its effectiveness when evaluated in community settings, although there is yet to be much work in that area that has been conducted by investigators with nonacademic affiliations. Several factors appear to predict CBT outcome, including the nature of SAD symptoms (generalized versus nongeneralized SAD), comorbidity with depression, outcome expectancies, and compliance with homework assignments, although considerable further research in these areas is warranted. Finally, there are many areas of knowledge that have simply not been well enough addressed. These include the utility of CBT for minority populations which have not been well represented in the treatment literature, who will and will not respond to treatment, and what to do for

patients who do not respond adequately to first attempts at treatment, whether it be with CBT, medication, or other modalities. The state of the science is good, but there remains much work to do.

References

Acarturk, C., Cuijpers, P., van Straten, A., & de Graaf, R. (2009). Psychological treatment of social anxiety disorder: A meta analysis. *Psychological Medicine, 39*, 241–254. doi:10.1017/S0033291708003590

Aderka, I. M. (2009). Factors affecting treatment efficacy in social phobia: The use of video feedback and individual vs. group formats. *Journal of Anxiety Disorders, 23*, 12–17. doi:10.1016/j.janxdis.2008.05.003

Alden, L. E., & Taylor, C. T. (2004). Interpersonal processes in social phobia. *Clinical Psychology Review, 24*, 857–882. doi:10.1016/j.cpr.2004.07.006

Alström, J. E., Nordlund, C. L., Persson, G., Hårding, M., & Ljungqvist, C. (1984). Effects of four treatment methods on social phobic patients not suitable for insight-oriented psychotherapy. *Acta Psychiatrica Scandinavica, 70*, 97–110. doi:10.1111/j.1600-0447.1984.tb01187.x

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

Bandelow, B. (2004). Sertraline and exposure therapy in social phobia. *British Journal of Psychiatry, 184*, 271. doi:10.1192/bjp.184.3.271

Beck, A. T., & Emery, G. (1985). *Anxiety disorders and phobias: A cognitive perspective*. New York, NY: Basic Books.

Blanco, C., Heimberg, R. G., Schneier, F. R., Fresco, D. M., Chen, H., Turk, C. L., . . . Liebowitz, M. R. (2010). A placebo-controlled trial of phenelzine, cognitive behavioral group therapy, and their combination for social anxiety disorder. *Archives of General Psychiatry, 67*, 286–295. doi:10.1001/archgenpsychiatry.2010.11

Blomhoff, S., Haug, T. T., Hellström, K., Holme, I., Humble, M., Madsbu, H. P., & Wold, J. E. (2001). Randomised controlled general practice trial of sertraline, exposure therapy and combined treatment in generalised social phobia. *British Journal of Psychiatry, 179*, 23–30. doi: 10.1192/bjp.179.1.23

Bögels, S. M., & Voncken, M. (2008). Social skills training versus cognitive therapy for social anxiety disorder characterized by fear of blushing, trembling, or sweating. *International Journal of Cognitive Therapy, 1*, 138–150. doi:10.1521/ijct.2008.1.2.138

Borge, F.-M., Hoffart, A., & Sexton, H. (2010). Predictors of outcome in residential cognitive and interpersonal treatment for social phobia: Do cognitive and social dysfunction moderate treatment outcome? *Journal of Behavior Therapy and Experimental Psychiatry, 41*, 212–219. doi:10.1016/j.jbtep.2010.01.005

Borge, F.-M., Hoffart, A., Sexton, H., Clark, D. M., Markowitz, J. C., & McManus, F. (2008). Residential cognitive therapy versus residential interpersonal therapy for social phobia: A randomized clinical trial. *Journal of Anxiety Disorders, 22*, 991–1010. doi:10.1016/j.janxdis.2007.10.002

Brown, E. J., Heimberg, R. G., & Juster, H. R. (1995). Social phobia subtype and avoidant personality disorder: Effect of severity of social phobia, impairment, and outcome of cognitive-behavioral treatment. *Behavior Therapy, 26*, 467–486. doi:10.1016/S0005-7894(05)80095-4

Butler, G. (1985). Exposure as treatment for social phobia: Some instructive difficulties. *Behaviour Research and Therapy, 23*, 651–657. doi:10.1016/0005-7967(85)90060-9

Butler, G., Cullington, A., Munby, M., Amies, P., & Gelder, M. (1984). Exposure and anxiety management in the treatment of social phobia. *Journal of Consulting and Clinical Psychology, 52*, 642–650. doi:10.1037/0022-006X.52.4.642

Chambless, D. L., Tran, G. Q., & Glass, C. R. (1997). Predictors of response to cognitive-behavioral group therapy for social phobia. *Journal of Anxiety Disorders, 11*, 221–240. doi:10.1016/S0887-6185(97)00008-X

Chen, J., Nakano, Y., Ietzugu, T., Ogawa, S., Funayama, T., Watanabe, N., . . . Furukawa, T. A. (2007). Group cognitive behavior therapy for Japanese patients with social anxiety disorder: Preliminary outcomes and their predictors. *BMC Psychiatry, 7*. doi:10.1186/1471-244X-7-69

Clark, D. M., Ehlers, A., Hackmann, A., McManus, F., Fennell, M., Grey, N., . . . Wild, J. (2006). Cognitive therapy versus exposure and applied relaxation in social phobia: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 74*, 568–578. doi:10.1037/0022-006X.74.3.568

Clark, D. M., Ehlers, A., McManus, F., Hackmann, A., Fennell, M., Campbell, H., . . . Louis, B. (2003). Cognitive therapy versus fluoxetine in generalized social phobia: A randomized placebo-controlled trial. *Journal of Consulting and Clinical Psychology, 71*, 1058–1067. doi:10.1037/0022-006X.71.6.1058

Clark, D. M., & Wells, A. (1995). A cognitive model of social phobia. In R. G. Heimberg, M. R. Liebowitz, D. A. Hope, & F. R. Schneier (Eds.), *Social phobia: Diagnosis, assessment, and treatment* (pp. 69–93). New York, NY: Guilford Press.

Couttraux, J., Note, I., Albuisson, E., Yao, S. N., Note, B., Mollard, E., . . . Coudert, A. J. (2000). Cognitive behavior therapy versus supportive therapy in social phobia: A randomized controlled trial. *Psychotherapy and Psychosomatics, 69*, 137–146.

Davidson, J. R. T., Foa, E. B., Huppert, J. D., Keefe, F. J., Franklin, M. E., Compton, J. S., . . . Gadde, K. M. (2004). Fluoxetine, comprehensive cognitive behavioral therapy, and placebo in generalized social phobia. *Archives of General Psychiatry, 61*, 1005–1013. doi:10.1001/archpsyc.61.10.1005

Davis, M., Ressler, K., Rothbaum, B. O., & Richardson, R. (2006). Effects of D-cycloserine on extinction: Translation from preclinical to clinical work. *Biological Psychiatry, 60*, 369–375. doi:10.1016/j.biopsych.2006.03.084

Dogaheh, E. R., Mohammadkhai, P., & Dolatshahi, B. (2011). Comparison of group and individual cognitive-behavioral therapy in reducing fear of negative evaluation. *Psychological Reports, 108*, 955–962. doi:10.2466/02.21.PR0.108.3.955-962

Edelman, R. E., & Chambless, D. L. (1995). Adherence during sessions and home cognitive behavior group treatment of social phobia. *Behaviour Research and Therapy, 33*, 573–577. doi:10.1016/0005-7967(94)00068-U

Erwin, B. A., Heimberg, R. G., Juster, H., & Mindlin, M. (2002). Comorbid anxiety and mood disorders among persons with social anxiety disorder. *Behaviour Research and Therapy, 40*, 19–35. doi:10.1016/S0005-7967(00)00114-5

Fink, C. M., Turner, S. M., & Beidel, D. C. (1996). Culturally relevant factors in the behavioral treatment of social phobia: A case study. *Journal of Anxiety Disorders, 10*, 201–209. doi:10.1016/0887-6185(96)00005-9

Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin, 99*, 20–35. doi:10.1037/0033-2909.99.1.20

Furmark, T., Tillfors, M., Marteinsdottir, I., Fischer, H., Pissiota, A., Långström, B., & Fredrikson, M. (2002). Common changes in cerebral blood flow in patients with social phobia

treated with citalopram or cognitive-behavioral therapy. *Archives of General Psychiatry, 59*, 425–433. doi:10.1001/archpsyc.59.5.425

Gaston, J. E., Abbott, M. J., Rapee, R. M., & Neary, S. A. (2006). Do empirically supported treatments generalize to private practice? A benchmark study of a cognitive-behavioural group treatment programme for social phobia. *British Journal of Clinical Psychology, 45*, 33–48. doi:10.1348/014466505X35146

Gelernter, C. S., Uhde, T. W., Cimbolic, P., Arnkoff, D. B., Vittone, B. J., Tancer, M. E., & Bartko, J. J. (1991). Cognitive-behavioral and pharmacological treatment of social phobia. *Archives of General Psychiatry, 48*, 938–945. doi:10.1001/archpsyc.1991.01810340070009

Goldin, P. R., & Gross, J. J. (2010). Effects of mindfulness-based stress reduction (MBSR) on emotion regulation in social anxiety disorder. *Emotion, 10*, 83–91. doi:10.1037/a0018441

Goldin, P. R., Manber, T., Hakimi, S., Canli, T., & Gross, J. J. (2009). Neural bases of social anxiety disorder: Emotional reactivity and cognitive regulation during social and physical threat. *Archives of General Psychiatry, 66*, 170–180. doi:10.1001/archgenpsychiatry.2008.525

Goldin, P. R., Ramel, W., & Gross, J. J. (2009). Mindfulness meditation training and self-referential processing in social anxiety disorder: Behavioral and neural effects. *Journal of Cognitive Psychotherapy, 23*, 242–257. doi:10.1891/0889-8391.23.3.242

Goldin, P. R., Ziv, M., Jazaieri, H., Hahn, K., Heimberg, R. G., & Gross, J. J. (2013). Impact of cognitive-behavioral therapy for social anxiety disorder on the neural dynamics of cognitive reappraisal of negative self-beliefs. *JAMA Psychiatry*. doi:10.1001/jamapsychiatry.2013.234

Goldin, P. R., Ziv, M., Jazaieri, H., Werner, K., Kraemer, H., Heimberg, R. G., & Gross, J. J. (2012). Cognitive reappraisal self-efficacy mediates the effects of individual cognitive-behavioral therapy for social anxiety disorder in a randomized controlled trial. *Journal of Consulting and Clinical Psychology, 80*(6), 1034–1040.

Guastella, A. J., Richardson, R., Lovibond, P. F., Rapee, R. M., Gaston, J. E., Mitchell, P., & Dadds, M. R. (2008). A randomized controlled trial of D-cycloserine enhancement of exposure therapy for social anxiety disorder. *Biological Psychiatry, 63*, 544–549. doi:10.1016/j.biopsych.2007.11.011

Harvey, A. G., Clark, D. M., Ehlers, A., & Rapee, R. M. (2000). Social anxiety and self-impression: Cognitive preparation enhances the beneficial effects of video feedback following a stressful social task. *Behaviour Research and Therapy, 38*, 1183–1192. doi:10.1016/S0005-7967(99)00148-5

Haug, T. T., Blomhoff, S., Hellstrøm, K., Holme, I., Humble, M., Madsbu, H. P., & Wold, J. E. (2003). Exposure therapy and sertraline in social phobia: 1-year follow-up of a randomised controlled trial. *British Journal of Psychiatry, 182*, 312–318. doi:10.1192/bjp.182.4.312

Hayes, S. A., Hope, D. A., VanDyke, M. M., & Heimberg, R. G. (2007). Working alliance for clients with social anxiety disorder: Relationship with session helpfulness and within-session habituation. *Cognitive Behaviour Therapy, 36*, 34–42. doi:10.1080/16506070600947624

Heimberg, R. G., & Becker, R. E. (2002). *Cognitive-behavioral group therapy for social phobia: Basic mechanisms and clinical strategies*. New York, NY: Guilford Press.

Heimberg, R. C., Brozovich, F. A., & Rapee, R. M. (2010). A cognitive behavioral model of social anxiety disorder: Update and extension. In S. G. Hofmann & P. M. DiBartolo (Eds.), *Social anxiety: Clinical, developmental, and social perspectives* (2nd ed., pp. 395–422). San Diego, CA: Elsevier Academic Press. doi:10.1016/B978-0-12-375096-9.00015-8

Heimberg, R. G., Dodge, C. S., Hope, D. A., Kennedy, C. R., Zollo, L., & Becker, R. E. (1990). Cognitive behavioral group treatment of social phobia: Comparison to a credible placebo control. *Cognitive Therapy and Research, 14*, 1–23. doi:10.1007/BF01173521

Heimberg, R. G., & Juster, H. R. (1995). Cognitive-behavioral treatment: Literature review. In Heimberg, M. Liebowitz, D. Hope, & F. R. Schneier (Eds.), *Social phobia: Diagnosis, assessment and treatment* (pp. 261–309). New York, NY: Guilford Press.

Heimberg, R. G., Liebowitz, M. R., Hope, D. A., Schneier, F. R., Holt, C. S., Welkowitz, L., . . . Klein, D. F. (1998). Cognitive-behavioral group therapy versus phenelzine in social phobia: 12-week outcome. *Archives of General Psychiatry, 55*, 1133–1141. doi:10.1001/archpsyc.55.12.1133

Heimberg, R. G., Salzman, D. G., Holt, C. S., & Blendell, K. A. (1993). Cognitive-behavioral group treatment for social phobia: Effectiveness at five-year follow up. *Cognitive Therapy and Research, 17*, 325–339. doi:10.1007/BF01177658

Herbert, J. D., Gaudiano, B. A., Rheingold, A. A., Myers, V., Dalrymple, K., & Nolan, E. M. (2005). Social skills training augments the effectiveness of cognitive behavioral group therapy for social anxiety disorder. *Behavior Therapy, 36*, 125–138. doi:10.1016/S0005-7894(05)80061-9

Hofmann, S. G., Meuret, A. E., Smits, J. A. J., Simon, N. M., Pollack, M. H., Eisenmenger, K., . . . Otto, M. W. (2006). Augmentation of exposure therapy with D-cycloserine for social anxiety disorder. *Archives of General Psychiatry, 63*, 298–304. doi:10.1001/archpsyc.63.3.298

Hofmann, S. G., Smits, J., Simon, N., Meuret, A., Asnaani, A., Sawyer, A., . . . Pollack, M. (2012, April). *Short-term and long-term efficacy of D-cycloserine augmentation of cognitive-behavioral group therapy in social anxiety disorder*. Paper presented at the annual meeting of the Anxiety Disorders Association of America, Arlington, VA.

Hofmann, S. G., & Suvak, M. (2006). Treatment attrition during group therapy for social phobia. *Journal of Anxiety Disorders, 20*, 961–972. doi:10.1016/j.janxdis.2006.03.005

Hope, D. A., Heimberg, R. G., & Turk, C. L. (2010). *Managing social anxiety: A cognitive-behavioral approach (client workbook)* (2nd ed.). New York, NY: Oxford University Press.

Hope, D. A., Herbert, J. D., & White, C. (1995). Diagnostic subtype, avoidant personality disorder, and efficacy of cognitive-behavioral group therapy for social phobia. *Cognitive Therapy and Research, 19*, 399–417. doi:10.1007/BF02230408

Jerremalm, A., Jansson, L., & Öst, L. (1986). Cognitive and physiological reactivity and the effects of different behavioral methods in the treatment of social phobia. *Behaviour Research and Therapy, 24*, 171–180. doi:10.1016/0005-7967(86)90088-4

Kabat-Zinn, J. (1990). *Full catastrophe living: Using the wisdom of your mind and body to face stress, pain, and illness*. New York, NY: Delacorte.

Kanter, N. J., & Goldfried, M. R. (1979). Relative effectiveness of rational restructuring and self-control desensitization in the reduction of interpersonal anxiety. *Behavior Therapy, 10*, 472–490. doi:10.1016/S0005-7894(79)80051-9

Kessler, R. C., Stang, P., Wittchen, H.-U., Stein, M., & Walters, E. E. (1999). Lifetime comorbidities between social phobia and mood disorders in the US National Comorbidity Survey. *Psychological Medicine, 29*, 555–567. doi:10.1017/S0033291799008375

Kim, H. Y., Lundh, L.-G., & Harvey, A. (2002). The enhancement of video feedback by cognitive preparation in the treatment of social anxiety: A single-session experiment. *Journal of Behavior Therapy and Experimental Psychiatry, 33*, 19–37. doi:10.1016/S0005-7916(02)00010-1

Klerman, G. L., Weissman, M. M., Rounsaville, B. J., & Chevron, E. S. (1984). *Interpersonal psychotherapy of depression*. New York, NY: Basic Books.

Koszycki, D., Benger, M., Shlik, J., & Bradwejn, J. (2007). Randomized trial of a meditation-based stress reduction program and cognitive behavior therapy in generalized social anxiety disorder. *Behaviour Research and Therapy, 45*, 2518–2526. doi:10.1016/j.brat.2007.04.011

Ledley, D. R., Heimberg, R. G., Hope, D. A., Hayes, S. A., Zaider, T. I., Van Dyke, M., . . . Fresco, D. M. (2009). Efficacy of a manualized and workbook-driven individual treatment for social anxiety disorder. *Behavior Therapy, 40*, 414–424. doi:10.1016/j.beth.2008.12.001

Ledley, D. R., Huppert, J. D., Foa, E. B., Davidson, J. R. T., Keefe, F. J., & Potts, N. L. S. (2005). Impact of depressive symptoms on the treatment of generalized social anxiety disorder. *Depression and Anxiety, 22*, 161–167. doi:10.1002/da.20121

Leung, A. W., & Heimberg, R. G. (1996). Homework compliance, perceptions of control, and outcome of cognitive-behavioral treatment of social phobia. *Behaviour Research and Therapy, 34*, 423–432. doi:10.1016/0005-7967(96)00014-9

Liebowitz, M. R., Heimberg, R. G., Schneier, F. R., Hope, D. A., Davies, S., Holt, C. S., . . . Klein, D. F. (1999). Cognitive-behavioral group therapy versus phenelzine in social phobia: Long term outcome. *Depression and Anxiety, 10*, 89–98. doi:10.1002/(SICI)1520-6394(1999)10:3<89::AID-DA1>3.0.CO;2-5

Lincoln, T. M., Rief, W., Hahlweg, K., Frank, M., von Witzleben, I., Schroeder, B., & Fiegenbaum, W. (2003). Effectiveness of an empirically supported treatment for social phobia in the field. *Behaviour Research and Therapy, 41*, 1251–1269. doi:10.1016/S0005-7967(03)00038-X

Lipsitz, J. D., Gur, M., Vermes, D., Petkova, E., Cheng, J., Miller, N., . . . Fryer, A., J. (2008). A randomized trial of interpersonal therapy versus supportive therapy for social anxiety disorder. *Depression and Anxiety, 25*, 542–553. doi:10.1002/da.20364

Lipsitz, J. D., Markowitz, J. C., Cherry, S., & Fyer, A. J. (1999). Open trial of interpersonal psychotherapy for the treatment of social phobia. *American Journal of Psychiatry, 156*, 1814–1816. doi:10.1002/(SICI)1520-6394(1999)10:3<105::AID-DA3>3.0.CO;2-X

Marzillier, J. S., Lambert, C., & Kellett, J. (1976). A controlled evaluation of systematic desensitization and social skills training for socially inadequate psychiatric patients. *Behaviour Research and Therapy, 14*, 225–238. doi:10.1016/0005-7967(76)90015-2

Mattick, R. P., Peters, L., & Clarke, J. C. (1989). Exposure and cognitive restructuring for social phobia: A controlled study. *Behavior Therapy, 20*, 3–23. doi:10.1016/S0005-7894(89)80115-7

McEvoy, P. M. (2007). Effectiveness of cognitive behavioural group therapy for social phobia in a community clinic: A benchmarking study. *Behaviour Research and Therapy, 45*, 3030–3040. doi:10.1016/j.brat.2007.08.002

McEvoy, P. M., Nathan, P., Rapee, R. M., & Campbell, B. N. C. (2012). Cognitive behavioural group therapy for social phobia: Evidence of transportability to community clinics. *Behaviour Research and Therapy, 50*, 258–265. doi:10.1016/j.brat.2012.01.009

McNeil, D. W., Lejuez, C. W., & Sorrel, J. T. (2010). Basic behavioral mechanisms and processes in social anxieties and social anxiety disorders. In S. G. Hofmann & P. M. DiBartolo (Eds.), *Social anxiety: Clinical, developmental, and social perspectives* (2nd ed., pp. 347–372). San Diego, CA: Elsevier Academic Press. doi:10.1016/B978-0-12-375096-9.00013-4

Mersch, P. P., Emmelkamp, P. M., Bögels, S. M., & Van der Sleen, J. (1989). Social phobia: Individual response patterns and the effects of behavioral and cognitive interventions. *Behaviour Research and Therapy, 27*, 421–434. doi:10.1016/0005-7967(89)90013-2

Mörtberg, E., Clark, D. M., & Bejerot, S. (2011). Intensive group cognitive therapy and individual cognitive therapy for social phobia: Sustained improvement at 5-year follow-up. *Journal of Anxiety Disorders, 25*, 994–1000. doi:10.1016/j.janxdis.2011.06.007

Moscovitch, D. A., Hofmann, S. G., Suvak, M. K., & In-Albon, T. (2005). Mediation of changes in anxiety and depression during treatment of social phobia. *Journal of Consulting and Clinical Psychology, 73*, 945–952. doi:10.1037/0022-006X.73.5.945

Öst, L. G. (1987). Applied relaxation: Description of a coping technique and review of controlled studies. *Behaviour Research and Therapy, 25*, 397–409. doi:10.1016/0005-7967(87)90017-9

Öst, L. G., Jerremalm, A., & Johansson, J. (1981). Individual response patterns and the effects of different behavioral methods in the treatment of social phobia. *Behaviour Research and Therapy, 19*, 1–16. doi:10.1016/0005-7967(81)90107-8

Otto, M. W., Pollack, M. H., Gould, R. A., Worthington, J. J., McArdle, E. T., Rosenbaum, J. F., & Heimberg, R. G. (2000). A comparison of the efficacy of clonazepam and cognitive-behavioral group therapy for the treatment of social phobia. *Journal of Anxiety Disorders, 14*, 345–358. doi:10.1016/S0887-6185(00)00027-X

Piet, J., Hougaard, E., Hecksher, M. S., & Rosenberg, N. K. (2010). A randomized pilot study of mindfulness-based cognitive therapy and group cognitive-behavioral therapy for young adults with social phobia. *Scandinavian Journal of Psychology, 51*, 403–410. doi:10.1111/j.1467-9450.2009.00801.x

Ponniah, K., & Hollon, S. D. (2008). Empirically supported psychological interventions for social phobia in adults: A qualitative review of randomized controlled trials. *Psychological Medicine, 38*, 3–14. doi:10.1017/S0033291707000918

Powers, M. B., Sigmarsson, S. R., & Emmelkamp, P. M. G. (2008). A meta-analytic review of psychological treatments for social anxiety disorder. *International Journal of Cognitive Therapy, 1*, 94–113. doi:10.1521/ijct.2008.1.2.94

Price, M., & Anderson, P. L. (2012). Outcome expectancy as a predictor of treatment response in cognitive behavioral therapy for public speaking fears within social anxiety disorder. *Psychotherapy, 49*, 173–179. doi:10.1037/a0024734

Rapee, R. M., & Hayman, K. (1996). The effects of video feedback on the self evaluation of performance in socially anxious subjects. *Behaviour Research and Therapy, 34*, 315–322. doi:10.1016/0005-7967(96)00003-4

Rapee, R. M., & Lim, L. (1992). Discrepancy between self- and observer ratings of performance in social phobics. *Journal of Abnormal Psychology, 101*, 728–731. doi:10.1037/0021-843X.101.4.728

Rathod, S., & Kingdon, D. (2009). Cognitive behavior therapy across cultures. *Psychiatry, 8*, 370–371. doi:10.1016/j.mppsy.2009.06.011

Ressler, K. J., Rothbaum, B. O., Tannenbaum, L., Anderson, P., Graap, K., Zimand, E., . . . Davis, M. (2004). Cognitive enhancers as adjuncts to psychotherapy: Use of D-cycloserine in phobic individuals to facilitate extinction of fear. *Archives of General Psychiatry, 61*, 1136–1144. doi: 10.1176/appi.ajp.2007.07121871

Rodebaugh, T. L., Heimberg, R. G., Schultz, L. T., & Blackmore, M. (2010). The moderated effects of video feedback for social anxiety disorder. *Journal of Anxiety Disorders, 24*, 663–671. doi:10.1016/j.janxdis.2010.04.007

Rodebaugh, T. L., & Rapee, R. M. (2005). Those who think they look worst respond best: Self-observer discrepancy predicts response to video feedback following a speech task. *Cognitive Therapy and Research, 29*, 705–715. doi:10.1007/s10608-005-9634-9

Safren, S. A., Heimberg, R. G., & Juster, H. R. (1997). Clients' expectancies and their relationship to pretreatment symptomatology and outcome of cognitive-behavioral group treatment for social phobia. *Journal of Consulting and Clinical Psychology, 65*, 694–698. doi:10.1037/0022-006X.65.4.694

Scholing, A., & Emmelkamp, P. M. G. (1999). Prediction of treatment outcome in social phobia: A cross-validation. *Behaviour Research and Therapy, 37*, 659–670. doi:10.1016/S0005-7967(98)00175-2

Segal, Z., Teasdale, J., & Williams, M. (2002). *Mindfulness-based cognitive therapy for depression*. New York, NY: Guilford Press.

Stangier, U., Heidenreich, T., Peitz, M., Lauterbach, W., & Clark, D. M. (2003). Cognitive therapy for social phobia: Individual versus group treatment. *Behaviour Research and Therapy, 41*, 991–1007. doi:10.1016/S0005-7967(02)00176-6

Stangier, U., Schramm, E., Heidenreich, T., Berger, M., & Clark, D. (2011). Cognitive therapy vs interpersonal psychotherapy in social anxiety disorder: A randomized controlled trial. *Archives of General Psychiatry, 68*, 692–700. doi:10.1001/archgenpsychiatry.2011.67

Stein, M. B., Goldin, P. R., Sareen, J., Eyler Zorrilla, L. T., & Brown, G. G. (2002). Increased amygdala activation to angry and contemptuous faces in generalized social phobia. *Archives of General Psychiatry, 59*, 1027–1034. doi:10.1001/archpsyc.59.11.1027

Stopa, L., & Clark, D. M. (1993). Cognitive processes in social phobia. *Behaviour Research and Therapy, 31*, 255–267. doi:10.1016/0005-7967(93)90024-O

Taylor, C. T., & Alden, L. E. (2005). Social interpretation bias and generalized social phobia: The influence of developmental experiences. *Behaviour Research and Therapy, 43*, 759–777. doi:10.1016/j.brat.2004.06.006

Toyokawa, T., & Nedate, K. (2006). Application of cognitive behavior therapy to interpersonal problems: A case study of a Japanese female client. *Cognitive and Behavioral Practice, 3*, 289–302. doi:10.1016/S1077-7229(96)80019-8

Trower, P., Yardley, K., Bryant, B. M., & Shaw, P. (1978). The treatment of social failure: A comparison of anxiety-reduction and skills-acquisition procedures on two social problems. *Behavior Modification, 2*, 41–60. doi:10.1177/014544557821003

Turner, S. M., Beidel, D. C., & Jacob, R. G. (1994). Social phobia: A comparison of behavior therapy and atenolol. *Journal of Consulting and Clinical Psychology, 62*, 350–358. doi:10.1037/0022-006X.62.2.350

Walsh, K., & Hope, D. A. (2010). LGB-affirmative cognitive-behavioral treatment for social anxiety: A case study applying evidence-based practice principles. *Cognitive and Behavioral Practice, 17*, 56–65. doi:10.1016/j.cbpra.2009.04.007

Weiss, B. J., Singh, S., & Hope, D. A. (2011). Cognitive-behavioral therapy for immigrants with social anxiety disorder: Two case studies. *Clinical Case Studies, 10*, 324–342. doi:10.1177/1534650111420706

Wells, A., Clark, D. M., Salkovskis, P., Ludgate, J., Hackmann, A., & Gelder, M. (1995). Social phobia: The role of in-situation safety-behaviors in maintaining anxiety and negative beliefs. *Behavior Therapy, 26*, 153–161. doi:10.1016/S0005-7894(05)80088-7

Wells, A., & Papageorgiou, C. (1998). Social phobia: Effects of external attention of anxiety, negative beliefs, and perspective taking. *Behavior Therapy, 29*, 357–370. doi:10.1016/S0005-7894(98)80037-3

Westra, H. A., & Dozois, D. J. A. (2006). Preparing clients for cognitive behavioral therapy: A randomized pilot study of motivational interviewing for anxiety. *Cognitive Therapy of Research, 30*, 481–498. doi:10.1007/s10608-006-9016-y

Westra, H. A., Dozois, D. J. A., & Marcus, M. (2007). Early improvement, expectancy, and homework compliance as predictors of outcome in cognitive behavioural therapy for anxiety. *Journal of Consulting and Clinical Psychology, 75*, 363–373. doi:10.1037/0022-006X.75.3.363

Wlazlo, Z., Schroeder-Hartwig, K., Hand, I., & Kaiser, G. (1990). Exposure *in vivo* vs social skills training for social phobia: Long-term outcome and differential effects. *Behaviour Research and Therapy, 28*, 181–193. doi:10.1016/0005-7967(90)90001-Y

Woody, S. R., & Adessky, R. S. (2002). Therapeutic alliance, group cohesion, and homework compliance during cognitive-behavioral group treatment for social phobia. *Behavior Therapy, 33*, 5–27. doi:10.1016/S0005-7894(02)80003-X

23

Cognitive-Behavioral Therapy for Social Anxiety Disorder

Applying the Approach

Karen Rowa[1,2], Irena Milosevic[1], and Martin M. Antony[3]

[1]*St. Joseph's Healthcare, Canada*
[2]*McMaster University, Canada*
[3]*Ryerson University, Canada*

There are a number of empirically supported treatments for social anxiety disorder (SAD), including both pharmacological and psychological approaches. Cognitive-behavioral therapy (CBT) is the most widely used psychological treatment for SAD. In this chapter we will discuss CBT strategies for SAD and how to apply these strategies when working with clients (see **Chapter 22** for a review of empirical support for CBT).

CBT for SAD is a time-limited, collaborative therapy in which the client and therapist work together to challenge the client's biased assumptions and to change unhelpful behavioral patterns. A typical course of CBT is between 10 and 15 weekly sessions. Sessions are typically 50–60 min when offered in individual format and 120 min when offered in groups, though some individual sessions may be longer when conducting an in-session exposure exercise. A sample 12-session protocol is found in Table 23.1. There is support for CBT offered in both individual (Taylor, 1996) and group formats (Heimberg, Dodge, Hope, & Kennedy, 2000). CBT typically involves the following core strategies (each of which is covered in detail later): (1) psychoeducation, (2) cognitive strategies, and (3) exposure-based strategies; CBT may also include additional strategies such as social skills training (SST), applied relaxation, and other methods.

Assessment

Before beginning CBT for SAD, it is important to conduct a thorough assessment. This topic is covered in detail in **Chapters 13 and 14**, so we will only highlight some of the most salient issues to consider, both before treatment begins and throughout the course of therapy. For an additional review of evidence-based assessment for SAD, see McCabe, Ashbaugh, and Antony (2010).

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

Table 23.1 Sample Protocol for a 12-Session Treatment

Session	*Content*
1	• Set agenda collaboratively with client • Treatment overview (e.g., session structure and frequency, expectations about homework completion, etc.) • Psychoeducation (e.g., CBT model of SAD) • Introduction to self-monitoring • Homework assignment: Complete monitoring forms; complete self-help readings on the nature and treatment of SAD
2	• Set agenda collaboratively with client • Homework review • Psychoeducation (e.g., expanded discussion of the role of thinking in triggering and maintaining social anxiety) • Homework assignment: Monitor anxiety-provoking thinking
3	• Set agenda collaboratively with client • Homework review • Psychoeducation: Strategies for shifting anxiety-related thinking • Homework assignment: Practice strategies for changing anxiety-related thinking (e.g., behavioral experiments; thought records)
4	• Set agenda collaboratively with client • Homework review (including challenging anxiety-provoking thinking in-session) • Psychoeducation: Introduction to exposure • Define goals for exposure and develop exposure hierarchy • In-session exposures, role-plays (time permitting) • Homework assignment: Practice strategies for changing anxiety-related thinking (e.g., behavioral experiments; thought records); conduct exposure practices
5–9	• Set agenda collaboratively with client • Homework review (including challenging anxiety-provoking thinking in-session) • In-session exposures, role-plays • Homework assignment: Practice strategies for changing anxiety-related thinking (e.g., behavioral experiments; thought records); conduct exposure practices
10	• Set agenda collaboratively with client • Homework review (including challenging anxiety-provoking thinking in-session) • Psychoeducation: Introduction to SST • In-session exposures and role-plays with emphasis on behavioral rehearsal of social skills • Homework assignment: Practice strategies for changing anxiety-related thinking (e.g., behavioral experiments; thought records); conduct exposure practices incorporating social skills rehearsal
11	• Set agenda collaboratively with client • Homework review (including challenging anxiety-provoking thinking in-session) • In-session exposures and role-plays with emphasis on behavioral rehearsal of social skills • Homework assignment: Practice strategies for changing anxiety-related thinking (e.g., behavioral experiments; thought records); conduct exposure practices incorporating social skills rehearsal
12	• Set agenda collaboratively with client • Homework review (including challenging anxiety-provoking thinking in-session) • Psychoeducation: Relapse prevention

Source: Adapted from Antony and Rowa (2008) and Bieling, McCabe, and Antony (2006). Reproduced with permission from Hogrefe and Huber Publishers and Guilford Press.

It is important to assess the situational cues that trigger the client's anxious reactions. These may include places (e.g., attending class, going to the mall), people (e.g., one's boss, strangers), and activities (e.g., playing team sports, making small talk). Awareness of these cues is essential for developing an exposure hierarchy and for planning exposure practices. Understanding these cues may also provide clues regarding the beliefs and predictions underlying the client's fear(s). When assessing triggers and avoidance, it is helpful to frame the discussion in terms of the client's therapy goals. Individuals with SAD often report extensive lists of triggers, cues, and avoided situations, all of which cannot be targeted in a time-limited therapy such as CBT. Therefore, it is helpful to encourage clients to select treatment targets that are most meaningful to them and most likely to facilitate reaching their treatment goals.

Although complete avoidance of feared situations is common among people with SAD, many individuals continue to enter feared situations by relying on more subtle avoidance strategies. These strategies, often termed *safety behaviors*, are common across the anxiety disorders and are designed to protect the individual, either from possible harm or from experiencing uncomfortable feelings when in a feared situation. Some common examples in SAD include drinking alcohol before attending a social event, avoiding eye contact with others, rehearsing what one will say before talking to someone, wearing clothing or makeup to disguise physical signs of anxiety (e.g., sweating, blushing), or staying close to a *safe person* (e.g., a significant other). Although research suggests that the judicious use of safety behaviors may facilitate early fear reduction during exposure therapy (Deacon, Sy, Lickel, & Nelson, 2010; Hood, Antony, Koerner, & Monson, 2010; Milosevic & Radomsky, 2008), clinicians and researchers generally agree that these behaviors should be phased out over the course of therapy.

It is also useful to assess the extent to which the client is frightened by physical symptoms of anxiety (often referred to as *anxiety sensitivity*). Research suggests that people with SAD often have elevated anxiety sensitivity (Taylor, Koch, & McNally, 1992), reacting negatively to physical cues such as blushing, sweating, shaking, or elevated heart rate. In cases of significantly elevated anxiety sensitivity, treatment may require the addition of techniques designed to reduce fear of physical anxiety symptoms (i.e., exposure to feared sensations, also known as *interoceptive exposure*).

It is also important to assess the predictions, assumptions, and beliefs associated with the client's anxiety, including beliefs about social situations, what others might be thinking, the sensations associated with anxiety, other people's reactions, etc. As described later in this chapter, the identification and subsequent countering of anxiety-provoking thoughts and assumptions is a core component of CBT, and examination of these cognitions should continue throughout therapy. Related to assessing the content of the client's thoughts, it is also helpful to assess the environmental, social, and familial factors that may contribute to the development and maintenance of biased or erroneous assumptions and beliefs. For example, studies have found a more frequent history of teasing and bullying in SAD as compared to other anxiety presentations (McCabe, Miller, Laugesen, Antony, & Young, 2010). As a therapist, it is important to be aware of events that may have contributed to the client's fear(s), both to better understand the underlying beliefs and to ensure that strategies used to challenge assumptions are sensitive to the client's experiences.

The therapist should assess cognitions that occur before entering social situations (e.g., "no one will talk to me at the party"), while in the situation (e.g., "I don't have anything interesting to say"), and after the person leaves the situation (e.g., "I'm sure that person thought I was stupid"). Negative interpretations that occur after the situation has ended are commonly known as *postevent processing*. It is important for the therapist to assess postevent processing because many clients will successfully endure a highly stressful situation only to then go home and ruminate about their performance after the fact. This process may trigger negative social memories and may even lead the individual to misinterpret ambiguous information as threatening upon review. Indeed, research suggests that postevent processing contributes to worsening self-evaluation over time (Cody & Teachman, 2011). If therapists do not ask about postevent processing, an entire course of CBT could be corrupted by this process.

As mentioned earlier, many clients with social anxiety have intact social skills. However, there are also clients who exhibit social skills deficits. Assessing objective signs of impaired social skills can be helpful in deciding whether to incorporate strategies for improving particular social skills (e.g., public speaking skills, dating skills, etc.) during treatment.

Psychoeducation

Psychoeducation (e.g., about the nature of social anxiety, description of treatment strategies) is typically the focus of the first treatment session, and continues throughout the course of therapy, particularly as new strategies are introduced. At the first session, basic information should be provided regarding the number of sessions, length and frequency of sessions, and the expectation that clients will complete homework between sessions. Psychoeducation also includes information about a core construct of social anxiety (i.e., fear of negative evaluation) and the high frequency of shyness and SAD in the general population (to normalize the experience of social anxiety and to let clients know that they are not alone). It is useful to highlight that anxiety is both a normal and adaptive emotion. For example, a *fight-or-flight* response is essential for survival in dangerous situations (e.g., to facilitate escape when being attacked). Similarly, some anxiety before an important presentation is useful for maintaining one's focus and ensuring adequate preparation. Social anxiety, in particular, can be advantageous at mild levels. When meeting a new group of people, it is helpful to be motivated to make a good impression. Of course, anxiety is unhelpful when it is more intense or more frequent than the situation warrants. Given the benefits of mild anxiety, and the fact that everyone experiences anxiety from time to time, it is useful to help the client to identify realistic and adaptive therapy goals (e.g., to reduce and better manage feelings of anxiety), rather than goals that are unlikely to be attained (e.g., to eliminate all anxiety).

CBT protocols for SAD typically encourage individuals to think about their anxiety in terms of three separate components: (1) physical symptoms of anxiety (e.g., blushing, sweating, heart palpitations, shaky hands, etc.); (2) cognitive aspects including beliefs, assumptions, predictions, and interpretations regarding fear cues, and information processing (e.g., attention, memory) biases that help to maintain

anxiety-provoking thoughts; and (3) behavioral responses, including avoidance and the use of safety behaviors. It may be useful to use specific illustrations from a client's experience to help the client identify examples of each of these three components. The client's examples can be used to illustrate the way that the components interact with each other to fuel increasing levels of anxiety. For example, noticing an increase in body temperature might cause a person to scan his body for further signs of anxiety. Noticing some sweating might make the person start to worry about how noticeable this is to others, which, in turn, might contribute to more sweating. This cycle, though problematic when a person is actively symptomatic, can be disrupted and changed through the use of CBT strategies. CBT will include strategies to challenge thoughts (cognitive strategies), to challenge behaviors (exposures and potentially skills training), and to challenge the physical aspects of anxiety (e.g., applied relaxation). By working on one component of anxiety, the benefits will be indirectly felt for the other components.

Cognitive Strategies

Cognitive strategies teach people to notice their anxiety-provoking assumptions and predications, and then to question whether these assumptions are valid, truthful, and helpful. Anxiety-provoking thoughts and assumptions experienced in SAD can often be categorized as one of the two main types: probability overestimations and catastrophic thinking. Probability overestimation involves exaggerating the likelihood of some negative event or consequence occurring (e.g., people will think I am stupid if I make a mistake; I will not have anything to say at my meeting; everyone finds me boring). Catastrophic thinking involves overestimating the impact of an event or consequence, or underestimating one's ability to cope with an event or consequence if it were to occur (e.g., it would be a disaster if the audience thought my presentation was less than perfect; it would be terrible if I were to ask someone out on a date and get rejected; I cannot cope with others judging me negatively). Clients with SAD may also hold a variety of unhelpful assumptions about social situations (e.g., it is important for everyone to like me; I must never make mistakes), and they also pay more attention to information that supports their anxiety-provoking beliefs than they do to disconfirming information. For example, they may take negative feedback very seriously, while ignoring compliments or positive feedback.

Challenging Anxiety-Provoking Thoughts

In cognitive therapy (Beck, Emery, & Greenberg, 1985), clients are encouraged to take a step back and observe their anxiety-provoking thoughts from a distance—to treat their thoughts as possibilities rather than as hard truths. The therapist helps the client to view feared situations from multiple perspectives, to examine evidence for whether assumptions are true, and to develop a greater tolerance for handling any outcomes that are less than desirable. One strategy for challenging anxiety-provoking thoughts is to use a *thought record*, on which clients record their assumptions and

predictions, and then write down information that counters or challenges their initial assumptions. Some examples of questions used to challenge anxiety-laden thoughts include:

- Are there any other ways of thinking about this situation?
- Are there any other ways this could turn out?
- Is this really as important as it feels?
- Even if my fears are true, how important will this be in a month? A year?
- If I actually do something embarrassing, how long will my embarrassment last? How long will other people think about me?
- How might someone without social anxiety think about this situation?
- What are all the possible things that other person might be thinking?

Once other perspectives have been considered, the person is asked to consider a more balanced way of viewing the situation. If the thought record reveals that the client was accurate in his or her negative assumptions (e.g., the boss really was displeased with the client's presentation), the therapist can work with the client on reducing the impact of what this negative feedback actually means (does that mean you are a terrible employee, or does this mean that you have some areas needing attention) as well as possibly engaging in problem solving or skills building (e.g., work on presentation skills). A sample thought record is found in Table 23.2.

Table 23.2 Sample Thought Record

Situation	*Initial anxiety (0–100)*	*Fearful thoughts and predictions*	*Rational responses and countering*	*Anxiety now (0–100)*
Arriving at a work meeting a few minutes late	85	Everyone will stare at me They will think I do not care about the meeting They will see how nervous I am	Everyone is late to a meeting at some point I have been late before and it is embarrassing, but not horrible They will probably be more focused on what is being discussed in the meeting than my late arrival Even if they notice me when I come in, they will only pay attention to me for a few seconds	60

Table 23.3 Behavioral Experiments

Fear/prediction	*Experiment*
Other people will stare and laugh if I do something embarrassing in public	Purposely drop something in public, make a loud noise, spill a glass of water
If I make a mistake at work, I will be fired	Purposely include a typo in an email or letter
I have nothing to talk about with others and everyone else has lots of interesting things to say	Eavesdrop on people talking in a public place and find out what they are talking about
I need to look my best or no one will want to talk to me	Go out without makeup or in sweat pants
It would be a disaster to lose my train of thought during a presentation	Purposely lose my train of thought for a few seconds while giving a presentation
People will stare at me if I go out	Look around when out and find out how many people are staring at you versus how many are attending to other things

Behavioral Experiments

Another important strategy for challenging anxiety-provoking thoughts is the use of behavioral experiments. See Butler and Hackmann (2004) for a more extensive discussion of behavioral experiments in SAD. Experiments allow an individual to collect information that may disconfirm negative predictions. Good behavioral experiments are under the person's control and are designed to test the validity of the client's specific anxiety-provoking prediction versus one or more alternative predictions. For example, a client who believes that attracting attention in public (e.g., by dropping keys) will lead to judgment by others might purposely drop their keys. Before carrying out the experiment, the client would be encouraged to make specific predictions (e.g., several people will laugh at me or tease me) and to generate alternative predictions (nobody will notice; perhaps one or two people will look in my direction, but they will not laugh or tease me). Next, the client would carry out the experiment and evaluate the outcome. This is a good experiment because (1) it is under the client's control (the client drops the keys) and (2) the client's predictions are specific and testable. A poorly designed behavioral experiment might include an activity that is out of a person's control (e.g., waiting for someone to talk to me) and predictions that cannot be tested (e.g., predicting that others are thinking bad things about me). Experiments may only need to occur once to gather the required information, or they may need to be repeated with different people or in different contexts to have an impact. Examples of possible experiments for SAD are found in Table 23.3.

Collecting New Data to Facilitate Cognitive Change

Social behavior and internal experiences vary widely from person to person, making it hard to know what is "normal" or common in the general population. For example, what do people enjoy talking about at a party? What do most people think if they

see someone do something embarrassing? How long do people talk about someone they have seen make a mistake? What are all the possible explanations that people have for physical symptoms of anxiety? Having the client survey a number of people can provide him/her with information about the range of responses that people may have, or can provide information that disconfirms strongly held ideas. Sometimes citing research results or providing education about social behavior is also helpful in generating alternative explanations for a person's assumptions. For example, research suggests that people with and without social anxiety have different explanations for noticeable symptoms of anxiety in others, with participants with social anxiety most likely attributing physical sensations (e.g., shaking hands) to anxiety, while participants without anxiety made more benign or medical attributions (Roth, Antony, & Swinson, 2001).

Core Beliefs

Cognitive theories suggest that cognitive distortions are fuelled by strongly held core beliefs. In CBT for SAD, it is sometimes necessary to work on core beliefs about oneself and others. Commonly held core beliefs about oneself include:

- I am worthless
- I am unlovable/unlikeable
- I am incapable
- I am boring
- I am a failure

Commonly held core beliefs about others include:

- Others are critical of me
- Others are mean
- Others will take advantage of me
- Others expect me to be perfect

Some clients can readily identify core beliefs, and examples of these may arise in monitoring forms or thought records. For other clients, the therapist may help to uncover negative core beliefs by noticing common themes across therapy sessions or by asking the client to consider the meaning of his or her anxiety-provoking thoughts. This strategy is often called the *downward arrow technique* and involves having the client articulate anxiety-provoking thoughts, then asking "if that was true, what would that mean about you/others" repeatedly until the client arrives at a relevant core belief.

Strategies to challenge core beliefs are similar to those used with other types of negative thinking (Greenberger & Padesky, 1995). These strategies include collecting evidence to challenge negative core beliefs, developing alternative core beliefs (e.g., "I am worthwhile"), using lifespan data from the client's life to challenge beliefs, and conducting experiments to challenge the validity of negative core beliefs. For example, if a client believes that he or she is completely incapable, a first assignment might be

to look for small daily examples of capability (e.g., getting dishes done, returning a call about an appointment, problem solving a minor repair). The client might then go on to identify an alternative core belief that feels more balanced and hopeful (e.g., "I am capable") and develop that through evidence gathering and behavioral tasks.

Exposure-Based Strategies

Exposure to feared situations is a highly effective method of fear reduction and a key behavioral component of CBT for SAD. There are a number of mechanisms by which exposure is thought to exert its effects (Moscovitch, Antony, & Swinson, 2009). According to the cognitive-behavioral model, one main mechanism is the acquisition of corrective information that contradicts the client's threat-related beliefs. For example, an individual who is fearful of running out of things to say during a casual conversation will likely learn that (1) the conversation continues for some time before running out of things to speak about; and (2) the consequences of running out of things to say are more manageable than anticipated, when it does happen.

Providing a Rationale for Exposure

Prior to initiating exposure, therapists should provide a sound rationale for this intervention, as most clients are likely to respond with anxiety when faced with the premise of facing their fears. It is helpful to ask clients about fears they might have had in childhood and have since overcome (e.g., fear of the dark, animals) to facilitate discussion of how repeated exposure helps us overcome fears. If a client is unable to volunteer any relevant examples for this exercise, the therapist can ask about how he or she might approach helping someone else overcome a specific fear and, again, highlight the role of exposure. Therapists should further inform clients that repeated exposure has the effect of reducing anxiety in the long term by way of:

- learning that feared consequences will generally not be realized and, in rare cases when negative outcomes do occur, they are manageable;
- increased comfort with physical sensations experienced in social situations;
- increased confidence about being in social situations and about tolerating anxiety; and
- increased awareness of surroundings in social situations (e.g., being able to focus on what someone else is saying during a conversation), as focus shifts away from symptoms of anxiety.

In addition, clients should be provided with a rationale for the need to reduce common maladaptive coping strategies during exposure practices, including avoidance, escape, and safety behaviors. These strategies hinder one's ability to learn that the situation is not as bad as anticipated, and they may promote the false belief that their use is essential to preventing feared outcomes (e.g., "She was only interested in me because I had a few beers, so I was able to be funny").

Table 23.4 Sample Hierarchy for Fear of Participating in Groups

Situation	*Fear*
11. Lead a book club discussion	100
10. Express a difference of opinion during a book club discussion	95
9. Join a book club in the community and present an idea during discussion of a book	90
8. Express a difference of opinion at a staff meeting at work	85
7. Present an idea or initiate a discussion at a staff meeting at work	80
6. Ask questions at a staff meeting at work	70
5. Express difference of opinion at a social gathering with a mix of friends, acquaintances, and unfamiliar individuals	65
4. Contribute a story at a social gathering with a mix of friends/ acquaintances and unfamiliar individuals	60
3. Ask questions or make comments at a social gathering with a mix of friends, acquaintances, and unfamiliar individuals	50
2. Express a difference of opinion at a social gathering with friends and acquaintances	40
1. Contribute a story at a social gathering with friends and acquaintances	35

Developing an Exposure Hierarchy

An exposure hierarchy is a rank-ordered list of situations commonly feared and avoided by the client, with the least threatening situation placed at the bottom of the list and the most threatening one at the top. The order of items is determined by the client's fear rating for each situation, typically using a scale ranging from 0 to 100. Clients may also provide separate avoidance ratings; however, these may not significantly contribute additional information given the high degree of association between fear and avoidance.

Hierarchies are commonly comprised of 10–15 steps. Hierarchy items should be generated collaboratively by the therapist and client and should reflect situations that are consistent with the client's goal(s) for treatment (e.g., to be more comfortable speaking in group settings). The description of the situations should be as specific as possible. For example, "Joining my co-workers for lunch in the cafeteria twice per week," is more appropriate than "Spending more time around people." Hierarchies should include items ranging in difficulty (including a number of moderately difficult items), and they should include mostly items that would be practical to arrange for possible exposure practices (e.g., "getting married" is not a good hierarchy item for an individual who is not currently in a relationship). A sample exposure hierarchy is provided in Table 23.4.

Guidelines for Exposure

Exposure hierarchies are used to facilitate exposure practices. Clients should begin with an item on the hierarchy that is challenging but not overwhelming. Often this

Table 23.5 Principles of Effective Exposure

Principle	*Description*
Planning	• The details of exposure practices should be planned in advance • Exposures should be scheduled for specific days and times • Possible obstacles to between-session exposures should be anticipated and resolved in advance (e.g., if social plans are cancelled, what else can the client do for exposure?)
Frequency	• Exposure practices are most effective when scheduled close together • Major exposure practices (e.g., drawing attention to oneself in public) should be conducted at least three to four times per week • More minor exposures (e.g., making eye contact with a passer-by) should be conducted as often as possible
Duration	• Longer exposures are more effective than shorter ones • Duration of exposure should be determined by one or both of the following: – Exposure may end once the client learns that feared outcomes will not occur – Exposure may end when anxiety has decreased to a manageable level (e.g., 50% of peak anxiety)
Attentional focus	• Clients should be encouraged to attend to all aspects of the situation, not just the most threatening ones (e.g., pay attention to all faces in the room rather than just individuals who appear bored during presentation)
Safety behavior	• Clients should be encouraged to gradually reduce their use of safety behaviors during exposure
Generalizability	• Whenever possible, exposures should be practiced across a variety of situations to maximize the generalizability of learning and anxiety reduction

will be a moderately difficult item (e.g., fear rating of around 40 or 50), though some clients may be willing to practice more difficult situations early in treatment. Often, people with SAD report that they have already been exposed to many of the situations on their hierarchies in their regular life, and that "exposure" has been ineffective in reducing their anxiety. In these cases, it is important to help the client to recognize the differences between their previous exposures and the therapeutic exposures that occur during treatment (Table 23.5).

Prior to initiating an exposure, clients should apply the cognitive countering skills already learned in treatment. Following exposure, the client should evaluate the outcomes of the practice in light of these predictions, continuing to challenge any negative assumptions (i.e., postevent processing). Success should be defined by completion of exposure to feared situations despite feeling anxious, rather than by the outcome of the situation.

Types of Exposure

Situational exposure. Situational or *in vivo* exposures involve being exposed to actual feared situations, including both interpersonal (e.g., dating, speaking on the phone, initiating conversations) and performance-related (e.g., giving a presentation, being interviewed) situations. One of the main challenges here is limited controllability and predictability inherent in social situations, and clients must therefore be prepared to manage these aspects of situational exposures. It is helpful to design hierarchies that include a variety of situations tapping into a common fear, so that a client has ample opportunity for exposure even if one situation does not work out (e.g., event is cancelled).

Simulated exposure. When situational exposures are not easily arranged or when clients prefer to begin exposure practices in a less threatening environment, simulated exposures or role-plays are a useful option. A further advantage of simulated exposures is their high degree of controllability. For example, their intensity or level of difficulty can be varied (e.g., more or less critical audience during a presentation, more or less engaged conversational partner). Role-plays require participation from others, which can include the therapist, therapist's colleagues, friends, family members, colleagues, and—in the case of group treatment—other clients. For role-plays in the context of group treatment, it is helpful to pair clients who do not have identical fears, so that the role-play partner's own anxiety does not influence his or her ability to participate effectively in the practice.

Interoceptive exposure. As reviewed earlier in the chapter, many individuals with SAD have concerns about the visibility of their anxiety-related physical symptoms while in the company of others. *Interoceptive* or *symptom exposure* involves exposure to feared anxiety-related physical symptoms and is a standard intervention for individuals with panic disorder (Craske & Barlow, 2006). This exposure method has been less studied in individuals with SAD, although it is commonly applied in cases where clients are fearful of experiencing particular symptoms in social situations. Interoceptive exposure practices should occur while in feared situations, and some examples include wearing heavy clothing to induce perspiration, purposely shaking one's hands in the presence of others (to simulate trembling), eating spicy foods to induce blushing, and exercising (e.g., running up stairs) to induce a pounding heart before entering a meeting.

Additional Strategies to Augment Treatment

Social Skills Training

Social skills training is comprised of a group of techniques that target behavioral deficits (e.g., poor eye contact) or excesses (e.g., excessive pauses during a presentation) that are believed to impact negatively upon performance in social situations. See **Chapter 17** for expanded discussion of this issue.

There is variability across CBT protocols for SAD regarding the inclusion of SST (Bieling et al., 2006; Heimberg & Becker, 2002), and evidence is mixed regarding

whether SST improves outcomes when included in CBT (for a review, see Antony & Rowa, 2008). While SST is unlikely to be a critical component of treatment for those who do not have deficits in skills, it may nevertheless promote therapeutic change for these individuals, as it includes elements of exposure (e.g., practicing skills in feared situations) and corrective feedback. There is greater consensus on its inclusion as a treatment adjunct for clients who demonstrate problems with effective communication.

Video Feedback

Video feedback (VF) is based on cognitive models of SAD, which stress the role of distorted self-perceptions and negative self-processing during social performance situations (Clark & Wells, 1995; Rapee & Heimberg, 1997). VF involves video recording socially anxious individuals while they are completing a social task (e.g., public speaking, engaging in conversation) and providing them with playback of their performance. It is anticipated that review of the recording corrects distorted self-evaluations, including underestimations of social skills.

VF is commonly preceded by *cognitive preparation* (CP), which has been shown to enhance its effectiveness in experimental studies (Kim, Lundh, & Harvey, 2002). Prior to VF, clients should be asked to (1) provide a detailed prediction of what they will observe in the video, (2) generate a vivid mental image of their performance in the situation, and (3) review the video objectively with emphasis on their performance rather than on their feelings.

Following VF, therapists should work with clients to identify any discrepancies between what was predicted and what was observed in the video, with emphasis on evidence that counters initial predictions of poor performance. This *cognitive review* (CR) phase should also highlight the difference between *feeling* anxious and *appearing* anxious, as socially anxious individuals tend to erroneously conflate the two. Adding CR to CP + VF has been shown to improve self-perceptions and expectations for future performances in socially anxious individuals (Orr & Moscovitch, 2010).

Clients may sometimes endorse dissatisfaction with aspects of their performance following VF (e.g., after observing oneself persistently averting eye contact, mumbling, shaking). Therapists should determine the extent to which this dissatisfaction is the result of a distortion in the client's processing of the video versus a more observable behavior. In the case of the former, cognitive strategies, as described earlier, are appropriate. The latter scenario may warrant exploration of whether any of their behavior serves as a safety behavior (e.g., mumbling to avoid a quavering voice), which should be gradually faded with additional exposures. When a client's poor performance is a function of a social skills deficit, SST is warranted.

Clinicians who do not have access to video equipment in their practice should note that many clients' mobile devices are likely to have video-recording capabilities. Audio feedback may also be a useful alternative and has been shown to be an effective cognitive intervention for negative self-evaluations (Nilsson, Lundhb, Faghihic, & Roth-Anderssond, 2011).

Applied Relaxation

Relaxation techniques target the physiological symptoms of social anxiety. In the context of CBT, applied relaxation is taught to clients to help them cope with autonomic arousal during exposures practices. Progressive muscle relaxation (systematic tensing and relaxing of different muscle groups), release-muscle relaxation (tension component is omitted), and cue-controlled relaxation (breathing-focused relaxation conditioned to a specific cue, such as the command to "relax") are among a number of techniques that clients may practice to be able to relax quickly in response to early signs of anxiety (Öst, 1987). Relaxation-based strategies for SAD have not been well studied and little is known regarding their utility in CBT, particularly whether they provide added benefit when combined with exposure. Research on other anxiety disorders suggests that relaxation training does not significantly improve the effects of exposure (Antony & Swinson, 2000).

Adaptations of Treatment

Group Versus Individual Format

Both individual and group formats of CBT for SAD have demonstrated efficacy, and current evidence does not conclusively support the superiority of one modality over the other (Powers, Sigmarsson, & Emmelkamp, 2008). The treatment formats cover the same therapeutic interventions (detailed descriptions of group treatment for SAD are available elsewhere; Bieling et al., 2006; Heimberg & Becker, 2002). However, inherent to group treatment is the opportunity for in-session exposure practices that involve other group members, whereas similar exposures (e.g., presenting a talk in front of a group) need to be arranged elsewhere in individual treatment. Both practical (e.g., availability of either form of treatment in a given setting, scheduling) and clinical (e.g., symptom severity, comorbidity, personality psychopathology) considerations should determine whether a client participates in group versus individual CBT.

Client preference is also an important factor when deciding on the treatment format. It is common for individuals with SAD to feel apprehensive about participating in a group treatment, and therapists should explore these concerns with them, stressing that all group participants will be anxious at the onset of treatment and that this anxiety is expected to decline.

Working with Diverse Populations

Children and adolescents. The CBT strategies described in this chapter can be applied to younger populations, but they may need to be adapted to be age appropriate. For example, "fear stepladders" and "fear thermometers" are common adaptations of exposure hierarchies and fear ratings, respectively, when working with children. Having young clients track their progress visually on a large chart or poster and providing rewards following the completion of exposure practices may help to reinforce their ongoing participation in exposure.

Including parents in treatment is an important consideration, as their participation can ensure the child's engagement in therapy and facilitate progress. Parents can be taught to reduce any of their own behaviors that might inadvertently reinforce their child's anxiety and avoidance (e.g., speaking on behalf of the child). Additionally, parents play an important role in facilitating adherence to homework assignments and ongoing practice of newly acquired skills, modeling nonfearful behavior, and helping the child to challenge threatening beliefs.

A number of excellent clinical resources offer more in-depth information on working with children and adolescents with anxiety disorders (Chorpita, 2007) and, specifically, with SAD (Albano & DiBartolo, 2007; Kearney, 2005).

Older adults. SAD continues to be a significant problem into late adulthood. The application of CBT to older adults warrants several considerations (for an in-depth discussion on this topic, see Cully & Stanley, 2008; Gallagher-Thompson, Steffen, & Thompson, 2008). First, medical comorbidities are common among older adults and have the potential to complicate treatment, as the physiological symptoms of anxiety parallel the symptoms of a number of medical illnesses (e.g., heart disease, lung disease). Clients should thus undergo a thorough medical examination before beginning CBT.

Greater rates of impairment in cognitive functioning in older adults may present challenges in terms of recall of session content and completion of homework assignments. Literature on CBT for generalized anxiety disorder in older adults suggests that homework compliance and treatment outcome can be enhanced with specific methods designed to circumvent problems with memory (e.g., frequent review of material presented in treatment, between-session phone calls; Mohlman et al., 2003). Simplification of the treatment rationale and therapeutic strategies may additionally be necessary for older clients struggling with cognitive impairment (Cully & Stanley, 2008).

Additional considerations include interpersonal losses (e.g., death of loved ones) and changes in one's social environment (e.g., move to retirement home or assisted living facility) that often accompany aging. Such transitions may amplify problems with social anxiety. The treatment approach for these concerns emphasizes the development of skills that will help clients to expand their social circles.

Adapting treatment across cultures. While SAD is evident across cultures, its lifetime prevalence rates vary widely across different groups (Hofmann, Asnaani, & Hinton, 2010). In terms of its presentation, SAD holds many similarities cross-culturally; however, there is also evidence of culturally specific expressions of the disorder, with commonly cited distinctions between Eastern and Western cultures (Stein, 2009). Most notably, the *offensive subtype* of *taijin kyofusho* (TKS), an intense fear of offending or embarrassing others through one's behavior or appearance, is prevalent in Japanese and Korean cultures, although features of this disorder have also been observed in North American patients with SAD (Choy, Schneier, Heimberg, Oh, & Liebowitz, 2008).

Current evidence does not support significant differences in response to psychological and pharmacological treatment of SAD as a function of culture (Hofmann et al., 2010). However, clinicians should be aware that the expression of symptoms related to social anxiety is influenced by cultural norms. For example, eye contact has widely

varying culturally ascribed meanings. In some cultures, avoidance of eye contact represents a normative sign of respect, whereas, in others, sustained eye contact signals respect. Culture also influences the degree of socially acceptable personal space, the preference for directiveness in treatment, and the level of acceptable assertiveness in one's social and family circles. Therapists must be sensitive to these variations and conduct ongoing assessment of the cultural influence on clients' beliefs and behaviors. It is equally imperative that clinicians do not over-attribute clients' presentations to cultural factors or make assumptions about clients based on cultural stereotypes. See **Chapter 11** for a detailed review of multicultural and diversity issues in SAD.

Presence of Comorbidity

Individuals with SAD often suffer from additional mental health problems, most commonly anxiety, mood, and substance use disorders on Axis I (Brown, Campbell, Lehman, Grisham, & Mancill, 2001; see also **Chapter 10**) and avoidant personality disorder (APD) on Axis II (Grant et al., 2005). However, there is scant research to inform the best approach for managing SAD in the context of comorbidity. Current evidence suggests that comorbidity with other anxiety disorders is unlikely to affect treatment outcome for SAD. On the other hand, comorbidity with major depression may have more significant implications for outcome, although this evidence is mixed (for a review, see Magee, Erwin, & Heimberg, 2009).

When comorbidity exists, therapy should be prioritized for the most significant problem, as defined both by the level of distress and impairment that it is causing and by the client's preference. Alternatively, the mood and anxiety disorders may be treated concurrently with a transdiagnostic approach (Norton, 2009), which rests on the observation of greater commonalities than differences across disorders and on the similarities between treatment strategies for individual disorders.

The use of substances to reduce anxiety symptoms or to manage skills deficits in social situations is common among individuals with SAD (Buckner, Eggleston, & Schmidt, 2006) and can complicate treatment. See **Chapter 25** for a detailed review of treatment implications for SAD patients with dual diagnoses, as well as discussion on the use of motivational enhancement therapy (MET; Miller, Zweben, DiClemente, & Rychtarik, 1992) for clients with ambivalence about reducing substance use.

Combining CBT with Medications

As the use of pharmacotherapy for SAD is reviewed elsewhere in this volume (see **Chapter 24**), the purpose of this section is to highlight issues relevant to the application of CBT in cases where clients are also taking psychotropic medications. In general, clinicians can expect many of their clients to be taking medication (for SAD or other disorders) while they are completing treatment. This raises several important considerations. First, for a variety of reasons, clients might need to adjust or switch medication during the course of CBT. However, it is helpful, when possible, for them to remain on a steady dosage of medication in order to reduce any interference of withdrawal or side effects with their engagement in CBT. Additionally, a change in

medication during the course of treatment may increase the likelihood that a client will over-attribute symptom improvements to medication.

Therapists will commonly see patients who are taking benzodiazepines to manage anxiety. This class of medications, which facilitates rapid short-term anxiety relief, has the potential to interfere with the effects of CBT (Otto, Smits, & Reese, 2005). Proposed mechanisms for this interference include a reduction in self-efficacy about one's ability to tolerate anxiety, increased attention to threat cues, and interference with memory and learning (Westra & Stewart, 1998). As needed or "prn" benzodiazepine use may serve as a safety behavior during exposure to anxiety-provoking situations. Therapists should assess how clients are using benzodiazepines, particularly in terms of exposure practices. They should also note that, should clients choose to reduce or discontinue their use of these medications, withdrawal symptoms often mimic those of anxiety. Overall, any changes in medication should be made only under medical supervision. A CBT protocol for benzodiazepine discontinuation is available (Otto & Pollack, 2009).

Relapse Prevention

Relapse prevention is the final component of CBT, with the goal of reducing the likelihood that the client's social anxiety (as well as the maladaptive thoughts and behaviors that maintain it) will return. At this point in treatment, clients should understand that one of the main goals of CBT is to foster the independent practice of skills after therapy has ended. This is particularly important, as many individuals with social anxiety continue to experience residual symptoms following the completion of treatment.

Therapists should provide education about the difference between a lapse and a relapse, with the former being a temporary increase in symptoms due to an acute stressor (i.e., a small slip or setback), whereas the latter is a sustained increase in symptoms. Lapses are not uncommon—indeed, they should be expected—and can be triggered by both positive (e.g., new baby, job promotion) and negative (e.g., loss of employment, physical illness, termination of relationship) stressors. A change in medications can also elicit a temporary increase in symptoms, as can termination of treatment.

A critical component in relapse prevention is how one responds to a lapse when it occurs. Clients should reflect on the types of stressors that may have previously exacerbated their social anxiety and develop a plan for managing similar stressors in the future. For many clients, maintaining a stable, healthy lifestyle that includes a balanced diet, physical activity, regular sleep, and limited use of substances may contribute positively to symptom management and relapse prevention. Clients should also be vigilant for declines in their practice of cognitive-behavioral skills as this, too, may increase risk of a lapse or relapse.

Being attuned to the first signs of an exacerbation in social anxiety will facilitate early intervention. It is helpful for clients to prepare a list of "red flags" or changes in symptoms that may signal a need for further action. Examples include an increase in negative thinking, changes in physical symptoms, and increase in avoidance of social

activities. Clients may wish to schedule periodic review of this list to "check-in" with how they are doing. Strategies to manage recurrence of symptoms may include any of those learned during the course of CBT. It might be helpful for clients to develop a specific action plan based on treatment elements that they found most effective.

Booster sessions are an additional relapse prevention strategy. These sessions serve to help clients review and reinforce ongoing practice of skills and to address any exacerbations in symptoms. Depending on a client's symptom severity at the end of treatment, sessions may be scheduled more (e.g., once per month) or less (e.g., every 6 months, annually, or as needed) frequently. Where resources permit, monthly group-based booster sessions, which treatment completers can attend on an as-needed basis, are an excellent way to facilitate review of cognitive-behavioral strategies and discussion of common "road-blocks."

Emerging CBT Treatments and Future Directions

In addition to the core components of CBT for SAD, there are several emerging treatments that may prove to be useful components of a CBT treatment program. These strategies are not commonly used in clinical settings at present, and further research is needed to support their inclusion; however, they do warrant continued research attention.

Virtual Reality Exposure Therapy

During the past decade, virtual reality exposure therapy (VRET) has become an increasingly popular treatment modality for anxiety disorders. VRET uses interactive computer-generated environments to simulate clients' feared situations. The principles of VRET parallel those of *in vivo* exposure treatment, with clients gradually and repeatedly being exposed to fear-provoking situations until their fear decreases. VRET has been provided both as a stand-alone treatment and as a component of broader behavioral or cognitive-behavioral interventions. In the case of SAD, it has most commonly been applied to fear of public speaking, and evidence from recent randomized controlled trials supports its effectiveness (Robillard, Bouchard, Dumoulin, Guitard, & Klinger, 2010; Wallach, Safir, & Bar-Zvi, 2009).

There are several advantages of using virtual environments for exposure practices, including full controllability of the frequency, pace, and intensity of exposures. The addition of VRET to individual CBT also provides convenient access to exposure situations that might otherwise be challenging to facilitate outside of group-based treatment, and it protects clients from possible loss of confidentiality during public exposures. Furthermore, VRET might appeal to clients who are too fearful to participate in real-life exposures.

A key drawback of VRET is the prohibitive cost to therapists and institutions of acquiring VR equipment and software. Additionally, whereas the high degree of controllability of the virtual environment has many benefits, the lower controllability of *in vivo* exposures can provide rich therapeutic material to help clients work toward tolerating unpredictable outcomes.

Cognitive Bias Training

Given the emphasis on biased interpretations in cognitive models of SAD, there have been several programs developed that attempt to directly modify cognitive biases. For example, *attention training* (ATT) aims to correct biased attentional processing commonly exhibited by individuals with SAD. Such biases are characterized by one's disproportionate allocation of attention to external (e.g., frowning faces) or internal (e.g., blushing) threat-related cues, as well as difficulty with disengaging from such cues. There are several ATT approaches, all of which train clients to shift their attention away from threatening stimuli. For example, Wells, White, and Carter (1997) have used auditory stimuli to reduce clients' self-focused attention by guiding them through phases of selective attention (focusing on a specific sound), attention switching (shifting attention from initial sound to a new one), and divided attention (simultaneously focusing on as many sounds as possible). A more recent ATT approach involves the application of computer-based dot-probe discrimination tasks that teach clients to attend to neutral visual stimuli and to disengage from anxiety-provoking stimuli (Schmidt, Richey, Buckner, & Timpano, 2009). ATT exercises are typically brief (e.g., 15 min) and are intended to be practiced regularly to facilitate improvement in attentional flexibility.

Interpretation training targets the tendency of people with SAD to make threatening interpretations of ambiguous scenarios. This procedure involves participants completing a computerized task in which they are asked to read an ambiguous sentence (e.g., "People laugh after something you said") and are reinforced for making nonthreatening interpretations of these sentences. Interpretation training has been found to be more effective than a control condition in reducing threat interpretations and reducing clinician-rated symptoms of SAD (Amir & Taylor, 2012).

Imagery Rescripting

For clients with SAD who have had unpleasant or traumatic experiences during childhood or adolescence, a recently developed technique, imagery rescripting, helps clients update their negative memories. Clients are asked to use cognitive countering skills in reference to a traumatic memory and then relive the memory from an adult perspective. Finally, the client is asked to relive the memory from the perspective of how old they actually were when it happened (e.g., 10 years old), and the adult self can intervene to aid the younger self. Imagery rescripting has received support for its effectiveness in reducing distress from socially traumatic memories and concerns about negative evaluation (Wild & Clark, 2011).

Conclusion

CBT for SAD consists of a group of interventions that are geared toward modifying maladaptive patterns of thoughts and behaviors that maintain anxiety. Core components include psychoeducation, cognitive strategies and behavioral strategies, and

direct targeting of anxiety-provoking thoughts and maladaptive coping strategies. Through modification of thoughts and behaviors, it is expected that clients will also experience a decrease in the physical symptoms of anxiety and enjoy an enhanced quality of life. While the core components of CBT are effective for achieving these goals, ongoing research into the psychopathology of SAD promises to provide ideas for new CBT strategies that will complement existing protocols.

References

Albano, A., & DiBartolo, P. M. (2007). *Cognitive-behavioral therapy for social phobia in adolescents: Stand up, speak out*. New York, NY: Oxford University Press.

Amir, N., & Taylor, C. T. (2012). Interpretation training in individuals with generalized social anxiety disorder: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 80*, 497–511. doi:10.1037/a0026928

Antony, M. M., & Rowa, K. (2008). *Social anxiety disorder: Psychological approaches to assessment and treatment*. Göttingen, Germany: Hogrefe.

Antony, M. M., & Swinson, R. P. (2000). *Phobic disorders and panic in adults: A guide to assessment and treatment*. Washington, DC: American Psychological Association.

Beck, A. T., Emery, G., & Greenberg, R. L. (1985). *Anxiety disorders and phobias: A cognitive perspective*. New York, NY: Basic Books.

Bieling, P. J., McCabe, R. E., & Antony, M. M. (2006). *Cognitive behavioral therapy in groups*. New York, NY: Guilford Press.

Brown, T. A., Campbell, L. A., Lehman, C. L., Grisham, J. R., & Mancill, R. B. (2001). Current and lifetime comorbidity of the DSM-IV anxiety and mood disorders in a large clinical sample. *Journal of Abnormal Psychology, 110*, 585–599. doi:10.1037//0021-843X.110.4.585

Buckner, J. D., Eggleston, A. M., & Schmidt, N. B. (2006). Social anxiety and problematic alcohol consumption: The mediating role of drinking motives and situations. *Behavior Therapy, 37*, 381–391. doi:10.1016/j.beth.2006.02.007

Butler, G., & Hackmann, A. (2004). Social anxiety. In J. Bennett-Levy, G. Butler, M. Fennell, A. Hackmann, M. Mueller, & D. Westbrook (Eds.), *Oxford guide to behavioural experiments in cognitive therapy* (pp. 141–158). New York, NY: Oxford University Press.

Chorpita, B. F. (2007). *Modular cognitive-behavioral therapy for childhood anxiety disorders*. New York, NY: Guilford Press.

Choy, Y., Schneier, F. R., Heimberg, R. G., Oh, K. S., & Liebowitz, M. R. (2008). Features of the offensive subtype of Taijin-Kyofu-Sho in US and Korean patients with DSM-IV social anxiety disorder. *Depression and Anxiety, 25*, 230–240. doi:10.1002/da.20295

Clark, D. M., & Wells, A. (1995). A cognitive model of social phobia. In R. G. Heimbert, M. R. Liebowitz, D. A. Hope, & F. R. Schneier (Eds.), *Social phobia: Diagnosis, assessment, and treatment* (pp. 69–93). New York, NY: Guilford Press.

Cody, M. W., & Teachman, B. A. (2011). Global and local evaluations of public speaking performance in social anxiety. *Behavior Therapy, 42*, 601–611. doi:10.1016/j.beth.2011.01.004

Craske, M. G., & Barlow, D. H. (2006). *Mastery of your anxiety and panic: Therapist guide* (4th ed.). New York, NY: Oxford University Press.

Cully, J. A., & Stanley, M. A. (2008). Assessment and treatment of anxiety in later life. In K. Laidlaw & B. Knight (Eds.), *Handbook of emotional disorders in later life: Assessment and treatment* (pp. 233–256). Oxford, UK: Oxford University Press.

Deacon, B. J., Sy, J. T., Lickel, J. J., & Nelson, E. A. (2010). Does the judicious use of safety behaviors improve the efficacy and acceptability of exposure therapy for claustrophobic fear? *Journal of Behavior Therapy and Experimental Psychiatry, 41*, 71–80. doi:10.1016/jbtep.2009.10.004

Gallagher-Thompson, D., Steffen, A., & Thompson, L. W. (Eds.). (2008). *Handbook of behavioral and cognitive therapies with older adults.* New York, NY: Springer.

Grant, B. F., Hasin, D. S., Stinson, F. S., Dawson, D. A., Chou, S. P., Ruan, W. J., & Huang, B. (2005). Co-occurrence of 12-month mood and anxiety disorders and personality disorders in the US: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Journal of Psychiatric Research, 39*, 1–9. doi:10.1016/j.jpsychires.2004.05.004

Greenberger, D., & Padesky, C. A. (1995). *Mind over mood: Change how you feel by changing the way you think.* New York, NY: Guilford Press.

Heimberg, R. G., & Becker, R. E. (2002). *Cognitive-behavioral group therapy for social phobia: Basic mechanisms and clinical strategies.* New York, NY: Guilford Press.

Heimberg, R. G., Dodge, C. S., Hope, D. A., & Kennedy, C. R. (2000). Cognitive behavioral group treatment for social phobia: Comparison with a credible placebo control. *Cognitive Therapy and Research, 14*, 1–23. doi:10.1007/bf01173521

Hofmann, S. G., Asnaani, A., & Hinton, D. E. (2010). Cultural aspects in social anxiety and social anxiety disorder. *Depression and Anxiety, 27*, 1117–1127. doi:10.1002/da.20759

Hood, H. K., Antony, M. M., Koerner, N., & Monson, C. M. (2010). Effects of safety behaviors on fear reduction during exposure. *Behaviour Research and Therapy, 48*, 1161–1169. doi:10.1016/j.brat.2010.08.006

Kearney, C. A. (2005). *Social anxiety and social phobia in youth: Characteristics, assessment, and psychological treatment.* New York, NY: Springer.

Kim, H.-Y., Lundh, L.-G., & Harvey, A. G. (2002). The enhancement of video feedback by cognitive preparation in the treatment of social anxiety. A single session experiment. *Journal of Behavior Therapy and Experimental Psychiatry, 33*, 19–37. doi:10.1016/S0005-7916(02)00010-1

Magee, L., Erwin, B. A., & Heimberg, R. G. (2009). Psychological treatment of social anxiety disorder and specific phobia. In M. M. Antony & M. B. Stein (Eds.), *Oxford handbook of anxiety and related disorders* (pp. 334–349). New York, NY: Oxford University Press.

McCabe, R. E., Ashbaugh, A. R., & Antony, M. M. (2010). Specific and social phobia. In M. M. Antony and D. H. Barlow (Eds.), *Handbook of assessment and treatment for psychological disorders* (2nd ed., pp. 186–223). New York, NY: Guilford Press.

McCabe, R. E., Miller, J. L., Laugesen, N., Antony, M. M., & Young., L. (2010). The relationship between anxiety disorders in adults and recalled childhood teasing. *Journal of Anxiety Disorders, 24*, 238–243. doi:10.1016/j.janxdis.2009.11.002

Miller, W. R., Zweben, A., DiClemente, C. C., & Rychtarik, R. G. (1992). *Motivational enhancement therapy manual: A clinical research guide for therapists treating individuals with alcohol abuse and dependence.* Rockville, MD: National Institute on Alcohol Abuse and Alcoholism.

Milosevic, I., & Radomsky, A. S. (2008). Safety behavior does not necessarily interfere with exposure therapy. *Behaviour Research and Therapy, 46*, 1111–1118. doi:10.1016/j.brat.2008.05.011

Mohlman, J., Gorenstein, E. E., Kleber, M., de Jesus, M., Gorman, J. M., & Papp, L. A. (2003). Standard and enhanced cognitive-behavior therapy for late-life generalized anxiety disorder: Two pilot investigations. *American Journal of Geriatric Psychiatry, 11*, 24–32.

Moscovitch, D. A., Antony, M. M., & Swinson, R. P. (2009). Exposure-based treatments for anxiety disorders: Theory and process. In M. M. Antony & M. B. Stein (Eds.), *Oxford handbook of anxiety and related disorders* (pp. 461–475). New York, NY: Oxford University Press.

Nilsson, J.-E., Lundhb, L.-G., Faghihic, S., & Roth-Anderssond, G. (2011). The enhancement of beneficial effects following audio feedback by cognitive preparation in the treatment of social anxiety. A single-session experiment. *Journal of Behavior Therapy and Experimental Psychiatry, 42*, 497–503. doi:10.1016/j.jbtep.2011.05.004

Norton, P. J. (2009). Integrated psychological treatment of multiple anxiety disorders. In M. M. Antony & M. B. Stein (Eds.), *Oxford handbook of anxiety and related disorders* (pp. 441–450). New York, NY: Oxford University Press.

Orr, E. M., & Moscovitch, D. A. (2010). Learning to re-appraise the self during video feedback for social anxiety: Does depth of processing matter? *Behaviour Research and Therapy, 48*, 728–737. doi:10.1016/j.brat.2010.04.004

Öst, L.-G. (1987). Applied relaxation: Description of a coping technique and review of controlled studies. *Behaviour Research and Therapy, 25*, 397–409. doi:10.1016/0005-7967(87)90017-9

Otto, M. W., & Pollack, M. H. (2009). *Stopping anxiety medication: Therapist guide* (2nd ed.). New York, NY: Oxford University Press.

Otto, M. W., Smits, J. A., & Reese, H. E. (2005). Combined psychotherapy and pharmacotherapy for mood and anxiety disorders in adults: Review and analysis. *Clinical Psychology: Science and Practice, 12*, 72–86. doi:10.1093/clipsy.bpi009

Powers, M. B., Sigmarsson, S. R., & Emmelkamp, P. M. (2008). A meta-analytic review of psychological treatments for social anxiety disorder. *International Journal of Cognitive Therapy, 1*, 94–113. doi:10.1680/ijct.2008.1.2.94

Rapee, R. M., & Heimberg, R. G. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy, 35*, 741–756. doi:10.1016/S0005-7967(97)00022-3

Robillard, G., Bouchard, S., Dumoulin, S., Guitard, T., & Klinger, E. (2010). Using virtual humans to alleviate social anxiety: Preliminary report from a comparative outcome study. *Studies in Health Technology and Informatics, 154*, 57–60. doi:10.3233/978-1-60750-561-7-57

Roth, D., Antony, M. M., & Swinson, R. P. (2001). Interpretations for anxiety symptoms in social phobia. *Behaviour Research and Therapy, 39*, 129–138. doi:10.1016/S0005-7967(99)—159-X

Schmidt, N. B., Richey, J. A., Buckner, J. D., & Timpano, K. R. (2009). Attention training for generalized social anxiety disorder. *Journal of Abnormal Psychology, 118*, 5–14. doi:10.1037/a0013643

Stein, D. J. (2009). Social anxiety disorder in the West and in the East. *Annals of Clinical Psychiatry, 21*, 109–117.

Taylor, S. (1996). Meta-analysis of cognitive-behavioral treatment for social phobia. *Journal of Behavioral Therapy and Experimental Psychiatry, 27*, 1–9. doi:10.1016/0005-7916(95)00058-5

Taylor, S., Koch, W. J., & McNally, R. J. (1992). How does anxiety sensitivity vary across the anxiety disorders? *Journal of Anxiety Disorders, 6*, 249–259. doi:10.1016/0887-6185(92)90037-8

Wallach, H. S., Safir, M. P., & Bar-Zvi, M. (2009). Virtual reality cognitive behavior therapy for public speaking anxiety: A randomized clinical trial. *Behavior Modification, 33*, 314–338. doi:10.1177/0145445509331926

Wells, A., White, J., Carter, K. (1997). Attention training: Effects on anxiety and beliefs in panic and social phobia. *Clinical Psychology and Psychotherapy, 4*, 226–232. doi:10.1002/(sici)1099-0879(199712)4:4<226::aid-cpp129>3.0.co;2-m

Westra, H. A., & Stewart, S. H. (1998). Cognitive behavioral therapy and pharmacotherapy: Complementary or contradictory approaches to the treatment of anxiety? *Clinical Psychology Review, 18*, 307–340. doi:10.1016/S0272-7358(97)00084-6

Wild, J., & Clark, D. M. (2011). Imagery rescripting of early traumatic memories in social phobia. *Cognitive and Behavioral Practice, 18*, 433–443. doi:10.1016/j.cbpra.2011.03.002

24

Pharmacological Treatment for Social Anxiety Disorder

Franklin R. Schneier, Laura B. Bragdon, Carlos Blanco, and Michael R. Liebowitz

Introduction

A variety of medication treatments have been established as efficacious for social anxiety disorder (SAD) over the past three decades. This chapter will review the evidence base for pharmacological treatment of SAD, with an emphasis on evidence from randomized clinical trials (RCTs). For this chapter, a PubMed search of published data was performed with the following search terms: "social anxiety disorder," "social phobia," "anxiety disorders," "pharmacotherapy," and "'medication." The search was conducted through May 2012 and only used articles published in English. The chapter concludes with an integration of this literature with our experience as practicing clinicians in a discussion of clinical issues in the pharmacotherapy of SAD.

The modern era of research in the pharmacological treatment of SAD dates back to the mid-1980s, after SAD was recognized as a distinct anxiety disorder in DSM-III (American Psychiatric Association, 1980). The selection of medications for testing in early clinical trials in SAD grew out of observations of efficacy for benzodiazepine anxiolytics in the treatment of anxiety symptoms in general; studies of β-adrenergic blockers in the treatment of stage fright in anxious performers; and evidence that monoamine oxidase inhibitors (MAOIs) had specific efficacy in "atypical" depression, a subtype defined in part by the feature of interpersonal sensitivity (Liebowitz, Gorman, Fyer, & Klein, 1985). The emergence of selective serotonin reuptake inhibitors (SSRIs) and serotonin norepinephrine reuptake inhibitors (SNRIs), and to a lesser extent the reversible inhibitors of monoamine oxidase A (RIMAs), then led to a large number of industry-sponsored RCTs in SAD, beginning in the mid-1990s. The contemporaneous development of a variety of cognitive-behavioral therapies (CBTs) has resulted in several comparative and combination trials with medication treatment. More recent pharmacological research has explored novel agents for treatment of SAD, including some agents specifically designed to enhance CBT.

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

The pharmacological treatment literature for SAD should be considered in the context of several methodological issues that have characterized these clinical trials. Most of these studies have limited recruitment to patients with a principal diagnosis of the generalized subtype of SAD, and most have excluded persons with current major depressive disorder, recent substance abuse, or lifetime psychosis. The generalizability of this literature to persons with only performance anxiety (nongeneralized SAD) or with prominent comorbidity is therefore unclear. This chapter will also address the few existing trials that have been conducted in patients with specific common comorbid conditions, such as alcohol dependence and major depressive disorder.

A majority of SAD trials have gradually escalated medication dosage to a prototypical antidepressant or anxiolytic dose over several weeks of treatment, and then adjusted the dose as clinically indicated. Only a few trials have compared fixed doses of SSRIs, so objective evidence for the relative efficacy of specific doses is limited. Similarly, a paucity of studies directly comparing active agents in the treatment of SAD has limited understanding of relative efficacy. Most trials have been of 8–12 weeks duration, with the goal of assessing acute response to treatment. A few, however, have assessed longer term outcome over periods ranging from 6 months to more than 2 years. Some of these studies have also examined the effects of medication discontinuation using randomized discontinuation designs.

The most commonly used outcome measure in SAD clinical trials has been the Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987), typically administered by a clinician. This instrument assesses fear and avoidance of 24 social or performance situations. Another common primary outcome measure has been the Clinical Global Impression (CGI) Change Scale, a 7-point scale, on which persons who are rated 2 (much improved) or 1 (very much improved) are typically considered responders (Zaider, Heimberg, Fresco, Schneier, & Liebowitz, 2003). Although a clear consensus on criteria for remission in SAD is lacking, some studies have reported remission rates in SAD, often defined by a CGI Change Scale score of 1 (very much improved). By this criterion, roughly half of pharmacotherapy responders in these studies (i.e., about 20–35% of some whole samples) have typically met this higher threshold representing remission.

Monotherapy

Selective Serotonin Reuptake Inhibitors and Serotonin Norepinephrine Reuptake Inhibitors

Over 20 placebo-controlled trials of SSRIs and 6 trials of the SNRI venlafaxine have consistently shown that SSRIs and SNRIs are efficacious in the treatment of SAD, and 7 meta-analyses have bolstered this finding. See Table 24.1 for individual study features and response rates for RCTs reviewed in this chapter. These findings of efficacy, along with a favorable side-effect profile and efficacy for depression (which is often comorbid with SAD), establish SSRIs and SNRIs as first-choice medications for SAD. All four of the medications that are Food and Drug Administration (FDA) approved for the treatment of SAD are in these classes: Paroxetine (both immediate and extended release), sertraline, extended-release fluvoxamine, and extended-release venlafaxine.

Table 24.1 Summary of Placebo-Controlled Studies in the Acute Treatment of Social Anxiety Disorder

Study population	*Drug class*	*Drug*	*Authors*	*Sample size*	*Duration (weeks)*	*Dose (mg/day)*	*Response rates (%)*	
							Drug	*Placebo*
Adults	MAOIs	Phenelzine[a]	Liebowitz et al. (1992)	51	8	45–90	64	23
		Phenelzine[b]	Gelernter et al. (1991)	64	12	30–90	69	20
		Phenelzine[c]	Versiani et al. (1992)	52	8	15–90	81	27
		Phenelzine	Heimberg et al. (1998)	64	12	15–75	52	27
		Phenelzine[d]	Blanco et al. (2010)	128	24	15–90	48	33
	RIMAs	Moclobemide[b]	Versiani et al. (1992)	52	8	100–600	65	20
		Moclobemide	Katschnig et al. (1997)	578	12	300–600	44	32
		Moclobemide	Noyes et al. (1997)	506	12	75–900	35	33
		Moclobemide	Schneier et al. (1998)	75	8	100–400	18	14
		Moclobemide	D. J. Stein, Cameron, et al. (2002)	390	12	450–750	43	30
		Brofaromine	van Vliet, den Boer, and Westenberg (1992)	30	12	50–150	80	14

(*continued*)

Table 24.1 (*Continued*)

Study population	*Drug class*	*Drug*	*Authors*	*Sample size*	*Duration (weeks)*	*Dose (mg/day)*	*Response rates (%)*	
							Drug	*Placebo*
		Brofaromine	Fahlen, Nilsson, Bog, Humble, and Pauli (1995)	77	12	150	78	23
		Brofaromine	Lott et al. (1997)	102	10	50–150	50	19
	Benzodiazepines	Clonazepam	Davidson et al. (1993)	75	10	0.5–3.0	78	20
		Clonazepam	Munjack et al. (1990)	23	8	0.5–6.0	90	10
		Clonazepam	Ontiveros (2008)	27	16	3.4, mean dose	65	30
		Bromazepam	Versiani, Nardi, Figueira, Mendlowicz, and Marques (1997)	60	12	3–27	83	20
		Alprazolam[b]	Gelernter (1991)	65	12	2.1–6.3	38	23
	SSRIs/SNRIs	Fluvoxamine	Van Vliet et al. (1993)	30	12	150	46	7
		Fluvoxamine	M. B. Stein et al. (1999)	86	12	202, mean dose	43	23
		Fluvoxamine (CR)	Westenberg et al. (2004)	300	12	100–300	48	44
		Fluvoxamine (CR)	Davidson, Yaryura-Tobias, et al. (2004)	279	12	100–300	34	17
		Paroxetine	M. B. Stein et al. (1998)	182	12	10–50	55	22

Paroxetine	Baldwin et al. (1999)	290	12	20–50	66	33
Paroxetine	Allgulander (1999)	92	12	20–50	70	8
Paroxetine	Liebowitz et al. (2002)	384	12	20–60	66	28
Paroxetine	D. J. Stein, Cameron, et al. (2002)	323	24	20–50	78	51
Paroxetine	Seedat and Stein (2004)	28	10	20–40	79	43
Paroxetine (CR)	Lepola et al. (2004)	370	12	12.5–37.5	57	30
Paroxetine	Allgulander et al. (2004)	434	12	20–50	66	36
Paroxetine	Lader et al. (2004)	839	24	20	80	66
Paroxetine	Wagner et al. (2004)	322	16	10–50	78	38
Paroxetine	Liebowitz et al. (2005)	440	12	25–50	63	36
Sertraline	Katzelnick et al. (1995)	12	10	50–200	50	9
Sertraline	Van Ameringen, Mancini, Farvolden, and Oakman (2000)	204	20	50–200	53	29
Sertraline	Walker et al. (2000)	50	24	50–200	96	64
Sertraline	Blomhoff et al. (2001)	387	24	50–150	40	24

(*continued*)

Table 24.1 (*Continued*)

Study population	*Drug class*	*Drug*	*Authors*	*Sample size*	*Duration (weeks)*	*Dose (mg/day)*	*Response rates (%)*	
							Drug	*Placebo*
		Sertraline	Liebowitz et al. (2003)	211	12	50–200	47	26
		Fluoxetine	Kobak et al. (2002)	60	8	20–60	40	30
		Fluoxetine	Davidson, Foa, et al. (2004)	295	14	10–60	51	32
		Fluoxetine	Clark et al. (2003)	60	16	20–60	33	16
		Escitalopram	Lader et al. (2004)	839	24	5–20	81	66
		Escitalopram	Kasper et al. (2005)	358	12	10–20	54	39
		Escitalopram	Montgomery et al. (2005)	371	24	10–20	78	50
		Citalopram	Furmark et al. (2005)	36	6	40	50	8.3
		Venlafaxine (ER)	Rickels et al. (2004)	272	12	75–225	50	34
		Venlafaxine (ER)	Allgulander et al. (2004)	434	12	75–225	69	36
		Venlafaxine (ER)	Liebowitz, Gelenberg, et al. (2005)	271	12	75–225	44	30
		Venlafaxine (ER)	Liebowitz, Mangano, et al. (2005)	440	12	75–225	59	36

	Venlafaxine (ER)	M. B. Stein, Pollack, Bystritsky, Kelsey, and Mangano (2005)	395	28	75–225	58	33
β-Blockers	Atenolol[a]	Liebowitz et al. (1992)	51	8	50–100	30	23
	Atenolol	Turner et al. (1994)	72	12	25–100	33	6
Other	Gabapentin	Pande et al. (1999)	69	14	900–3600	38	14
	Pregabalin	Pande et al. (2004)	135	10	150–600	43	22
	Pregabalin	Feltner et al. (2011)	329	11	300, 450	30	20
	Buspirone	Van Vliet et al. (1997)	30	12	15–30	27	13
	Buspirone	Clark and Agras (1991)	34	6	32, mean dose	57	60
	Nefazodone	Van Ameringen et al. (2007)	105	14	300–600	31	24
	Mirtazapine	Muehlbacher et al. (2005)	66	10	30	26	5.4
	Mirtazapine	Schutters et al. (2010)	60	12	30–45	13	13
	Levetiracetam	Zhang et al. (2005)	18	7	500–3000	22	14
Atypical antipsychotics	Olanzapine	Barnett et al. (2002)	12	8	5–20	60	0
	Quetiapine	Vaishnavi et al. (2007)	15	8	400	20	0

(*continued*)

Table 24.1 (*Continued*)

Study population	*Drug class*	*Drug*	*Authors*	*Sample size*	*Duration (weeks)*	*Dose (mg/day)*	*Response rates (%)*	
							Drug	*Placebo*
Pediatric	Benzodiazepines	Alprazolam	Simeon et al. (1992)	30	4	0.25–3.5	88	61
		Clonazepam	Graae et al. (1994)	15	8	0.25–2.0	75	25
	SSRIs/SNRIs	Fluoxetine	Black and Uhde (1994)	15	12	0.6	67	11
		Fluoxetine	Birmaher et al. (2003)	74	12	20	61	35
		Fluoxetine	Beidel et al. (2007)	122	12	40	36	6
		Sertraline	Rynn et al. (2001)	22	9	25–50	90	10
		Paroxetine	Wagner et al. (2004)	322	16	10–50	77.6	38.3
		Venlafaxine (ER)	March et al. (2007)	293	16	37.5–225	56	37

MAOIs, monoamine oxidase inhibitors; RIMAs, reversible inhibitors of monoamine oxidase A; SSRI, selective serotonin reuptake inhibitors; fluvoxamine (CR), fluvoxamine controlled release; venlafaxine (ER), venlafaxine extended release.

[a]Study had three arms: phenelzine, atenolol, and placebo; [b]Study had three arms: phenelzine, alprazolam, and placebo; [c]Study had three arms: phenelzine, moclobemide, and placebo; [d]Study had four arms: cognitive-behavioral group therapy (CBGT), phenelzine, combined CBGT and phenelzine, and placebo.

Paroxetine Paroxetine, in immediate-release and extended-release forms, has been found to be superior to placebo for treatment of SAD in all 11 published placebo-controlled studies (Allgulander, 1999; Baldwin, Bobes, Stein, Scharwachter, & Faure, 1999; Lader, Stender, Burger, & Nil, 2004; Lepola, Bergtholdt, St. Lambert, Davy, & Ruggiero, 2004; Liebowitz, Gelenberg, & Munjack, 2005; Liebowitz et al., 2002; Seedat & Stein, 2004; M. B. Stein, Liebowitz, Lydiard, & Pitts, 1998; D. J. Stein, Stein, Pitts, Kumar, & Hunter, 2002; D. J. Stein, Versiani, Hair, & Kumar, 2002; Wagner et al., 2004). Paroxetine has also been found superior to placebo in the prevention of relapse over an additional 24 weeks of double-blind discontinuation treatment in patients who had demonstrated an acute response after an initial 12-week course (D. J. Stein, Cameron, Amrein, & Montgomery, 2002).

Fluvoxamine Results from four double-blind studies investigating the efficacy of fluvoxamine in immediate-release and extended-release forms identify it as superior to placebo for reduction of SAD symptoms (Davidson, Yaryura-Tobias, et al., 2004; M. B. Stein, Fyer, Davidson, Pollack, & Wiita, 1999; van Vliet, den Boer, & Westenberg, 1994; Westenberg, Stein, Yang, Li, & Barbato, 2004).

Sertraline Five placebo-controlled trials have demonstrated the efficacy of sertraline (Blomhoff et al., 2001; Katzelnick et al., 1995; Liebowitz et al., 2003; Van Ameringen et al., 2001; Walker et al., 2000). Sertraline has also been found to be more effective than placebo in preventing relapse (Walker et al., 2000).

Escitalopram and citalopram Results from placebo-controlled trials of escitalopram have found it to be superior to placebo in SAD symptom reduction and relapse prevention (Kasper, Stein, Loft, & Nil, 2005; Lader et al., 2004; Montgomery, Nil, Durr-Pal, Loft, & Boulenger, 2005). A small placebo-controlled study also found that citalopram was well tolerated and superior to placebo (Furmark et al., 2005).

Fluoxetine Among the SSRIs and SNRIs, fluoxetine is the only medication to have shown mixed results in clinical trials for SAD. The largest study (Davidson, Foa, et al., 2004), a collaboration between expert pharmacological and cognitive-behavioral sites, found fluoxetine to be superior to placebo and equivalent to group CBT, but two smaller trials in adults did not find fluoxetine efficacy to differ from placebo (Clark et al., 2003; Kobak, Griest, Jefferson, & Katzelnick, 2002).

Venlafaxine Five placebo-controlled trials have supported the efficacy of extended-release venlafaxine, a SNRI, for SAD (Allgulander et al., 2004; Liebowitz, Gelenberg, et al., 2005; Liebowitz, Mangano, Bradweijn, & Asnis, 2005; Rickels, Mangano, & Khan, 2004; M. B. Stein, Pollack, Bystritsky, Kelsey, & Mangano, 2005). One of these trials also compared venlafaxine to paroxetine and did not detect a difference in efficacy (Liebowitz, Gelenberg, et al., 2005). In a fixed dose comparison study, there were no significant differences between high (150–225 mg/day) and low (75 mg/day) dosages, suggesting that the therapeutic mechanism of action is similar to that of SSRIs and generally not dependent upon the inhibition of norepinephrine reuptake that is recruited at higher dosages (M. B. Stein et al., 2005).

Time course of treatment response The time course of initial response to SSRIs and SNRIs appears similar to that seen in depression, with onset of benefit typically beginning within the first 4 weeks, such that persons who have shown no benefit after the first 4 weeks of treatment have only a 20% chance of responding by the end of a 12-week trial (Baldwin, Stein, Dolberg, & Bandelow, 2009). On the other hand, once improvement begins, it may progress more gradually than is typically reported for treatment of depression. For example, across three studies of paroxetine, only 28% of nonresponders after 8 weeks of treatment (Clinical Global Improvement score of no better than "minimally improved") were shown to achieve response by week 12 (D. J. Stein, Cameron, et al., 2002).

Long-term treatment/discontinuation Three studies have randomized patients who had responded to an acute 12–20-week trial of SSRI or SNRI to either continuation of active treatment or double blind switch to placebo (Montgomery et al., 2005; D. J. Stein, Versiani, et al., 2002; Walker et al., 2000). These discontinuation studies demonstrate the benefit of continuation treatment: Response in these trials was well maintained during 6 months of continuation treatment, with relapse rates under 25%—significantly lower than the relapse rates of 36–50% among persons switched to placebo. Conversely, the same studies demonstrate that 50–64% of persons switched to placebo do not relapse over 6 months, suggesting that many SSRI responders can discontinue treatment and do well for at least this period of time. Predictors of relapse, however, are not well established.

Monoamine Oxidase Inhibitors

MAOIs were among the first medications to be widely studied as a treatment for SAD, and they are the second most-studied class of medications for SAD after SSRIs/SNRIs. Five double-blind, placebo-controlled trials have consistently demonstrated the efficacy of the irreversible MAOI phenelzine at doses of 30–90 mg/day in the treatment of SAD (Blanco et al., 2010; Gelernter et al., 1991; Heimberg et al., 1998; Liebowitz et al., 1992; Versiani et al., 1992). Another irreversible MAOI, tranylcypromine, was also shown to be an effective treatment for SAD in a placebo-controlled trial of patients with comorbid SAD and panic disorder (Nardi et al., 2010). Despite their efficacy, clinical use of irreversible MAOIs has been limited by their side-effect profile and particularly the risk of hypertensive crisis if a low-tyramine diet and related precautions are not strictly followed.

Reversible Inhibitors of Monoamine Oxidase A

RIMAs, which bind reversibly to monoamine oxidase A, and therefore confer a significantly lower risk than irreversible MAOIs of potentiating the dangerous pressor effect of tyramine, were developed with the hope of providing the efficacy of the older MAOIs with less risk. Although RIMAs do allow for relaxation or total elimination of dietary restrictions, they also appear to be less effective than standard MAOIs.

Moclobemide is the only marketed RIMA, available in much of the world although it has never been approved for use in the United States. Five double-blind placebo-controlled studies of moclobemide have produced mixed results, suggesting modest efficacy in the treatment of SAD (Katschnig, Stein, & Buller, 1997; Noyes et al., 1997; Schneier, Goetz, Campeas, Marshall, & Liebowitz, 1998; D. J. Stein, Cameron, et al., 2002; Versiani et al., 1992). The results of these studies, including one head-to-head comparison with phenelzine, indicate that whereas moclobemide appears better tolerated and safer than phenelzine, it is less efficacious in the treatment of SAD. The RIMA brofaromine appeared efficacious in three RCTs, but it has not been marketed.

Other Antidepressants

Mirtazapine, a presynaptic adrenoceptor antagonist, has been found efficacious in one of two placebo-controlled trials for the treatment of SAD (Muehlbacher et al., 2005; Schutters, Van Megen, Van Veen, Denys, & Westenberg, 2010). The only published placebo-controlled study of nefazodone, which has both 5-HT reuptake and 5-HT_{2A} receptor blockade properties, had negative results (Van Ameringen et al., 2007). Neither bupropion nor tricyclic antidepressants have been shown to be effective in the treatment of SAD (Emmanuel, Lydiard, & Ballenger, 1991; Simpson et al., 1998).

Benzodiazepines

Although benzodiazepines are among the most widely used medications for anxiety in general, they have been relatively little studied for SAD. Three studies of the high-potency benzodiazepine clonazepam prescribed on a standing-dose basis at 2–3 mg/day have reported significant improvement as compared to placebo (Davidson et al., 1993; Munjack, Baltazar, Bohn, Cabe, & Appleton, 1990; Ontiveros, 2008). One of these studies evidenced one of the larger effects in the SAD literature, with 78% of the clonazepam group classified as responders versus 20% of the placebo group (Davidson et al., 1993). In the only published placebo-controlled study of alprazolam for SAD, there was no significant difference in response rate between groups (Gelernter et al., 1991). Use of bromazepam, a benzodiazepine marketed outside the United States, has also been reported to be efficacious for the treatment of SAD in a controlled trial (Versiani, Amrein, & Montgomery, 1997).

Prescription of benzodiazepines has been widely discouraged due to potential adverse effects such as impaired cognition, sedation and falls, potential for abuse and dependency, and withdrawal effects upon discontinuation. A double-blind study of clonazepam discontinuation, however, showed that most SAD patients who had responded to clonazepam could be safely tapered off the medication. Tapering clonazepam at the rate of 0.25 mg/day every 2 weeks resulted in low rates of withdrawal symptoms (28%) and of relapse (21%) over the next 5 months (Connor et al., 1998).

In summary, double-blind studies of clonazepam and bromazepam, but not alprazolam, have found these medications superior to placebo. Benzodiazepines are also

often used clinically on an as-needed basis for performance anxiety, although this is supported only by small, older studies in nonclinical samples and anecdotal clinical evidence in SAD.

β-Adrenergic Antagonists

The use of β-adrenergic antagonists (β-blockers) in SAD is similarly based on an old literature showing efficacy in small samples of anxious performers. Research showing a connection between anxiety, signs and symptoms of peripheral arousal, and increased plasma levels of norepinephrine led to early trials of β-blockers in nonclinical samples of performers with high levels of anxiety (Hartley, Ungapen, David, & Spencer, 1983; Neftel et al., 1982). The results of those trials indicated that β-blockers are successful in decreasing the autonomic manifestations of anxiety in performance situations.

Anecdotal experience also suggests that β-blockers are effective for nongeneralized, circumscribed performance anxiety. However, modern controlled trials of *daily use* of β-blockers in patients diagnosed primarily with the *generalized* subtype of SAD have not demonstrated efficacy superior to placebo in this population (Liebowitz et al., 1992; Turner, Beidel, & Jacob, 1994). Thus, the utility of β-blockers for SAD appears confined to as-needed use for performance anxiety, although controlled data to validate that clinical impression are lacking.

Other Medications

Buspirone Buspirone is an azaspirone that acts as a full agonist on the serotonin 1A ($5HT_{1A}$) autoreceptor and as a partial agonist on the postsynaptic 5-HT_{1A} receptor. Neither of the two controlled trials of buspirone, up to 30 mg/day, in SAD demonstrated efficacy for it as monotherapy (van Vliet, den Boer, Westenberg, & Pian, 1997). An open trial of buspirone (up to 60 mg/day) found a response rate of 47% overall, but 67% among patients who tolerated a dose of 45–60 mg/day. Efficacy of these higher doses has not been assessed in controlled trials (Schneier et al., 1994).

Anticonvulsants Gabapentin and pregabalin have been reported to act at the alpha delta calcium channel to reduce glutamatergic activity. In the single published placebo-controlled trial of gabapentin, at 900–3600 mg/day, SAD patients showed a significantly higher response rate than the placebo group (Pande et al., 1999).

In two randomized double-blind trials, pregabalin 600 mg/day (but not 150 mg/day) was superior to placebo for SAD (Feltner, Liu-Dumaw, Schweizer, & Bielski, 2011; Pande et al., 2004). A controlled discontinuation trial found that 450 mg/day pregabalin significantly lowered the rate of relapse as compared to placebo (Greist, Liu-Dumaw, Schweizer, & Feltner, 2011). Further research will be needed to define the optimal dose, magnitude of the effect, and long-term effects of pregabalin for SAD.

Levetiracetam is a novel anticonvulsant that modulates voltage-gated calcium channels in the central nervous system. A small randomized placebo-controlled study

(Zhang, Conner, & Davidson, 2005) found no differences in efficacy as compared to placebo. Other anticonvulsants, including tiagabine, valproic acid, and topiramate have not been impressive in small open trials (Dunlop et al., 2007; Van Ameringen, Mancini, Pipe, Oakman, & Bennett, 2004).

Atypical antipsychotics Several antipsychotics, including olanzapine (Barnett, Kramer, Casat, Connor, & Davidson, 2002), quetiapine (Schutters, van Megen, & Westenberg, 2005; Vaishnavi, Alamy, Zhang, Connor, & Davidson, 2007), and risperidone (Simon et al., 2006), have been studied in small open trials for the treatment of SAD. A small ($N = 15$) controlled study of quetiapine found no significant group differences; however, 40% of quetiapine patients and 0% of placebo patients showed much or very much improvement (Vaishnavi et al., 2007). Another small RCT of quetiapine reported that a single 25 mg dose taken 1 hr before a public speaking challenge was not effective in preventing SAD symptoms in persons with fear of public speaking (Donahue et al., 2009). Notably, there have also been multiple case reports of the *emergence* of SAD symptoms during treatment of other disorders with antipsychotic medications (Scahill, Leckman, Schultz, Katsovich, & Peterson, 2003). Further studies will be needed to clarify whether these medications have a role in the treatment of SAD, especially given their potential side-effect burdens of weight gain and metabolic syndrome.

Cannabidiol A recent placebo-controlled study assessed the effects of cannabidiol, an investigative cannabinoid, during a simulated public speaking test. Results showed the cannabidiol group had significantly lower ratings on anxiety, cognitive impairment, and discomfort measures (Bergamaschi, Querioz, Chagas, Oliverira, & Martinis, 2011), suggesting need for further research of this novel class of agents.

Meta-analyses

Several meta-analyses have examined the efficacy of medication for the treatment of SAD in adults. Here we will focus on the most recent reports. Blanco, Schneier, et al. (2003) conducted a meta-analysis of the placebo-controlled studies of pharmacotherapy for SAD using studies published between January 1980 and June 2001. Effect sizes for SSRIs, phenelzine, and clonazepam were not significantly different from each other, but stability of findings for classes other than SSRIs was limited by the small number of trials. There were also no significant differences in efficacy between the three SSRIs that had been tested in placebo-controlled studies: paroxetine, sertraline, and fluvoxamine (see Table 24.2).

Hedges and colleagues investigated the efficacy of SSRIs for the treatment of adult SAD and included 15 published randomized double-blind placebo-controlled trials (Hedges, Brown, Shwalb, Godfrey, & Larcher, 2007), confirming that all SSRIs studied were significantly more efficacious than placebo. Furthermore, no significant differences were found between LSAS outcome scores for the drugs paroxetine, sertraline, fluvoxamine, and fluoxetine.

Table 24.2 Meta-analytic Results of Controlled Psychopharmacological Trials in SAD

Study	*Drug*	*Number of studies*	*Results*	
Blanco, Schneier, et al. (2003)			Effect size based on LSAS[a] (95% CI)	Effect size based on the CGI[b] (95% CI)
	SSRIs	6	0.65 (0.50–0.81)	4.1 (2.01–8.41)
	Benzodiazepines	2	1.54 (–0.03 to 3.32)	16.61 (10.18–27.39)
	Phenelzine	3	1.02 (0.50–1.02)	5.53 (2.56–11.94)
	Moclobemide	4	0.30 (0.00–0.6)	1.84 (0.89–3.82)
	Brofaromine	3	0.66 (0.38–0.94)	6.96 (2.39–20.29)
	Gabapentin	1	0.78 (0.29–1.27)	3.78 (1.88–7.54)
	Atenolol	2	0.10 (–0.44 to 0.64)	1.36 (0.87–2.12)
	Buspirone[c]	1	0.02 (–0.70 to 0.73)	–
Canton, Scott, and Glue, (2012)			Odds ratio M-H (95% CI)[d]	
	SSRIs	17	2.78 (2.32–3.32)	
	SNRIs	4	2.42 (1.92–3.06)	
	MAOIs	4	7.22 (2.90–17.97)	
	RIMAs	9	2.96 (1.78–4.91)	
	A2Ds	3	3.11 (1.92–5.04)	

SSRIs, selective serotonin reuptake inhibitors; SNRIs, selective norepinephrine reuptake inhibitors; MAOIs, irreversible monoamine oxidase inhibitors; RIMAs, reversible inhibitors of monoamine oxidase A; A2Ds, alpha-2-delta ligands. Odds ratios of response, for most studies, were based on assessments on Clinical Global Impression Change Scale. [a]LSAS, Liebowitz Social Anxiety Scale; [b]CGI, Clinical Global Impression Scale; [c]Study did not use the CGI; [d]M-H (95% CI), Mantel–Haenszel 95% confidence interval.

Hansen et al. (2008) focused on the comparative efficacy of SSRIs and venlafaxine. Confirming findings from previous meta-analyses, they did not find significant differences in the efficacy of these medications. The most recent meta-analysis, by Canton and colleagues, included 41 pharmacological, psychotherapeutic, and combined treatment randomized controlled trials (Canton, Scott, & Glue, 2012). The mean odds ratio for response to SSRIs versus placebo was 2.73 (see Table 24.2).

Combination, Augmentation, and Switching of Treatments

Medications

Although even the most efficacious monotherapies for SAD yield only partial responses for many patients, there has been little study of augmentation and combination treatments. Combined treatment with a benzodiazepine plus an SSRI was studied in a small controlled trial. Seedat and Stein (2004) randomized 28 patients to paroxetine 20–40 mg/day plus clonazepam 1–2 mg/day or paroxetine plus placebo. More clonazepam patients (79%) than placebo patients (43%) were classified as CGI responders, but this effect only approached significance ($p = 0.06$) in this small sample.

Combined treatment with pindolol, a β-adrenergic blocker with $5HT_{1A}$ agonist effects, and the SSRI paroxetine was not found to be superior to paroxetine alone

in a placebo-controlled study by Stein and colleagues (M. B. Stein, Sareen, Hami, & Chao, 2001).

Buspirone augmentation of SSRI treatment nonresponders appeared promising in a preliminary open trial. Ten treatment-resistant patients with generalized SAD had buspirone added to their existing SSRI treatment for 8 weeks, and seven (70%) patients were considered responders (Van Ameringen, Mancini, & Wilson, 1996).

Open trials of switching SSRI nonresponders to either escitalopram or venlafaxine suggest that these may be useful strategies (Altamura, Pioli, Vitto, & Mannu, 1999; Pallanti & Quercioli, 2006). It is not clear, however, whether these results represent properties specific to escitalopram and/or venlafaxine switches or a more general principle of differential responsivity to individual medications within the SSRI/SNRI class.

Medications and Psychotherapy

As CBT and pharmacotherapy treatments for SAD are presumed to work by different mechanisms, a natural progression in the study of treatment of SAD has been to compare the efficacy of these modalities and their combinations. Several controlled studies have examined the combination of CBT and SSRI treatments, and some have also compared CBT to SSRI treatments in SAD. In a comparison of CBT to self-exposure plus fluoxetine and self-exposure plus placebo, after 16 weeks there was significant improvement for all three treatment groups, and CBT was superior to both fluoxetine and placebo groups at post-treatment and 12-month follow-up. Fluoxetine response at 12 months did not differ from response to placebo in this study (Clark et al., 2003).

Another study compared a different form of CBT to fluoxetine, placebo, and their combinations (CBT + fluoxetine and CBT + placebo) in a generalized SAD sample in a collaboration of pharmacological- and CBT-oriented research groups (Davidson, Foa, et al., 2004). After 14 weeks of treatment, all active treatments were superior to placebo, with no differences among them. Combined treatment was not superior to either of the monotherapies.

Blomhoff et al. (2001) compared sertraline, exposure therapy, their combination, and placebo. Patients received either sertraline or placebo for 24 weeks, and half of those in each group also received eight sessions of exposure therapy during the first 12 weeks. Results indicated that patients given only sertraline and those given sertraline plus exposure therapy did not differ from each other in response, but both were significantly more improved than the control group. Reanalysis of these data (Blanco et al., 2010) showed an ordering of response, with combined treatment being superior overall to the monotherapies. In a 1-year follow-up of this trial, patients in the sertraline-only group and the combined group demonstrated greater deterioration than those in the exposure-only group, whereas those in the combined group had outcomes that were superior to both other treatments at week 24 but similar to exposure-alone group at week 52 (Haug et al., 2003).

Two studies have included both MAOIs and CBT in the treatment of SAD. In another two-site collaboration of pharmacological- and CBT-oriented research groups, the MAOI phenelzine, CBT delivered in a group format, an educational

support group, or pill placebo were compared. After 12 weeks of treatment, CBT and phenelzine produced similar proportions of treatment responders (75% and 77%, respectively) and both active treatments had higher proportions of responders than the placebo or educational support conditions (Heimberg et al., 1998). However, phenelzine patients were significantly more improved than CBT patients on some dimensional measures of social anxiety. Over the course of 6 months maintenance treatment and 6 months follow-up, patients treated with CBT were significantly less likely to relapse than were patients treated with phenelzine (0% vs. 50%) (Liebowitz et al., 1999). Thus, phenelzine may provide somewhat more immediate relief, but CBT may provide greater protection against relapse. A subsequent study at the same sites compared the combination of CBT plus phenelzine, to CBT, phenelzine, and placebo in 128 patients with SAD (Blanco et al., 2010). The combined treatment outperformed placebo on all measures, fared better than phenelzine on some measures of social anxiety, and consistently did better than CBT alone. No follow-up data have been reported for this trial.

Another small study suggested that adding psychodynamic group therapy to clonazepam increased efficacy (Knijnik et al., 2008). Some other comparative studies have limited importance for understanding relative efficacy because they compared CBT to medications that were not themselves superior to placebo (Clark & Agras, 1991; Turner et al., 1994) or allowed some CBT procedures in both CBT and medication (non-CBT) groups (Gelernter et al., 1991).

Several studies have examined novel medication approaches designed to enhance response to CBT. D-Cycloserine, a partial agonist at the NMDA receptor, is not believed to be anxiolytic in its own right, but it has been shown in animal models to enhance extinction learning, the same process believed crucial to the mechanism of the exposure element of CBT in humans. In a variant on this approach, the investigational neuromodulator oxytocin, which has been shown to reduce social threat perception and improve processing of positive social cues, has also been studied as a CBT augmentation strategy. Studies of D-cycloserine and oxytocin are reviewed in detail in **Chapters 18 and 21**, respectively.

Treatment of SAD with Comorbid Disorders

A few studies have addressed pharmacotherapy of persons who present with SAD and comorbid conditions. In patients with SAD and comorbid depression, depressive symptoms were observed to respond more rapidly than SAD symptoms during an open trial of SSRI treatment with citalopram (Schneier et al., 2003). Persons with SAD and hyperhidrosis were shown to benefit from augmentation of SSRI treatment with botulinum toxin treatment for sweating in an RCT (Connor, Cook, & Davidson, 2006). In an RCT of patients with SAD and comorbid alcohol use disorders, paroxetine was efficacious for symptoms of SAD, although it did not reduce overall alcohol consumption (Book, Thomas, Randall, & Randall, 2008). A large multicenter RCT in adults with SAD comorbid with attention-deficit/hyperactivity disorder (ADHD) found that the ADHD treatment atomoxetine improved both ADHD and SAD symptoms more than placebo (Adler et al., 2009).

Pharmacotherapy of SAD in Children and Adolescents

Nonpharmacological approaches are particularly preferred in children, and relatively few studies of pediatric SAD have examined the efficacy of pharmacotherapy. Some of these pediatric randomized placebo-controlled studies relevant to SAD have included children with a range of anxiety disorders, in addition to SAD. Only two studies have investigated the efficacy of benzodiazepines in pediatric SAD and neither reported results that differentiated medication from placebo (Graae, Milner, Rizzotto, & Klein, 1994; Simeon et al., 1992).

More data are available on the efficacy of SSRIs and SNRIs from several placebo-controlled trials in children and adolescents 6–17 years of age (Beidel et al., 2007; Birmaher et al., 2003; Ginsburg et al., 2011; March, Entusah, Rynn, Albano, & Tourian, 2007; Rynn, Siqueland, & Rickels, 2001; Wagner et al., 2004; Walkup et al., 2008; Williams & Miller, 2003). These studies have largely supported the efficacy of SSRIs and SNRIs in the treatment of SAD in children/adolescents, and they are summarized in Table 24.1. Reports of an increased risk of suicidal ideation among adolescents treated with SSRIs or SNRIs, primarily in studies of depression, led the FDA to add a warning in regard to the use of antidepressants in this population (Bridge et al., 2007). The increase in suicidal ideation has been reported to be more linked to children with depressive disorders rather than anxiety (Gibbons, Hur, Bhaumik, & Mann, 2006).

Clinical Approach to Medication Treatment of the Patient with SAD

Assessment

The clinical approach to the patient with SAD begins with a thorough clinical assessment. Decisions about whether to treat, to treat with medication, and to treat with a specific medication all will depend on the outcome of a variety of aspects of the evaluation, including diagnosis, history of prior treatments, psychosocial supports and stressors, the patient's beliefs about their symptoms, their short- and long-term goals, and their attitudes about medication treatment.

Diagnostic assessment must, in addition to establishing whether SAD is present, identify any relevant psychiatric and medical comorbidity. Presence of significant comorbid unipolar depression, for example, favors use of an antidepressant medication that is indicated for both conditions, whereas comorbid bipolar disorder would mitigate against the use of agents that could precipitate mania. As described above, there is evidence that in comorbid patients depression may respond more quickly than SAD, and persons with comorbid ADHD may benefit from treatment directed at that condition alone. Comorbid alcohol, cannabis, or other substance abuse and dependence usually require concomitant treatment directed at the substance use disorder, and the comorbidities would influence choice of medication for SAD away from classes with potential for abuse, such as benzodiazepines. Avoidant personality disorder is very common in generalized SAD, at least partially due to overlapping

diagnostic criteria (see **Chapters 6 and 12** for extended discussion on this issue), and clinical experience suggests that it generally does not require specific modification of pharmacotherapy approach.

Concomitant *medical* conditions can also influence the decision to treat SAD with medication and the choice of medication. Asthma and some forms of heart block are contraindications to β-adrenergic blockers. SNRIs have potential to increase blood pressure at higher doses. MAOIs cannot be used in conjunction with several types of medications, including decongestants, epinephrine that is commonly administered in dental anesthesia, and some narcotics. History of allergic reactions to specific agents needs to be considered as well.

Many of the patients seeking treatment for SAD are women of childbearing potential. Since nonmedication treatments are generally preferable during pregnancy and breast-feeding, assessment of women for treatment must consider their plans regarding pregnancy and their use of contraceptive methods.

Assessment of clinical features within the diagnosis of SAD is also essential. Key issues include determining the scope of feared situations, quality of symptoms, assessing for suicidality, and extent of impairment. Feared situations may range from highly specific and predictable public performance situations to global fears of any interpersonal contact. Understanding the specific nature of the physical, emotional, and cognitive symptoms and the resulting types of impairment is essential to understanding the patient and being able to monitor the effects of treatment.

Finally, it is critical to take a treatment history and to explore the patient's ideas about and preferences for treatment. Obtaining a treatment history may not only identify prior treatments to return to or to avoid, but it may offer clues to the shaping of the patient's treatment preferences and potential obstacles such as history of noncompliance with treatment. Treatment history of family members can offer additional guidance in these areas. Patients with SAD are often reticent about expressing disagreement or conflict, and they will be more likely to simply avoid treatment recommendations that they find unacceptable. Initiating a discussion about treatment options and preferences is therefore important for increasing likelihood of compliance.

Choice of Treatment

Treatment selection for adults with SAD should include a discussion of what is known about the short- and long-term efficacy and risks of established treatments with medication and CBT. A majority of patients can expect significant improvement over a several-month course of acute treatment with either modality, but full remission is less common. The potential long-term benefits of CBT and absence of side-effect risks are advantages, but in some cases medications or combined treatment may be more effective, may work more rapidly, or may be more acceptable to some patients. Pharmacotherapy that is undertaken without concomitant formal CBT should be accompanied by encouragement to actively confront feared situations as treatment progresses.

SSRIs and SNRIs are currently considered first-line pharmacotherapies for SAD based on the large body of data showing their efficacy and safety, but superiority of any particular agent within this class has not been established. Pharmacotherapy

for persons with circumscribed predictable feared situations may be most efficiently pursued using as-needed medication with β-adrenergic blockers or benzodiazepines. Use of trial doses at home can establish tolerability of the as-needed medication prior to use in feared situations. Effective pharmacotherapy of the generalized subtype of SAD, in which persons fear most social situations, however, requires use of standing-dose medications, such as SSRIs.

Patients should be educated that, while improvement of symptoms during SSRI treatment can sometimes occur in the first 2 weeks, an 8–12-week course of treatment is needed to fully assess the effect of treatment. Since optimal dosage may vary widely between individuals, a typical strategy is to achieve a dose at the lower end of the effective range (e.g., 20 mg of paroxetine) within the first week and to increase the dose further in persons who are not showing marked improvement after 4 weeks of treatment. In persons who either do not respond to or do not tolerate an 8–12-week course of SSRI/SNRI treatment, options include (1) switching to another SSRI/SNRI, a benzodiazepine, mirtazapine, gabapentin, or pregabalin, a RIMA or MAOI, or CBT; or particularly in the case of partial response to the SSRI/SNRI, (2) augmenting treatment with a benzodiazepine, mirtazapine, gabapentin, or pregabalin, buspirone, or CBT. Because no clear guidelines for choice among second-line treatments have been established, selection of a strategy should be determined on an individual basis related to prior treatment history, clinical judgment, and patient preference.

Treatment Monitoring and Discontinuation

Treatment should be monitored most closely in the initial weeks of treatment, when side effects are most likely to emerge. Assessment of treatment outcome should consider improvement in physical and cognitive symptoms of social anxiety, avoidance of social situations and avoidance of social interaction within those situations, and ancillary symptoms, such as depression, and self-medication with alcohol. In persons who respond to an acute trial of medication, it is reasonable to continue medication for at least another 6 months or longer if improvement has not yet reached a plateau. Clinical experience suggests that patients with persistent symptoms may be at greater risk of relapse upon medication discontinuation than patients who are in full remission. Partial responders should therefore have their dose adjusted or their medication augmented in service of the goal of remission. When medication is discontinued, it should be done gradually, and this is especially true of relatively short-acting SSRI/SNRIs such as paroxetine and venlafaxine, as well as the benzodiazepines, to minimize the risk of acute withdrawal symptoms.

Conclusions

In summary, a substantial body of evidence now supports the acute efficacy of medication treatment for SAD. Medication and CBT are first-line treatments that appear to be roughly comparable in respect to acute efficacy, and they are sometimes most effective in combination. Among medication treatments for SAD, SSRIs and SNRIs are well-established first-line approaches due to good evidence for efficacy and tolerability

in over 25 RCTs. Alternative medication treatments that have appeared efficacious in controlled trials include benzodiazepines, mirtazapine, moclobemide, gabapentin, and pregabalin. β-Blockers are commonly used in the treatment of performance anxiety in nongeneralized SAD. Future directions for research should include development of predictors of pharmacotherapy response, clarification of treatments for nonresponders, and further assessment of novel cognitive enhancers that may offer synergies with CBT (also see **Chapters 18 and 21**).

References

Adler, L. A., Liebowitz, M., Kronenberger, W., Qiao, M., Rubin, R., Hollandbeck, M., et al. (2009). Atomoxetine treatment in adults with attention-deficit/hyperactivity disorder and comorbid social anxiety disorder. *Depression and Anxiety, 26*, 212–221.

Allgulander, C. I. (1999). Paroxetine in social anxiety disorder: A randomized placebo-controlled study. *Acta Psychiatrica Scandinavica, 100*, 196–198.

Allgulander, C. I., Mangano, R., Zhang, J., Dahl, A., Lepola, U., Sjodin, I., et al. (2004). Efficacy of Venlafaxine ER in patients with social anxiety disorder: A double-blind, placebo-controlled, parallel-group comparison with paroxetine. *Human Psychopharmacology: Clinical and Experimental, 9*, 387–396.

Altamura, A. C., Pioli, R., Vitto, M., & Mannu, P. (1999). Venlafaxine in social phobia: A study in selective serotonin reuptake inhibitor non-responders. *International Journal of Clinical Psychopharmacology, 14*, 239–245.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders.* Washington, DC: Author.

Baldwin, D., Bobes, J., Stein, D. J., Scharwaechter, I., & Faure, M. (1999). Paroxetine in social phobia/social anxiety disorder: Randomized, double-blind, placebo-controlled study. *The British Journal of Psychiatry, 175*, 120–126.

Baldwin, D. S., Stein, D. J., Dolberg, O. T., & Bandelow, B. (2009). How long should a trial of escitalopram treatment be in patients with major depressive disorder, generalized anxiety disorder or social anxiety disorder? An exploration of the randomized controlled trial database. *Human Psychopharmacology, 24*, 269–275.

Barnett, S. D., Kramer, M. L., Casat, C. D., Connor, K. M., & Davidson, J. R. (2002). Efficacy of olanzapine in social anxiety disorder: A pilot study. *Journal of Psychopharmacology, 16*, 365–368.

Beidel, D. C., Turner, S. M., Sallee, F. R., Ammerman, R. T., Crosby, L. A., & Pathak, S. (2007). SET-C versus fluoxetine in the treatment of childhood social phobia. *Journal of the American Academy of Child & Adolescent Psychiatry, 46*, 1622–1632.

Bergamaschi, M. M., Queiroz, R. H., Chagas, M. H., Oliveira, D. C., & Martinis, B. S. (2011). Cannabidiol reduces the anxiety induced by simulated public speaking in treatment-naïve social phobia patients. *Neuropsychopharmacology, 36*, 1219–1226.

Birmaher, B., Axelson, D. A., Monk, K., Kalas, C., Clark, D. B., Ehmann, M., et al. (2003). Fluoxetine for the treatment of childhood anxiety disorders. *Journal of the American Academy of Child & Adolescent Psychiatry, 42*, 415–423.

Black, B., & Uhde, T. W. (1994). Treatment of elective mutism with fluoxetine: A double-blind placebo-controlled study. *Journal of the American Academy of Child & Adolescent Psychiatry, 33*, 1000–1006.

Blanco, C., Heimberg, R. G., Schneier, F. R., Fresco, D. M., Chen, H., Turk, C. L., et al. (2010). A placebo-controlled trial of phenelzine, cognitive behavioral group therapy, and

their combination for social anxiety disorder. *Archives of General Psychiatry, 67*, 286–295.

Blanco, C., Raza, M. S., Schneier, F. R., & Liebowitz, M. R. (2003). The evidence-based pharmacological treatment of social anxiety disorder. *International Journal of Neuropsychopharmacology, 6*, 427–442.

Blanco, C., Schneier, F. R., Schmidt, A. B., Blanco-Jerez, C. R., Marshall, R. D., Sanchez-Lacay, A., et al. (2003). Pharmacological treatment of social anxiety disorder: A meta-analysis. *Depression and Anxiety, 18*, 29–40.

Blomhoff, S., Haug, T. T., Hellstrom, K., Holme, I., Humble, M., Madsbu, H. P., et al. (2001). Randomised controlled general practice trial of sertraline, exposure therapy and combined treatment in generalised social phobia. *The British Journal of Psychiatry, 179*, 23–30.

Book, S. W., Thomas, S. E., Randall, P. K., & Randall, C. L. (2008). Paroxetine reduces social anxiety in individuals with a co-occurring alcohol use disorder. *Journal of Anxiety Disorders, 22*, 310–318.

Bridge, J. A., Iyengar, S., Salary, C. B., Barbe, R. P., Birmaher, B., Pincus, H. A., et al. (2007). Clinical response and risk for reported suicidal ideation and suicide attempts in pediatric antidepressant treatment. *The Journal of the American Medical Association, 297*, 1683–1696.

Canton, J., Scott, K. M., & Glue, P. (2012). Optimal treatment of social phobia: A systematic review and meta-analysis. *Neuropsychiatric Disease and Treatment, 8*, 203–215.

Clark, D., & Agras, W. S. (1991). The assessment and treatment of performance anxiety in musicians. *American Journal of Psychiatry, 148*, 598–605.

Clark, D. M., Ehlers, A., McManus, F., Hackmann, A., Fennell, M., Campbell, H., et al. (2003). Cognitive therapy versus fluoxetine in generalized social phobia: A randomized placebo-controlled trial. *Journal of Consulting and Clinical Psychology, 71*, 1058–1067.

Connor, K. M., Cook, J. L., & Davidson, J. R. (2006). Botulinum toxin treatment of social anxiety disorder with hyperhidrosis: A placebo-controlled double-blind trial. *Journal of Clinical Psychiatry, 67*, 30–36.

Connor, K. M., Davidson, J. R., Potts, N. L., Tupler, L. A., Miner, C. M., Malik, M. L., et al. (1998). Discontinuation of clonazepam in the treatment of social phobia. *Journal of Clinical Psychopharmacology, 18*, 373–378.

Davidson, J. R., Foa, E. B., Huppert, J. D., Keefe, F. J., Franklin, M. E., Compton, J. S., et al. (2004). Fluoxetine, comprehensive cognitive behavioral therapy, and placebo in generalized social phobia. *Archives of General Psychiatry, 61*, 1005–1013.

Davidson, J. R., Potts, N., Richichi, E., Krishnan, R., Ford, S. M., Smith, R., et al. (1993). Treatment of social phobia with clonazepam and placebo. *Journal of Clinical Psychopharmacology, 13*, 423–428.

Davidson, J. R., Yaryura-Tobias, J., DuPont, R., Stallings, L., Barbato, L. M., van der Hoop, R. G., et al. (2004). Fluvoxamine-controlled release formulation for the treatment of generalized social anxiety disorder. *Journal of Clinical Psychopharmacology, 24*, 118–125.

Donahue, C. B., Kushner, M. G., Thuras, P. D., Murphy, T. G., Van Demark, J. B., & Adson, D. E. (2009). Effect of Quetiapine vs. placebo on response to two virtual public speaking exposures in individuals with social phobia. *Journal of Anxiety Disorders, 23*, 362–368.

Dunlop, B. W., Papp, L., Garlow, S. J., Weiss, P. S., Knight, B. T., & Ninan, P. T. (2007). Tiagabine for social anxiety disorder. *Human Psychopharmacology, 22*, 241–244.

Emmanuel, N. P., Lydiard, R. B., & Ballenger, J. C. (1991). Treatment of social phobia with bupropion. *Journal of Clinical Psychopharmacology, 11*, 276–277.

Fahlen, T., Nilsson, H. L., Bog, K., Humble, H., & Pauli, U. (1995). Social phobia: The clinical efficacy and tolerability of the monoamine oxidase-A and serotonin uptake inhibitor brofaromine. *Acta Psychiatrica Scandinavica, 92*, 351–358.

Feltner, D. E., Liu-Dumaw, M., Schweizer, E., & Bielski, R. (2011). Efficacy of pregabalin in generalized social anxiety disorder: Results of a double-blind, placebo-controlled, fixed-dose study. *International Clinical Psychopharmacology, 26*, 213–220.

Furmark, T., Appel, L., Michelgård, A., Wahlstedt, K., Ahs, F., Zancan, S, et al. (2005). Cerebral blood flow changes after treatment of social phobia with the neurokinin-1 antagonist GR205171, citalopram, or placebo. *Biological Psychiatry, 58*, 132–142.

Gelernter, C. S., Uhde, T. W., Cimbolic, P., Arnkoff, D. B., Vittone, B. J., Tancer, M. E., et al. (1991). Cognitive-behavioral and pharmacological treatments of social phobia: A controlled study. *Archives of General Psychiatry, 48*, 938–945.

Gibbons, R., Hur, K., Bhaumik, D. K., & Mann, J. J. (2006). The relationship between antidepressant prescription rates and rate of early adolescent suicide. *American Journal of Psychiatry, 163*, 1898–1904.

Ginsburg, G. S., Kendall, P. C., Sakolsky, D., Compton, S. N., Piacentini, J., Albano, A., et al. (2011). Remission after acute treatment in children and adolescents with anxiety disorders: Findings from the CAMS. *Journal of Consulting and Clinical Psychology, 76*, 806–813.

Graae, F., Milner, J., Rizzotto, L., & Klein, R. G. (1994). Clonazepam in childhood anxiety disorders. *Journal of the American Academy of Children & Adolescent Psychiatry, 33*, 372–376.

Greist, J. H., Liu-Dumaw, M., Schweizer, E., & Feltner, D. (2011). Efficacy of pregabalin in preventing relapse in patients with generalized social anxiety disorder: Results of a double-blind, placebo-controlled 26-week study. *International Clinical Psychopharmacology, 26*, 243–251.

Hansen, R. A., Gaynes, B. N., Gartlehner, G., Moore, C. G., Tiwari, R., & Lohr, K. N. (2008). Efficacy and tolerability of second-generation antidepressants in social anxiety disorder. *International Clinical Psychopharmacology, 23*, 170–179.

Hartley, L. R., Ungapen, S., Davie, I., & Spencer, D. J. (1983). The effect of beta adrenergeric blocking drugs on speakers' performance and memory. *British Journal of Psychiatry, 142*, 512–517.

Haug, T. T., Blomhoff, S., Hellstrom, K., Holme, I, Humble, M, Madsbu, H. P., et al. (2003). Exposure therapy and sertraline in social phobia: 1-year follow-up of a randomized controlled trial. *British Journal of Psychiatry, 182*, 312–318.

Hedges, D. W., Brown, B. L., Shwalb, D. A., Godfrey, K., & Larcher, A. M. (2007). The efficacy of selective serotonin reuptake inhibitors in adult social anxiety disorder: A meta-analysis of double-blind, placebo-controlled trials. *Journal of Psychopharmacology, 21*, 102–111.

Heimberg, R. G., Liebowitz, M. R., Hope, D. A., Schneier, F. R., Holt, C. S., Welkowitz, L. A, et al. (1998). Cognitive-behavioral group therapy vs phenelzine therapy for social phobia. *Archives of General Psychiatry, 55*, 1133–1141.

Kasper, S., Stein, D. J., Loft, H., & Nil, R. (2005). Escitalopram in the treatment of social anxiety disorder: Randomised, placebo-controlled, flexible-dosage study. *The British Journal of Psychiatry, 186*, 222–226.

Katschnig, H., Stein, M. B., & Buller, R. (1997). Moclobemide in social phobia. A double-blind, placebo-controlled clinical study. *European Archives of Psychiatry and Clinical Neuroscience, 247*, 71–80.

Katzelnick, D. J., Kobak, K. A., Greist, J. H., Jefferson, J. W., Mantle, J. M., & Serlin, R. C. (1995). Sertraline for social phobia: A double-blind, placebo-controlled cross over study. *American Journal of Psychiatry, 152*, 1368–1371.

Knijnik, D. Z., Blanco, C., Salum, G., Moraes, C. U., Mombvach, C., Almeida, E., et al. (2008). A pilot study of clonazepam versus psychodynamic group therapy plus clonazepam in the treatment of generalized social anxiety disorder. *European Psychiatry, 23*, 567–574.

Kobak, K. A., Griest, J. H., Jefferson, J. W., & Katzelnick, D. J. (2002). Fluoxetine in social phobia: A double-blind placebo controlled pilot study. *Journal of Clinical Psychopharmacology, 22*, 257–262.

Lader, M., Stender, K., Burger, V., & Nil, R. (2004). Efficacy and tolerability of escitalopram in 12- and 24-week treatment of social anxiety disorder: Randomised, double-blind, placebo-controlled, fixed-dose study. *Depression and Anxiety, 19*, 241–248.

Lepola, U., Bergtholdt, B., St. Lambert, J., Davy, K. L., & Ruggiero, L. (2004). Controlled-release paroxetine in the treatment of patients with social anxiety disorder. *Journal of Clinical Psychiatry, 65*, 222–229.

Liebowitz, M. R. (1987). Social phobia. *Modern Problems of Pharmacopsychiatry, 22*, 141–173.

Liebowitz, M. R., DeMartinis, N. A., Weihs, K., Londborg, P. D., Smith, W. T., Chung, H., et al. (2003). Efficacy of sertraline in severe generalized social anxiety disorder: Results of a double-blind, placebo-controlled study. *Journal of Clinical Psychiatry, 64*, 785–792.

Liebowitz, M. R., Gelenberg, A. J., & Munjack, D. (2005). Venlafaxine extended release vs placebo and paroxetine in social anxiety disorder. *Archives of General Psychiatry, 62*, 190–198.

Liebowitz, M. R., Gorman, J. M., Fyer, A. J., & Klein, D. F. (1985). Social phobia. Review of a neglected anxiety disorder. *Archives of General Psychiatry, 42*, 729–736.

Liebowitz, M. R., Heimberg, R. G., Schneier, F. R., Hope, D. A., Davies, S., Holt, C. S., et al. (1999). Cognitive-behavioral group therapy versus phenelzine in social phobia: Long-term outcome. *Depression and Anxiety, 10*, 89–98.

Liebowitz, M. R., Mangano, R. M., Bradwejn, J., & Asnis, G. (2005). A randomized controlled trial of venlafaxine extended release in generalized social anxiety disorder. *Journal of Clinical Psychiatry, 66*, 238–247.

Liebowitz, M. R., Schneier, F. R., Campeas, R., Hollander, E., Hatterer, J., Fyer, A., et al. (1992). Phenelzine vs atenolol in social phobia: A placebo-controlled comparison. *Archives of General Psychiatry, 49*, 290–300.

Liebowitz, M. R., Stein, M. B., Tancer, M., Carpenter, D., Oakes, R., & Pitts, C. D. (2002). A randomized, double-blind, fixed-dose comparison of paroxetine and placebo in the treatment of generalized social anxiety disorder. *Journal of Clinical Psychiatry, 63*, 66–74.

Lott, M., Greist, J., Jefferson, J. W., Kobak, K. A., Katzelnick, D. J., Katz, R. J., et al. (1997). Brofaromine for social phobia: A multicenter, placebo-controlled, double-blind study. *Journal of Clinical Psychopharmacology, 17*, 255–260.

March, J. S., Entusah, A. R., Rynn, M., Albano, A. M., & Tourian, K. A. (2007). A randomized controlled trial of venlafaxine ER versus placebo in pediatric social anxiety disorder. *Biological Psychiatry, 62*, 1149–1154.

Montgomery, S. A., Nil, R., Durr-Pal, N., Loft, H., & Boulenger, J. P. (2005). A 24-week randomized, double-blind, placebo-controlled study of escitalopram for the prevention of generalized social anxiety disorder. *Journal of Clinical Psychiatry, 66*, 1270–1278.

Muehlbacher, M., Nickel, M. K., Nickel, C., Kettler, C., Lahmann, C., Pedrosa, F., et al. (2005). Mirtazapine treatment of social phobia in women: A randomized, double-blind, placebo-controlled study. *Journal of Clinical Psychopharmacology, 25*, 580–583.

Munjack, D. J., Baltazar, P. L., Bohn, P. B., Cabe, D. D., & Appleton, A. A. (1990). Clonazepam in the treatment of social phobia: A pilot study. *Journal of Clinical Psychiatry, 51*, 35–53.

Nardi, A. E., Lopes, F. L., Valenca, A. M., Freire, R. C., Nascimento, I., Veras, A. B., et al. (2010). Double-blind comparison of 30 and 60 mg tranylcypromine daily in patients

with panic disorder comorbid with social anxiety disorder. *Psychiatry Research, 175*, 260–265.

Neftel, K. A., Adler, R. H., Kappeli, L., Rossi, M., Dolder, M., Kaser, H. E.,... Vorkauf, H. (1982). Stage fright in musicians: A model illustrating the effect of beta blockers. *Psychosomatic Medicine, 44*, 461–469.

Noyes, R., Moroz, G., Davidson, J., Liebowitz, M. R., Davidson, A., Siegel, J., et al. (1997). Moclobemide in social phobia: A controlled dose-response. *Journal of Clinical Psychopharmacology, 17*, 247–254.

Ontiveros, J. A. (2008). Double-blind controlled study with clonazepam and placebo in social anxiety disorder. *Salud Mental, 31*, 299–306.

Pallanti, S., & Quercioli, L. (2006). Resistant social anxiety disorder response to escitalopram. *Clinical Practice and Epidemiology in Mental Health, 2*, 35.

Pande, A. C., Davidson, R. T., Jefferson, J. W., Janney, C. A., Katzelnick, D. J., Weisler, R. H., et al. (1999). Treatment of social phobia with gabapentin: A placebo controlled study. *Journal of Clinical Psychopharmacology, 19*, 341–348.

Pande, A. C., Feltner, D. E., Jefferson, J. W., Davidson, J. R., Pollack, M., Stein, M. B., et al. (2004). Efficacy of the novel anxiolytic pregabalin in social anxiety disorder: A placebo-controlled, multicenter study. *Journal of Clinical Psychopharmacology, 24*,141–149.

Rickels, K., Mangano, R., & Khan, A. (2004). A double-blind, placebo-controlled study of a flexible dose of venlafaxine ER in adult outpatients with generalized social anxiety disorder. *Journal of Clinical Psychopharmacology, 24*, 488–496.

Rynn, M. A., Siqueland, L., & Rickels, K. (2001). Placebo-controlled trial of sertraline in the treatment of children with generalized anxiety disorder. *American Journal of Psychiatry, 158*, 2008–2014.

Scahill, L., Leckman, J. F., Schultz, R. T., Katsovich, L., & Peterson, B. S. (2003). A placebo-controlled trial of risperidone in Tourette syndrome. *Neurology, 60*, 1130–1135.

Schneier, F. R., Blanco, C., Campeas, R., Lewis-Fernandez, R., Lin, S. H., Marshall, R., et al. (2003). Citalopram treatment of social anxiety disorder and comorbid major depression. *Depression and Anxiety, 17*, 191–196.

Schneier, F. R., Goetz, D., Campeas, R., Marshall, R., & Liebowitz, M. R. (1998). A placebo-controlled trial of moclobemide in social phobia. *The British Journal of Psychiatry, 172*, 70–77.

Schneier, F. R., Liebowitz, M. R., Garfinkel, R., Campeas, R., Fallon, B., Gitow, A., et al. (1994). Disability in work and social functioning and social phobia. *Journal of Clinical Psychiatry, 55*, 322–331.

Schneier, F. R., Saoud, J. B., Campeas, R., Fallon, B. A., Hollander, E., Coplan, J., et al. (1993). Buspirone in social phobia. *Journal of Clinical Psychopharmacology, 13*, 251–256.

Schutters, S. I., Van Megen, H. J., Van Veen, J. F., Denys, D. A., & Westenberg, H. G. (2010). Mirtazapine in generalized social anxiety disorder: A randomized, double-blind, placebo-controlled study. *International Journal of Clinical Psychopharmacology, 25*, 302–304.

Schutters, S. I., van Megen, H. J., & Westenberg, H. G. (2005). Efficacy of quetiapine in generalized social anxiety disorder: Results from an open-label study. *Journal of Clinical Psychiatry, 66*, 540–542.

Seedat, S., & Stein, M. B. (2004). Double-blind, placebo-controlled assessment of combined clonazepam with paroxetine compared with paroxetine monotherapy for generalized social anxiety disorder. *Journal of Clinical Psychiatry, 65*, 244–248.

Simeon, J. G., Ferguson, H. B., Knott, V., Roberts, N., Gauthier, B., Dubois, C., et al. (1992). Clinical, cognitive and neurophysiological effects of alprazolam in children and adolescents with overanxious and avoidant disorders. *Journal of the American Academy of Children & Adolescent Psychiatry, 31*, 29–33.

Simon, N. M., Hoge, E. A., Fischmann, D., Worthington, J. J., Christian, K. M., Kinrys, G., et al. (2006). An open-label trial of risperidone augmentation for refractory anxiety disorders. *Journal of Clinical Psychiatry, 67*, 381–385.

Simpson, H. B., Schneier, F. R., Campeas, R. B., Marshall, R. D., Fallon, B. A., Davis, S., et al. (1998). Imipramine in the treatment of social phobia. *Journal of Clinical Psychopharmacology, 18*, 132–135.

Stein, D. J., Cameron, A., Amrein, R., & Montgomery, S. A. (2002). Moclobemide is effective and well tolerated in the long-term pharmacotherapy of social anxiety disorder with or without comorbid anxiety disorder. *International Clinical Psychopharmacology, 17*, 161–170.

Stein, D. J., Stein, M. B., Pitts, C. D., Kumar, R., & Hunter, B. (2002). Predictors of response to pharmacotherapy in social anxiety disorder, an analysis of 3 placebo-controlled paroxetine trial. *Journal of Clinical Psychiatry, 63*, 152–155.

Stein, D. J., Versiani, M., Hair, T., & Kumar, R. (2002). Efficacy of paroxetine for relapse prevention in social anxiety disorder: A 24-week study. *Archives of General Psychiatry, 59*, 1111–1118.

Stein, M. B., Fyer, A. J., Davidson, J. R., Pollack, M. H., & Wiita, B. (1999). Fluvoxamine treatment of social phobia (social anxiety disorder): A double-blind placebo-controlled study. *American Journal of Psychiatry, 156*, 756–760.

Stein, M. B., Liebowitz, M. R., Lydiard, R. B., Pitts, C. D., Bushnell, W., & Gergel, I. (1998). Paroxetine treatment of generalized social phobia (social anxiety disorder): A randomized, double-blind, placebo-controlled study. *Journal of the American Medical Association, 280*, 708–713.

Stein, M. B., Pollack, M. H., Bystritsky, A., Kelsey, J. E., & Mangano, R. M. (2005). Efficacy of low and higher dose extended-release venlafaxine in generalized social anxiety disorder: A 6-month randomized controlled trial. *Psychopharmacology, 177*, 280–288.

Stein, M. B., Sareen, J., Hami, S., & Chao, J. (2001). Pindolol potentiation of paroxitine for generalized social phobia. A double-blind, placebo-controlled, cross-over study. *American Journal of Psychiatry, 158*, 1725–1727.

Turner, S. M., Beidel, D. C., & Jacob, R. G. (1994). Social phobia: A comparison of behavior therapy and atenolol. *Journal of Consulting Clinical Psychology, 62*, 350–358.

Vaishnavi, S., Alamy, S., Zhang, W., Connor, K. M., & Davidson, J. R. (2007). Quetiapine as monotherapy for social anxiety disorder: A placebo-controlled study. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 31*, 1464–1469.

Van Ameringen, M. A., Lane, R. M., Walker, J. R., Bowen, R. C., Chokka, P. R., Golderner, E. M., et al. (2001). Sertraline treatment of generalized social phobia: A 20-week double-blind, placebo-controlled study. *American Journal of Psychiatry, 158*, 275–281.

Van Ameringen, M. A., Mancini, C., Farvolden, P., & Oakman, J. (2000). Drugs in development for social anxiety disorder: More to social anxiety than meets SSRI. *Expert Opinion on Investigational Drugs, 9*, 2215–2231.

Van Ameringen, M. A., Mancini, C., Oakman, J., Walker, J., Kjernisted, K., Chokka, P. R., et al. (2007). Nefazodone in the treatment of generalized social phobia: A randomized, placebo-controlled trial. *Journal of Clinical Psychiatry, 68*, 288–295.

Van Ameringen, M. A., Mancini, C., Pipe, B., Oakman, J., & Bennett, M. (2004). An open trial of topiramate in the treatment of generalized social phobia. *Journal of Clinical Psychiatry, 65*, 1674–1678.

Van Ameringen, M. A., Mancini, C., & Wilson, C. (1996). Buspirone augmentation of selective serotonin reuptake inhibitors (SSRIs) in social phobia. *Journal of Affective Disorders, 39*, 115–121.

van Vliet, I. M., den Boer, J. A., Westenberg, G. A., & Ho Pian, K. L. (1997). Clinical effects of buspirone in social phobia. A double-blind, placebo-controlled study. *Journal of Clinical Psychiatry, 58*, 164–168.

van Vliet, I. M., den Boer, J. A., & Westenberg, H. G. (1992). Psychopharmacological treatment of social phobia: Clinical and biochemical effects of brofaromine, a selective MAO-A inhibitor. *European Neuropsychopharmacology, 2*, 21–29.

van Vliet, I. M., den Boer, J. A., & Westenberg, H. G. (1994). Psychopharmacological treatment of social phobia: A double blind controlled study with fluvoxamine. *Psychopharmacology, 115*, 128–134.

van Vliet, I. M., Westenberg, H. G., & Den Boer, J. A. (1993). MAO inhibitors in panic disorder: Clinical effects of treatment with brofaromine: A double blind placebo controlled study. *Psychopharmacology, 112*, 483–489.

Versiani, M., Amrein, R., & Montgomery, S. A. (1997). Social phobia: Long-term treatment outcome and prediction of response–A moclobemide study. *International Clinical Psychopharmacology, 12*, 239–254.

Versiani, M., Nardi, A. E., Figueira, I., Mendlowicz, M., & Marques, C. (1997). Double-blind placebo controlled trials with bromazepam. *Jornal Brasileiro de Psiquiatria, 46*, 167–171.

Versiani, M., Nardi, A. E., Mundim, F. D., Alves, A. B., Liebowitz, M. R., & Amrein, R. (1992). Pharmacotherapy of social phobia: A controlled study with moclobemide and phenelzine. *The British Journal of Psychiatry, 161*, 353–360.

Wagner, K. D., Berard, R., Stein, M. B., Wetherhold, E., Carpenter, D. J., Perera, P., et al. (2004). A multicenter, randomized, double blind, placebo-controlled trial of Paroxetine in children and adolescents with social anxiety disorder. *Archives of General Psychiatry, 61*, 1153–1162.

Walker, J. R., Van Ameringen, M. A., Swinson, R., Bowen, R. C., Chokka, P. R., Goldner, E., et al. (2000). Prevention of relapse in generalized social phobia: Results of a 24-week study in responders to 20 weeks of sertraline treatment. *Journal of Clinical Psychopharmacology, 20*, 636–644.

Walkup, J. T., Albano, A. M., Piacentini, J., Birmaher, B., Compton, S. N., Sherrill, J. T., et al. (2008). Cognitive behavioral therapy, sertraline, or a combination in childhood anxiety. *New England Journal of Medicine, 359*, 2753–2766.

Westenberg, H. G., Stein, D. J., Yang, H., Li, D., & Barbato, L. (2004). A double-blind placebo-controlled study of controlled release fluvoxamine for the treatment of generalized social anxiety disorder. *Journal of Clinical Psychopharmacology, 24*, 49–55.

Williams, T. P., & Miller, B. D. (2003). Pharmacologic management of anxiety disorders in children and adolescents. *Current Opinion in Pediatrics, 15*, 483–490.

Zaider, T. I., Heimberg, R. G., Fresco, D. M., Schneier, F. R., & Liebowitz, M. R. (2003). Evaluation of the clinical global impression scale among individuals with social anxiety disorder. *Psychological Medicine, 33*, 611–622.

Zhang, W., Connor, K. M., & Davidson, J. R. (2005). Levetiracetam in social phobia: A placebo controlled pilot study. *Journal of Psychopharmacology, 19*, 551–553.

25

Dual Diagnosis Cases

Treating Comorbid Social Anxiety Disorder and Substance Abuse or Dependence

Julia D. Buckner
Louisiana State University, USA

Individuals with elevated social anxiety appear especially vulnerable to experiencing substance-related problems including substance use disorders. The co-occurrence of social anxiety disorder (SAD) with substance use disorders is related to greater distress and impairment than either disorder alone. Further, this comorbidity is associated with poorer treatment outcomes. Thus, researchers have begun to develop and evaluate personalized treatments specifically for these dually diagnosed patients. Although outcomes are promising, there remain many challenges to treating these patients.

The majority of research in this area has focused on the relations between social anxiety and alcohol-related impairment (for recent reviews see Battista, Stewart, & Ham, 2010; Tran & Smith, 2008). This chapter will extend extant reviews by reviewing the literature on tobacco use and illicit substance use disorders in addition to alcohol use. Further, the focus of this chapter is on ways in which the extant literature informs treatment recommendations. This chapter provides an overview of the scope of the problem (i.e., high rates of comorbid SAD and substance use disorders, poor treatment response of these patients), reviews the extant literature on treatment outcomes for treatments developed for and tested with SAD patients with substance use disorders, and provides treatment recommendations and suggestions for additional work in this area.

Rates of Comorbid SAD and Substance Use Disorders

Social anxiety is related to substance-related problems. In the National Epidemiologic Survey on Alcohol and Related Conditions, 48% of individuals with a lifetime

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

diagnosis of SAD also met criteria for a lifetime diagnosis of an alcohol-use disorder (Grant et al., 2005). The 12-month prevalence of alcohol-use disorders among individuals with SAD was 13.1% (Grant et al., 2005) compared to 8.5% among the general population (Grant, Dawson, et al., 2004). In the National Comorbidity Survey, SAD was associated with higher rates of alcohol-use disorders relative to most other anxiety disorders (Kessler, Crum, et al., 1997) and remained related to alcohol-use disorders after controlling for other Axis I disorders (Buckner, Timpano, Zvolensky, Sachs-Ericsson, & Schmidt, 2008). SAD, but not other anxiety disorders or depression, remained significantly, prospectively related to onset of alcohol dependence in multivariate analyses (Buckner, Schmidt, et al., 2008). Among college students, a group at particular risk for substance-related problems (see Larimer, Kilmer, & Lee, 2005), social anxiety is positively correlated with alcohol-related problems (e.g., Buckner, Ecker, & Proctor, 2011; Buckner, Eggleston, & Schmidt, 2006; Buckner & Heimberg, 2010; Buckner & Matthews, 2012; Buckner & Schmidt, 2009c; Gilles, Turk, & Fresco, 2006; Lewis & O'Neill, 2000; Lewis et al., 2008; Stewart, Morris, Mellings, & Komar, 2006).

Although less work has investigated the relations between social anxiety and illicit substance use disorders, emerging data suggest that socially anxious individuals are especially vulnerable to cannabis-related impairment. In the National Comorbidity Survey, 29% of individuals with lifetime cannabis dependence met criteria for a lifetime diagnosis of SAD, a rate higher than other anxiety disorders whose rates ranged from 6.9% (panic disorder) to 18.5% (PTSD) (Agosti, Nunes, & Levin, 2002). Relative to adolescents without SAD, those with SAD were nearly five times more likely to develop cannabis dependence as young adults after controlling for other internalizing and externalizing disorders (Buckner, Schmidt, et al., 2008). SAD appears more strongly related to cannabis dependence than abuse (Buckner, Heimberg, Schneier, et al., 2012; Buckner, Schmidt, et al., 2008). Among cannabis users, SAD was also related to transition from first use to cannabis-related problems among male users (Buckner, Heimberg, Schneier, et al., 2012; Marmorstein, White, Loeber, & Stouthamer-Loeber, 2010). The relation between SAD and cannabis dependence and cannabis-related problems remains robust after controlling for other relevant Axis I disorders (Buckner, Heimberg, Schneier, et al., 2012; Buckner & Schmidt, 2009b; Buckner, Schmidt, et al., 2008).

Social anxiety may also be related to tobacco smoking and nicotine dependence. In the National Comorbidity Survey Replication, SAD was associated with greater risk of nicotine dependence, current and lifetime smoking, and unsuccessful quit attempts relative to those without SAD after controlling for co-occurring anxiety disorders, depression, and other substance use disorders (Cougle, Zvolensky, Fitch, & Sachs-Ericsson, 2010). Among adolescents and young adults, the relation between social fears and nicotine dependence remained after controlling for depression (Sonntag, Wittchen, Hofler, Kessler, & Stein, 2000). However, it appears as though individuals with anxiety disorders generally (rather than those with SAD specifically) suffer from co-occurring nicotine dependence, as individuals with SAD report rates of nicotine dependence that are comparable to or somewhat lower than individuals with some other anxiety disorders (e.g., panic disorder, generalized anxiety disorder [GAD], PTSD) (Cougle et al., 2010; Grant, Hasin, Chou, Stinson, & Dawson, 2004; McCabe et al., 2004).

Rates of Comorbid SAD and Substance Use Disorders: Treatment Populations

Rates of co-occurring social anxiety and substance use disorders are even higher among treatment-seeking populations. Approximately 25% of patients in alcohol treatment have clinically elevated social anxiety (Kushner et al., 2005; Terlecki, Buckner, Larimer, & Copeland, 2011; Terra et al., 2006; Thomas, Thevos, & Randall, 1999), and 55% of patients seeking detoxification for alcohol dependence demonstrate at least moderate social anxiety (Liappas, Paparrigopoulos, Tzavellas, & Christodoulou, 2003). Among patients seeking intensive outpatient treatment of substance dependence (alcohol and/or illicit substance dependence), 37% exhibited clinically elevated social anxiety (Book, Thomas, Dempsey, Randall, & Randall, 2009). Among those seeking smoking cessation treatment, patients with SAD reported higher levels of nicotine dependence than those with no history of anxiety disorder (Piper, Cook, Schlam, Jorenby, & Baker, 2011). In fact, level of nicotine dependence among patients with SAD was comparable to those reported among patients with histories of panic attacks or GAD.

Temporal Relations

Social anxiety tends to onset prior to substance use problems. Typical age of onset of SAD precedes that of alcohol-use disorders (e.g., Buckner, Timpano, et al., 2008; Courbasson & Nishikawa, 2010; Kessler, Anthony, et al., 1997; Randall, Johnson, et al., 2001; Randall, Thomas, & Thevos, 2001; Schneier, Martin, Liebowitz, Gorman, & Fyer, 1989). Prospective data also suggest that social anxiety is related to greater risk of alcohol-use disorders. In a 13-year longitudinal investigation (Crum & Pratt, 2001), individuals with subclinical SAD (i.e., excessive fear of social situations without clinically significant avoidance or impairment) showed a greater risk for alcohol-use disorders relative to individuals without a history of social fears. Subclinically elevated fear of scrutiny also prospectively predicted onset of alcohol-use disorders (Buckner & Schmidt, 2009c). SAD in adolescence predicted greater rates of alcohol dependence by age 30 (Buckner, Schmidt, et al., 2008). Among young women, SAD (but not other anxiety disorders, depression, or conduct disorder) was related to developing alcohol-use disorders over a 3-year period in a sample that was predominately Hispanic/Latina and/or African American (Buckner & Turner, 2009).

SAD onset also appears to occur prior to onset of other substance use disorders. SAD in adolescence predicted greater rates of cannabis dependence by age 30 (Buckner, Schmidt, et al., 2008), and the majority of SAD individuals with comorbid cannabis-use disorder report SAD onset prior to the cannabis-use disorder (Buckner, Heimberg, Schneier, et al., 2012). Nearly all patients with comorbid SAD and cocaine dependence reported SAD onset prior to cocaine dependence (Myrick & Brady, 1997). Epidemiological data indicate that over 80% of nicotine users with SAD report that SAD was present prior to smoking initiation (Cougle et al., 2010). Social anxiety also

appears to be related to greater odds of developing nicotine dependence (Sonntag et al., 2000).

Public Health Implications of Co-occurring Social Anxiety and Substance Use Disorders

The high rate of co-occurrence between social anxiety and substance-related problems is a substantial concern given that this co-occurrence is associated with greater impairment than either condition alone. Among treatment-seeking patients with an alcohol-use disorder, those with a lifetime history of SAD experienced more severe alcohol dependence and reported more major depressive episodes, less peer social support, and lower occupational status than alcohol-use disorder patients without SAD (Thevos, Thomas, & Randall, 1999; Thomas et al., 1999). Similarly, relative to SAD patients with no history of alcohol-use disorders, SAD patients with a history of alcohol-use disorders exhibit more severe SAD, greater psychiatric comorbidity, more health problems, and greater interpersonal functioning deficits (Buckner, Timpano, et al., 2008; Schneier et al., 1989; Schneier et al., 2010). Among patients receiving inpatient treatment for alcohol dependence, clinically elevated social anxiety is related to greater history of suicide attempt (Evren, Sar, Dalbudak, Oncu, & Cakmak, 2009).

The co-occurrence of SAD with illicit substance use disorders is also related to greater impairment than either disorder alone. Among persons with a cannabis-use disorder, those with comorbid SAD were less educated, reported lower incomes and poorer physical health, were likely to require financial assistance, used more illicit (non-cannabis) drugs, and were more likely to suffer from additional psychiatric disorders (Buckner, Heimberg, Schneier, et al., 2012). Among daily cannabis users (but not less frequent users), higher social anxiety is related to greater suicidality (Buckner, Joiner, Schmdit, & Zvolensky, 2012). Among those with cocaine dependence, those with comorbid SAD were less likely to be married and more likely to suffer from a co-occurring Axis I disorder and to experience greater depression, suicidal ideation, paranoia related to use, and polysubstance use compared to those without SAD (Myrick & Brady, 1997).

The greater impairment and distress associated with the co-occurrence of SAD with substance use disorders represents an important public health concern. Thus, efforts to improve prevention and treatment protocols for these patients remain an important goal with clear public health benefits.

Patients with Comorbid SAD and Substance Use Disorders Have Poorer Treatment Outcomes

Unfortunately, the impact of substance use and use-related problems on SAD treatment has received very little empirical attention. Most randomized control trials of SAD treatment exclude patients with substance use disorders, presumably because

researchers believe that the co-occurrence of substance use disorders will negatively impact outcomes. The impact of social anxiety on treatment for substance use disorders has received some attention and research suggests it can negatively impact treatment adherence as well as outcomes.

Social Anxiety Related to Poorer Treatment Adherence

Patients with comorbid SAD were less likely to participate in 12-step groups following outpatient treatment for cocaine dependence (Myrick & Brady, 1997). Anxiety sensitivity (i.e., fear of the consequences of anxiety-like sensations) is higher among patients who prematurely terminate from court-mandated residential treatment for heroin and/or crack/cocaine dependence (Lejuez et al., 2008). Notably, those with higher anxiety sensitivity-social concerns (i.e., fears regarding the social consequences of anxiety) were more likely to prematurely terminate than those who did not have these social fears. In fact, social concerns (but not cognitive or physical concerns) were significantly, incrementally related to drop-out after controlling for contract duration (i.e., treatment duration determined at admission), legal obligation, alcohol frequency, and depressive symptoms.

Anxiety Is Related to Greater Relapse

Although comorbid anxiety disorders are related to greater rates of relapse following treatment for alcohol-use disorders (Driessen et al., 2001; Kushner et al., 2005), SAD was the only anxiety disorder to uniquely predict relapse to drinking after completing an inpatient alcohol treatment program (Kushner et al., 2005). Subclinically elevated social anxiety is also associated with greater relapse following alcohol detoxification programs (Hull, Young, & Jouriles, 1986). Among heavy-drinking students undergoing *Brief Alcohol Screening and Intervention for College Students* (BASICS; Dimeff, Baer, Kivlahan, & Marlatt, 1999), clinically elevated social anxiety is related to heavier drinking at baseline and follow-up (Terlecki et al., 2011). In fact, at follow-up, those with clinically elevated social anxiety reported drinking, on average, twice the number of drinks during a typical drinking period relative to those with lower social anxiety.

Anxiety Remains at Clinical Levels Following Substance Use Disorder Treatment

Patients with anxiety disorders continue to experience clinically elevated anxiety following an alcohol detoxification program consisting of group cognitive behavioral therapy (CBT) for alcohol-related problems plus a 3-week inpatient motivation-based treatment (Driessen et al., 2001). This elevated anxiety may play a role in difficulty maintaining abstinence of these patients if they use alcohol to help them cope with their elevated anxiety.

Mechanisms Underlying Poorer Treatment Outcomes

Researchers have begun to investigate reasons for poorer outcomes among patients with co-occurring social anxiety and substance-related problems. Three factors appear especially relevant to socially anxious patients: (1) fear of participating in treatment, (2) use to cope with negative affect, and (3) socially motivated use.

Performance Fears

Socially anxious patients report that their "shyness" interferes with their ability to participate in the various aspects of treatment for substance use disorders, including talking to a therapist, talking in group treatment, attending alcoholics or narcotics anonymous (AA/NA) meetings, and asking for an AA/NA sponsor (Book et al., 2009). Thus, the nature of social anxiety itself may place comorbid patients at risk of not benefiting from treatment for substance use disorders if their fear of negative evaluation leads to less participation in available treatments.

Coping-Motivated Use

Social anxiety is related to using substances in situations involving negative affect (Buckner et al., 2006) and explicitly to cope with negative affect (Buckner, Bonn-Miller, Zvolensky, & Schmidt, 2007; Lewis et al., 2008; Stewart et al., 2006). Socially anxious individuals are also more likely to use substances to cope in social situations and to avoid social situations if substances are not available (Buckner & Heimberg, 2010; Buckner, Heimberg, Matthews, & Silgado, 2012; Thomas, Randall, & Carrigan, 2003; Watson, VanderVeen, Cohen, DeMarree, & Morrell, 2012). Coping-motivated use at least partially accounts for the relations between social anxiety and substance-related problems (Buckner & Heimberg, 2010; Buckner, Heimberg, Matthews, et al., 2012; Buckner, Zvolensky, & Schmidt, 2012; Lewis et al., 2008; Stewart et al., 2006), suggesting that patients who continue to rely on substances to manage anxiety may be at greater risk for relapse and use-related problems.

Socially Motivated Use

Individuals with elevated social anxiety may also use substances to avoid scrutiny from substance-using peers and/or because they believe substances such as alcohol, cannabis, and/or cigarettes are a common (and thus socially acceptable) strategy for dampening state social anxiety. In partial support of this hypothesis, social anxiety moderated the relation between normative beliefs (i.e., beliefs about how much alcohol peers consume) and drinking behaviors, such that among students with higher (but not lower) social anxiety, normative beliefs were positively related to drinking (Neighbors et al., 2007). Elevated social anxiety is also related to consuming alcohol in response to social pressure to drink (Buckner et al., 2006) as well as to avoid social scrutiny and to socialize (Stewart et al., 2006).

Thus, socially anxious patients may not respond as well to cognitive restructuring because of fear that they may be evaluated for not conforming to what they believe are normative substance use behaviors. Terlecki, Buckner, Larimer, & Copeland, (2012) tested whether change in normative beliefs was related to BASICS outcomes (see *Anxiety Is Related to Greater Relapse* above) among heavy-drinking socially anxious patients. Higher social anxiety was related to less change in normative beliefs after BASICS. Change in normative beliefs moderated the relation between social anxiety and follow-up drinking. Specifically, among students with smaller change in normative beliefs after BASICS, higher social anxiety was related to heavier drinking at 1-month follow-up. These findings suggest that socially anxious patients may be less responsive to cognitive restructuring efforts aimed at changing normative beliefs, indicating a need for additional work aimed at improving these techniques with these patients.

Treatment for Co-occurring SAD and Substance Use Disorders

Despite the high rates of co-occurring SAD and substance use disorders, very little empirical attention has been directed toward treatment when these conditions co-occur. One known study examined the utility of SAD treatment with patients seeking treatment for SAD who also had an alcohol-use disorder and reported that they use alcohol to cope with their anxiety (Book, Thomas, Randall, & Randall, 2008; Thomas, Randall, Book, & Randall, 2008). Patients were randomly assigned to either paroxetine (a selective serotonin reuptake inhibitor efficacious for SAD; see **Chapter 24** for a review) or placebo in this 16-week double-blind randomized controlled trial. The majority of patients (80%) were compliant with the medication protocol. The paroxetine condition reported greater decreases in social anxiety over the course of the trial relative to those who received placebo. Specifically, social anxiety scores were reduced by 53% for the paroxetine group compared to 32% in the placebo condition. The paroxetine condition was associated with greater declines in both drinking to cope within social situations and avoidance of social situations if they could not drink compared to placebo.

Although these results are promising, they suggest that paroxetine may not be an efficacious stand-alone treatment for comorbid SAD and alcohol-use disorders. Social anxiety scores remained in the clinical range in both groups, which is concerning given that elevated anxiety increases alcohol-use disorder relapse risk (Driessen et al., 2001; Kushner et al., 2005). Also, paroxetine did not have a significant impact on quantity or frequency of alcohol use. In fact, overall alcohol use did not decrease for either condition. Although patients in both conditions reported reductions in days in which alcohol was used to cope with social anxiety, paroxetine did not produce greater reductions in days alcohol was used to cope with social anxiety than placebo. Also, patients receiving paroxetine reported avoiding social situations in which they could not drink 35% of the time by week 16. Although this is less avoidance than patients in the placebo condition (who reported avoidance 68% of the time alcohol was

unavailable), avoidance of one-third of social situations in which alcohol is unavailable is concerning. Thus, additional work is necessary to determine whether teaching patients skills to help them better manage their drinking behaviors in conjunction with paroxetine treatment for SAD can further improve outcomes.

These data, combined with evidence of poorer outcomes reported above, suggest the need to explicitly address both SAD and the co-occurring substance use disorder in treatment. There are at least three approaches to the treatment of co-occurring anxiety and substance use disorders (Stewart & Conrod, 2008): (1) *sequential*, in which therapists treat one problem before treating the other problem (common in clinical practice); (2) *parallel*, in which two treatments (one for SAD, one for the substance use disorder) are used simultaneously but not in an integrated manner; and (3) *integrated*, in which the intervention is a "hybrid" of the two treatments (one for SAD, one for the substance use disorder), addressing the reciprocal nature of the relation between social anxiety and substance use.

Parallel Treatments

Most empirical work thus far has tested parallel treatments for comorbid SAD and substance use disorders. One common method of treating dually diagnosed patients is to provide psychosocial treatment for the substance use disorder and treat the co-occurring disorder pharmacologically. Liappas et al. (2003) examined whether treating SAD symptoms with the antidepressant mirtazapine, administered in parallel to treatment for alcohol-use disorders (alcohol detoxification with vitamin replacement and diazepam followed by 4–5 weeks of CBT for alcohol-use disorders), would produce better outcomes than the alcohol-use disorder treatment alone among patients receiving inpatient alcohol-use disorder treatment. Patients were unselected for social anxiety (although mean social anxiety scores were in the clinical range at intake). Social anxiety decreased over time for both conditions. Encouragingly, the parallel treatment condition evinced significantly less social anxiety than the control condition following treatment.

Although these findings are promising, additional work is necessary to determine the utility of this intervention. First, patients were unselected for social anxiety. Although over half the sample reported clinically significant social anxiety, it is unclear whether those patients demonstrated clinically meaningful reductions in social anxiety. Second, mean social anxiety scores remained in the clinical range for both groups (as per the cut-scores identified by Mennin et al., 2002). Third, the authors did not report the impact of the parallel treatment on alcohol relapse rates, and future work is necessary to determine whether this treatment had an impact on drinking behaviors. Fourth, the parallel treatment produced greater reductions in depression. However, the authors did not report whether observed reductions in social anxiety were related to reductions in depression. Fifth, future work is necessary to examine the impact of mirtazapine discontinuation on anxiety and drinking outcomes.

Randall, Thomas, et al. (2001) evaluated a parallel psychosocial treatment specifically designed for patients seeking treatment for alcohol-use disorders. All patients indicated that they used alcohol to cope with anxiety and met DSM-III-R criteria for

SAD and alcohol dependence. Patients were randomly assigned to receive 12 individual sessions of either CBT for alcohol-use disorders or parallel CBT for alcohol-use disorders plus CBT for SAD. CBT for alcohol-use disorders consisted of the following topics (see Kadden et al., 1992): core sessions included (1) coping with cravings, (2) managing thoughts about alcohol, (3) problem-solving, (4) refusal skills, (5) planning for emergencies, and (6) seemingly irrelevant decisions; elective topics included (1) awareness of anger, (2) anger management, (3) increasing pleasant activities, (4) managing depression, and (5) spouse/partner session. Alcohol-only CBT sessions lasted 60 min and patients were discouraged from discussing social anxiety. The parallel treatment sessions lasted 90 min and addressed both drinking and social anxiety. The first half of each parallel treatment session consisted of CBT for alcohol and the second half consisted of CBT for social anxiety. CBT for social anxiety topics included (1) relaxation training, (2) construction of fear hierarchy, (3) *in vivo* exposures, (4) managing thoughts about social anxiety, and (4) managing emergencies.

Patients in the parallel treatment were somewhat more likely to complete treatment (43% compared to 32%). Both treatments produced decreases in drinking and in social anxiety. However, the parallel CBT resulted in *worse* drinking outcomes at 3-month follow-up relative to alcohol-only CBT. Both treatment conditions exhibited clinically elevated social anxiety after treatment which is problematic, as elevated anxiety may place these patients at risk for drinking relapse. The authors proposed several hypotheses that might account for these surprising findings. For instance, patients in the parallel condition may have engaged in more social situations following treatment, thereby increasing the likelihood that they would drink (although the authors stated that this explanation seems unlikely given the lack of difference in social avoidance between the two conditions at post-treatment). It is also possible that the longer parallel treatment session resulted in inability for patients to attend to and/or retain information discussed in sessions (although the lack of difference in drop-out rates between conditions suggests that this explanation is unlikely). The worse drinking outcomes observed in the parallel treatment may have been due to patients drinking to cope with out-of-session anxiety exposure exercises, suggesting the need to teach patients ways to cope with out-of-session anxiety without relying on alcohol.

Another study (Schadé et al., 2005) examined a parallel treatment for inpatients with primary alcohol dependence and secondary anxiety disorder (67% of patients met criteria for SAD). After undergoing alcohol detoxification, patients were randomly assigned to either alcohol treatment as usual (TAU) or parallel TAU plus CBT for anxiety disorders (with optional fluvoxamine, a selective serotonin reuptake inhibitor). TAU consisted of 25 hr of group therapy over 12–16 weeks. Patients received disulfiram and psychotherapy topics included (1) psychoeducation; (2) self-control training (i.e., training in functional analysis, alternative coping skills, and self-monitoring); (3) social skills training; (4) covert sensitization (aversion therapy in which patients' imaginations are used to develop conditioned avoidance of alcohol); and (5) as needed assistance with job management, financial budgeting and housing, as well as marital or family therapy. Patients were offered individual weekly follow-up sessions on an outpatient basis for up to 32 weeks following discharge. Patients in the parallel treatment condition received TAU plus CBT for anxiety disorders (Beck & Emery, 1985), 12 sessions of 60-min individual treatment consisting primarily of cognitive

restructuring (including restructuring thoughts related to drinking). Half the sessions occurred during inpatient treatment, half during follow-up outpatient treatment.

The addition of CBT for anxiety disorders did not improve alcohol outcomes, and scores on the social phobia assessment remained in the clinical range at follow-up for both conditions. Further, although 94% of the sample completed TAU, only 43% of patients completed the CBT portion of the parallel treatment. However, interpretation of results is complicated given that the parallel treatment group reported more heavy-drinking days (drinking at least five drinks per day) at baseline.

Bowen, D'Arcy, Keegan, and Senthilselvan (2000) tested whether CBT for panic disorder administered in parallel with TAU would produce better outcomes among patients with panic disorder undergoing inpatient alcohol-use disorder treatment. They assessed the impact of the parallel treatment on social anxiety symptoms as well as panic and agoraphobia. After undergoing at least 1 week of alcohol detoxification, patients were assessed for eligibility and then randomly assigned to TAU or TAU plus CBT for panic disorder. CBT for panic consisted of six 2-hr group sessions. The addition of CBT for panic was unrelated to social anxiety or drinking outcomes (and was unrelated to panic and depression outcomes). Specifically, fear of negative evaluation decreased during the course of treatment for both treatment conditions, with no significant differences between treatment conditions. Treatment conditions also did not differ on alcohol-related outcomes.

Integrated Treatments

The disappointing results of studies of parallel treatments for comorbid anxiety and substance use disorders have led to a call for the development of treatments for dually diagnosed patients that treat the anxiety and substance use disorders in an integrated fashion (Stewart & Conrod, 2008). To date, two known integrated treatments have been developed and empirically tested for patients with co-occurring social anxiety and substance use.

Courbasson and Nishikawa (2010) conducted an uncontrolled study of a partially integrated treatment for SAD and substance use disorders with 59 patients (61% of whom had primary SAD). The treatment consisted of 10 2-hr sessions of group CBT for SAD (Heimberg & Becker, 2002) modified to include social skills training and discussion of the relations between anxiety and substance use disorders. CBT techniques included (1) psychoeducation of SAD and possible relations between anxiety and substance use (e.g., beliefs about substances as a means to cope with anxiety); (2) cognitive restructuring, including restructuring maladaptive thoughts regarding the relations between anxiety and substance use; (3) *in vivo* exposures during which patients were encouraged to abstain from using substances; and (4) social skills training. Although there were clear decreases in social anxiety, scores remained in the clinical range post-treatment, and only 44% of patients completed treatment. Unfortunately, the authors did not report the impact of the treatment on substance use behaviors. These results show promise in that they provide support for the ability to integrate skills to address social anxiety and substance use for anxiety management in a manner that results in decreases in social anxiety. Future work, however, is necessary

to examine the impact of these techniques on substance use behaviors as well as to determine ways to improve treatment completion and outcomes for these patients.

Tran and colleagues developed a brief (three-session) intervention for socially anxious drinkers known as *Brief Intervention for Socially Anxious Drinkers* (BISAD; see Black et al., 2012). BISAD includes (1) psychoeducation regarding the potential negative consequences of drinking and positive aspects of moderate drinking; (2) personalized feedback on patient's drinking and social anxiety behaviors; (3) motivational interviewing techniques to explore and resolve ambivalence about changing drinking and social anxiety-related behaviors; (4) discussion of drinking to cope with anxiety; and (5) CBT strategies to help patients better manage social anxiety. In a pilot study of the utility of BISAD, participants were randomly assigned to either BISAD or to alcohol psychoeducation only. Participants were heavy-drinking college students (who reported engaging in at least two binge drinking episodes and at least two alcohol-related problems in the past month) who reported subclinically elevated social anxiety. Both groups exhibited decreases in drinking, drinking-related problems, and positive expectancies regarding alcohol's effects in social situations. Further, both groups reported increases in self-efficacy to refuse alcohol in social situations. Thus, results suggest BISAD may be useful in changing drinking behaviors among socially anxious college students who experience alcohol-related problems.

However, BISAD did not produce better outcomes than psychoeducation control. To illustrate, at 4-month follow-up, both groups reported drinking comparable drinks in the past month, comparable number of binge drinking days, and comparable severity of alcohol-related problems. The BISAD condition did evince significant decreases in drinking and the control group did not. Yet the BISAD condition also reported more drinking at baseline, making it difficult to delineate whether observed decreases were due to regression to the mean, especially in light of the comparable outcomes observed between groups. It is also noteworthy that only 54% of the sample completed the 4-month follow-up. Thus, replication with a larger sample size (there were approximately 20 subjects per condition at baseline) is necessary to determine whether BISAD produces meaningful decreases in social anxiety, drinking, and drinking-related problems.

Recommendations

Given the preliminary nature of the extant data in this area, recommendations regarding treatment for patients with comorbid social anxiety and substance use disorders are necessarily tentative. Therefore, presented here are preliminary recommendations based on the extant literature.

Assessment

Given the high rates of clinically elevated social anxiety among patients presenting for treatment for substance use disorders (as well as the high rates of substance use and use-related problems among socially anxious persons), it is recommended that behavioral health professionals assess patients for both social anxiety and

substance use behaviors at intake and monitor these behaviors during the course of treatment.

Unfortunately, SAD appears to be underdiagnosed in substance use disorder treatment facilities. Among patients presenting for outpatient substance dependence treatment, 50% evinced clinically elevated social anxiety on a self-report measure; yet, only 3% were diagnosed with SAD by psychiatrists and drug counselors conducting intake assessment interviews (El-Sayegh, Fattal, & Muzina, 2006). Data such as these suggest that efforts should be undertaken to ensure proper assessment and treatment of SAD among patients presenting for substance use disorder treatment.

In our Anxiety and Addictive Behaviors Clinic, we have found that when dually diagnosed patients present for treatment of SAD, they often do not present for treatment of substance use. In fact, frequently they believe that their substance use is an adaptive method to cope with their pathological anxiety. Thus, we have found that it is necessary to assess for substance use and use-related problems (regardless of whether patients present for substance-related treatment) to detect the presence of risky or problematic substance use.

Given the high rates of suicidality among these patients (Buckner, Joiner, et al., 2012; Evren et al., 2009), it is also recommended that dually diagnosed patients undergo a thorough suicide assessment (e.g., Joiner, Walker, Rudd, & Jobes, 1999) at intake. Suicidality should be monitored and addressed as necessary during the course of treatment (see Rudd, Joiner, & Rajab, 2001).

Diagnostic Feedback

Dually diagnosed patients presenting for SAD treatment may be surprised to learn they have a co-occurring substance use disorder. Similarly, patients seeking substance use disorder treatment (i.e., not seeking treatment for SAD) are often surprised to learn that they have co-occurring SAD. Although these patients usually acknowledge that they have always been "shy," they often do realize that their shyness is pathological. In our clinic, we have found it helpful to provide thorough diagnostic feedback to patients to provide a rationale for a treatment plan that includes targeting both social anxiety and substance use. Therapists provide structured diagnostic feedback (as per Holm-Denoma et al., 2008) that includes provision of diagnoses, discussion of symptoms that meet criteria for each diagnosis, and answering questions that patients may have about the diagnoses. Therapists provide patients psychoeducation about the relationships between social anxiety and substance use disorders, usually in the form of data illustrating the high rates of comorbidity between these disorders. This psychoeducation is designed to normalize the patient's experiences by illustrating that social anxiety frequently co-occurs with substance-related problems.

Feedback for dually diagnosed patients also includes a discussion of the relationship between social anxiety and substance use. Therapists first ask patients to describe any links that they have noticed between their anxiety and their substance use. Therapists then provide patients with psychoeducational materials that include a discussion of the "vicious cycle" that can occur between anxiety and substance use (e.g., substance use for anxiety management can lead to reliance on substances to manage anxiety which

can increase anxiety via craving, withdrawal, fear of evaluation for substance-related problems, etc.). Therapists encourage patients to ask questions about the diagnoses and the relationships between the disorders. We believe it is important that patients understand their diagnoses and the relationships between the disorders so that they are more likely to agree to treatment of both disorders.

Integrated Treatment

In light of the poor outcomes of parallel treatments (e.g., Randall, Thomas, et al., 2001), it is recommended that dually diagnosed patients receive treatment that addresses social anxiety and substance use in an integrated manner. Given the limited empirical support for extant integrated treatments, one promising approach is to combine motivation enhancement therapy (MET) for substance use disorders (Miller, Zweben, DiClemente, & Rychtarik, 1992) with CBT for SAD (e.g., Heimberg et al., 1990; Hope, Heimberg, & Turk, 2010a, 2010b) to treat comorbid SAD and substance use disorders. Mounting evidence suggests that motivational interviewing techniques can be used successfully with patients with SAD to effect other types of behavioral changes. A motivation enhancement therapy that combined personalized feedback about social anxiety with MET techniques to explore and resolve ambivalence about seeking CBT for SAD resulted in 73% of nontreatment seekers with SAD indicating a willingness to engage in therapist contact compared to just 33% of those in a feedback-only control group (Buckner & Schmidt, 2009a). In another study, patients seeking CBT for anxiety disorders (31% of whom had SAD) were randomly assigned to either a pre-CBT motivational interviewing or waitlist control (Westra & Dozois, 2006). Patients who received the pre-CBT motivational interviewing reported a greater increase in their expectations that CBT would help them change their anxiety (the size of this effect was especially large for patients with SAD). Further, 84% of the motivational interviewing condition completed CBT compared to 63% of controls, and motivational interviewing patients reported completing significantly more CBT homework. Taken together, these data suggest that incorporating a motivational component addressing problematic substance use could be combined with CBT for SAD, a treatment with demonstrated efficacy for SAD (for review, see Heimberg, 2002), to treat comorbid SAD and substance use disorders in an integrated fashion that addresses motivation to change substance use behaviors while simultaneously teaching patients more adaptive skills to manage anxiety and other negative affective states.

There is preliminary support from case studies for this approach. An overview of this treatment approach with one patient with SAD and alcohol abuse and one patient with SAD and cannabis abuse are presented below.

Buckner, Ledley, Heimberg, and Schmidt (2008) integrated MET for alcohol-use disorders with individual CBT for SAD (Hope, Heimberg, Juster, & Turk, 2000; Hope, Heimberg, & Turk, 2006) in the case of an adult male patient with generalized SAD and alcohol abuse disorder. The integration of MET and CBT resulted in decreased alcohol-related problems and social anxiety (including the remission of both disorders and increases in quality of life). The first three sessions included

discussion of the ways in which the patient's alcohol use and social anxiety were related. MET was used to help him consider changing his alcohol-use behaviors. Homework included reading psychoeducational materials developed by the authors regarding the functional relationships between SAD and alcohol abuse. At session 4, a *change plan* was developed that included daily monitoring of alcohol use. The patient's goal was to reduce his drinking so that he no longer drank for social anxiety management. Sessions 5–19 consisted primarily of CBT for SAD with continued monitoring of drinking. When the patient used alcohol, past-week drinking was discussed to determine whether his use remained consistent with his treatment goals. During session 13, the patient suggested that he would attend a party without drinking. After this successful exposure, he developed other exposures to practice anxiety management skills in situations in which he would have previously used alcohol to cope. From session 13 until termination (session 19), he denied drinking to manage anxiety and reported no drinking-related problems. He also experienced clinically significant improvement in fear of negative evaluation, from a maximum score on the measure (60) to a score (32) in the range achieved by nonclinical controls (as per Weeks et al., 2005). Also, he denied current (past-month) alcohol-related problems. He was considered in remission for both SAD and alcohol abuse. Six months after termination, the patient reported that his social anxiety continued to decline and he continued to deny current alcohol-related problems. He also reported improvements in quality of life (e.g., obtaining a desired higher paying job, making new friends).

MET-CBT has also been used to treat comorbid generalized SAD and cannabis abuse with an adult male patient. The patient attended 21 MET-CBT sessions which resulted in cannabis abstinence and decreases in social anxiety and avoidance. In the first two sessions, MET was used to help him consider changing his cannabis-use behaviors. Session 1 included personalized feedback regarding his social anxiety and cannabis use, combined with discussion of the ways in which his cannabis use and social anxiety were related. Homework included reading psychoeducational materials regarding the functional relationships between SAD and cannabis use. During session 2, the patient stated that his goal was to no longer use cannabis to manage social anxiety and to abstain from using cannabis before or while driving. A change plan was developed that included daily monitoring of cannabis use. During session 3, the patient reported an "experiment" in which he engaged in a social anxiety-evoking situation without using cannabis. After this successful exposure, he remained cannabis abstinent. He also attended social events (without using substances) in which he would have used cannabis to manage his social anxiety in the past. Sessions 3–21 consisted primarily of CBT for SAD with continued monitoring of cannabis use. In session 7, the patient reported a score of 10 on the Social Anxiety Session Change Index (Hayes, Miller, Hope, Heimberg, & Juster, 2008), indicating reduced fears of evaluation and avoidance of social situations (scores of 4–15 indicate improvement). By session 17, the patient's score had decreased to 5. Further, he reported feeling proud about his 3-month abstinence. During session 21, he reported feeling proud of all of accomplishments including becoming involved in a romantic relationship, employment in his desired career, daily exposures, and attending social events without feeling his usual "paralyzing" anxiety.

Importantly, patient reported abstaining from cannabis when involved in these social situations.

Of course, recommendations regarding the use of MET-CBT with dually diagnosed patients are necessarily tentative until a large-scale clinical trial can be conducted to test the efficacy of this approach with a larger number of patients. However, these preliminary data suggest that this approach may be useful, particularly for SAD patients with comorbid substance abuse. Additional work is necessary to determine whether these strategies would be beneficial for patients with substance dependence.

Future Directions

In light of the poorer outcomes experienced by patients with co-occurring social anxiety and substance use disorders, combined with limited data regarding the efficacy of extant treatments designed specifically to target these comorbid disorders, there remains much more work to be done to determine the best way to treat these dually diagnosed patients. In addition to research aimed at testing the utility of integrated treatments (including combined motivation enhancement therapy with CBT), the below may yield promising avenues for future work in this area.

Dually Diagnosed Patients May Benefit from Individual (as Opposed to Group) Treatment

Post hoc analyses of data from Project MATCH (Project MATCH Research Group, 1993), a multisite study of alcohol treatments, indicated that female alcoholics with SAD responded worse to 12-step facilitation than to individual CBT for alcohol-use disorders (the reverse was true of alcoholics without SAD) (Thevos, Roberts, Thomas, & Randall, 2000). Also, dually diagnosed patients are less likely to attend 12-step groups than patients with substance dependence and no SAD (Myrick & Brady, 1997), and social anxiety seems to interfere with patients' ability to participate in group therapy for substance use disorders (Book et al., 2009). Thus, it may be beneficial to examine the utility of individual treatments for comorbid SAD and substance use disorders.

Dually Diagnosed Patients May Benefit from Learning Skills to Help Them Manage High-Risk Social Situations

Nontreatment seekers attempting voluntary, self-guided cannabis quit attempts reported that situations involving negative affect and in which others are using cannabis are among the situations in which users report the most difficulty maintaining abstinence (Hughes, Peters, Callas, Budney, & Livingstone, 2008). Among adolescents in substance dependence treatment, the majority of relapse occurs in social situations, especially when socializing with pretreatment friends (Brown, Vik, & Creamer, 1989). These data suggest that socially anxious patients may benefit from assertiveness training and/or refusal skills to help them manage these high-risk social situations.

Dually Diagnosed Patients May Benefit from Specific Cognitive Restructuring Techniques

Less change in normative beliefs (i.e., beliefs about how much other students drink) was related to poorer drinking outcomes among heavy-drinking, socially anxious college students undergoing a brief motivation intervention (Terlecki et al., 2012). However, among those with greater change in normative beliefs, socially anxious students did not differ from those with less social anxiety on drinking outcomes. Thus, it may be important to develop and test the efficacy of techniques aimed at changing normative beliefs held by socially anxious patients, who may be reluctant to change their substance use if they fear such a change could result in negative evaluation by others for deviating from what they perceive to be normative use. Additional work could also benefit from identifying other maladaptive cognitions that play a role in poorer outcomes for dually diagnosed patients.

Concluding Remarks

Persons with elevated social anxiety appear especially vulnerable to substance-related problems. They also appear less likely to benefit from extant social anxiety and/or substance use disorder treatments. Researchers are beginning to develop and evaluate personalized treatments for these at-risk patients. Results are promising in that they suggest that techniques aimed at reducing social anxiety and substance use can be combined to treat dually diagnosed patients. However, more work is necessary to determine the best ways to combine these strategies to improve outcomes. Future work may be necessary to determine whether specific strategies need to be developed to address the specific concerns of socially anxious patients.

References

Agosti, V., Nunes, E., & Levin, F. (2002). Rates of psychiatric comorbidity among U.S. residents with lifetime cannabis dependence. *American Journal of Drug and Alcohol Abuse, 28*, 643–652. doi:10.1081/ADA-120015873

Battista, S. R., Stewart, S. H., & Ham, L. S. (2010). A critical review of laboratory-based studies examining the relationships of social anxiety and alcohol intake. *Current Drug Abuse Reviews, 3*, 3–22.

Beck, A. T., & Emery, G. (1985). *Anxiety disorders and phobias: A cognitive perspective*. New York, NY: Basic Books.

Black, J. J., Tran, G. Q., Goldsmith, A. A., Thompson, R. D., Smith, J. P., & Welge, J. A. (2012). Alcohol expectancies and social self-efficacy as mediators of differential intervention outcomes for college hazardous drinkers with social anxiety. *Addictive Behaviors, 37*, 248–255.

Book, S. W., Thomas, S. E., Dempsey, J. P., Randall, P. K., & Randall, C. L. (2009). Social anxiety impacts willingness to participate in addictions treatment. *Addictive Behaviors, 34*, 474–476. doi:10.1016/j.addbeh.2008.12.011

Book, S. W., Thomas, S. E., Randall, P. K., & Randall, C. L. (2008). Paroxetine reduces social anxiety in individuals with a co-occurring alcohol use disorder. *Journal of Anxiety Disorders, 22*, 310–318.

Bowen, R. C., D'Arcy, C., Keegan, D., & Senthilselvan, A. (2000). A controlled trial of cognitive behavioral treatment of panic in alcoholic inpatients with comorbid panic disorder. *Addictive Behaviors, 25*, 593–597. doi:10.1016/s0306-4603(99)00017-9

Brown, S. A., Vik, P. W., & Creamer, V. A. (1989). Characteristics of relapse following adolescent substance abuse treatment. *Addictive Behaviors, 14*, 291–300. doi:10.1016/0306-4603(89)90060-9

Buckner, J. D., Bonn-Miller, M. O., Zvolensky, M. J., & Schmidt, N. B. (2007). Marijuana use motives and social anxiety among marijuana-using young adults. *Addictive Behaviors, 32*, 2238–2252. doi:10.1016/j.addbeh.2007.04.004

Buckner, J. D., Ecker, A. H., & Proctor, S. L. (2011). Social anxiety and alcohol problems: The roles of perceived descriptive and injunctive peer norms. *Journal of Anxiety Disorders, 25*, 631–638. doi:10.1016/j.janxdis.2011.02.003

Buckner, J. D., Eggleston, A. M., & Schmidt, N. B. (2006). Social anxiety and problematic alcohol consumption: The mediating role of drinking motives and situations. *Behavior Therapy, 37*, 381–391. doi:10.1016/j.beth.2006.02.007

Buckner, J. D., & Heimberg, R. G. (2010). Drinking behaviors in social situations account for alcohol-related problems among socially anxious individuals. *Psychology of Addictive Behaviors, 24*, 640–648. doi:10.1037/a0020968

Buckner, J. D., Heimberg, R. G., Matthews, R. A., & Silgado, J. (2012). Marijuana-related problems and social anxiety: The role of marijuana behaviors in social situations. *Psychology of Addictive Behaviors, 26*, 151–156. doi:10.1037/a0025822

Buckner, J. D., Heimberg, R. G., Schneier, F. R., Liu, S., Wang, S., & Blanco, C. (2012). The relationship between cannabis use disorders and social anxiety disorder in the national epidemiological study of alcohol and related conditions (NESARC). *Drug and Alcohol Dependence, 124*, 128–134. doi:10.1016/j.drugalcdep.2011.12.023

Buckner, J. D., Joiner, T. E., Jr., Schmdit, N. B., & Zvolensky, M. J. (2012). Daily marijuana use and suicidality: The unique impact of social anxiety. *Addictive Behaviors, 37*, 387–392. doi:10.1016/j.addbeh.2011.11.019

Buckner, J. D., Ledley, D. R., Heimberg, R. G., & Schmidt, N. B. (2008). Treating comorbid social anxiety and alcohol use disorders: Combining motivation enhancement therapy with cognitive-behavioral therapy. *Clinical Case Studies, 7*, 208–223.

Buckner, J. D., & Matthews, R. A. (2012). Social impressions while drinking account for the relationship between alcohol-related problems and social anxiety. *Addictive Behaviors, 37*, 533–536. doi:10.1016/j.addbeh.2011.11.026

Buckner, J. D., & Schmidt, N. B. (2009a). A randomized pilot study of motivation enhancement therapy to increase utilization of cognitive-behavioral therapy for social anxiety. *Behaviour Research and Therapy, 47*, 710–715. doi:10.1016/j.brat.2009.04.009

Buckner, J. D., & Schmidt, N. B. (2009b). Social anxiety disorder and marijuana use problems: The mediating role of marijuana effect expectancies. *Depression and Anxiety, 26*, 864–870. doi:10.1002/da.20567

Buckner, J. D., & Schmidt, N. B. (2009c). Understanding social anxiety as a risk for alcohol use disorders: Fear of scrutiny, not social interaction fears, prospectively predicts alcohol use disorders. *Journal of Psychiatric Research, 43*, 477–483. doi:10.1016/j.jpsychires.2008.04.012

Buckner, J. D., Schmidt, N. B., Lang, A. R., Small, J. W., Schlauch, R. C., & Lewinsohn, P. M. (2008). Specificity of social anxiety disorder as a risk factor for alcohol and cannabis dependence. *Journal of Psychiatric Research, 42*, 230–239. doi:10.1016/j.jpsychires.2007.01.002

Buckner, J. D., Timpano, K. R., Zvolensky, M. J., Sachs-Ericsson, N., & Schmidt, N. B. (2008). Implications of comorbid alcohol dependence among individuals with social anxiety disorder. *Depression and Anxiety, 25*, 1028–1037. doi:10.1002/da.20442

Buckner, J. D., & Turner, R. J. (2009). Social anxiety disorder as a risk factor for alcohol use disorders: A prospective examination of parental and peer influences. *Drug and Alcohol Dependence, 100*, 128–137. doi:10.1016/j.drugalcdep.2008.09.018

Buckner, J. D., Zvolensky, M. J., & Schmidt, N. B. (2012). Cannabis-related impairment and social anxiety: The roles of gender and cannabis use motives. *Addictive Behaviors, 37*, 1294–1297. doi:10.1016/j.addbeh.2012.06.013

Cougle, J. R., Zvolensky, M. J., Fitch, K. E., & Sachs-Ericsson, N. (2010). The role of comorbidity in explaining the associations between anxiety disorders and smoking. *Nicotine and Tobacco Research, 12*, 355–364.

Courbasson, C. M., & Nishikawa, Y. (2010). Cognitive behavioral group therapy for patients with co-existing social anxiety disorder and substance use disorders: A pilot study. *Cognitive Therapy and Research, 34*, 82–91. doi:10.1007/s10608-008-9216-8

Crum, R. M., & Pratt, L. A. (2001). Risk of heavy drinking and alcohol use disorders in social phobia: A prospective analysis. *American Journal of Psychiatry, 158*, 1693–1700. doi:10.1176/appi.ajp.158.10.1693

Dimeff, L. A., Baer, J. S., Kivlahan, D. R., & Marlatt, G. A. (1999). *Brief alcohol screening and intervention for college students: A harm reduction approach.* New York, NY: Guilford Press.

Driessen, M., Meier, S., Hill, A., Wetterling, T., Lange, W., & Junghanns, K. (2001). The course of anxiety, depression and drinking behaviours after completed detoxification in alcoholics with and without comorbid anxiety and depressive disorders. *Alcohol and Alcoholism, 36*, 249–255. doi:10.1093/alcalc/36.3.249

El-Sayegh, S., Fattal, O., & Muzina, D. J. (2006). Is social anxiety disorder unrecognized in patients with substance dependence? *Addictive Disorders and Their Treatment, 5*, 145–151. doi:10.1097/01.adt.0000210714.87821.98

Evren, C., Sar, V., Dalbudak, E., Oncu, F., & Cakmak, D. (2009). Social anxiety and dissociation among male patients with alcohol dependency. *Psychiatry Research, 165*, 273–280.

Gilles, D. M., Turk, C. L., & Fresco, D. M. (2006). Social anxiety, alcohol expectancies, and self-efficacy as predictors of heavy drinking in college students. *Addictive Behaviors, 31*, 388–398. doi:10.1016/j.addbeh.2005.05.020

Grant, B. F., Dawson, D. A., Stinson, F. S., Chou, S. P., Dufour, M. C., & Pickering, R. P. (2004). The 12-month prevalence and trends in DSM-IV alcohol abuse and dependence: United States, 1991-1992 and 2001-2002. *Drug and Alcohol Dependence, 74*, 223–234. doi:10.1016/j.drugalcdep.2004.02.004

Grant, B. F., Hasin, D. S., Blanco, C., Stinson, F. S., Chou, S. P., Goldstein, R. B., . . . Huang, B. (2005). The epidemiology of social anxiety disorder in the United States: Results from the national epidemiologic survey on alcohol and related conditions. *Journal of Clinical Psychiatry, 66*, 1351–1361.

Grant, B. F., Hasin, D. S., Chou, S. P., Stinson, F. S., & Dawson, D. A. (2004). Nicotine dependence and psychiatric disorders in the United States. *Archives of General Psychiatry, 61*, 1107–1115. doi:10.1001/archpsyc.61.11.1107

Hayes, S. A., Miller, N. A., Hope, D. A., Heimberg, R. G., & Juster, H. R. (2008). Assessing client progress session-by-session in the treatment of social anxiety disorder: The Social Anxiety Session Change Index. *Cognitive and Behavioral Practice, 15*, 203–211. doi:10.1016/j.cbpra.2007.02.010

Heimberg, R. G. (2002). Cognitive-behavioral therapy for social anxiety disorder: Current status and future directions. *Biological Psychiatry, 51*, 101–108. doi:10.1016/S0006-3223(01)01183-0

Heimberg, R. G., & Becker, R. E. (2002). *Cognitive-behavioral group therapy for social phobia: Basic mechanisms and clinical strategies.* New York, NY: Guilford Press.

Heimberg, R. G., Dodge, C. S., Hope, D. A., Kennedy, C. R., Zollo, L., & Becker, R. E. (1990). Cognitive behavioral group treatment of social phobia: Comparison to a credible placebo control. *Cognitive Therapy and Research, 14*, 1–23. doi:10.1007/BF01173521

Holm-Denoma, J. M., Gordon, K. H., Donohue, K. F., Waesche, M. C., Castro, Y., Brown, J. S., … Joiner, T. E., Jr. (2008). Patients' affective reactions to receiving diagnostic feedback. *Journal of Social and Clinical Psychology, 27*, 555–575. doi:10.1521/jscp.2008.27.6.555

Hope, D. A., Heimberg, R. G., Juster, H. R., & Turk, C. L. (2000). *Managing social anxiety: A cognitive-behavioral therapy approach (client workbook)*. New York, NY: Oxford University Press.

Hope, D. A., Heimberg, R. G., & Turk, C. L. (2006). *Therapist guide for managing social anxiety: A cognitive-behavioral therapy approach.* New York, NY: Oxford University Press.

Hope, D. A., Heimberg, R. G., & Turk, C. L. (2010a). *Managing social anxiety: A cognitive-behavioral therapy approach (client workbook, 2nd ed.)*. New York, NY: Oxford University Press.

Hope, D. A., Heimberg, R. G., & Turk, C. L. (2010b). *Managing social anxiety: A cognitive-behavioral therapy approach (therapist guide, 2nd ed.)*. New York, NY: Oxford University Press.

Hughes, J. R., Peters, E. N., Callas, P. W., Budney, A. J., & Livingstone, A. E. (2008). Attempts to stop or reduce marijuana use in non-treatment seekers. *Drug and Alcohol Dependence, 97*, 180–184. doi:10.1016/j.drugalcdep.2008.03.031

Hull, J. G., Young, R. D., & Jouriles, E. (1986). Applications of the self-awareness model of alcohol consumption: Predicting patterns of use and abuse. *Journal of Personality and Social Psychology, 51*, 790–796. doi:10.1037/0022-3514.51.4.790

Joiner, T. E., Jr., Walker, R. L., Rudd, M. D., & Jobes, D. A. (1999). Scientizing and routinizing the assessment of suicidality in outpatient practice. *Professional Psychology: Research and Practice, 30*, 447–453. doi:10.1037/0735-7028.30.5.447

Kadden, R., Carroll, K. M., Donovan, D., Cooney, N., Monti, P. M., Abrams, D., … Hester, R. (1992). *Cognitive-behavioral coping skills therapy manual: A clinical research guide for therapists treating individuals with alcohol abuse and dependence* (Vol. *3*). Washington, DC: U.S. Government Printing Office.

Kessler, R. C., Anthony, J. C., Blazer, D. G., Bromet, E., Eaton, W. W., Kendler, K., … Zhao, S. (1997). The US national comorbidity survey: Overview and future directions. *Epidemiologia e Psichiatria Sociale, 6*, 4–16.

Kessler, R. C., Crum, R. M., Warner, L. A., Nelson, C. B., Schulenberg, J., & Anthony, J. C. (1997). Lifetime co-occurrence of DSM-III-R alcohol abuse and dependence with other psychiatric disorders in the national comorbidity survey. *Archives of General Psychiatry, 54*, 313–321.

Kushner, M. G., Abrams, K., Thuras, P., Hanson, K. L., Brekke, M., & Sletten, S. (2005). Follow-up study of anxiety disorder and alcohol dependence in comorbid alcoholism treatment patients. *Alcoholism: Clinical and Experimental Research, 29*, 1432–1443. doi:10.1097/01.alc.0000175072.17623.f8

Larimer, M. E., Kilmer, J. R., & Lee, C. M. (2005). College student drug prevention: A review of individually-oriented prevention strategies. *Journal of Drug Issues, 35*, 431–456.

Lejuez, C. W., Zvolensky, M. J., Daughters, S. B., Bornovalova, M. A., Paulson, A., Tull, M. T., … Otto, M. W. (2008). Anxiety sensitivity: A unique predictor of dropout among inner-city heroin and crack/cocaine users in residential substance use treatment. *Behaviour Research and Therapy, 46*, 811–818. doi:10.1016/j.brat.2008.03.010

Lewis, B. A., & O'Neill, H. K. (2000). Alcohol expectancies and social deficits relating to problem drinking among college students. *Addictive Behaviors, 25*, 295–299. doi:10.1016/s0306-4603(99)00063-5

Lewis, M. A., Hove, M. C., Whiteside, U., Lee, C. M., Kirkeby, B. S., Oster-Aaland, L., … Larimer, M. E. (2008). Fitting in and feeling fine: Conformity and coping motives as mediators of the relationship between social anxiety and problematic drinking. *Psychology of Addictive Behaviors, 22*, 58–67. doi:10.1037/0893-164X.22.1.58

Liappas, J., Paparrigopoulos, T., Tzavellas, E., & Christodoulou, G. (2003). Alcohol detoxification and social anxiety symptoms: A preliminary study of the impact of mirtazapine administration. *Journal of Affective Disorders, 76*, 279–284. doi:10.1016/S0165-0327(02)00094-0

Marmorstein, N. R., White, H. R., Loeber, R., & Stouthamer-Loeber, M. (2010). Anxiety as a predictor of age at first use of substances and progression to substance use problems among boys. *Journal of Abnormal Child Psychology, 38*, 211–224. doi:10.1007/s10802-009-9360-y

McCabe, R. E., Chudzik, S. M., Antony, M. M., Young, L., Swinson, R. P., & Zolvensky, M. J. (2004). Smoking behaviors across anxiety disorders. *Journal of Anxiety Disorders, 18*, 7–18. doi:10.1016/j.janxdis.2003.07.003

Mennin, D. S., Fresco, D. M., Heimberg, R. G., Schneier, F. R., Davies, S. O., & Liebowitz, M. R. (2002). Screening for social anxiety disorder in the clinical setting: Using the Liebowitz Social Anxiety Scale. *Journal of Anxiety Disorders, 16*, 661–673. doi:10.1016/s0887-6185(02)00134-2

Miller, W. R., Zweben, A., DiClemente, C. C., & Rychtarik, R. G. (1992). *Motivational enhancement therapy manual*. Rockville, MD: National Institute on Alcohol Abuse and Alcoholism [NIAAA].

Myrick, H., & Brady, K. T. (1997). Social phobia in cocaine-dependent individuals. *American Journal on Addictions, 6*, 99–104. doi:10.3109/10550499709137020

Neighbors, C., Fossos, N., Woods, B. A., Fabiano, P., Sledge, M., & Frost, D. (2007). Social anxiety as a moderator of the relationship between perceived norms and drinking. *Journal of Studies on Alcohol and Drugs, 68*, 91–96.

Piper, M. E., Cook, J. W., Schlam, T. R., Jorenby, D. E., & Baker, T. B. (2011). Anxiety diagnoses in smokers seeking cessation treatment: Relations with tobacco dependence, withdrawal, outcome and response to treatment. *Addiction, 106*, 418–427. doi:10.1111/j.1360-0443.2010.03173.x

Project MATCH Research Group. (1993). Project MATCH: Rationale and methods for a multisite clinical trial matching patients to alcoholism treatment. *Alcoholism: Clinical and Experimental Research, 17*, 1130–1145. doi:10.1111/j.1530-0277.1993.tb05219.x

Randall, C. L., Johnson, M. R., Thevos, A. K., Sonne, S. C., Thomas, S. E., Willard, S. L., … Davidson, J. R. T. (2001). Paroxetine for social anxiety and alcohol use in dual-diagnosed patients. *Depression and Anxiety, 14*, 255–262. doi:10.1002/da.1077

Randall, C. L., Thomas, S. E., & Thevos, A. K. (2001). Concurrent alcoholism and social anxiety disorder: A first step toward developing effective treatments. *Alcoholism: Clinical and Experimental Research, 25*, 210–220. doi:10.1111/j.1530-0277.2001.tb02201.x

Rudd, M. D., Joiner, T. E., & Rajab, M. H. (2001). *Treating suicidal behavior: An effective, time-limited approach*. New York, NY: Guilford Press.

Schadé, A., Marquenie, L. A., van Balkom, A. J. L. M., Koeter, M. W. J., de Beurs, E., van den Brink, W., & van Dyck, R. (2005). The effectiveness of anxiety treatment on alcohol-dependent patients with a comorbid phobic disorder: A randomized controlled trial. *Alcoholism: Clinical and Experimental Research, 29*, 794–800.

Schneier, F. R., Foose, T., Hasin, D. S., Heimberg, R. G., Liu, S.-M., Grant, B. F., & Blanco, C. (2010). Social anxiety disorder and alcohol use disorder co-morbidity in the national epidemiologic survey on alcohol and related conditions. *Psychological Medicine, 40*, 977–988. doi:10.1017/S0033291709991231

Schneier, F. R., Martin, L. Y., Liebowitz, M. R., Gorman, J. M., & Fyer, A. J. (1989). Alcohol abuse in social phobia. *Journal of Anxiety Disorders, 3*, 15–23. doi:10.1016/0887-6185(89)90025-X

Sonntag, H., Wittchen, H. U., Hofler, M., Kessler, R. C., & Stein, M. B. (2000). Are social fears and DSM-IV social anxiety disorder associated with smoking and nicotine dependence in adolescents and young adults? *European Psychiatry, 15*, 67–74.

Stewart, S. H., & Conrod, P. J. (2008). Anxiety disorder and substance use disorder co-morbidity: Common themes and future directions. In S. H. Stewart & P. J. Conrod (Eds.), *Anxiety and substance use disorders: The vicious cycle of comorbidity.* New York, NY: Springer.

Stewart, S. H., Morris, E., Mellings, T., & Komar, J. (2006). Relations of social anxiety variables to drinking motives, drinking quantity and frequency, and alcohol-related problems in undergraduates. *Journal of Mental Health, 15*, 671–682. doi:10.1080/09638230600998904

Terlecki, M. A., Buckner, J. D., Larimer, M. E., & Copeland, A. L. (2011). The role of social anxiety in a brief alcohol intervention for heavy drinking college students. *Journal of Cognitive Psychotherapy, 25*, 7–21. doi:10.1891/0889-8391.24.4.5

Terlecki, M. A., Buckner, J. D., Larimer, M. E., & Copeland, A. L. (2012). Brief motivational intervention for college drinking: The synergistic impact of social anxiety and perceived drinking norms. *Psychology of Addictive Behaviors, 26*, 917–923. doi:10.1037/a0027982

Terra, M. B., Barros, H. M., Stein, A. T., Figueira, I., Athayde, L. D., Spanemberg, L., … da Silveira, D. X. (2006). Does co-occurring social phobia interfere with alcoholism treatment adherence and relapse? *Journal of Substance Abuse Treatment, 31*, 403–409. doi:10.1016/j.jsat.2006.05.013

Thevos, A. K., Roberts, J. S., Thomas, S. E., & Randall, C. L. (2000). Cognitive behavioral therapy delays relapse in female socially phobic alcoholics. *Addictive Behaviors, 25*, 333–345. doi:10.1016/S0306-4603(99)00067-2

Thevos, A. K., Thomas, S. E., & Randall, C. L. (1999). Baseline differences in social support among treatment-seeking alcoholics with and without social phobia. *Substance Abuse, 20*, 107–121.

Thomas, S. E., Randall, C. L., & Carrigan, M. H. (2003). Drinking to cope in socially anxious individuals: A controlled study. *Alcoholism: Clinical and Experimental Research, 27*, 1937–1943. doi:10.1097/01.ALC.0000100942.30743.8C

Thomas, S. E., Randall, P. K., Book, S. W., & Randall, C. L. (2008). The complex relationship between co-occurring social anxiety and alcohol use disorders: What effect does treating social anxiety have on drinking? *Alcoholism: Clinical and Experimental Research, 32*, 77–84. doi:10.1111/j.1530-0277.2007.00546.x

Thomas, S. E., Thevos, A. K., & Randall, C. L. (1999). Alcoholics with and without social phobia: A comparison of substance use and psychiatric variables. *Journal of Studies on Alcohol, 60*, 472–479.

Tran, G. Q., & Smith, J. P. (2008). Co-morbidity of social phobia and alcohol use disorders: A review of psychopathology research findings. In S. H. Stewart & P. J. Conrod (Eds.), *Anxiety and Substance Use Disorders.* New York, NY: Springer.

Watson, N. L., VanderVeen, J. W., Cohen, L. M., DeMarree, K. G., & Morrell, H. E. R. (2012). Examining the interrelationships between social anxiety, smoking to cope,

and cigarette craving. *Addictive Behaviors, 37*, 986–989. doi:10.1016/j.addbeh.2012.03.025

Weeks, J. W., Heimberg, R. G., Fresco, D. M., Hart, T. A., Turk, C. L., Schneier, F. R., & Liebowitz, M. R. (2005). Empirical validation and psychometric evaluation of the brief Fear of Negative Evaluation Scale in patients with social anxiety disorder. *Psychological Assessment, 17*, 179–190. doi:10.1037/1040-3590.17.2.179

Westra, H. A., & Dozois, D. J. A. (2006). Preparing clients for cognitive behavioral therapy: A randomized pilot study of motivational interviewing for anxiety. *Cognitive Therapy and Research, 30*, 481–498. doi:10.1007/s10608-006-9016-y

26

Internet-Delivered Treatments for Social Anxiety Disorder

Gerhard Andersson[1], Per Carlbring[2], and Tomas Furmark[3]

[1]*Linköping University, Sweden*
[2]*Stockholm University, Sweden*
[3]*Uppsala University, Sweden*

Introduction to Internet-Delivered Interventions

Using the internet to deliver evidence-based psychological treatments has a rather short history as the first programs were developed in the mid-1990s, not long after the introduction of the internet in general public. To date, cognitive behavioral therapy (CBT) programs have dominated (G. Andersson, 2009), even if there are recent examples of other approaches such as psychodynamic internet treatment (G. Andersson, Paxling, Roch-Norlund, et al., 2012; Johansson et al., 2012). Internet-delivered treatments were predated by research on computerized interventions (Marks, Shaw, & Parkin, 1998), often presented via CD-ROM, and research on bibliotherapy in which self-help books are used in the treatment with minimal guidance from a clinician (Watkins & Clum, 2008). In many ways internet interventions can be viewed as a mixture of these two approaches, with the addition of ease of contact (i.e., e-mail, chat, online discussion forum) and with therapist contact spanning from real-time chats, off-line contact (e.g., e-mail), to being fully automated (e.g., computer-generated reminders). One commonly used approach in research on internet interventions has been defined as follows by our research group:

> "... a therapy that is based on self-help books, guided by an identified therapist which gives feedback and answers to questions, with a scheduling that mirrors face to face treatment, and which also can include interactive online features such as queries to obtain passwords in order to get access to treatment modules" (G. Andersson et al., 2008, p. 164).

Another description was provided by representatives from the International Society for Research on Internet Interventions (ISRII):

> "Internet interventions are treatments, typically behaviorally based, that are operationalized and transformed for delivery via the Internet. Usually, they are highly structured; self-guided or partly self-guided; based on effective face-to-face interventions;

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

personalized to the user; interactive; enhanced by graphics, animations, audio, and video; and tailored to provide follow-up and feedback" (Ritterband, Andersson, Christensen, Carlbring, & Cuijpers, 2006, p. 1).

While the focus of this chapter is on SAD it is worth mentioning that there are now several controlled trials suggesting that guided internet-delivered CBT can be regarded as an evidence-based treatment that often is as effective as face-to-face CBT for a range of disorders (Andrews, Cuijpers, Craske, McEvoy, & Titov, 2010). Within the anxiety disorder spectrum, large effects have been found in studies on panic disorder (Bergström et al., 2010; Carlbring et al., 2006), severe health anxiety (Hedman, Andersson, Ljótsson, Andersson, Rück, Asmundson, et al., 2011), obsessive–compulsive disorder (E. Andersson et al., 2012), generalized anxiety disorder (Paxling et al., 2011; Titov, Andrews, Robinson, et al., 2009), specific phobia (G. Andersson et al., 2009), posttraumatic stress disorder (Spence et al., 2011) and also mixed anxiety (Carlbring et al., 2010). In addition, these promising findings have largely been replicated by independent research groups. There are also studies on depression (Johansson & Andersson, 2012) and health conditions (Cuijpers, van Straten, & Andersson, 2008), all pointing in the same direction with promising outcomes. However, one important limitation is that unguided treatment tends to yield smaller effects and larger dropouts than therapist-guided treatment (Farvolden, Denisoff, Selby, Bagby, & Rudy, 2005), even if there are exceptions to this as will be reviewed later on in this chapter. The rest of the chapter will focus on SAD and how the internet can be used in the assessment and treatment of SAD. First, we will begin with a note on how the internet and SAD may be related.

Social Anxiety Disorder and the Internet

SAD by definition involves avoidance of social interactions where there is a perceived risk of scrutiny and anxiety symptoms. Hence, investigators and clinicians have noted that persons with SAD may be prone to use the internet as it does not necessarily involve direct contact. Indeed, many persons with SAD are likely to seek information on the internet and there are also several web sites with information about SAD (Khazaal, Fernandez, Cochand, Reboh, & Zullino, 2008). Erwin, Turk, Heimberg, Fresco, and Hantula (2004) investigated a sample of 434 individuals who responded to an internet-based survey (posted on an anxiety disorder clinic web site). They found that 92% of the respondents met the criteria for SAD by means of self-report. There were some negative consequences of internet use in this group. For example, participants reported that internet use had made them more passive. They also reported that they used the internet because they experienced more comfort interacting on the internet than face-to-face. As a consequence, socially anxious individuals are reported to be particularly susceptible to problematic internet use because online communication is perceived by some individuals as a safer means of interacting with decreased risk of negative evaluation (Lee & Stapinski, 2012). Indeed, the anxiety level has been shown to be lower in online as compared to real-life interaction in a sample of 2348 college students (Yen et al., 2012). On a more positive note, respondents in the

study by Erwin et al. (2004) reported that they had acquired new information about SAD through internet use and had learned about psychotherapy and medication treatments. With a large proportion of individuals in the Western world using the internet (www.internetworldstats.com), and with the more recent rapid expansion of online social networking (e.g., Facebook), it has become harder to discern how the internet is used by persons with SAD. For example, modern Smartphones make the internet present in practically all settings, and a clinical observation is that some persons with SAD used their mobile phones when engaging in *safety behaviors* (see **Chapter 1** for definition). In sum, there are both pros and cons with internet use and relatively little research on how SAD and internet use may be related.

Treatment Programs

In this section we will provide an overview of the different treatment programs that have been developed and tested specifically for SAD. There are at least eight different internet-based treatment protocols for SAD that have been tested in research. We begin with our own program, called SOFIE, which was the first to be investigated in a randomized controlled trial. The treatment approach can be described as *guided net-bibliotherapy* (Marks, Cavanagh, & Gega, 2007), in that the program mainly involves text that can either be downloaded as a pdf file from the internet and then printed (as recommended in our early trials) or read directly on the screen as plain text with few illustrations. A more recent version of the program is currently being tested which is a briefer adaption for presentation on Smartphones, but data are not yet available. This revised version also includes video, but most of the material is presented as text. A feature of internet-delivered treatments that is not always commented on is that most use online screening and questionnaire measures when conducting assessments. There are studies suggesting that online administration of commonly used measures in research on SAD replicate well and that psychometric properties are maintained (Hedman et al., 2010; Hirai, Vernon, Clum, & Skidmore, 2011). The SOFIE program was developed in 2003 and is based on evidence-based CBT techniques (G. Andersson et al., 2006). An overview of the program is presented in the Appendix. In later sections we will provide a review of the evidence for the SOFIE program when delivered via the internet. Here it can be mentioned that the material has also been tested as pure bibliotherapy with the text presented in book form only (Furmark et al., 2009). Indeed, guided bibliotherapy has also been tested and found to be effective in a small study by Abramowitz, Moore, Braddock, and Harrington (2009).

A research group in Switzerland has also developed an internet-based treatment for SAD (Berger, Hohl, & Caspar, 2009). This was probably the second to be developed, even if the trial was published later than the work conducted in Australia by Titov, Andrews, Schwencke, Drobny, and Einstein (2008). The program by Berger is similar to the SOFIE program in content but has more interactive features such as making an exposure hierarchy online. The treatment lasts for 10 weeks and consists of a total of 57 web pages that are divided into five sessions. In contrast to the SOFIE program, this is less like a self-help book and thus takes more advantage of the interactivity on the internet. In common with other programs, Berger and coworkers

have used collaborative online group elements (e.g., online discussion groups), where participants have the opportunity to share their experiences with other participants.

The third internet program for SAD was developed in Australia by Titov and Andrews and their coworkers (Titov, Andrews, Schwencke, Drobny, et al., 2008). This program, called *Shyness*, is distinctly different from the Swedish text-based program and is presented as online lessons. There are six online lessons supported by a therapist, with parts of the content presented as an illustrated story about a young man with SAD. Lessons 1 and 2 include psychoeducation regarding SAD; Lesson 3: developing an exposure hierarchy; Lessons 4 and 5: cognitive restructuring; and Lesson 6: information on relapse prevention. In common with the two previous programs, there are homework assignments and the possibility to participate in a secure online discussion forum.

There are some other programs that have been described in the research literature. In Spain, Botella et al. (2010) have developed and tested a program that focuses on fear of public speaking. We will not cover this program in detail, but both in terms of content and effects, the program appears to be similar to the three previously described programs for SAD.

A fifth internet-based treatment program that has been tested is *applied relaxation* (Furmark et al., 2009; Öst, 1987), which is a treatment program that is distinct from the CBT programs developed by the Swedish, Swiss, and Australian research groups. Internet-delivered applied relaxation has been tested as a stand-alone program for panic disorder (Carlbring, Ekselius, & Andersson, 2003), but is a major ingredient in the generalized anxiety disorder program developed by Paxling et al. (2011). Briefly, the applied relaxation program aimed for SAD consists of a total of nine weekly modules, with two being aimed at psychoeducation and treatment rationale. Modules 3–7 include relaxation exercises (*progressive, conditioned, differential,* and *quick* relaxation), and modules 8 and 9 include applied relaxation exercises in actual phobic situations (i.e., *in vivo* exposure) and relapse prevention. In the trial by Furmark et al. (2009) internet-delivered applied relaxation had a large effect (within-group Cohen's $d = 0.99$).

While the Swedish research groups have focused on tailoring internet treatments specific to SAD (Carlbring et al., 2010; Johansson et al., 2012), the Australian group has tested a unified treatment protocol in several trials in which persons with SAD have been included, along with persons with other anxiety disorders. As SAD has not been the specific target in these latter trials, we will not cover the program in more detail here, but the program includes basic CBT ingredients for treating anxiety disorders (Titov, Andrews, Johnston, Robinson, & Spence, 2010).

The final category of internet-delivered treatments for SAD builds on recent promising findings on the effects of attention training for SAD (Amir et al., 2009; Schmidt, Richey, Buckner, & Timpano, 2009). Briefly, attention training consists of a computer program that is used to practice attention with an adapted dot probe paradigm (see **Chapter 15** for additional details). Two research groups have transferred attention training for SAD to the internet, and in two separate trials the treatment has not been found to work better than a placebo condition (Boettcher, Berger, & Renneberg, 2012a; Carlbring et al., 2012). We will not comment further on this treatment approach in this chapter.

Security Issues

A few words should be said about the role of internet security. Communication between a client and a therapist should never be sent as a plain e-mail. Instead, web portals should include a secure e-mail service that handles security issues with two-factor authentication in order to decrease the probability that the requestor is presenting false evidence of his/her identity. This information is not always specified clearly in trials. State-of-the-art treatment programs require login by use, first by a personal anonymous user name (e.g., "1234abcd") in combination with a strong password including letters, numbers, and special characters (e.g., "175uVc4&"). In addition to this, a unique one-time password is required (e.g., "660610"). These passwords are either sent by SMS or are mailed to the participant by surface mail before the treatment commences. This process provides an extra layer of online security beyond merely a username and password. In addition to preventing identity theft, it is important that the communication between the client and the server is encrypted in order to minimize the risk of eavesdropping. That is typically done by the use of Transport Layer Security (TLS), which automatically encrypts the connection. Finally, any data stored in a database should be adequately encrypted and continuous monitoring should be used to detect any hacking attempts.

Effects of Internet-Delivered Treatments

In this section we will provide a brief review of the treatment studies that have been conducted to date on the effects of internet treatments for SAD. We will review the literature for each of the programs separately and in the order of their development, beginning with our own SOFIE program. More specific research findings will be covered in later sections (such as long-term effects, the role of therapist support, and comparisons between internet and face-to-face treatment).

In the first SOFIE trial, two *in vivo* group-based exposure sessions were included (G. Andersson et al., 2006). The treatment consisted of a 9-week program and therapist support was provided through e-mail. All participants in the trial ($n = 64$) were diagnosed with SAD in a live interview, and 70% were of the generalized subtype. Relatively large effects were observed, with overall within- and between-group Cohen's d effect sizes being 0.87 and 0.70, respectively, when compared against a waiting-list control group. Treatment gains were maintained at 1-year follow-up.

In the second SOFIE trial, no live meetings were included, and brief weekly telephone calls were added to the internet treatment instead. A diagnostic telephone interview was also included before and after the intervention. The trial included 57 participants and showed equally good outcomes as the first SOFIE trial, with an average between-group (ICBT vs. wait-list) effect size across measures of $d = 0.95$ (Carlbring et al., 2007).

The third controlled trial on the SOFIE program was smaller ($n = 38$) and targeted university students specifically (Tillfors et al., 2008). The aim of the trial was to investigate the added value of extra *in vivo* exposure sessions, with participants randomized to either zero or five live exposure sessions. There were no between-group differences, but the within-group effects ($d = 1.0$) were in line with the previous two SOFIE trials.

The fourth and fifth SOFIE trials were reported in the same paper (Furmark et al., 2009) and included a total of 235 participants. The trial included five conditions to which participants were randomized in two separate studies: guided internet treatment, unguided bibliotherapy, bibliotherapy with a discussion group, guided applied relaxation, and a waiting-list control group. Overall, the active treatments were superior to the control condition and equally effective.

The sixth SOFIE study was a large trial ($n = 204$) in which the roles of therapist experience and knowledge acquisition were investigated (G. Andersson, Carlbring, & Furmark on behalf of SOFIE research group, 2012). Treatment was compared to a waiting-list control condition in which participants were encouraged to use a moderated discussion forum. Results showed a large between-group effect size at posttreatment (Hedges' g effect size = 0.75), and effects were maintained at 1-year follow-up. Knowledge about SAD also increased following treatment for the ICBT group. There were no differences between experienced and novice therapists in terms of effects, but the more experienced therapists spent less time when guiding the participant. In addition to the six SOFIE trials, an adapted version of the same program has been tested in a small controlled trial (ICBT vs. wait-list) on high school students ($n = 19$; Tillfors et al., 2011). That study showed large between-group effects as in the previous SOFIE trials ($d = 1.38$), but the number of completed modules was low, suggesting that a smaller treatment dose was needed in this younger sample. Finally, the SOFIE program has been tested in a comparative trial against face-to-face group treatment (Hedman, Andersson, Ljótsson, Andersson, Rück, Mörtberg, et al., 2011) and this trial will be commented on in the section—*Are internet-delivered treatments as effective as face-to-face treatment for SAD?*.

Berger and coworkers have published two controlled trials on their program. In the first trial, they randomized 52 individuals who were diagnosed with SAD following an interview either in person or over the phone (Berger et al., 2009). Results on the main social anxiety outcome measures showed a between-group effect size of $d = 0.82$ (treament vs. wait-list). In their second trial (Berger et al., 2011), they randomized 81 individuals diagnosed with SAD into three conditions: *unguided treatment*, *guided treatment,* and *flexible support,* the latter of which was stepped up according to need. This trial will be commented on in a later section (see *The roles of guidance and support*), but here it suffices to say that all three conditions led to reductions in SAD symptoms (within-group $d = 1.47$) with small differences between the groups.

Several controlled treatment trials on SAD have been conducted by Titov and Andrews and their coworkers, with most being in the form of guided internet treatment and with diagnostic telephone interviews before inclusion in the trials. In their first trial, *Shyness 1,* they included 105 individuals with SAD who were interviewed over the phone to confirm diagnosis and then randomized to treatment or waiting list (Titov, Andrews, Schwencke, Drobny, et al., 2008). The between-group effect size at posttreatment was $d = 0.95$. In their second trial, *Shyness 2*, they included 88 persons with SAD and replicated their first promising results with a between-group effect size of $d = 1.20$ (Titov, Andrews, & Schwencke, 2008). In *Shyness 3*, the researchers investigated the differences between guided and unguided treatment ($n = 98$) (Titov, Andrews, Choi, Schwencke, & Mahoney, 2008), and guided treatment was found to be superior (see below for additional comments). The same group also tested the effects of unguided treatment (Titov, Andrews, Johnston,

Schwencke, & Choi, 2009) in the *Shyness 4* study, in which they included 163 individuals with SAD, and compared the self-guided treatment to a *self-guided plus support condition* wherein participants were called on a weekly basis by a research assistant in addition to the online treatment. Adherence and outcomes were better in the group who received telephone reminders. *Shyness 5* was an open effectiveness trial which will be commented on in the section *Effectiveness in clinical settings* (Aydos, Titov, & Andrews, 2009). In their next study—*Shyness 6*—they went further, and studied the effects of their program in a controlled trial with 82 individuals with SAD who were randomized to either treatment and telephone calls from a technician, or to treatment plus regular access to a clinician-moderated discussion forum (Titov, Andrews, Choi, Schwencke, & Johnston, 2009). The two forms of support were equally effective as attested by the large within-group effect sizes ($d = 1.31$ and 1.54 for technician and forum groups, respectively). In the *Shyness 7* study, Titov, Andrews, Schwencke, et al. (2010) included 108 individuals with SAD, and this time compared self-guided treatment versus self-guided plus motivational enhancement strategies. Large mean within-group effect sizes were found for both the unguided only and unguided plus motivational enhancement groups (Cohen's ds $= 1.10$ and 0.95, respectively). It should be mentioned, however, that automated reminders were included for both groups.

Titov and coworkers did not include follow-up data in their original SAD trials, but subsequently published 6-month follow-up data (Titov, Andrews, Johnston, et al., 2009) from the first two *Shyness* studies. The research group has also published a separate report in which they showed that comorbidity is reduced following treatment with the *Shyness* program (Titov, Gibson, Andrews, & McEvoy, 2009).

While there have been previous reviews of the ICBT literature (G. Andersson & Carlbring, 2011), there is only one meta-analytic summary. It should be noted that this summary was preliminary as it only included eight studies on SAD (Tulbure, 2011). Tulbore reported an average between-group effect size of $d = 0.86$ for SAD symptoms, with very low heterogeneity of effects. This effect size can be compared with the effect size from a meta-analysis of CBT for SAD which was $d = 0.70$ based on 29 trials (Acarturk, Cuijpers, van Straten, & de Graaf, 2009).

Long-Term Outcome Studies

Some of the published trials have included 1-year follow-up data in the original report (G. Andersson et al., 2006; Carlbring et al., 2007; Furmark et al., 2009). There are, however, also longer term follow-ups. Carlbring, Bergman-Nordgren, Furmark, and Andersson (2009) did a 30-month follow-up where they contacted 57 participants from a previous study. A total of 77% (44/57) responded to the internet-administered outcome measures, and 67% (38/57) completed a telephone interview. Results showed large pretreatment to follow up within-group effect sizes for the primary outcome measures (Cohen's d ranged from 1.10–1.71), and a majority (68%; 26/38) reported clinically significant improvements in the diagnostic interview. In another follow-up study, Hedman, Furmark, et al. (2011) conducted a 5-year follow-up study of 80 persons with SAD who had undergone internet-based CBT. A large proportion of the sample responded, with 89% (71/80) completing a diagnostic telephone interview and 80% (64/80) responding to the online questionnaire package.

The effect sizes on the SAD measures were large (Cohen's *d* ranged from 1.30–1.40; 95% CI: 0.77–1.90), and it was found that the improvements gained at the previous 1-year follow-up were sustained 5 years after participants had completed treatment. This is probably the largest and longest follow-up of ICBT for an anxiety disorder, and compares well with what has been found in follow-up studies of face-to-face CBT for SAD (Heimberg, Salzman, Holt, & Blendell, 1993).

The Roles of Guidance and Support

In the research on ICBT, there has been a longstanding interest in the roles of therapist support and guidance, as unguided programs tend to yield smaller effects and larger dropouts across the anxiety and mood disorders (Klein, Meyer, Austin, & Kyrios, 2011). The importance of support is far from established in terms of how, when, and where the support needs to be provided to guide a person through a treatment program delivered over the internet. First, support may occur before the treatment begins during telephone screening or even live interviews (Johansson & Andersson, 2012). Second, even if there may be no contact during the actual treatment, there may be a clear deadline when the research participant is called for an interview that has been scheduled in advance (Nordin, Carlbring, Cuijpers, & Andersson, 2010). In other words, the participant is aware of the fact that contact with the research team will occur and also knows that it is possible to contact the clinician if needed. This is different from a totally unguided intervention with no contact at all with the researchers. However, some ICBT programs can be accessed without any human contact (Christensen, Griffiths, Groves, & Korten, 2006), and there may be automatic reminders in the system that more or less mimic the role of a therapist.

With regard to SAD, there have been a number of studies in which the role of guidance during the treatment has been studied. Rapee, Abbott, Baillie, and Gaston (2007) found that unguided bibliotherapy for SAD was not effective. As mentioned in the *Shyness 3* study (Titov, Andrews, Choi, et al., 2008), it was found that guided treatment was superior to unguided. For example, more persons in the guided treatment completed all lessons in the program (77%) compared with those in the unguided treatment (33%). Compared to an untreated wait-list control group, the guided treatment was superior ($d = 1.04$), whereas the difference between the unguided treatment and the control group was not statistically significant ($d = 0.38$). The authors did, however, report that persons in the unguided treatment who completed all lessons did improve; but overall, the study clearly showed that guided treatment was superior. In a second study on the same topic—*Shyness 4*—Titov, Andrews, Johnston, et al. (2009) found that unguided treatment enhanced with automatic reminders was inferior to a condition where they phoned participants on a weekly basis. Results showed better adherence in the guided (81%) versus the unguided (56%) groups, and within-group effect sizes were also better in the guided group ($d = 1.15$ vs. $d = 0.86$ in the unguided group). However, it should be noted that improvements in the unguided group were substantial, and that the automatic reminders probably improved both adherence and outcome. Both of the studies by the Titov group included initial contact with the research staff during the assessment.

As mentioned earlier, Berger et al. (2011) randomized 81 participants to either unguided treatment, guided treatment, or flexible support, and found improvements in all three groups. In fact, between-group effect sizes were very small (as mentioned above, the within-group effects were large). However, the unguided treatment involved participation in an online discussion forum, and all participants were in contact with the research staff during the recruitment phase. Boettcher, Berger, and Renneberg (2012b) did a study where they compared the effects of having a telephone interview before the unguided treatment. A total of 109 participants were randomized to either an interview group ($n = 53$) or to a noninterview group who were only screened online with self-reported symptoms ($n = 56$). Overall, outcome did not differ between the groups, with large within-group effect sizes.

In a study by our group, Furmark et al. (2009) investigated the difference between guided ICBT and unguided bibliotherapy, and found no group differences. One possible reason for the large effects of the unguided treatment could be that weekly assessments of SAD symptoms could have served as a motivational enhancer, although this did not affect the waiting-list group in the trial. Another caveat for this review is that it was not pure ICBT, as the unguided treatment consisted of a book sent to the participants; furthermore, this study included contact with the research staff during the assessment phase (telephone interview).

Klein et al. (2011) did a large uncontrolled study on a fully automated open-access program with no contact with a clinician. In that study, 602 individuals commenced their SAD online program; only 50 completed the treatment and the posttreatment assessment. However, only 27 opted to drop out. Within-group effect sizes were moderate for the completers, ranging from $d = 0.85$ for clinical disorder severity ratings to $d = 0.61$ for the Kessler Scale (Kessler et al., 2002). This study is in line with most open-access programs, showing large attrition rates from assessments and probably smaller effects than the controlled studies.

Overall, the controlled studies on unguided treatments for SAD are inconclusive, as there has been contact with clinicians during the assessments and also after the treatment. In one uncontrolled study in which there was no contact at all with the researchers, dropout was substantial. More research is hence needed.

Who is the Therapist?

Given that guidance can boost the effects of ICBT, the question then becomes whether it matters who provides the support. There are some studies in which this question has been investigated. First, a study on the role of therapist factors in ICBT for anxiety disorders including SAD did not identify any significant differences between therapists (Almlöv et al., 2011). Second, Titov and colleagues have investigated if support can be provided from a mainly technical point of view (Johnston, Titov, Andrews, Spence, & Dear, 2011; Robinson et al., 2010). In the *Shyness 6* study mentioned previously, it was found that a nonlicensed technician could provide support with maintained effects (Titov, Andrews, Choi, et al., 2009), and a similar finding was observed in a study on transdiagnostic treatment for a subgroup with SAD (Johnston et al., 2011).

The role of therapist experience was investigated in the SOFIE 6 trial G. Andersson, Carlbring, et al. (2012), and there were no differences between

experienced therapists ($n = 7$) versus therapists with no previous experience of internet treatment ($n = 6$). Overall, these findings indicate that it may be possible to guide a client through ICBT without being an experienced therapist. On the other hand, the therapists in the above-mentioned studies have not been totally inexperienced. In most cases, they were psychologists in training, or had instructions to adhere to the protocol and refrain from using therapeutic advice when guiding the client.

Are Internet-Delivered Treatments as Effective as Face-to-Face Treatment for SAD?

There are at least three randomized controlled trials directly comparing guided ICBT to face-to-face treatment. The first published study in this area was the previously mentioned Spanish trial (Botella et al., 2010). While they focused on fear of public speaking, all had a diagnosis of SAD. There were three groups in the trial, with 62 participants being randomized to ICBT, 36 to live therapy and 29 to a waiting-list group (unbalanced design). Results showed improvements in both treatment groups relative to the control group, with effects being sustained at 12-month follow-up. The between-group effect sizes were small between the two active treatments. A small Australian study compared guided ICBT ($n = 23$) with face-to-face CBT ($n = 14$) (Andrews, Davies, & Titov, 2011), again with unbalanced design. The researchers found large within-group effects and no difference between the two conditions. For example, on the Social Interaction Anxiety Scale (Mattick & Clarke, 1998; see **Chapter 14** for additional details) the between-group difference was $d = 0.00$. The most recent study on live therapy versus ICBT for SAD was conducted by a Swedish group, and this is the largest direct comparison to date (Hedman, Andersson, Ljótsson, Andersson, Rück, Mörtberg, et al., 2011). Participants were randomized to either guided ICBT ($n = 64$) or to cognitive behavioral group therapy (CBGT) ($n = 62$). Results showed that both groups made large improvements that were maintained at 6-month follow-up. At posttreatment and follow-up, Cohen's *d* between-group effect sizes were 0.41 and 0.36, respectively and favored ICBT, although this was not a significant difference. This trial was also followed by a separate report on the cost-effectiveness of ICBT versus live treatment (Hedman, Andersson, Ljótsson, Andersson, Rück, Andersson, et al., 2011). Costs were investigated from a societal perspective, where both direct and indirect costs were included. Results showed that the gross total costs were significantly reduced at 6-month follow-up, compared to pretreatment costs for both treatment conditions. However, since ICBT was associated with less treatment costs, that treatment became more cost-effective. This finding is in line with a previous study by Titov, Andrews, Johnston, et al. (2009), which also included a comparison of the costs of face-to-face treatment (although this latter study was not based on a randomized controlled trial or a societal perspective).

Overall, there are now studies to suggest that guided ICBT can be as effective as face-to-face treatment. The Hedman et al. study contrasted guided ICBT with group therapy (Hedman, Andersson, Ljótsson, Andersson, Rück, Mörtberg, et al., 2011) according to the Heimberg manual (Heimberg & Becker, 2002), and the Andrews et al. (2011) trial also compared ICBT with group treatment. The Botella et al. (2010) trial compared ICBT with individual CBT, but had a focus on public speaking fear. It

is yet unclear how guided ICBT would compare to the Clark et al. (2003) individual treatment, which has had the largest effects published to date, and which may be a more effective treatment than group CBT (Ponniah & Hollon, 2008).

Effectiveness in Real Clinical Settings

There is an imbalance between the number of efficacy and effectiveness trials in the literature on ICBT for SAD, as most studies have been conducted with participants recruited via advertisement. In contrast to face-to-face trials, research participants completing ICBT do not need to live close to the research clinic. It is, however, important to study how well ICBT works when it is implemented in regular care. The group versus internet study by Hedman, Andersson, Ljótsson, Andersson, Rück, Andersson, et al. (2011) was conducted in a regular clinic, in which ICBT was delivered regularly and therapists were working as clinicians in the clinic. Moreover, participants had to be able to come to the clinic, and were recruited from the Stockholm county region. Hence, it is probably accurate to describe this trial as an effectiveness trial (Shadish, Matt, Navarro, & Philips, 2000), even if ICBT is not widely disseminated and the internet psychiatry unit is a specialized treatment facility.

Another much smaller effectiveness study was reported by Aydos et al. (2009). This was an open study with 17 participants who were seen at a regular outpatient clinic. Results of ICBT were in line with the results from the efficacy studies on the *Shyness* program, with within-group effect sizes of $d = 1.06$ and $d = 0.77$ for two SAD measures. Although six patients failed to complete the posttreatment measures, overall findings suggested that ICBT for SAD works in the clinic. This is also in line with our clinical experience and unpublished findings from the internet psychiatry unit in Stockholm, Sweden where a large number of persons have been treated with ICBT.

While there are yet few studies on how ICBT works under clinically representative conditions, there are clear indications from trials on related conditions such as panic disorder (Bergström et al., 2009), and clinical observations, which suggest that ICBT is an effective treatment format.

Moderators of Outcome (What Works for Whom?)

There are few studies on moderators and mediators of outcome in ICBT, as the majority of studies have focused on establishing ICBT as an efficacious treatment. However there are a few studies on predictors, and associations between pretreatment characteristics and outcome are sometimes mentioned in the original reports of randomized trials. Nordgreen et al. (2012) used data from four previously published SOFIE trials and included a total of 245 participants who had received either guided or unguided treatment. The results showed that intensity of baseline SAD symptoms was associated with treatment outcome, in the sense that more symptoms were associated with more room for improvement, whereas less intensive symptoms were associated with higher change of remission. These findings are in line with face-to-face studies. Adherence to unguided treatment was predicted by how high the treatment credibility was rated by participants at pretreatment (Nordgreen et al., 2012), but overall, few treatment-specific findings were found. Hedman et al. (2012) investigated

clinical and genetic predictors and moderators of treatment outcome for their Hedman, Andersson, Ljótsson, Andersson, Rück, Andersson, et al. (2011) trial, where guided ICBT was compared with face-to-face group treatment. As there were two groups in the trial, specific moderators of outcome could be investigated. None of the genetic polymorphisms examined (i.e., 5-HTTLPR, COMTval158met, and BDNF-val66met) were found to predict the outcome in either group. However, working full time (75–100% vs. less work or no work at all), having children, less depressive symptoms, higher expectancy of treatment effectiveness, and greater adherence to treatment predicted positive outcome. Comorbid generalized anxiety disorder and depression were also found to moderate outcome; more specifically, lower levels were associated with better treatment response in ICBT but not in CBGT.

The literature on the process of change and predictors of outcome in ICBT for SAD is too limited to draw any firm conclusions, but it is probably the case that traditional predictors such as the therapeutic alliance (G. Andersson, Paxling, Wiwe, et al., 2012) are not of significant importance in ICBT for SAD. Other aspects such as the number of treatment modules completed are probably more important (G. Andersson et al., 2006).

Discussion

As evident from this chapter, there is a surprising amount of research on the effects of ICBT for SAD, and this is not restricted to efficacy studies only. Indeed, since the first study was published in 2006 (G. Andersson et al., 2006), there have been numerous randomized controlled trials, making ICBT one of the most evidence-based treatments for SAD. One way to interpret the finding that ICBT appears to be as effective as face-to-face CBT in the treatment of SAD is to view it as just being a change in delivery format. Some therapists may, however, then wonder if their training was done all in vain, because therapist skills, albeit not unimportant, play a lesser role in ICBT. We do not believe this to be the case. There are probably specific gains from ICBT for some patients. This could, for example, be that they are probably more relaxed when learning about their conditions (e.g., psychoeducation) and the tasks they are encouraged to perform (e.g., exposure). Moreover, the instructions delivered over the internet are also probably less intrusive and can be paced at the patient's own convenience. But here is also a potential risk with ICBT. Patients may need the extra help from a therapist in session. While we have no data to support this notion, it is rather likely that some patients may be more or less suitable for either format. Finally, the combination of face-to-face sessions and internet treatment is likely to become more commonplace in the future. At the Karolinska internet psychiatry unit, all patients with SAD are seen for assessment at the clinic before starting their internet treatment, which facilitates integration with other face-to-face services (e.g., regular CBT). Using the internet to provide evidence-based psychological treatment is a recent innovation but is likely to stay. The internet may, however, change and we have recently begun investigating the use of Smartphones as an adjunct to ICBT. A second arena for research is the use of social media among persons with SAD. We have not found any clear indications that adding online discussion boards improve the outcomes of ICBT in SAD, but clearly, social media is something to consider

when conducting treatment over the internet. Finally, many patients with SAD suffer from comorbidities (e.g., see **Chapter 10**), and we believe there is a role for tailored interventions to cover those conditions such as mood disorders and insomnia. While research has been done on unified "transdiagnostic" treatments where persons with SAD have been included, there is no study to date on tailored ICBT with the exception of a few patients who have been included in previous trials (Carlbring et al., 2010). In other words it is not clear if persons with SAD with comorbid problems would benefit more from tailored ICBT versus treatment that targets SAD only. On a related note, more research is needed regarding adaption of ICBT for SAD in different target groups such as children and adolescents and older persons.

Conclusions

We conclude that ICBT for SAD is a promising new treatment option and that effects are boosted by guidance from a clinician. The outcomes are in line with what is seen in face-to-face CBT, and effects tend to be stable over long-term follow-up. ICBT for SAD is also a cost-effective treatment option that has been found to work in clinically representative settings. More research is needed, however, on the treatment mechanisms involved in ICBT.

References

Abramowitz, J. S., Moore, E. L., Braddock, A. E., & Harrington, D. L. (2009). Self-help cognitive-behavioral therapy with minimal therapist contact for social phobia: A controlled trial. *Journal of Behavior Therapy and Experimental Psychiatry, 40*, 98–105. doi:10.1016/j.jbtep.2008.04.004

Acarturk, C., Cuijpers, P., van Straten, A., & de Graaf, R. (2009). Psychological treatment of social anxiety disorder: A meta-analysis. *Psychological Medicine, 39*, 241–254. doi:10.1017/S0033291708003590

Almlöv, J., Carlbring, P., Källqvist, K., Paxling, B., Cuijpers, P., & Andersson, G. (2011). Therapist effects in guided internet-delivered CBT for anxiety disorders. *Behavioural and Cognitive Psychotherapy, 39*, 311–322. doi:10.1017/S135246581000069X

Amir, N., Beard, C., Taylor, C. T., Klumpp, H., Elias, J., Burns, M., & Chen, X. (2009). Attention training in individuals with generalized social phobia: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 77*, 961–973. doi:10.1037/a0016685

Andersson, E., Enander, J., Andrén, P., Hedman, E., Ljótsson, B., Hursti, T., . . . Rück, C. (2012). Internet-based cognitive behaviour therapy for obsessive-compulsive disorder: A randomised controlled trial. *Psychological Medicine, 42*(10), 2193–2203. doi:10.1017/S0033291712000244

Andersson, G. (2009). Using the internet to provide cognitive behaviour therapy. *Behaviour Research and Therapy, 47*, 175–180. doi:10.1016/j.brat.2009.01.010.

Andersson, G., Bergström, J., Buhrman, M., Carlbring, P., Holländare, F., Kaldo, V., . . . Waara, J. (2008). Development of a new approach to guided self-help via the internet. The Swedish experience. *Journal of Technology in Human Services, 26*, 161–181. doi:10.1080/15228830802094627

Andersson, G., & Carlbring, P. (2011). Social phobia (social anxiety disorder). In W. T. O'Donohue & C. R. Draper (Eds.), *Stepped care and e-Health: Practical applications to behavioral disorders* (pp. 99–114). New York, NY: Springer.

Andersson, G., Carlbring, P., & Furmark, T.; on behalf of SOFIE Research Group. (2012). Therapist experience and knowledge acquisition in internet-delivered CBT for social anxiety disorder: A randomized controlled trial. *PLoS ONE, 7*(5), e37411. doi:10.1371/journal.pone.0037411

Andersson, G., Carlbring, P., Holmström, A., Sparthan, E., Furmark, T., Nilsson-Ihrfelt, E., . . . Ekselius, L. (2006). Internet-based self-help with therapist feedback and in-vivo group exposure for social phobia: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 74*, 677–686. doi:10.1037/0022-006X.74.4.677

Andersson, G., Paxling, B., Roch-Norlund, P., Östman, G., Norgren, A., Almlöv, J., . . . Silverberg, F. (2012). Internet-based psychodynamic vs. cognitive behavioural guided self-help for generalized anxiety disorder: A randomised controlled trial. *Psychotherapy and Psychosomatics, 81*, 344–355. doi:10.1159/000339371

Andersson, G., Paxling, B., Wiwe, M., Vernmark, K., Bertholds Felix, C., Lundborg, L., . . . Carlbring, P. (2012). Therapeutic alliance in guided internet-delivered cognitive behavioral treatment of depression, generalized anxiety disorder and social anxiety disorder. *Behaviour Research and Therapy, 50*, 544–550. doi:10.1016/j.brat.2012.05.003

Andersson, G., Waara, J., Jonsson, U., Malmaeus, F., Carlbring, P., & Öst, L. G. (2009). Internet-based self-help vs. one-session exposure in the treatment of spider phobia: A randomized controlled trial. *Cognitive Behaviour Therapy, 38*, 114–120. doi:10.1080/16506070902931326

Andrews, G., Cuijpers, P., Craske, M. G., McEvoy, P., & Titov, N. (2010). Computer therapy for the anxiety and depressive disorders is effective, acceptable and practical health care: A meta-analysis. *PLoS ONE, 5*(10), e13196. doi:10.1371/journal.pone.0013196

Andrews, G., Davies, M., & Titov, N. (2011). Effectiveness randomized controlled trial of face to face versus internet cognitive behaviour therapy for social phobia. *Australian and New Zealand Journal of Psychiatry, 45*, 337–340. doi:10.3109/00048674.2010.538840

Aydos, L., Titov, N., & Andrews, G. (2009). Shyness 5: The clinical effectiveness of internet-based clinician-assisted treatment of social phobia. *Australasian Psychiatry, 17*, 488–492. doi:10.1080/10398560903284943

Berger, T., Caspar, F., Richardson, R., Kneubühler, B., Sutter, D., & Andersson, G. (2011). Internet-based treatment of social phobia: A randomized controlled trial comparing unguided with two types of guided self-help. *Behaviour Research and Therapy, 48*, 158–169. doi:10.1016/j.brat.2010.12.007

Berger, T., Hohl, E., & Caspar, F. (2009). Internet-based treatment for social phobia: A randomized controlled trial. *Journal of Clinical Psychology, 65*, 1021–1035. doi:10.1002/jclp.20603

Bergström, J., Andersson, G., Karlsson, A., Andreewitch, S., Rück, C., Carlbring, P., & Lindefors, N. (2009). An open study of the effectiveness of internet treatment for panic disorder delivered in a psychiatric setting. *Nordic Journal of Psychiatry, 63*, 44–50. doi:10.1080/08039480802191132

Bergström, J., Andersson, G., Ljótsson, B., Rück, C., Andréewitch, S., Karlsson, A., . . . Lindefors, N. (2010). Internet- versus group-administered cognitive behaviour therapy for panic disorder in a psychiatric setting: A randomised trial. *BMC Psychiatry, 10*, 54. doi:10.1186/1471-244X-10-54

Boettcher, J., Berger, T., & Renneberg, B. (2012a). Internet-based attention training for social anxiety: A randomized controlled trial. *Cognitive Therapy and Research, 36*(5), 522–536. doi:10.1007/s10608-011-9374-y

Boettcher, J., Berger, T., & Renneberg, B. (2012b). Does a pre-treatment diagnostic interview affect the outcome of internet-based self-help for social anxiety disorder? A randomized

controlled trial. *Behavioural and Cognitive Psychotherapy, 40*(5), 513–528. doi: 10.1017/S1352465812000501

Botella, C., Gallego, M. J., Garcia-Palacios, A., Guillen, V., Banos, R. M., Quero, S., & Alcaniz, M. (2010). An internet-based self-help treatment for fear of public speaking: A controlled trial. *Cyberpsychology, Behavior and Social Networking, 13*, 407–421. doi:10.1089/cyber.2009.0224

Carlbring, P., Apelstrand, M., Sehlin, H., Amir, N., Rousseau, A., Hofmann, S., & Andersson, G. (2012). Internet-delivered attention training in individuals with social anxiety disorder—a double blind randomized controlled trial. *BMC Psychiatry, 12*, 66. doi:10.1186/1471-244X-12-66

Carlbring, P., Bergman-Nordgren, L., Furmark, T., & Andersson, G. (2009). Long term outcome of internet delivered cognitive-behavioural therapy for social anxiety disorder: A 30-month follow-up. *Behaviour Research and Therapy, 47*, 848–850. doi:10.1016/j.brat.2009.06.012

Carlbring, P., Bohman, S., Brunt, S., Buhrman, M., Westling, B. E., Ekselius, L., & Andersson, G. (2006). Remote treatment of panic disorder: A randomized trial of internet-based cognitive behavioral therapy supplemented with telephone calls. *American Journal of Psychiatry, 163*, 2119–2125. doi:10.1176/appi.ajp.163.12.2119

Carlbring, P., Ekselius, L., & Andersson, G. (2003). Treatment of panic disorder via the internet: A randomized trial of CBT vs. applied relaxation. *Journal of Behavior Therapy and Experimental Psychiatry, 34*, 129–140. doi:10.1016/S0005-7916(03)00026-0

Carlbring, P., Gunnarsdóttir, M., Hedensjö, L., Andersson, G., Ekselius, L., & Furmark, T. (2007). Treatment of social phobia: Randomized trial of internet delivered cognitive behaviour therapy and telephone support. *British Journal of Psychiatry, 190*, 123–128. doi:10.1192/bjp.bp.105.020107

Carlbring, P., Maurin, L., Törngren, C., Linna, E., Eriksson, T., Sparthan, E., . . . Andersson, G. (2010). Individually tailored internet-based treatment for anxiety disorders: A randomized controlled trial. *Behaviour Research and Therapy, 49*, 18–24. doi:10.1016/j.brat.2010.10.002

Christensen, H., Griffiths, K., Groves, C., & Korten, A. (2006). Free range users and one hit wonders: Community users of an internet-based cognitive behaviour therapy program. *Australian and New Zealand Journal of Psychiatry, 40*, 59–62. doi:10.1111/j.1440-1614.2006.01743.x

Clark, D. M., Ehlers, A., McManus, F., Hackmann, A., Fennell, M., Campbell, H., . . . Louis, B. (2003). Cognitive therapy versus fluoxetine in generalized social phobia: A randomized placebo-controlled trial. *Journal of Consulting and Clinical Psychology, 71*, 1058–1067. doi:10.1037/0022-006X.71.6.1058

Clark, D. M., & Wells, A. (1995). A cognitive model of social phobia. In R. G. Heimberg, M. Leibowitz, D. A. Hope & F. R. Schneider (Eds.), *Social phobia: Diagnosis, assessment and treatment* (pp. 63–93). New York, NY: Guilford press.

Cuijpers, P., van Straten, A. M., & Andersson, G. (2008). Internet-administered cognitive behavior therapy for health problems: A systematic review. *Journal of Behavioral Medicine, 31*, 169–177. doi:10.1007/s10865-007-9144-1

Erwin, B. A., Turk, C. L., Heimberg, R. G., Fresco, D. M., & Hantula, D. A. (2004). The internet: Home to a severe population of individuals with social anxiety disorder? *Journal of Anxiety Disorders, 18*, 629–646.

Farvolden, P., Denisoff, E., Selby, P., Bagby, R. M., & Rudy, L. (2005). Usage and longitudinal effectiveness of a web-based self-help cognitive behavioral therapy program for panic disorder. *Journal of Medical Internet Research, 7*(1), e7. doi:10.2196/jmir.7.1.e7

Furmark, T., Carlbring, P., Hedman, E., Sonnenstein, A., Clevberger, P., Bohman, B., . . . Andersson, G. (2009). Guided and unguided self-help for social anxiety disorder: Randomised controlled trial. *British Journal of Psychiatry, 195*, 440–447. doi:10.1192/bjp.bp.108.060996

Hedman, E., Andersson, E., Ljótsson, B., Andersson, G., Andersson, E. M., Schalling, M., . . . Rück, C. (2012). Clinical and genetic outcome determinants of internet- and group-based cognitive behavior therapy for social anxiety disorder. *Acta Psychiatrica Scandinavica, 126*, 126–136. doi:10.1111/j.1600-0447.2012.01834.x

Hedman, E., Andersson, E., Ljótsson, B., Andersson, G., Rück, C., & Lindefors, N. (2011). Cost-effectiveness of internet-based cognitive behavior therapy vs. cognitive behavioral group therapy for social anxiety disorder: Results from a randomized controlled trial. *Behaviour Research and Therapy, 49*, 729–736. doi:10.1016/j.brat.2011.07.009

Hedman, E., Andersson, G., Ljótsson, B., Andersson, E., Rück, C., Asmundson, G. J. G., & Lindefors, N. (2011). Internet-based cognitive-behavioural therapy for severe health anxiety: Randomised controlled trial. *British Journal of Psychiatry, 198*, 230–236. doi:10.1192/bjp.bp.110.086843

Hedman, E., Andersson, G., Ljótsson, B., Andersson, E., Rück, C., Mörtberg, E., & Lindefors, N. (2011). Internet-based cognitive behavior therapy vs. cognitive behavioral group therapy for social anxiety disorder: A randomized controlled non-inferiority trial. *PLoS ONE, 6*(3), e18001. doi:10.1371/journal.pone.0018001

Hedman, E., Furmark, T., Carlbring, P., Ljótsson, B., Rück, C., Lindefors, N., & Andersson, G. (2011). Five-year follow-up of internet-based cognitive behaviour therapy for social anxiety disorder. *Journal of Medical Internet Research, 13*(2), e39. doi:10.2196/jmir.1776

Hedman, E., Ljótsson, B., Rück, C., Furmark, T., Carlbring, P., Lindefors, N., & Andersson, G. (2010). Internet administration of self-report measures commonly used in research on social anxiety disorder: A psychometric evaluation. *Computers in Human Behavior, 26*, 736–740. doi:10.1016/j.chb.2010.01.010

Heimberg, R. G., & Becker, R. E. (2002). *Cognitive-behavioral group therapy for social phobia. Basic mechanisms and clinical strategies.* New York, NY: Guilford Press.

Heimberg, R. G., Salzman, D. G., Holt, C. S., & Blendell, K. A. (1993). Cognitive-behavioral group treatment for social phobia: Effectiveness at five-year follow-up. *Cognitive Therapy and Research, 17*, 325–339. doi:10.1007/BF01177658

Hirai, M., Vernon, L. L., Clum, G. A., & Skidmore, S. T. (2011). Psychometric properties and administration measurement invariance of social phobia symptom measures: Paper-pencil vs. internet administrations. *Journal of Psychopathology and Behavioral Assessment, 33*, 470–479. doi:10.1007/s10862-011-9257-2

Johansson, R., & Andersson, G. (2012). Internet-based psychological treatments for depression. *Expert Review of Neurotherapeutics, 12*(7), 861–869.

Johansson, R., Ekbladh, S., Hebert, A., Lindström, M., Möller, S., Petitt, E., . . . Andersson, G. (2012). Psychodynamic guided self-help for adult depression through the internet: A randomised controlled trial. *PLoS ONE, 7*(5), e38021. doi:10.1371/journal.pone.0038021

Johnston, L., Titov, N., Andrews, G., Spence, J., & Dear, B. F. (2011). A RCT of a transdiagnostic internet-delivered treatment for three anxiety disorders: Examination of support roles and disorder-specific outcomes. *PLoS ONE, 6*(11), e28079. doi:10.1371/journal.pone.0028079 PONE-D-11-04313

Kessler, R., Andrews, G., Colpe, L., Hiripi, E., Mroczek, D., Walters, E. E., & Zaslavsky, A. M. (2002). Short screening scales to monitor population prevalences and trends in non-specific psychological distress. *Psychological Medicine, 32*, 959–976. doi:10.1017/S0033291702006074

Khazaal, Y., Fernandez, S., Cochand, S., Reboh, I., & Zullino, D. (2008). Quality of web-based information on social phobia: A cross-sectional study. *Depression and Anxiety, 25*, 461–465. doi:10.1002/da.20381

Klein, B., Meyer, D., Austin, D. W., & Kyrios, M. (2011). Anxiety online—A virtual clinic: Preliminary outcomes following completion of five fully automated treatment programs for anxiety disorders and symptoms. *Journal of Medical Internet Research, 13*, e89. doi:10.2196/jmir.1918

Lee, B. W., & Stapinski, L. A. (2012). Seeking safety on the internet: Relationship between social anxiety and problematic internet use. *Journal of Anxiety Disorders, 26*, 197–205. doi:10.1016/j.janxdis.2011.11.001

Marks, I., Shaw, S., & Parkin, R. (1998). Computer-assisted treatments of mental health problems. *Clinical Psychology: Science and Practice, 5*, 151–170. doi:10.1111/j.1468-2850.1998.tb00141.x

Marks, I. M., Cavanagh, K., & Gega, L. (2007). *Hands-on help: Computer-aided psychotherapy* [Maudsley Monograph no. 49]. Hove, UK: Psychology Press.

Mattick, R. P., & Clarke, J. C. (1998). Development and validation of measures of social phobia scrutiny fear and social interaction anxiety. *Behaviour Research and Therapy, 36*, 455–470. doi:10.1016/S0005-7967(97)10031-6

Nordgreen, T., Havik, O. E., Öst, L. G., Furmark, T., Carlbring, P., & Andersson, G. (2012). Outcome predictors in guided and unguided self-help for social anxiety disorder. *Behaviour Research and Therapy, 50*, 13–21. doi:10.1016/j.brat.2011.10.009

Nordin, S., Carlbring, P., Cuijpers, P., & Andersson, G. (2010). Expanding the limits of bibliotherapy for panic disorder. Randomized trial of self-help without support but with a clear deadline. *Behavior Therapy, 41*, 267–276. doi:10.1016/j.beth.2009.06.001

Öst, L. G. (1987). Applied relaxation: Description of a coping technique and review of controlled studies. *Behaviour Research and Therapy, 25*, 379–409. doi:10.1016/0005-7967(87)90017-9

Paxling, B., Almlöv, J., Dahlin, M., Carlbring, P., Breitholtz, E., Eriksson, T., & Andersson, G. (2011). Guided internet-delivered cognitive behavior therapy for generalized anxiety disorder: A randomized controlled trial. *Cognitive Behaviour Therapy, 40*, 159–173. doi:10.1080/16506073.2011.576699

Ponniah, K., & Hollon, S. D. (2008). Empirically supported psychological interventions for social phobia in adults: A qualitative review of randomized controlled trials. *Psychological Medicine, 38*, 3–14. doi:110.1017/S00332911707000918

Rapee, R. M., Abbott, M. J., Baillie, A. J., & Gaston, J. E. (2007). Treatment of social phobia through pure self-help and therapist-augmented self-help. *British Journal of Psychiatry, 191*, 246–252. doi:10.1192/bjp.bp.106.028167

Ritterband, L. M., Andersson, G., Christensen, H. M., Carlbring, P., & Cuijpers, P. (2006). Directions for the International Society for Research on Internet Interventions (ISRII). *Journal of Medical Internet Research, 8*, e23. doi:10.2196/jmir.8.3.e23

Robinson, E., Titov, N., Andrews, G., McIntyre, K., Schwencke, G., & Solley, K. (2010). Internet treatment for generalized anxiety disorder: A randomized controlled trial comparing clinician vs. technician assistance. *PLoS ONE, 5*, e10942. doi:10.1371/journal.pone.0010942

Schmidt, N. B., Richey, J. A., Buckner, J. D., & Timpano, K. R. (2009). Attention training for generalized social anxiety disorder. *Journal of Abnormal Psychology, 118*, 5–14. doi:10.1037/a0013643

Shadish, W. R., Matt, G. E., Navarro, A. M., & Philips, G. (2000). The effects of psychological therapies under clinically representative conditions: A meta-analysis. *Psychological Bulletin, 126*, 512–529. doi:10.1037//0033-2909.126.4.512

Spence, J., Titov, N., Dear, B. F., Johnston, L., Solley, K., Lorian, C., . . . Schwencke, G. (2011). Randomized controlled trial of internet delivered cognitive behavioural therapy for post-traumatic stress disorder. *Depression and Anxiety, 28*, 541–550. doi:10.1002/da.20835

Tillfors, M., Andersson, G., Ekselius, L., Furmark, T., Lewenhaupt, S., Karlsson, A., & Carlbring, P. (2011). A randomized trial of internet delivered treatment for social anxiety disorder in high school students. *Cognitive Behaviour Therapy, 40*, 147–157. doi:10.1080/16506073.2011.555486

Tillfors, M., Carlbring, P., Furmark, T., Lewenhaupt, S., Spak, M., Eriksson, A., . . . Andersson, G. (2008). Treating university students with social phobia and public speaking fears: Internet delivered self-help with or without live group exposure sessions. *Depression and Anxiety, 25*, 708–717. doi:10.1002/da.20416

Titov, N., Andrews, G., Choi, I., Schwencke, G., & Johnston, L. (2009). Randomized controlled trial of web-based treatment of social phobia without clinical guidance. *Australian and New Zealand Journal of Psychiatry, 43*, 913–919. doi:10.1080/00048670903179160

Titov, N., Andrews, G., Choi, I., Schwencke, G., & Mahoney, A. (2008). Shyness 3: Randomized controlled trial of guided versus unguided internet-based CBT for social phobia. *The Australian and New Zealand Journal of Psychiatry, 42*, 1030–1040. doi:10.1080/00048670802512107

Titov, N., Andrews, G., Johnston, L., Robinson, E., & Spence, J. (2010). Transdiagnostic internet treatment for anxiety disorders: A randomized controlled trial. *Behaviour Research and Therapy, 48*(9), 890–899. doi:10.1016/j.brat.2010.05.014

Titov, N., Andrews, G., Johnston, L., Schwencke, G., & Choi, I. (2009). Shyness programme: Longer term benefits, cost-effectiveness, and acceptability. *The Australian and New Zealand Journal of Psychiatry, 43*, 36–44.

Titov, N., Andrews, G., Robinson, E., Schwencke, G., Johnston, L., Solley, K., & Choi, I. (2009). Clinician-assisted internet-based treatment is effective for generalized anxiety disorder: Randomized controlled trial. *Australian and New Zealand Journal of Psychiatry, 43*, 905–912. doi:10.1080/00048670903179269

Titov, N., Andrews, G., & Schwencke, G. (2008). Shyness 2: Treating social phobia online: Replication and extension. *The Australian and New Zealand Journal of Psychiatry, 42*, 595–605. doi:10.1080/00048670802119820

Titov, N., Andrews, G., Schwencke, G., Drobny, J., & Einstein, D. (2008). Shyness 1: Distance treatment of social phobia over the internet. *The Australian and New Zealand Journal of Psychiatry, 42*, 585–594. doi:10.1080/00048670802119762

Titov, N., Andrews, G., Schwencke, G., Robinson, E., Peters, L., & Spence, J. (2010). Randomized controlled trial of internet cognitive behavioural treatment for social phobia with and without motivational enhancement strategies. *Australian and New Zealand Journal of Psychiatry, 44*, 938–945. doi:10.3109/00048674.2010.493859

Titov, N., Gibson, M., Andrews, G., & McEvoy, P. (2009). Internet treatment for social phobia reduces comorbidity. *The Australian and New Zealand Journal of Psychiatry, 43*(8), 754–759. doi:10.1080/00048670903001992

Tulbure, B. T. (2011). The efficacy of internet-supported intervention for social anxiety disorder: A brief meta-analytic review. *Procedia—Social and Behavioral Sciences, 30*, 552–557. doi:10.1016/j.sbspro.2011.10.108

Watkins, P. L., & Clum, G. A. (Eds.). (2008). *Handbook of self-help therapies*. New York, NY: Routledge.

Yen, J. Y., Yen, C. F., Chen, C. S., Wang, P. W., Chang, Y. H., & Ko, C. H. (2012). Social anxiety in online and real-life interaction and their associated factors. *Cyberpsychology, Behavior, and Social Networking, 15*, 7–12. doi:10.1089/cyber.2011.0015

Appendix: Description of the SAD Internet Treatment Used in the Swedish Studies

The SOFIE treatment consists of 186 pages and is divided into nine modules. The first module introduces the program and portrays SAD, its symptoms, and proposed possible etiological factors; and describes facts about CBT. The second module outlines a model for SAD (Clark & Wells, 1995), and the relationship between thoughts, feelings, behavior, and cognitive symptoms. It also defines automatic thoughts and explains how to register them. The third module provides a basic outline of thinking errors/cognitive distortions, the registration of automatic thoughts, and information about how to challenge these. Work with automatic thoughts continues in the fourth module and behavioral experiments are introduced. Formulation of specific therapeutic goals is also included in this module. The fifth module covers the principles behind exposure and reality testing while the sixth module concerns self-focus, shifting of focus, attention training, and safety behaviors. The seventh module continues the previous work with exposure. It focuses on problems that are commonly encountered during exposure and suggests behavioral experiments. The eighth module concerns the art of listening and conversing, nonverbal communication, the ability to say "no," and assertiveness (social skills). The final module informs the reader about the role of perfectionism, procrastination, and self-confidence as well as relapse prevention. The entire program is then summarized.

Each module includes information and exercises, and ends with three to eight essay questions. Participants are asked to: explain, in their own words, the most important sections of the module they have just completed; provide thought records; and describe their experience with and outcome of their exposure exercises. The questions are intended to promote learning and to enable the online therapists to assess whether the participants have assimilated the material, and completed their homework. Also included in each module is a multiple-choice quiz that the participants need to get 95% correct in order to proceed. Finally, in each module the participants are required to post a message in a discussion forum about a specific topic.

Personal feedback on the homework is usually given within 24 hours after participants have sent their answers via e-mail. On the basis of these e-mails, an assessment is made of whether the participant is ready to continue; if so, the password to the next module is sent. If not, the participant receives instructions on what needs to be completed before proceeding to the next module.

27

Acceptance and Mindfulness-Based Therapies for Social Anxiety Disorder

Current Findings and Future Directions

James D. Herbert, Marina Gershkovich, and Evan M. Forman

Drexel University, USA

Once labeled "the neglected anxiety disorder" (Liebowitz et al., 1985) due to the paucity of research attention it had received relative to other anxiety and mood disorders, social anxiety disorder (SAD; also known as social phobia) is certainly no longer neglected. The past three decades have witnessed a great deal of work focused on describing, understanding, assessing, and treating this common condition. Much progress has been made, with scientific consensus emerging around several themes. For example, it is generally accepted that both the degree of functional impairment and the number of phobic situations occur dimensionally rather than categorically. The content of social anxiety-related cognitions is well documented. Both cognitive behavior therapy (CBT) programs and antidepressant medications show short-term benefit, with the former also demonstrating strong maintenance of gains over follow-up assessments. In fact, there is a perception by some that the progress in treating SAD has been so successful that the condition no longer represents an especially high priority for clinical research efforts.

Despite these advances, if we step back and evaluate the state of the field, it quickly becomes apparent that we really know very little about SAD and its treatment, and that much work remains to be done. Many studies of SAD have excluded patients with common comorbid conditions, including mood disorders, other anxiety disorders, and substance abuse, so little is known about how best to treat such individuals. Attrition is unacceptably high. For example, Hofmann and Suvak (2006) found attrition of 25.6% in one study, and attrition of up to 45% has been reported in other studies (Ossman, Wilson, Storaasli, & McNeill, 2006). Even when treated, many patients fail to recover fully (Hofmann & Bögels, 2006; Juster & Heimberg, 1995; Turner, Beidel, & Wolff, 1994). And even after successful treatment, ratings of quality of life typically show

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

only limited improvement, and do not reach normative levels (Eng, Coles, Heimberg, & Safren, 2001, 2005). Relapse is especially problematic following discontinuation of pharmacotherapy (Haug et al., 2003; Stein, Versiani, Hair, & Kumar, 2002; Walker et al., 2000).

Moreover, even when treatment is effective, little is known about specific active ingredients, nor about the mechanisms or processes that are actually driving change. The neurobiological underpinnings and the mechanisms of pharmacotherapy for SAD are poorly understood, including even issues as fundamental as how much of the variance is attributable to psychological expectancies and how much is actually associated with biochemical factors. Moreover, recent findings are raising disturbing questions about the widespread long-term use of pharmacotherapy more generally (Whitaker, 2010). Established CBT programs consist of multicomponent packages, and the specific treatment components responsible for their effects remain unclear.

In addition, most patients with SAD do not receive any treatment at all, much less a scientifically supported, evidence-based psychotherapy. In fact, more than 80% of persons with SAD receive no treatment whatsoever, more than other anxiety or mood disorders (Grant et al., 2005). Coles, Turk, Jindra, and Heimberg (2004) similarly reported that only 15% of individuals who initially contacted an anxiety disorders specialty clinic actually proceeded to obtain treatment. There are a number of reasons that so many individuals with SAD do not obtain treatment, including failure to disseminate successfully evidence-based treatments to many front-line providers, the absence of clinicians with specific expertise in the disorder in many communities, and the very nature of the disorder itself, in which concerns over social evaluation paradoxically prevent many from seeking treatment (Nelson & Velasquez, 2011; Olfson et al., 2000).

Thus, despite advances over the past 30 years, there remains much work to be done in understanding, treating, and ultimately preventing SAD. In this chapter we focus on one area that we believe represents an especially promising area for further development: the incorporation of mindfulness principles and intervention strategies into CBT, and into the CBT treatment of SAD in particular.

CBT programs are by far the most studied and best-established treatments for SAD. CBT is an umbrella term that represents a broad family of psychotherapy models that are rooted in learning theory, incorporate behavioral interventions, address cognition in one way or another, and share a commitment to empirical evaluation. In fact, the mindfulness-based models themselves can be thought of as newer members of the CBT family (Herbert & Forman, 2011). Traditional CBT programs posit that negative cognitions are the root cause of SAD, and intervention efforts focus largely on various cognitive restructuring strategies and techniques (Jørstad-Stein & Heimberg, 2009). Nevertheless, it remains unclear to what degree negative cognitions cause social anxiety and avoidance, or rather simply represent concurrent symptoms. Studies of cognitive mediation have been equivocal, with some positive findings (Hofmann, 2004) and other null findings (McManus, Clark, & Hackmann, 2000). Moreover, component control studies have generally not supported the idea that direct cognitive change strategies, which represent the distinctive feature of traditional CBT programs for SAD, accrue incremental benefits beyond standard behavioral interventions, which all CBT programs include (Dimidjian et al., 2006; Fedoroff & Taylor, 2001; Gould,

Buckminster, Pollack, Otto, & Yap, 1997). For example, two meta-analyses (Feske & Chambless, 1995; Powers, Sigmarsson, & Emmelkamp, 2008) found no differences between exposure-only programs and those that combined exposure with cognitive restructuring.

The Emergence of Mindfulness-Based Perspectives

Despite the dominance of traditional CBT programs in the treatment of SAD, the equivocal findings regarding cognitive interventions and treatment processes, along with theoretical questions about the best way to approach cognition with respect to behavior change technologies, have caused some scholars to explore if other theoretical models and treatment strategies might be fruitful. In particular, emerging work has focused on strategies that incorporate mindful awareness and psychological acceptance of distressing symptoms in the service of behavior change.

The concept of mindfulness, which dates back to ancient Hindu and Buddhist traditions, has been discussed by psychologists from various perspectives over the past century (Williams & Lynn, 2010), but only gained significant traction within the CBT community over the past decade (Herbert & Forman, 2011, 2013). Consensus has yet to emerge on a single definition of mindfulness, although the most commonly cited description was offered by Jon Kabat-Zinn (1994, p. 4): "paying attention in a particular way: on purpose, in the present moment, and nonjudgmentally." This definition highlights three key features of the concept: the active, intentional embracing of experience rather than mere passive acquiescence; a heightened sense of awareness of ongoing psychological experience; and acceptance of that experience fully and nonjudgmentally, without attempting to escape or modify it.

Within CBT, in contrast to cultural and spiritual traditions from which it was derived, mindfulness is a means to an end, rather than an end in and of itself (Herbert, Forman, & England, 2009). The ultimate goal is behavior change and, more broadly, living a meaningful, fulfilled life. Secular mindfulness-based CB therapists share the hypothesis that efforts to enhance mindfulness, or at least some of the constituents of mindfulness, will foster these broader goals. In addition, mindfulness is best viewed as a psychological state, rather than any particular method designed to foster that state (Marchand, 2012). This is a common point of confusion, as mindfulness is often used synonymously with contemplative meditative practices. Although meditation is a common strategy to foster mindfulness, it is by no means the only such strategy.

Newer mindfulness- and acceptance-oriented approaches to CBT integrate aspects of mindfulness with the values, clinical strategies, and techniques of traditional behavior therapy. Although none of these approaches was originally developed for SAD in particular, several have recently been applied to the treatment of the condition.

Mindfulness-based approaches in general can be grouped into two broad categories: (a) meditation-based programs, which include but are not limited to metacognitive approaches that remain grounded in traditional cognitive theories, and (b) modern behavior analytic (or contextual behavioral science) approaches that adopt fundamentally different assumptions and methods. What these approaches share is a de-emphasis on direct attempts to change negative thoughts and beliefs about specific social

situations, and a corresponding emphasis on promoting behavior change despite distressing cognition and affect.

Meditation-Based Programs

Mindfulness-Based Stress Reduction

The first modern program to bring mindfulness sensitivities to Western medicine and psychotherapy was mindfulness-based stress reduction (MBSR; Kabat-Zinn, 1990). The program was originally developed as a treatment for chronic pain (Kabat-Zinn, 2005), but has since been expanded to a number of different areas. MBSR is a transdiagnostic, weekly, group-based program that primarily focuses on mindfulness meditation training. Various mindfulness-fostering exercises, including meditation, body scanning, and Hatha yoga are conducted in session and for homework. Psychological distress (including anxiety and depression) is understood as reactions to one's internal experience. MBSR has been shown to reduce anxiety in a wide range of nonclinical and clinical populations (for reviews, see Baer, 2003; Vøllestad, Nielsen, & Nielsen, 2012). MBSR has been associated with decreases in emotional reactivity (Ramel, Goldin, Carmona, & McQuaid, 2004) and improvements in self-regulation (Lykins & Baer, 2009). MBSR has been found to be more effective than wait-list control conditions (Fjorback, Arendt, Ornbol, Fink, & Walach, 2011). However, as reviewed below, its efficacy in the treatment of specific anxiety disorders, including SAD, appears to be mixed.

The effect of MBSR on patients with anxiety disorders was recently investigated in a randomized trial comparing MBSR to a wait-list control (Vøllestad, Sivertsen, & Nielsen, 2011). The sample was heterogeneous and consisted of individuals with panic disorder with or without agoraphobia, SAD, and generalized anxiety disorder. The intervention was delivered in groups and consisted of eight weekly sessions. The individuals who completed the intervention showed improvements on all outcome measures, including anxiety (moderate to large effect sizes, $d = 0.53–0.97$) and depression. Furthermore, treatment gains were maintained at 6-month follow-up. There were, however, several limitations to the study. Although the findings were promising, the absence of active comparison conditions and behavioral measures limit any conclusions that can be drawn about specific efficacy for SAD.

There is some evidence to suggest that MBSR may be effective in interrupting ingrained and maladaptive self-views that are often characteristic of SAD. In a unique neural imaging study, Goldin, Ramel, and Gross (2009) explored neurological effects of MBSR training in adults with SAD. Patients demonstrated an increase in attention regulation and a decrease in negative self-referential processing as measured by neural activity in an fMRI task. In a different study that involved a breath-focusing task in the fMRI scanner, MBSR training was similarly associated with reduced emotional reactivity and enhanced emotional regulation (Goldin & Gross, 2010).

In a randomized trial of MBSR specifically targeting generalized SAD, Koszycki, Benger, Shlik, and Bradwejn (2007) compared the efficacy of 8 weekly sessions of MBSR to a gold-standard treatment of 12 weekly sessions of cognitive-behavioral

group therapy (CBGT; Heimberg & Becker, 2002). Participants in both treatment groups experienced clinically significant improvement in measures of anxiety, mood, and quality of life. The attrition rates in the two groups were equivalent and suggest similar acceptability. The CBGT condition, however, had greater decreases on measures of social anxiety, specifically fear and avoidance, as measured by clinician and self-report. Additionally, remission rates were significantly greater with CBGT. The authors concluded that CBGT remains the treatment of choice for SAD, as it was more effective in decreasing symptoms of SAD; MBSR may be better utilized as a supplement to standard CBT.

In a recent comparative trial, participants with SAD were randomized to receive 8 weeks of MBSR or aerobic exercise (Jazaieri, Goldin, Werner, Ziv, & Gross, 2012). The authors speculated that the exercise condition would be an especially appropriate active comparison group to assess the efficacy of MBSR, as it involves an active intervention that does not have overlapping "active ingredients" with MBSR. Both groups experienced comparable improvements on measures of social anxiety and well-being, with effects maintained at 3-month follow-up. Following the intervention, 22.5% of participants in the MBSR group met threshold for clinical significance on the LSAS-SR relative to 29.5% of those in the AE group; however, this difference was not significant. The authors hypothesize that the aerobic exercise condition was more socially active than originally anticipated, as participants were engaging in social situations by attending the designated facility.

In summary, preliminary research suggests that MBSR has positive effects on SAD, but may not be as effective as the best existing treatments. It is possible that the approach may be useful for some patients as an emotion regulation strategy, but the absence of structured exposure exercises may limit its efficacy with respect to SAD. Future research should explore the integration of MBSR, or at least elements of MBSR, within CBT programs.

Although MBSR originally developed outside the larger CBT tradition, other metacognitive models are extensions of traditional cognitive approaches to CBT, most notably the work of Beck and colleagues (Beck, 1976). These models emphasize metacognition, or beliefs about the function of other cognitions, rather than the content of specific thoughts and beliefs. The two most prominent models are metacognitive therapy (MCT; Wells, 2008, 2011), and mindfulness-based cognitive therapy (MBCT; Segal, Williams, & Teasdale, 2001).

Metacognitive Therapy

MCT distinguishes two types of metacognitive beliefs: positive metacognitive beliefs, which refer to the presumed benefits of monitoring and controlling negative thoughts, and negative metacognitive beliefs, which concern beliefs about the danger of certain thoughts and the uncontrollability of experience. Biases in both types of beliefs lead to emotional dysregulation. Individuals can overreact to transient negative thoughts and feelings through maladaptive efforts to control their experience, setting up a pattern of intense rumination and self-focused attention. MCT does not target specific negative thoughts, but rather attempts to change these metacognitive beliefs by means of various strategies, including mindfulness-oriented techniques. Wells and King (2006)

reported large effects in an open trial of MCT for generalized anxiety disorder. Wells (2007) discussed the application of MCT to SAD, but no data have been presented to date.

Mindfulness-Based Cognitive Therapy

MBCT was originally developed as a treatment to prevent the relapse of chronic depression (Segal, Williams, & Teasdale, 2001). The approach developed from the observation that individuals susceptible to depression, relative to those who were not, experience mildly depressive situations with patterns of greater negative thinking and affect (Teasdale et al., 2002). In addition, the inability to describe one's experience from a more neutral, detached perspective, which the authors term *metacognitive awareness*, also predicted depressive relapse among at-risk individuals. MBCT aims to foster an open, detached perspective on one's experience, and emphasizes mindfulness meditation practices, many of which are based on MBSR. MBCT has recently been applied to generalized anxiety disorder in particular (Craigie, Rees, Marsh, & Nathan, 2008; Evans et al., 2008; Kim et al., 2009; Semple & Lee, 2008; Wong et al., 2011).

Two studies have evaluated MBCT programs for SAD (Bögels, Sijbers, & Voncken, 2006; Piet, Hougaard, Hecksher, & Rosenberg, 2010). Bögels et al. (2006) conducted a small pilot study ($n = 9$) evaluating the combination of MBCT and task concentration training (i.e., teaching patients how to redirect attention away from internal thoughts and feelings and toward the task at hand) for SAD. Treatment consisted of nine individual sessions delivered weekly. The results were promising, with a pre- to posttreatment effect size of $d = 0.85$ on self-report measures of social anxiety. The improvements were maintained at 2-month follow-up. In another pilot study, Piet et al. (2010) randomly assigned young adults (ages 18–25) with SAD to receive sequential courses of 8 group sessions of MBCT and 12 sessions of group CBT, in counterbalanced order, using a cross-over design. Overall, MBCT resulted in moderately large gains ($d = 0.78$), which were somewhat smaller, although not significantly different from, those produced by CBT ($d = 1.15$). However, these group trends may simply be a result of differences in the number of sessions between the two conditions.

The results of these studies are consistent with the conclusions of two recent meta-analyses of mindfulness-based interventions for anxiety disorders. Hofmann, Sawyer, Witt, and Oh (2010) examined the efficacy of 39 mindfulness-based interventions (based on either MBSR or MBCT) for a range of psychological and medical conditions, focusing on measures of mood and anxiety. Among patients with anxiety disorders, a large within-group mean effect size was reported (Hedges' $g = 0.97$). Hofmann et al. concluded that the positive effects of mindfulness-based interventions for various mood and anxiety disorders, among other conditions, suggest that they may target transdiagnostic rather than disorder-specific processes. A second meta-analysis was recently conducted by Vøllestad, Nielsen, and Nielsen (2012), and focused not only on meditation-based programs such as MBSR and MBCT, but also on multicomponent intervention programs such as acceptance and commitment therapy (described below). They identified 19 studies of samples meeting diagnostic criteria for anxiety disorders. Mean treatment-related effect sizes for anxiety symptoms were large (Hedges' $g = 1.08$). Interestingly, neither of these meta-analyses found significant

moderating effects for factors such as specific intervention type, dosage, or study design, although Vøllestad et al. observed a trend toward larger effects of individual over group treatment.

Modern Behavior Analytic Approaches

A more fundamental break with traditional, cognitively oriented CBT models is represented by modern behavior analytic approaches. Contrary to popular misconception, the branch of modern behavior analysis known as contextual behavioral science recognizes the important role of cognitive processes in human behavior, and in fact argues that earlier behavior analytic perspectives did not sufficiently appreciate the importance of verbal/cognitive factors. Unlike traditional cognitive theories, however, this approach emphasizes the environmental contexts (both historical and current) that give rise to cognitions, as well as the contexts that result in cognitions influencing other behaviors. Thinking, sensing, perceiving, believing, and remembering are all considered behaviors, broadly conceptualized as things that an intact organism does. Behaviors (including these private or covert behaviors) may participate in causal relationships with other behaviors, but these relationships are themselves contextually controlled. For example, in some environmental contexts, thoughts of harm may contribute to behavioral avoidance, whereas in other contexts, these same thoughts may not do so. At first glance, this perspective may not seem fundamentally different from metacognitive models that focus on metabeliefs about the truth value, dangerousness, and/or necessity to change negative thoughts in order to change affective reactions and overt behavior. But there is an important distinction: Contextual behavior analytic perspectives insist on understanding the environmental determinants that govern the relationship between cognitions and other behaviors, whereas cognitive models, including metacognitive models, ascribe causal status to hypothetical constructs such as metabeliefs without insisting on elucidating the contextual determinants of those constructs or of their relationships with behavior. This difference relates to fundamental theoretical differences between the two approaches, which in turn are tied to even deeper philosophical distinctions.

Contextual behavioral science is based on a pragmatic philosophy of science known as functional contextualism (Gifford & Hayes, 1999; Hayes, 1993, 2004). Although a review of this philosophy is well beyond the scope of this chapter, one point is important to appreciate. Within this system, ontology (the study of the fundamental categories of nature) is de-emphasized, and a correspondence theory of truth (in which the goal of science is to create increasingly accurate models that represent the pre-existing nature of the world) is rejected. Instead, "truth" is defined pragmatically as what works in a given context. This requires that scientific goals be specified *a priori*, because successful working can only be evaluated with respect to some specific goal. Functional contextualism asserts that the goals of psychology include not only prediction, but also the ability to impact the phenomenon of interest. The only way to impact behavior, including private behaviors like thinking and feeling, is via some environmental manipulation (where "environment" is broadly conceptualized). For example, even if thinking in a particular way increases the likelihood of another

behavior such as social avoidance in a given context, the only way to change this thinking, or to change its relationship with the avoidant behavior, is by changing something in the environment (e.g., changing the socio-verbal context through psychotherapy, or even taking a medication). It is in this sense that the behavior analyst's insistence on environmental causation is not arbitrary (Hayes, Barnes-Holmes, & Wilson, 2012; Herbert & Forman, in press). Modern behavior analysts believe that by clarifying the contextual control of cognitions on problem behaviors, interventions can more directly target relevant processes that cause and maintain the behaviors (see Lohr, Lilienfeld, & Rosen, 2012, for a discussion of the importance of linking treatment strategies to underlying psychopathological processes in anxiety disorders). Regardless of the degree to which this turns out to be the case, at a minimum, the modern behavior analytic perspective has led to novel clinical developments that provide potentially useful alternatives to traditional CBT approaches.

Acceptance and Commitment Therapy

The approach within this tradition that has attracted by far the most clinical and research attention is acceptance and commitment therapy (ACT; Hayes, Strosahl, & Wilson, 1999, 2012). ACT is explicitly rooted in functional contextualism (Hayes, 2004) and a behavioral theory of language and cognition known as relational frame theory (Hayes, Barnes-Holmes, & Roche, 2001; Törneke, 2010); taken together, the integration of this philosophy, theory, and associated technologies is known as contextual behavioral science (Hayes et al., 2012). The ACT model holds that attempts to alter distressing private experiences are often not only unnecessary, but even counterproductive and paradoxically harmful. ACT seeks to cultivate the psychological flexibility to engage one's experience without judgment or defense, while articulating and then behaving consistently with personally derived valued goals. Metaphors and experiential exercises are integrated into traditional behavioral interventions in an effort to change the psychological context in which distressing experience operates without emphasizing explicit, rule-driven instructions. Although ACT treatments typically do result in symptom improvement, the model de-emphasizes attempts to alter symptoms *per se* in favor of a focus on fostering flexible patterns of behavior with respect to broad life values.

ACT is broadly applicable, and has been successfully used to treat a wide range of psychopathology. It is supported by a robust and growing literature, including over 60 RCTs (for reviews, see Hayes et al., 2012; Hayes, Levin, Plumb-Vilardaga, Villatte, & Pistorello, 2013; Hayes, Luoma, Bond, Masuda, & Lillis, 2006; Powers, Zum Vorde Sive Vording, & Emmelkamp, 2009; Pull, 2009). ACT programs have also been shown to produce gains of comparable or larger magnitude relative to traditional CBT in a sample of patients with various anxiety disorders (Arch et al., 2012), and in clinic samples that include patients with primary complaints of anxiety (Forman, Herbert, Moitra, Yeomans, & Geller, 2007; Lappalainen et al., 2007).

In addition to treatment outcome, the ACT theoretical model is specifically supported by a growing body of empirical studies that have obtained support, through mediational analyses, for mechanisms postulated to drive acceptance and mindfulness-based treatments, for example, experiential acceptance and metacognitive distancing

(Hayes et al., 2006). A meta-analysis of 12 studies with mediational findings obtained support for the mediating role of cognitive defusion (i.e., the ability to see one's experience from a detached perspective), experiential avoidance, and mindfulness (Hayes, Levin, Yadavaia, & Vilardaga, 2007). Several studies have obtained evidence for the mediating role of experiential avoidance in treatments of anxiety (Bond & Bunce, 2000; Woods, Wetterneck, & Flessner, 2006; Zettle, 2003), including one that tracked changes in mediators and outcomes over time (Forman et al., 2012). A meta-analysis of 66 laboratory experiments (i.e., tightly controlled analog studies conducted in the laboratory that focused on isolating mechanisms of action) concluded that psychological acceptance, defusion, present-moment awareness, values, and mindfulness are all independently efficacious over and above various comparison components (i.e., inert conditions such as reading a magazine, or theoretically distinct conditions such as attention control, cognitive reappraisal, or distraction) (Levin, Hildebrandt, Lillis, & Hayes, 2012).

ACT for Social Anxiety

Studies have begun to evaluate ACT programs for the treatment of SAD. Four studies have examined public speaking anxiety, the most common form of the specific subtype of SAD. Seven other studies have focused on the more generalized form of SAD. Finally, one study has recently examined ACT programs for mixed anxiety disorder samples that include a large percentage of patients with SAD.

Block and Wulfert (2000) semi-randomly assigned (due to scheduling restraints) undergraduates ($n = 11$) reporting public speaking anxiety to four weekly sessions of group ACT ($n = 3$), group traditional CBT ($n = 4$), or a wait-list control ($n = 4$). Although both treatments were effective relative to the wait-list condition, the very small sample size precluded statistical analyses. In an extension of this study, Block (2003) again compared group ACT and traditional CBT to a no-treatment control for public speaking anxiety in college students ($n = 39$). There were significant improvements for both treatment groups on anxiety measures, although only the participants who received ACT significantly increased their speech length relative to the wait-list condition.

In the first study of public speaking phobia using a clinical population, England et al. (2012) recently evaluated the acceptability, feasibility, and preliminary effectiveness of a 6-week group ACT-based exposure treatment relative to a standard habituation-based exposure program. Forty-five participants were enrolled, and 35 (78%) completed treatment. Both groups showed large and equivalent improvements in observer-rated speech performance, and in self-rated measures of confidence and anxiety, and both interventions were rated as equally acceptable and credible. However, those in the ACT condition were more likely to achieve diagnostic remission by 6-week follow-up; in fact, none of the individuals who received ACT continued to meet diagnostic criteria for SAD, relative to 17% in the standard treatment. Baseline levels of defusion moderated changes in state anxiety for the ACT condition only.

In a subsequent study from our group, Glassman et al. (2012) compared brief, 90-min ACT versus traditional CBT interventions in a clinical sample of 21 patients

with public speaking anxiety. The effects of treatment on prefrontal brain activation, using functional near-infrared spectroscopy, were also examined. Results indicated that participants in the ACT condition experienced greater improvements in observer-rated performance relative to those in the traditional CBT condition. Individuals in the ACT condition also exhibited greater reductions in oxygenated hemoglobin and blood volume in the right dorsolateral prefrontal cortex following the intervention. Such prefrontal asymmetry, specifically increased activity in the right hemisphere in comparison to the left, is noteworthy because it has been associated with socially anxious states (see Freitas-Ferrari et al., 2010, for a review).

Seven studies have evaluated ACT-based programs specifically for more generalized SAD. In an exploratory study, Ossman et al. (2006) evaluated a group-based ACT program for SAD ($n = 22$). Although the authors did not explicitly state if the sample met diagnostic criteria for the generalized subtype of SAD, it is clear from the description of their sample that the participants had anxiety and avoidance across multiple life domains. The intervention consisted of 10 sessions, each lasting two hours, and comprised standard ACT interventions (e.g., highlighting the futility of direct efforts to control distressing thoughts and feelings, fostering cognitive defusion, enhancing psychological acceptance, and values clarification). Of the 22 participants who were enrolled in the study, only 12 completed treatment; this 45% attrition rate is substantially higher than most other group-based CBT programs, which typically report attrition in the range of 20–25% (Heimberg et al., 1990; Otto et al., 2000). The program resulted in large reductions in social anxiety symptoms, with pretreatment to 3-month follow-up effect sizes of $d = 0.83$ for treatment completers (and $d = 0.56$ for the intent-to-treat sample). Participants' ratings of experiential avoidance also decreased significantly, and were associated with symptom decreases.

We have developed an acceptance-based treatment for generalized SAD that integrates ACT principles and techniques with the behavioral components of traditional CBT programs (specifically graduated, in-session *in vivo* and simulated exposure exercises). Based on a mindfulness-based model of SAD (Herbert & Cardaciotto, 2005), the program was designed to be delivered in an individual treatment format over 12 weeks (Herbert, Forman, & Dalrymple, 2009). Although focused on social anxiety and avoidance, it is designed to accommodate individuals with comorbid problems, especially other anxiety and mood disorders. In an initial evaluation of the feasibility, acceptability, and efficacy of the program, we treated 19 patients with generalized SAD using this protocol (Dalrymple & Herbert, 2007). Two participants (10%) dropped out of treatment. Participants rated the treatment as highly acceptable, with 94% having reported that they were "highly satisfied" with the program. The results revealed large and significant improvement from pretreatment to 3-month follow-up on self-report and clinician-rated measures of social anxiety symptoms, as well as self-report measures of quality of life and experiential avoidance. The mean pre-to-follow-up effect size for the symptom measures was very large ($d = 1.29$), and was even larger for the primary measure of social anxiety ($d = 1.41$). Behavioral assessments, using standard role-played interactions with trained confederates that were video-recorded and rated by observers blinded to assessment occasion, also showed significant improvements in the quality of social behavior and in observer-rated anxiety. Independent assessor ratings and clinician ratings also showed significant improvements. Exploratory process

analyses revealed that decreases in experiential avoidance, but not in negative cognitions, during the first half of treatment were associated with symptom reductions over the last half of treatment, consistent with the ACT model.

This study provided initial support for the efficacy of ACT for generalized SAD. An obvious limitation was the absence of control or comparison conditions. It is possible that some of the observed improvement was independent of the treatment, and reflected phenomena such as regression to the mean, maturation effects, spontaneous recovery, and so forth; definitively ruling out these alternatives would require a no-treatment control condition. Such concerns are mitigated, however, by two considerations. First, there exists a substantial literature demonstrating that SAD tends not to improve without treatment (Davidson, Hughes, George, & Blazer, 1993; Ruscio et al., 2008; Schneier, Johnson, Hornig, Liebowitz, & Weissman, 1992; Yonkers, Dyck, & Keller, 2001). Second, Dalrymple and Herbert (2007) assessed participants before and after a 4-week pretreatment baseline, and found no changes, suggesting that the subsequent improvement was in fact related to treatment. Of greater concern is the absence of a comparison with existing evidence-based treatments. We are currently conducting a randomized clinical trial of the program versus traditional CBT.

Two subsequent pilot studies have utilized this program in remote, internet-based treatment studies. Yuen et al. (2013) treated 14 patients with generalized SAD using the virtual world Second Life. Patients and therapists never met face-to-face, but were represented by digital avatars, which they controlled using their computer keyboard and mouse. They also communicated verbally using voice-over-internet protocol headset devices. Therapists met in a virtual therapy room weekly for 12 weekly individual sessions. Two patients (14%) dropped out of treatment. The results were surprisingly strong, with very large pretreatment to 3-month follow-up effect sizes ($d = 1.50$ on the primary dependent measure). Yuen et al. (2012) followed up this study by a similar trial, this time using the videoconferencing program Skype to offer treatment to 24 patients, again for 12 consecutive weekly sessions of individual treatment. Attrition was 17%. The results were even stronger, with very large pretreatment to follow-up effect sizes ($d = 2.10$ on the primary dependent measure).

Kocovski, Fleming, and Rector (2009) evaluated a newly developed mindfulness and acceptance-based group therapy for SAD. This program combined instruction and practice in mindfulness meditation derived from MBSR with concepts and exercises derived from ACT, as well as exposure exercises. Treatment consisted of an open trial of 12 two-hour group sessions and a subsequent 3-month follow-up. Forty-two participants with generalized SAD were enrolled, but attrition was relatively high, with 29 (69%) completing treatment. Participants experienced significant improvements in social anxiety, depression, and rumination, and increases in mindfulness and acceptance. Pretreatment to follow-up effect sizes on social anxiety measures ranged from 1.00 to 1.17 for treatment completers, and 0.65 to 0.76 in the intent-to-treat sample. Kocovski, Fleming, and Antony (2012) conducted a subsequent randomized clinical trial comparing this program with CBGT and wait-list control ($n = 137$). Attrition was again high, with 30% for the mindfulness treatment and 40% for CBGT. Both treatments were more effective than the wait-list condition, and did not differ from one another. Mediational analyses suggested that cognitive reappraisal was a

mechanism for CBGT but not for the mindfulness-based treatment, whereas psychological acceptance, decentering, rumination, and mindfulness emerged as mediators for both treatments.

In an unpublished study, Harai and Okajima (2010) used a quasi-experimental design to compare five treatment conditions for 49 patients with SAD: (1) high dose of antidepressant medication, (2) low-dose antidepressant medication, (3) pill placebo, (4) CBT combined with flexible dose antidepressant medication, and (5) a group ACT program. They reported that the average decrease in social anxiety symptoms was 60% for high-dose antidepressants, 20% for low-dose antidepressants, 14% for placebo, 50% for combined CBT and antidepressants, and 76% for ACT. Although intriguing, little information is available about the details of the study, and it has not yet undergone peer review.

Finally, one study recently randomized 128 anxiety patients, including 25 with a primary diagnosis of SAD, to either ACT or standard CBT (Arch et al., 2012). Both treatments were designed to be broadly applicable to various anxiety disorders, and were delivered in 12 weekly individual sessions. At posttreatment, ACT and traditional CBT were shown to be equally effective, with no differences on self-report or clinician-rated measures of anxiety or quality of life. However, differences did emerge by the 6-month follow-up assessment, with the ACT condition showing greater improvements across all participants (including those with SAD) on clinical severity ratings and measures of psychological flexibility.

Conclusions and Future Directions

Traditional CBT programs remain the current gold standard for the treatment of social anxiety. A substantial body of work has demonstrated the effectiveness of both group and individual CBT for SAD in various settings. However, these programs are far from perfect, with relatively high refusal and attrition rates, and many patients receiving only limited benefits. Concurrent with the recent growth of acceptance and mindfulness-oriented approaches to CBT, specific interventions have been developed for anxiety disorders, including SAD in particular, and studies have begun to examine their effectiveness. The results thus far are preliminary, but highly promising. More definitive conclusions about the effectiveness of these approaches, both in absolute terms and relative to established evidence-based treatments, must await further research.

There are many questions that remain unanswered. First, the essential active ingredients of these programs await clarification. It is widely assumed among scholars of SAD that systematic exposure is the foundation of any effective treatment program. Indeed, arguably the most innovative feature of Heimberg and Becker's (2002) CBT program for SAD is their skillful and creative incorporation of in-session exposure exercises, which is linked to exposure homework assignments. Most CBT programs—whether traditional programs focusing on cognitive change or newer mindfulness and acceptance-based programs—continue this practice, and place simulated, *in vivo*, and sometimes imaginal exposure exercises as the central focus of treatment. Indeed, the question of whether additional treatment components add incrementally to the presumed powerful effects of exposure remains unresolved.

Despite the consensus on the importance of exposure, the results of the meta-analysis by Hofmann et al. (2010), as well as other recent research, raise an intriguing possibility. Recall that Hofmann et al. found large, positive effects on various mood and anxiety disorders of intervention programs that focused on the practice of mindfulness meditation. Their meta-analysis explicitly excluded studies of multicomponent programs like ACT that featured exposure as a central component. Similarly, individual studies that have focused on mindfulness training without explicit exposure have likewise found positive effects in anxiety disorders (Vøllestad et al., 2011), and SAD in particular (Bögels et al., 2006; Jazaieri et al., 2012; Piet et al., 2010). In the Kocovski et al. (2009) study described above, reductions in social anxiety symptoms were evident by Session 6 of the 12-week program, even though formal exposure did not begin until Session 7. Even among patients with obsessive–compulsive disorder, in which exposure is almost universally considered a necessary part of any effective behavioral treatment, Twohig et al. (2010) found that an ACT program in which exposure was explicitly omitted was effective.

These findings raise the possibility that treatments that focus on enhancing mindfulness, or at least certain components of mindfulness such as psychological acceptance, may be therapeutic for SAD even without explicit exposure training. Given that some patients are unwilling to undergo exposure therapy due to the perceived stress involved, it may be that mindfulness-based programs could serve as a viable alternative in these cases.

Another possibility is that mindfulness and acceptance interventions may potentiate the effects of exposure, and vice versa (see Treanor, 2011, for an interesting discussion of how mindfulness may enhance extinction learning). Indeed, this is the hypothesis that we have followed in developing our acceptance-based behavioral treatment for SAD, which combines the exposure exercises of traditional CBT with the focus on defusion, psychological acceptance, values clarification, and other interventions and sensitivities derived from ACT. Again, however, the degree to which any of these components (including even exposure) have unique effects awaits further research.

Another unresolved question concerns the degree to which a highly tailored, case formulation-based approach might fare relative to a more standardized, protocol-based intervention. There is a widespread assumption that highly tailored interventions are more effective than more standardized ones (Nezu, Nezu, & Lombardo, 2004; Persons, 1991). This assumption operates in the cases of both traditional CBT and newer mindfulness-based approaches. For example, the ACT model of psychopathology highlights several interrelated processes, and intervention strategies and techniques target these processes. The ACT therapist is encouraged to carefully track the patient's particular needs and to apply interventions flexibly on that basis, rather than to intervene using standard techniques in a fixed sequence. Indeed, the emphasis on this individualized approach has increased over time (Hayes et al., 2012). Despite the wide consensus on the superiority of individualized case formulation-based approaches, there are surprisingly few data supporting this assertion. Moreover, there are problems with highly individualized approaches that may limit their utility. These include poor interassessor reliability of case formulations and the related problem of the absence of explicit decision rules regarding which interventions to use at any given time. In addition, a danger in the case of exposure-based interventions

is that greater flexibility may result in patients and therapists inadvertently colluding to avoid conducting anxiety-provoking exposures, even when they are clearly indicated, in favor of therapeutic interventions that are less distressing (but also less likely to be helpful). Engaging in exposure therapy can be stressful for patients and also challenging for some therapists as well (see Waller, 2009, for a relevant discussion of therapist drift in CBT, and Deacon & Farrell, in press, for a review of barriers to the use of exposure treatments). For example, conducting exposures may demand more effort of the therapist during the session, and some may experience discomfort in seeing their patient in distress. Given the increased license afforded by less structured treatment protocols, therapists may therefore avoid conducting exposures in order to escape the discomfort they would entail. It may turn out that a more structured approach is at least as effective, or even more so, than a highly tailored treatment program. Of course, even highly structured, manualized interventions are more tailored than is often appreciated, so the question involves the ideal degree of structure versus flexibility.

Mindfulness and acceptance-based models of CBT represent an exciting development. Their application to SAD has resulted in some novel clinical interventions, as well as the reframing of the purpose and understanding of common established techniques such as exposure. Preliminary research is promising, and a great deal more work is needed. It is critical to keep an open mind about these developments, while simultaneously insisting that they be subjected to rigorous research and not accepted just because of their popular appeal.

References

Arch, J. J., Eifert, G. H., Davies, C., Vilardaga, J. C., Rose, R. D., & Craske, M. G. (2012). Randomized clinical trial of cognitive behavioral therapy (CBT) versus acceptance and commitment therapy (ACT) for mixed anxiety disorders. *Journal of Consulting and Clinical Psychology, 80*(5), 750–765. doi:10.1037/a0028310

Baer, R. A. (2003). Mindfulness training as a clinical intervention: A conceptual and empirical review. *Clinical Psychology: Science and Practice, 10*, 125–143. doi:10.1093/clipsy.bpg015

Beck, A. T. (1976). *Cognitive therapy and the emotional disorders.* New York, NY: International Universities Press.

Block, J. A. (2003). Acceptance or change of private experiences: A comparative analysis in college students with public speaking anxiety. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 63*(9-B), 4361.

Block, J. A., & Wulfert, E. (2000). Acceptance and change: Treating socially anxious college students with ACT or CBGT. *Behavior Analyst Today, 1*(2), 3–10.

Bögels, S. M., Sijbers, G., & Voncken, M. (2006). Mindfulness and task concentration training for social phobia: A pilot study. *Journal of Cognitive Psychotherapy, 20*, 33–44.

Bond, F. W., & Bunce, D. (2000). Mediators of change in emotion-focused and problem-focused worksite stress management interventions. *Journal of Occupational Health Psychology, 5*, 156–163.

Coles, M. E., Turk, C. L., Jindra, L., & Heimberg, R. G. (2004). The path from initial inquiry to initiation of treatment for social anxiety disorder in an anxiety disorders specialty clinic. *Journal of Anxiety Disorders, 18*(3), 371–383. doi:10.1016/S0887-6185(02)00259-1

Craigie, M. A., Rees, C. S., Marsh, A., & Nathan, P. (2008). Mindfulness-based cognitive therapy for generalized anxiety disorder: A preliminary evaluation. *Behavioural and Cognitive Psychotherapy, 36*(5), 553–568. doi:10.1017/S135246580800458X

Dalrymple, K. L., & Herbert, J. D. (2007). Acceptance and commitment therapy for generalized social anxiety disorder: A pilot study. *Behavior Modification, 31*, 543–568.

Davidson, J. R., Hughes, D. L., George, L. K., & Blazer, D. G. (1993). The epidemiology of social phobia: Findings from the duke epidemiological catchment area study. *Psychological Medicine, 23*(3), 709–718.

Deacon, B. J., & Farrell, N. (in press). Therapist barriers in the dissemination of exposure therapy. In E. Storch & D. McKay (Eds.), *Treating variants and complications in anxiety disorders.* New York, NY: Springer.

Dimidjian, S., Hollon, S. D., Dobson, K. S., Schmaling, K. B., Kohlenberg, R. J., Addis, M. E., . . . Jacobson, N. S. (2006). Randomized trial of behavioral activation, cognitive therapy, and antidepressant medication in the acute treatment of adults with major depression. *Journal of Consulting and Clinical Psychology, 74*(4), 658–670.

Eng, W., Coles, M. E., Heimberg, R. G., & Safren, S. A. (2001). Quality of life following cognitive behavioral treatment for social anxiety disorder: Preliminary findings. *Depression and Anxiety, 13*, 192–193.

Eng, W., Coles, M. E., Heimberg, R. G., & Safren, S. A. (2005). Domains of life satisfaction in social anxiety disorder: Relation to symptoms and response to cognitive-behavioral therapy. *Journal of Anxiety Disorders, 19*, 143–156.

England, E. L., Herbert, J. D., Forman, E. M., Rabin, S. J., Juarascio, A., & Goldstein, S. P. (2012). Acceptance-based exposure therapy for public speaking anxiety. *Journal of Contextual Behavioral Science, 1*(1–2), 66–72.

Evans, S., Ferrando, S., Findler, M., Stowell, C., Smart, C., & Haglin, D. (2008). Mindfulness-based cognitive therapy for generalized anxiety disorder. *Journal of Anxiety Disorders, 22*(4), 716–721. doi:10.1016/j.janxdis.2007.07.005

Fedoroff, I. C., & Taylor, S. (2001). Psychological and pharmacological treatments of social phobia: A meta-analysis. *Journal of Clinical Psychopharmacology, 21*(3), 311–324.

Feske, U., & Chambless, D. L. (1995). Cognitive behavioral versus exposure only treatment for social phobia: A meta-analysis. *Behavior Therapy, 26*(4), 695–720.

Fjorback, L. O., Arendt, M., Ornbol, E., Fink, P., & Walach, H. (2011). Mindfulness-based stress reduction and mindfulness-based cognitive therapy: A systematic review of randomized controlled trials. *Acta Psychiatrica Scandinavica, 124*(2), 102–119. doi:10.1111/j.1600-0447.2011.01704.x

Forman, E. M., Chapman, J. E., Herbert, J. D., Goetter, E. M., Yuen, E. K., & Moitra, E. M. (2012). Using session-by-session measurement to compare mechanisms of action for acceptance and commitment therapy and cognitive therapy. *Behavior Therapy, 43*, 341–354.

Forman, E. M., Herbert, J. D., Moitra, E., Yeomans, P. D., & Geller, P. A. (2007). A randomized controlled effectiveness trial of acceptance and commitment therapy and cognitive therapy for anxiety and depression. *Behavior Modification, 31*, 772–799.

Freitas-Ferrari, M. C., Hallak, J. E. C., Trzesniak, C., Filho, A. S., Machado-de-Sousa, J. P., Chagas, M. H. N., . . . Crippa, J. A. (2010). Neuroimaging in social anxiety disorder: A systematic review of the literature. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 34*(4), 565–580.

Gifford, E. V., & Hayes, S. C. (1999). Functional contextualism: A pragmatic philosophy for behavioral science. In W. O'Donohue & R. Kitchener (Eds.), *Handbook of behaviorism* (pp. 285–327). San Diego, CA: Academic Press.

Glassman, L. H., Forman, E. M., Herbert, J. D., Bradley, L., Izzetoglu, M., Ruocco, A., & Foster, E. (2012). *The effects of brief acceptance and commitment therapy vs. traditional cognitive behavior therapy for public speaking anxiety: Differential effects on performance and neurophysiology*. Manuscript under review.

Goldin, P., & Gross, J. J. (2010). Effects of mindfulness-based stress reduction (MBSR) on emotion regulation in social anxiety disorder. *Emotion, 10*(1), 83–91. doi:10.1037/a0018441

Goldin, P., Ramel, W., & Gross, J. J. (2009). Mindfulness meditation training and self-referential processing in social anxiety disorder: Behavioral and neural effects. *Journal of Cognitive Psychotherapy, 23*, 242–257.

Gould, R. A., Buckminster, S., Pollack, M. H., Otto, M. W., & Yap, L. (1997). Cognitive-behavioral and pharmacological treatment for social phobia: A meta-analysis. *Clinical Psychology: Science and Practice, 4*(4), 291–306.

Grant, B. F., Hasin, D. S., Blanco, C., Stinson, F. S., Chou, S. P., Goldstein, R. B., . . . Huang, B. (2005). The epidemiology of social anxiety disorder in the United States: Results from the national epidemiologic survey on alcohol and related conditions. *The Journal of Clinical Psychiatry, 66*(11), 1351–1361.

Harai, H., & Okajima, M. (2010, June). *Acceptance Commitment Therapy for social anxiety disorder: Comparing the treatment outcome with cognitive behavior therapy and antidepressants*. Paper presented at the World Congress of Behavioral and Cognitive Therapies, Boston, MA.

Haug, T. T., Blomhoff, S., Hellstrom, K., Holme, I., Humble, M., Madsbu, H. P., & Wold, J. E. (2003). Exposure therapy and sertraline in social phobia: I-year follow-up of a randomized controlled trial. *The British Journal of Psychiatry : The Journal of Mental Science, 182*, 312–318.

Hayes, S. C. (1993). Analytic goals and varieties of scientific contextualism. In S. C. Hayes, L. J. Hayes, H. W. Reese, & T. R. Sarbin (Eds.), *Varieties of Scientific Contextualism* (pp. 11–27). Reno, NV: Context Press.

Hayes, S. C. (2004). Acceptance and commitment therapy, relational frame theory, and the third wave of behavioral and cognitive therapies. *Behavior Therapy, 35*, 639–665.

Hayes, S. C., Barnes-Holmes, D., & Roche, B. (2001). *Relational frame theory: A post-Skinnerian account of human language and cognition*. New York, NY: Kluwer Academic/Plenum.

Hayes, S. C., Barnes-Holmes, D., & Wilson, K. G. (2012). Contextual behavioral science: Creating a science more adequate to the challenge of the human condition. Manuscript under review. *Journal of Contextual Behavioral Science, 1*, 1–16.

Hayes, S. C., Levin, M. E., Plumb-Vilardaga, J., Villatte, J. L., & Pistorello, J. (2013). Acceptance and commitment therapy and contextual behavioral science: Examining the progress of a distinctive model of behavioral and cognitive therapy. *Behavior Therapy, 44*(2), 180–198.

Hayes, S. C., Levin, M., Yadavaia, J. E., & Vilardaga, R. V. (2007, November). *ACT: Model and processes of change*. Paper presented at the annual meeting of the Association for Behavioral and Cognitive Therapies, Philadelphia, PA.

Hayes, S. C., Luoma, J. B., Bond, F. W., Masuda, A., & Lillis, J. (2006). Acceptance and commitment therapy: Model, processes and outcomes. *Behavior Research and Therapy, 44*, 1–25.

Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (1999). *Acceptance and commitment therapy: An experimental approach to behavior change*. New York, NY: Guilford.

Hayes, S. C., Strosahl, K., & Wilson, K. G. (2012). *Acceptance and commitment therapy: The process and practice of mindful change* (2nd ed.). New York, NY: Guilford.

Heimberg, R. G., & Becker, R. E. (2002). *Cognitive-behavioral group therapy for social phobia: Basic mechanisms and clinical strategies*. New York, NY: Guilford.

Heimberg, R. G., Dodge, C. S., Hope, D. A., Kennedy, C. R., Zollo, L., & Becker, R. E. (1990). Cognitive-behavioral group treatment of social phobia: Comparison to a credible placebo control. *Cognitive Therapy and Research, 14*, 1–23.

Herbert, J. D., & Cardaciotto, L. (2005). A mindfulness and acceptance-based perspective on social anxiety disorder. In S. Orsillo & L. Roemer (Eds.), *Acceptance and mindfulness-based approaches to anxiety: Conceptualization and treatment* (pp. 189–212). New York, NY: Springer.

Herbert, J. D., & Forman, E. M. (2011). The evolution of cognitive behavior therapy: The rise of psychological acceptance and mindfulness. In J. D. Herbert & E. M. Forman (Eds.), *Acceptance and mindfulness in cognitive behavior therapy: Understanding and applying the new therapies* (pp. 3–25). Hoboken, NJ: John Wiley & Sons, Ltd.

Herbert, J. D., & Forman, E. M. (2013). Caution: The differences between CT and ACT may be larger (and smaller) than they appear. *Behavior Therapy, 44*(2), 218–223.

Herbert, J. D., & Forman, E. M. (in press). Mindfulness and acceptance techniques. In D. J. A. Dozois (Ed.), *Cognitive behavioral therapy: A complete reference*. Hoboken, NJ: Wiley-Blackwell.

Herbert, J. D., Forman, E. M., & Dalrymple, K. L. (2009). *Acceptance-based behavior therapy for social anxiety disorder*. Unpublished Therapy Manual. Philadelphia, PA: Drexel University.

Herbert, J. D., Forman, E. M., & England, E. L. (2009). Psychological acceptance. In W. O'Donohue & J. E. Fisher (Eds.), *General principles and empirically supported techniques of cognitive behavior therapy* (pp. 77–101). Hoboken, NJ: John Wiley & Sons, Ltd.

Hofmann, S. G. (2004). Cognitive mediation of treatment change in social phobia. *Journal of Clinical and Consulting Psychology, 72*(3), 393–399.

Hofmann, S. G., & Bögels, S. M. (2006). Recent advances in the treatment of social phobia: Introduction to the special issue. *Journal of Cognitive Psychotherapy, 20*, 3–5.

Hofmann, S. G., Sawyer, A. T., Witt, A. A., & Oh, D. (2010). The effect of mindfulness-based therapy on anxiety and depression: A meta-analytic review. *Journal of Consulting and Clinical Psychology, 78*, 169–183. doi:10.1037/a0018555

Hofmann, S. G., & Suvak, M. (2006). Treatment attrition during group therapy for social phobia. *Journal of Anxiety Disorders, 20*(7), 961–972.

Jazaieri, H., Goldin, P. R., Werner, K., Ziv, M., & Gross, J. J. (2012). A randomized trial of MBSR versus aerobic exercise for social anxiety disorder. *Journal of Clinical Psychology, 68*(7), 715–731. doi:10.1002/jclp.21863

Jørstad-Stein, E. C., & Heimberg, R. C. (2009). Social phobia: An update on treatment. *Psychiatric Clinics of North America, 32*, 641–663. doi:10.1016/j.psc.2009.05.003

Juster, H. R., & Heimberg, R. G. (1995). Social phobia: Longitudinal course and long-term outcome of cognitive-behavioral treatment. *Psychiatric Clinics of North America, 18*, 821–842.

Kabat-Zinn, J. (1990). *Full catastrophe living: Using the wisdom of your mind to face stress, pain and illness*. New York, NY: Dell Publishing.

Kabat-Zinn, J. (1994). *Wherever you go, there you are: Mindfulness meditation in everyday life*. New York, NY: Hyperion.

Kabat-Zinn, J. (2005) *Coming to our senses: Healing ourselves and the world through mindfulness*. New York, NY: Hyperion.

Kim, Y. W., Lee, S.-H., Choi, T. K., Suh, S. Y., Kim, B., Kim, C. M., . . . Yook, K. H. (2009). Effectiveness of mindfulness-based cognitive therapy as an adjuvant to pharmacotherapy

in patients with panic disorder or generalized anxiety disorder. *Depression and Anxiety, 26*(7), 601–606. doi:10.1002/da.20552

Kocovski, N. L., Fleming, J. E., & Antony, M. M. (2012). *Mindfulness and acceptance-based versus cognitive behavioral group therapy for social anxiety disorder: A randomized controlled trial.* Manuscript under review.

Kocovski, N. L., Fleming, J. E., & Rector, N. A. (2009). Mindfulness and acceptance-based group therapy for social anxiety disorder: An open trial. *Cognitive and Behavioral Practice, 16*, 276–289.

Koszycki, D., Benger, M., Shlik, J., & Bradwejn, J. (2007). Randomized trial of a meditation-based stress reduction program and cognitive behavior therapy in generalized social anxiety disorder. *Behaviour Research and Therapy, 45*, 2518–2526.

Lappalainen, R., Lehtonen, T., Skarp, E., Taubert, E., Ojanen, M., & Hayes, S. C. (2007). The impact of CBT and ACT models using psychology trainee therapists: A preliminary controlled effectiveness trial. *Behavior Modification, 31*, 488–511. doi:10.1177/0145445506298436

Levin, M. E., Hildebrandt, M. J., Lillis, J., & Hayes, S. C. (2012). The impact of treatment components suggested by the psychological flexibility model: A meta-analysis of laboratory-based component studies. *Behavior Therapy, 43*, 741–756.

Liebowitz, M. R., Fyer, A. J., Gorman, J. M., Dillon, D., Davies, S., Stein, J. M., . . . Klein, D. E. (1985). Specificity of lactate infusions in social phobia versus panic disorders. *American Journal of Psychiatry, 142*, 947–950.

Lohr, J. M., Lilienfeld, S. O., & Rosen, G. M. (2012). Anxiety and its treatment: Promoting science-based practice. *Journal of Anxiety Disorders, 26*, 719–727.

Lykins, E. L. B., & Baer, R. A. (2009). Psychological functioning in a sample of long-term practitioners of mindfulness meditation. *Journal of Cognitive Psychotherapy, 23*, 226–241.

Marchand, W. R. (2012). Mindfulness-based stress reduction, mindfulness-based cognitive therapy, and Zen meditation for depression, anxiety, pain, and psychological distress. *Journal of Psychiatric Practice, 18*(4), 233–252.

McManus, F., Clark, D. M., & Hackmann, A. (2000). Specificity of cognitive biases in social phobia and their role in recovery. *Behavioural and Cognitive Psychotherapy, 28*, 201–209.

Nelson, E., & Velasquez, S. E. (2011). Implementing psychological services over televideo. *Professional Psychology: Research and Practice, 42*(6), 535–542.

Nezu, A. M., Nezu, C. M., & Lombardo, E. R. (2004). *Cognitive-behavioral case formulation and treatment design: A problem-solving approach.* New York, NY: Springer.

Olfson, M., Guardino, M., Struening, E., Schneier, F. R., Hellman, F., & Klein, D. F. (2000). Barriers to the treatment of social anxiety. *American Journal of Psychiatry, 157*(4), 521–527.

Ossman, W. A., Wilson, K. G., Storaasli, R. D., & McNeill, J. W. (2006). A preliminary investigation of the use of acceptance and commitment therapy in group treatment for social phobia. *International Journal of Psychology and Psychological Therapy, 6*, 397–416.

Otto, M. W., Pollack, M. H., Gould, R. A., Worthington, J. J., McArdle, E. T., Rosenbaum, J. F., & Heimberg, R. G. (2000). A comparison of the efficacy of clonazepam and cognitive behavioral group therapy for treatment of social phobia. *Journal of Anxiety Disorders, 14*, 345–358.

Persons, J. B. (1991). Psychotherapy outcome studies do not accurately represent current models of psychotherapy: A proposed remedy. *American Psychologist, 46*, 99–106.

Piet, J., Hougaard, E., Hecksher, M. S., & Rosenberg, N. K. (2010). A randomized pilot study of mindfulness-based cognitive therapy and group cognitive-behavioral therapy for young adults with social phobia. *Scandinavian Journal of Psychology, 51*, 403–410. doi:10.1111/j.1467-9450.2009.00801.x

Powers, M. B., Sigmarsson, S. R., & Emmelkamp, P. M. G. (2008). A meta–analytic review of psychological treatments for social anxiety disorder. *International Journal of Cognitive Therapy, 1*, 94–113.

Powers, M. B., Zum Vorde Sive Vording, M. B., & Emmelkamp, P. M. (2009). Acceptance and commitment therapy: A meta-analytic review. *Psychotherapy and Psychosomatics, 78*(2), 73–80. doi:10.1159/000190790

Pull, C. B. (2009). Current empirical status of acceptance and commitment therapy. *Current Opinion in Psychiatry, 22*(1), 55–60. doi:10.1097/YCO.0b013e32831a6e9d

Ramel, W., Goldin, P. R., Carmona, P. E., & McQuaid, J. R. (2004). The effects of mindfulness meditation on cognitive processes and affect in patients with past depression. *Cognitive Therapy and Research, 28*, 433.

Ruscio, A. M., Brown, T. A., Chiu, W. T., Sareen, J., Stein, M. B., & Kessler, R. C. (2008). Social fears and social phobia in the USA: Results from the national comorbidity survey replication. *Psychological Medicine, 38*(1), 15–28. doi:10.1017/S0033291707001699

Schneier, F., Johnson, J., Hornig, C., Liebowitz, M., & Weissman, M. (1992). Social phobia: Comorbidity and morbidity in an epidemiologic sample. *Archives of General Psychiatry, 49*, 282–288.

Segal, Z. V., Williams, J. M. G., & Teasdale, J. D. (2001). *Mindfulness-based cognitive therapy for depression: A new approach to preventing relapse*. New York, NY: Guilford.

Semple, R. J., & Lee, J. (2008). Treating anxiety with mindfulness: Mindfulness-based cognitive therapy for children. In L. A. Greco & S. C. Hayes (Eds.), *Acceptance and mindfulness interventions for children, adolescents, and families* (pp. 94–134). Oakland, CA: Context Press/New Harbinger Publications.

Stein, D. J., Versiani, M., Hair, T., & Kumar, R. (2002). Efficacy of paroxetine for relapse prevention in social anxiety disorder: A 24-week study. *Archives of General Psychiatry, 59*(12), 1111–1118.

Teasdale, J. D., Moore, R. G., Hayhurst, H., Pope, M., Williams, S., & Segal, Z. V. (2002). Metacognitive awareness and prevention of relapse in depression: Empirical evidence. *Journal of Consulting and Clinical Psychology, 70*, 275–287.

Törneke, N. (2010). *Learning RFT: An introduction to relational frame theory and its clinical application*. Oakland, CA: New Harbinger.

Treanor, M. (2011). The potential impact of mindfulness on exposure and extinction learning in anxiety disorders. *Clinical Psychology Review, 31*, 617–625. doi:10.1016/j.cpr.2011.02.003

Turner, S. M., Beidel, D. C., & Wolff, P. L. (1994). A composite measure to determine improvement following treatment for social phobia: The Index of Social Phobia Improvement. *Behaviour Research and Therapy, 4*, 471–476.

Twohig, M. P., Hayes, S. C., Plumb, J. C., Pruitt, L. D., Collins, A. B., Hazlett-Stevens, H., & Woidneck, M. R. (2010). A randomized clinical trial of acceptance and commitment therapy vs. progressive relaxation training for obsessive-compulsive disorder. *Journal of Consulting and Clinical Psychology, 78*, 705–716.

Vøllestad, J., Nielsen, M. B., & Nielsen, G. H. (2012). Mindfulness- and acceptance-based interventions for anxiety disorders: A systematic review and meta-analysis. *British Journal of Clinical Psychology, 51*(3), 239–260. doi:10.1111/j.2044-8260.2011.02024.x

Vøllestad, J., Sivertsen, B., & Nielsen, G. H. (2011). Mindfulness-based stress reduction for patients with anxiety disorders: Evaluation in a randomized controlled trial. *Behaviour Research and Therapy, 49*(4), 281–288. doi:10.1016/j.brat.2011.01.007

Walker, J. R., Van Ameringen, M. A., Swinson, R., Bowen, R. C., Chokka, P. R., Goldner, E., . . . Lane, R. M. (2000). Prevention of relapse in generalized social phobia: Results of

a 24-week study in responders to 20 weeks of sertraline treatment. *Journal of Clinical Psychopharmacology, 20*(6), 636–644.

Waller, G. (2009). Evidence-based treatment and therapist drift. *Behaviour Research and Therapy, 47*, 119–127. doi:10.1016/j.brat.2008.10.018

Wells, A. (2007). Cognition about cognition: Metacognitive therapy and change in generalized anxiety disorder and social phobia. *Cognitive and Behavioural Practice, 14*, 18–25.

Wells, A. (2008). *Metacognitive therapy: A practical guide*. New York, NY: Guilford.

Wells, A. (2011). Metacognitive therapy. In J. D. Herbert & E. M. Forman (Eds.), *Acceptance and mindfulness in cognitive behavior therapy* (pp. 83–108). Hoboken, NJ: John Wiley & Sons, Ltd.

Wells, A., & King, P. (2006). Metacognitive therapy for generalized anxiety disorder: An open trial. *Journal of Behavior Therapy and Experimental Psychiatry, 37*(3), 206–212. doi:10.1016/j.jbtep.2005.07.002

Whitaker, R. (2010). *Anatomy of an epidemic*. New York, NY: Broadway Paperbacks.

Williams, J. C., & Lynn, S. J. (2010). Acceptance: An historical and conceptual review. *Imagination, Cognition, & Personality, 30*, 5–56.

Wong, S. Y., Mak, W. W., Cheung, E. Y., Ling, C. Y., Lui, W. W., Tang, W., . . . Ma, H. S. (2011). A randomized, controlled clinical trial: The effect of mindfulness-based cognitive therapy on generalized anxiety disorder among Chinese community patients: Protocol for a randomized trial. *BMC Psychiatry, 11*, 187. doi:10.1186/1471-244X-11-187

Woods, D. W., Wetterneck, C. T., & Flessner, C. A. (2006). A controlled evaluation of acceptance and commitment therapy plus habit reversal for trichotillomania. *Behaviour Research and Therapy, 44*, 639–656.

Yonkers, K. A., Dyck, I. R., & Keller, M. B. (2001). An eight-year longitudinal comparison of clinical course and characteristics of social phobia among men and women. *Psychiatric Services, 52*, 637–643.

Yuen, E. K., Herbert, J. D., Forman, E. M., Goetter, E. M., Comer, R., & Bradley, J. (2013). Treatment of social anxiety disorder using online virtual environments in Second Life. *Behavior Therapy, 44*(1), 51–61. (published on line at: http://dx.doi.org/10.1016/j.beth.2012.06.001)

Yuen, E. K., Herbert, J. D., Forman, E. M., Goetter, E. M., Juarascio, A. S., Rabin, S., . . . Bouchard, S. (2012). *Acceptance-based behavior therapy for social anxiety disorder through videoconferencing*. Manuscript under review.

Zettle, R. D. (2003). Acceptance and commitment therapy vs. systematic desensitization in treatment of mathematics anxiety. *The Psychological Record, 53*(2), 197–215.

Index

acceptance and commitment therapy (ACT) 597–8
  social anxiety 598–601
acceptance therapy 590–2
adolescents *see* children and adolescents
β-adrenergic antagonists 532
adrenocorticotropic hormone (ACTH) 455
affiliative psychology 40–1
  emotion regulation 42–3
  shame 41–2
affirmation 276
age of onset of SAD 182
agoraphobia 204
alcohol use 551
alcohol use disorder 212
alprazolam 483, 524, 528, 531
Alzheimer's disease 161
ambivalence 277
amygdala 69, 79, 91
  hyperreactivity 395–7
  response before and after SAD treatment 398–9
anticonvulsants 532–3
antisocial personality disorder (ASPD) 210–12
Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV) 282–3, 308–9
anxiety physiological correlates 90–2
anxiety-related behaviors 357
anxiety sensitivity 97, 500
applied relaxation (AR) 479, 480–1
  Internet-based 574
Approach-Avoidance Task (AAT) 348–9, 414
archetypal disposition 25
arginine vasopressin (AVP) 74–5
Asperger's Disorder (AD) 378
atenolol 527, 534
attachment processes 187–8
attachment theory 187–8
attention bias modification (ABM) programs 420
attention deficit hyperactivity disorder (ADHD) 135
attention training (ATT) 516
attentional deployment 418
attractiveness 33–4, 40–1
atypical antipsychotics 533
atypical depression 206, 521
audacious trust 165
automatic thoughts, positive and negative 442
avoidance 167
avoidance of social interactions 446
avoidant personality disorder (APD/AVPD) 111, 125, 208–10, 249–50
  clinical interviews 271, 279

The Wiley Blackwell Handbook of Social Anxiety Disorder, First Edition. Edited by Justin W. Weeks.

basal hyperarousal 94
behavioral approach system (BAS) 14–15
behavioral deviations 360
  evolution of SAD and behavioral biases 344–6
  model of cognition and behaviour in SAD 355–9
  models of biased processing 346–8
  social devaluation behaviors 348
    anxious behavior 354–5
    avoidance during social interactions 348–9
    facial mimicry 350–1
    interpersonal distance and personal space 349–50
    social skills and social behavior 352–4
behavioral inhibition (BI) 59–60, 92–3, 133
  assessment questionnaire 144–6
  association with anxiety disorders
    cross-sectional studies and retrospective studies 139–40
    depression 140
    family studies 136
    prospective studies 136–9
    symptoms other than social anxiety 139
  children and adolescents 185–7
  cultural considerations 140–1
  definition 133–4
  future directions 147–8
  genetic underpinnings 135–6
  neurobiological underpinnings
    neuroimaging studies 134–5
    physiologic reactivity 134
  preventive intervention 142–7
  risk factor for SAD 141–2
behavioral inhibition system (BIS) 14–15
benzodiazepines 531–2
beta-blockers 532
bipolar disorder 208
bivalent fear of evaluation model 434
blood oxygen-level-dependent (BOLD) responses 395
blushing 57, 91–2, 96–7
brain-derived neurotrophic factor (BDNF) 401
Brief Intervention for Socially Anxious Drinkers (BISAD) 559
Brief Social Phobia Scale (BSPS) 285–6
brofaromine 524, 534
bromazepam 524, 531–2
buspirone 527, 532, 534, 535

cannabidiol 533
cannabis use 213–14, 550
care giving 28
care seeking/receiving 28
cautious trust 165
children and adolescents 181, 194
  developmental psychology
    developmental psychopathology 184–5
    developmental theory 182–4
  implications for research and practice 194–5
  pathways to SAD 185
    attachment processes 187–8
    behavioral inhibition (BI) 185–7
    emotion regulation 188–91
    parenting practices 191–2
    peer relationships 192–4
  pharmacotherapy 537
  social anxiety disorder 181–2
circumplex models 162–3
  interpersonal circumplex theory 119–21
citalopram 526, 529
Clark–Wells model of SAD 8–9, 347
Clinical Global Impression (CGI) Change Scale 522
clinical interviews for SAD 271–2
  conceptualization 272–4
  conducting the interview
    approaches 275
    behaviors to consider during interview 277–8
    cultural and developmental considerations 280–1
    differential diagnosis 279–80
    environment 274
    motivational interviewing 275–7
    subtypes and pervasiveness of SAD 278–9
  recommendations 286–7
  structured and semi-structured interviews 282
    diagnostic interviews 282–4
    symptom severity assessment 284–6

clonazepam 483, 524, 528, 531–2
cocaine use 551
cognitive behavioral models of SAD 3
  Clarke and Wells (1995) cognitive model 3
    dysfunctional processes 4–5
  comparison between models 7–9
  future directions 20
  other models
    evolutionary/psychobiological models 16–17
    interpersonal model of SAD 15–16
    Reinforcement Sensitivity Theory (RST) 13–15
    self-presentation model of SAD 12–13
  overview of models
    discrepancy as the key 18
    etiology and developmental perspectives 17–18
    social anxiety as adaptive and normative 18–19
    SAD as intrapersonal and interpersonal disorder 19
  Rapee and Heimberg (1997) cognitive–behavioral model 5
    dysfunctional processes 5–7
  recent models
    Hofmann (2007) cognitive factors that maintain SAD 9–10
    Moscovitch (2009) proposed core fear in SAD 10–11
    importance of the self in understanding SAD 11–12
cognitive behavioral therapy (CBT) for SAD 477, 490–1, 498, 516–17
  see also treatment for SAD
  adaptations of treatment
    comorbidities 513
    diverse populations 511–13
    group versus individual 511
    medication 513–14
  assessment 498–501
  cognitive strategies 502
    behavioral experiments 504
    challenging anxiety-provoking thoughts 502–4
    core beliefs 505–6
    new data collection 504–5
  compared with other therapies
    interpersonal psychotherapy (IPT) 482–3
    mindfulness-based therapies 481–2
  comparisons and combinations with pharmacotherapy 483–4
  diversity considerations 233–7
  emerging treatments 515
    cognitive bias training 516
    imagery rescripting 516
    virtual reality exposure therapy (VRET) 515
  exposure 477–9
  exposure-based strategies 506
    guidelines 507–8
    hierarchy development 507
    rationale for exposure 506
    types of exposure 509
  exposure combined with cognitive restructuring 478–9
  exposure combined with nontraditional pharmacotherapy 484–5
  factors affecting outcome
    comorbidity 486–7
    homework compliance
    outcome expectancy 487
    subtype of SAD 486
    supplementary treatment components 487–8
    treatment modality 487
  further study issues
    nonresponders to treatment 489–90
    understudied populations 488–9
  neuroimaging findings 485
  psychoeducation 501–2
  relapse prevention 514–15
  relaxation techniques 480–1, 511
  single case studies 237–8
  social skills training (SST) 479–80, 509–10
  video feedback 510
cognitive bias modification (CBM) 326
cognitive biases 323–4
  future research 335–6
  implicit associations 333–4
  interpretations and judgment 324–6
  post-event processing (PEP) and memory 331–3

cognitive biases (*Continued*)
selective attention bias 326–7
conscious 329–31
unconscious 327–8
cognitive change 418
cognitive preparation 334
adaptations
combining with medications 513–14
comorbidities 513
diverse populations 511–13
group versus individual format 511
assessment 498–501
augmentation strategies
applied relaxation 511
social skills training 509–10
video feedback 510
cognitive strategies 502
behavioral experiments 504
challenging anxiety-provoking thoughts 502–4
core beliefs 505–6
new data collection 504–5
combination of nontraditional pharmacotherapy and exposure 484–5
comparison and combination with pharmacotherapy 483–4
comparison with other psychotherapies
interpersonal psychotherapy (IPT) 482–3
mindfulness-based therapies 481–2
emerging treatments and future directions 515
cognitive bias training 516
imagery rescripting 516
virtual reality exposure therapy (VRET) 515
exposure 477–8
exposure and cognitive restructuring combined 478–9
exposure-based strategies 506
exposure guidelines 507–8
exposure hierarchy 507
exposure types 509
rationale 506
factors affecting outcome
comorbidities 486–7
expectancy 487
homework compliance 488
subtypes 486
supplementary treatment components 487–8
treatment modality 487
further study
treatment nonresponders 489–90
understudied populations 488–9
neuroimaging findings 485
psychoeducation 501–2
relapse prevention 514–15
relaxation techniques 480–1
session protocol 499
social skills training 479–80
combined cognitive bias hypothesis 358
common method variance (CMV) 303, 307–8
communicative behaviours 357
comorbidities with SAD 201–2, 215
anxiety disorders
generalized anxiety disorder (GAD) 202–3
obsessive–compulsive disorder (OCD) 203–4
panic disorder with/without agoraphobia 204
posttraumatic stress disorder (PTSD) 205
CBT outcomes 486–7, 513
mood disorders
bipolar disorder 208
dysthymic disorder 207–8
major depressive disorder 205–7
personality disorders
avoidant personality disorder (APD) 208–10, 249–50
conduct disorder (CD) and antisocial personality disorder (ASPD) 210–12
substance use disorders 212
alcohol 212
cannabis 213–14
other illicit substances 214–15
tobacco 213
compassion 44–5
self-compassion 45–6
competition and evolutionary change 31–2
resource competition, group living and social hierarchies 33

competitive behavior 28
complementarity 162
conditioned response (CR) 456
conditioned stimulus (CS) 456
conduct disorder (CD) 210–12
confirmatory factor analysis (CFA) 116, 302
conflicts 29, 34
construct validity 300
contextual behavioral science 596–7
convergent validity 302
cooperative behavior 28
corticotrophin-releasing hormone (CRH) 59–60, 135, 136
cortisol 393
critical parenting style 191
cultural aspects of SAD 101–4
  behavioral inhibition (BI) 140–1
Curse of the Self 26

D-cycloserine (DCS) 392–3, 484–5
depression
  atypical depression 206, 521
  behavioral inhibition (BI) 140
  fear of positive evaluation (FPE) 439
  major depressive disorder 205–7
developmental contextualism 183–4
diagnosis
  differential diagnosis 279–80
  interviews 282–4
discriminant validity 302
disqualification of positive social outcomes (DPSO) 438, 441–2, 448
diversity considerations 223–5, 240–1
  assessment 231–3
  case conceptualization 238–40
  CBT 511–13
  cultural variation in presentation
    influences on presentation 230–1
    specific presentations 229–30
  minority stress and SAD theories 225
    linking models 228–9
    minority stress models 225–8
  treatment
    cognitive behavioral therapy (CBT) 233–7
    pharmacotherapy 237
    single case CBT studies 237–8
dorsal anterior cingulate cortex (dACC) 160
dorsal medial prefrontal cortex (DMPFC) 397
dorsolateral prefrontal cortex (DLPFC) 397
Dot probe task 328, 330
downward arrow technique 505
dysthymic disorder 207–8

ecological momentary assessment (EMA) 112, 308
ego self-identity goals 44
electromyography (EMG) 351
embodiment 357
emotion regulation 31, 32, 188–91
emotion systems, functional analysis of 30–1
emotional reactivity 413
endogenous glucocorticoids 455–6
endophenotypes of SAD 400
  brain-derived neurotrophic factor (BDNF) 401
  oxytocin receptor (OXTR) gene 401
  serotonin transporter (5-HTT) gene 400–1, 2
erythrophobia 101
escitalopram 526, 529
estradiol 462
  endogenous release 463
  exogenous administration 464–5
event reactivity 413
evolutionary models of SAD 16–17, 24, 46
  competition and evolutionary change dynamics 31–2
    resource competition, group living and social hierarchies 33
  competition and social rank and subordination 33–6
    low rank and positive evaluation 38–40
    paranoia and SAD 37–8
    self-blame and self-criticism 36–7
  developing compassion 44–5
  functional analysis of emotion systems 30–1
  general evolutionary processes in SAD 24–5
    old and new brain mechanisms 25
    social mentalities 26–30
  self-compassion 45–6

evolutionary models of SAD (*Continued*)
social attractiveness and affiliative psychology 40–1
emotion regulation 42–3
shame 41–2
treatment implications 43–4
exogenous glucocorticoid administration 456–7
experiential avoidance 29
exploratory factor analysis (EFA) 302
exposure guidelines 507–8
exposure hierarchy 507
external shame 42
external validity 307
extroversion 58–9

facial mimicry 350–1
factorial discriminant validity 302
fear of negative evaluation (FNE) 433, 437
Fear of Negative Evaluation (FNE) scale 354, 377
fear of positive evaluation (FPE) 7, 433, 437–8, 450
clinical implications 447–9
cognitive–behavioral components 447
behavior 446–7
cognitions 441–2
emotions and arousal 442–5
future directions 449–50
theoretical overview
assessment of FPE 436
basic properties 435–6
distinctions 437–41
overall construct validity 436–7
positivity impairment 434
psycho-evolutionary considerations 434
fear of success 439–40
feared social situation types 250
fear of displaying physical signs of social anxiety 252–3
fear of offending others 253
performance fears 250–1
social interaction and observation fears 252
fearful temperament 92–3
fight–flight–freeze system (FFFS) 14–15, 91, 501
five-factor theory (FFT) of personality 116–18
fluoxetine 483, 484, 526, 528, 529
fluvoxamine 524, 529
friendship development models 163–5, 168–9
fusiform cortex 70–1
fuzzy-clustering method 55

gabapentin 527, 532, 534
gamma-aminobutyric acid (GABA) 458
gaze avoidance 446–7
generalized anxiety disorder (GAD) 171, 202–3
generalized social anxiety disorder (GSAD) 247–9
genetic factors in SAD 53
behavioral inhibition (BI) 135–6
family and twin evidence 54
future directions 60–1, 74–5
genetic association studies 56
behavioral inhibition (BI) and selective mutism 59–60
blushing 57
extroversion 58–9
neuroticism 57–8
shyness 57
social anxiety disorder 56–7
genetic linkage studies 54–6
genome-wide association studies (GWAS) 58, 59
glucocorticoids, endogenous 455–6
glucocorticoids, exogenous 456–7
gonadal hormones 461
endogenous release 462–3
exogenous administration 463–5
synthesis and sites of action 462
group cognitive behavioral therapy (GCBT) 171, 207
group living 33
guided net-bibliotherapy 573

heterogeneity in SAD 247, 261
diagnostic subtypes
comorbid avoidant personality disorder (APD) 249–50
generalized social anxiety disorder (GSAD) 247–9

feared social situation types 250
performance fears 250–1
social interaction and observation fears 252
functioning, heterogeneity 255
demographic variables 256–7
symptom severity 255–6
personality considerations 253–4
understudied contexts and groups 257
Internet users 259–61
medical settings 257–8
veterans 258–9
variations in focus of social fears 252
fear of displaying physical signs of social anxiety 252–3
fear of offending others 253
high social anxiety (HSA) 323
future research 335–6
implicit associations 333–4
judgment biases 324–6
post-event processing (PEP) and memory 331–3
selective attention bias 326–7
conscious 329–31
unconscious 327–8
Hofmann model of SAD 9–10
hormones, gonadal 461
endogenous release 462–3
exogenous administration 463–5
synthesis and sites of action 462
hyperarousal 93, 94–6
hypervigiliance 345, 357
hypothalamic–pituitary–adrenal (HPA) axis 160–1, 454–5, 458, 465
hypothalamic–pituitary–gonadal (HPG) axis 462, 465
hypothalamus 91

ideal self 122
immune function and SAD 75–6
impact message 163
implicit association test (IAT) 310, 333
impression management 167
insecure attachment 142, 188
insula 69–70
interference effect 329
internal shame 41–2
Internet-delivered treatments for SAD 571–2, 583
discussion 582–3
SAD and the Internet 572–3
effectiveness 580–1
effectiveness in real clinical settings 581
effects of treatment 575–7
guidance and support roles 578–9
long-term outcome studies 577–8
moderators of outcome 581–2
security issues 575
therapist identity 579–80
treatment programs 573–4
interoceptive exposure 500, 509
interpersonal circumplex theory 119–21
interpersonal distance 349–50
interpersonal functioning 255
interpersonal interactions and SAD 166
dysfunctional behavior as relational strategies 166–8
friendship development 168–9
intimate relationships 169–71
interpersonal model of SAD 15–16
interpersonal psychotherapy (IPT) 482–3
interpersonal transaction cycle 163
interpretation bias modification (IBM) programs 421
interpretation training 516
intimate relationships 169–71
isolation 449

James–Lange theory of emotion 73
judging audience 41

labeled praises 275
lesbian, gay, bisexual (LGB) individuals 225, 228, 232–3, 236
cognitive–behavioural therapy (CBT) 489
levetiracetam 527, 532–3
Liebowitz Social Anxiety Scale (LSAS) 284–5, 446, 522
life satisfaction 256
LOD scores 54–5
loneliness 160
looking glass self 40

major depressive disorder (MDD) 205–7, 279
metacognitive awareness 595

metacognitive therapy 594–5
mindfulness-based cognitive therapy (MBCT) 481–2, 595–6
mindfulness-based stress reduction (MBSR) 481, 593–4
  neuroimaging studies 485
mindfulness-based therapies 481–2, 590–2
  ACT for social anxiety 598–601
  emergence 592–3
  future directions 601–3
  meditation-based programs
    metacognitive therapy 594–5
    mindfulness-based cognitive therapy (MBCT) 595–6
    mindfulness-based stress reduction (MBSR) 593–4
  modern behavior analytic approaches 596–7
    acceptance and commitment therapy (ACT) 597–8
minorities *see* diversity considerations
minority stress theory 223
mirtazapine 527, 531
moclobemide 523, 531, 534
monoamine oxidase inhibitors (MAOIs) 521, 530
Moscovitch model of SAD 10–11
motivation enhancement therapy (MET) 561–3
motivational interviewing 275–7, 487–8
mutual influence 163

need to belong 345–6
nefazodone 527, 531
negative affect syndrome 125
negative feedback, preference for 440–1
neuroendocrinology of SAD 454
  future directions 465–6
  gonadal hormones 461
    endogenous release 462–3
    exogenous administration 463–5
    synthesis and sites of action 462
  social neuropeptides 457–8
    endogenous release 458–9
    exogenous manipulation and release 459
    oxytocin administration 459–60
    synthesis, storage and site of action 458
    vasopressin administration 461
  stress response 454–5
    endogenous glucocorticoids 455–6
    exogenous glucocorticoid administration 456–7
neuropeptides, social 457–8
  endogenous release 458–9
  exogenous manipulation and release 459
  oxytocin administration 459–60
  synthesis, storage and site of action 458
  vasopressin administration 461
neuroticism 57–8
nonverbal communication 277

observation fears 252
obsessive–compulsive disorder (OCD) 203–4
occupational functioning 255–6
olanzapine 527, 533
orbitofrontal cortex (OFC) 72, 397
ought self 122
over-identification 449
oxytocin 43, 74–5, 393–4, 458
  administration 459–60
  withdrawal behaviors 459
oxytocin receptor (OXTR) gene 401

panic disorder with/without agoraphobia 136, 204
paranoia and SAD 37–8
paraphrasing 276
parasympathetic anxiety 94–6
parental criticism 142
parental overprotectiveness 142
parent–child relationship 187
parenting practices 191–2
paroxetine 524, 525, 528, 529, 534–5
pathophysiology of SAD 90
  cultural aspects 101–4
  future directions 104–5
  physiological aspects to etiology/maintenance of SAD 97–9
  physiological correlates of anxiety 90–2
  physiological correlates of fearful temperament 92–3

physiological correlates of SAD 93
blushing 96–7
hyperarousal, sympathetic and parasympathetic anxiety 94–6
physiology of SAD subtypes 99–101
peer relationships 192–4
performance fears 250–1
personal space 349–50
personality and SAD 112, 125–6
clinical implications 125
future directions 124–5
personality theories
common themes 123–4
five-factor theory (FFT) 116–18
interpersonal circumplex theory 119–21
self-regulation theory 121–3
trait–affect theory 118–19
theory 112–16
personality considerations 253–4
personality disorders, assessing 280
pharmacotherapy 521–2, 539–40
children and adolescents 537
clinical approach
assessment 537–8
choice of treatment 538–9
monitoring and discontinuation of treatment 539
combination, augmentation and switching of treatments
medication 534–5
medications and psychotherapy 535–6
diversity considerations 237
meta-analyses 533–4
monotherapy
β-adrenergic antagonists 532
benzodiazepines 531–2
monoamine oxidase inhibitors (MAOIs) 530
other antidepressants 531
other medications 532–3
reversible inhibitors of monoamine oxidase A (RIMAs) 530–1
SSRIs and SNRIs 522–30
treating SAD with comorbid disorders 536
phenelzine 483, 523, 530, 534
physiologic reactivity 134
physiological arousal 445
positive affect stimulation and sustainment (PASS) 422
Positive and Negative Affect Schedule (PANAS) 118–19, 311, 411
positivity impairment as a feature of SAD 409–11, 434
causal status of positive affect 419
attention bias modification (ABM) programs 420
cognitive modification training studies 420
interpretation bias modification (IBM) programs 421
longitudinal studies 419–20
implications and future directions 422
core features 421–2
treatment 422
reactivity to positive signals 412–13
attention 413
cognitive biases 415
event reactivity 416–17
interpretation and evaluation 414
memory 414–15
smiles 415–16
stimulus reactivity 413
regulation of positive emotions 417–19
scope 411–12
post-event processing (PEP) 7, 331–3, 501
posttraumatic stress disorder (PTSD) 139, 205
preference for negative feedback 440–1
prefrontal cortex (PFC) 71–2, 76–7
pregabalin 527, 532
preparedness 25
preventive intervention 142–7
pride 411–12
progressive muscle relaxation (PMR) 479
prosocial concepts 166
psychobiological models of SAD 16–17

quasi-behavioral measures 310
quetiapine 527, 533

Rapee–Heimberg model of SAD 5–7, 347
reflection 276
regulatory focus theory 122

Reinforcement Sensitivity Theory (RST) 13–15
relational processes in SAD 159–60, 172
  circumplex models 162–3
  friendship development models 163–5
  interpersonal interactions 166
    dysfunctional behavior as relational strategies 166–8
    friendship development 168–9
    intimate relationships 169–71
  relational theories 162
  relationships 161–2
  risk-regulation models 165–6
  social bonds and human wellbeing 160–1
  underexplored areas 171–2
relational schema 15
relaxation techniques 480–1, 511
resource competition 33
response modification 418
reversible inhibitors of monoamine oxidase A (RIMAs) 521, 530–1
risk-regulation models 165–6
risperidone 533

safety behaviors 4, 167, 500
Schedule for Affective Disorders and Schizophrenia (SADS) 283–4
screening for BI 143
secure attachments 187
security 165, 187
selective attention bias 326–7
  conscious 329–31
  unconscious 327–8
selective mutism 60
selective norepinephrine reuptake inhibitors (SNRIs) 521, 522–30
selective serotonin reuptake inhibitors (SSRIs) 521, 522–30
self-blame 36–7
self-compassion 45–6
self-concealment 167
self-conscious emotions 159
self-construal 231
self-control theory 123
self-criticism 36–7
self-disclosure 276
self-judgment 449
self-kindness 449
self-presentation model of SAD 12–13
self-reappraisal 76–7
self-reference 76–7
self-regulation theory 121–3
self-report assessment 292–8, 312
  evaluating assessment tools 298
    reliability 299
    validity 299–303
  future of self-report assessment 311
    multiple types of tools 311–12
    psychometric analyses 312
  practical guide to assessment selection 303–4
    assessment of related constructs 304
    assessments at different levels of the model 304–5
    sample measure analysis 305–6
  real problems, illusory problems and problematic solutions 306–7
    behavioral measures 309–10
    limits to self-report 307–8
    psychophysiological measures 310–11
    some solutions 308
    utility of interviews 308–9
  recommended self-report assessment tools 293–7
self-reported submissive behaviors 446
self-verification theory 440
serotonin transporter (5-HTT) gene 400–2
sertraline 484, 525, 526, 528, 529
sexual attractiveness 33–4
sexual orientation 223
shame 41–2
shyness 57
Shyness (program) 574
simulated exposure 509
situation modification 417
situation selection 417
situational exposure 509
social attention–holding power (SAHP) 26
social attractiveness 40–1
  emotion regulation 42–3
  shame 41–2
social behavior 352–4
Social Behavior and Anxious Appearance Rating Scale 352
social bonds 160–1
social comparisons 442

social hierarchies 33
Social Interaction Anxiety Scale (SIAS) 232, 305–6
social interaction fears 252
social isolation 76, 160
social mentality 27
social neuropeptides 457–8
  endogenous release 458–9
  exogenous manipulation and release 459
  oxytocin administration 459–60
  synthesis, storage and site of action 458
  vasopressin administration 461
social neuroscience of SAD 67–8, 79–80
  current research 69
    brain imaging of socioemotional processing 69–72
    physiological and behavioral data 72–3
    summary 73–4
  future research directions 74
    genetics 74–5
    immune function 75–6
    insight from other disorders 79
    limited scope of social stimuli 77–9
    self-reference, self-reappraisal and social others 76–7
    social isolation 76
  scope 68–9
social others 76–7
Social Phobia and Anxiety Inventory (SPAI) 376–7
social rank 33–6
  low rank and positive evaluation 38–40
  paranoia and SAD 37–8
  self-blame and self-criticism 36–7
social reprisal fears 442
social skills 352–4
social skills literature 366–7
  future directions 382–3
  problems
    assessment measures 381–2
    comparison groups 378–9
    defining social skills 376
    laboratory tasks 379–81
    sample selection 376–8
  studies 368–75
  treating social anxiety by targeting social skills 382
social skills training (SST) 382, 479–80, 509–10
socially anxious individuals (SAIs) 344
  biased processing and behavior 346–8
  cognition and behavior 355–9
  social devaluation behaviors 348
    anxious behavior 354–5
    avoidance during social interactions 348–9
    behavioral and facial mimicry 350–1
    interpersonal distance and personal space 349–50
    social skills and social behavior 352–4
socially anxious temperament 111–12, 125–6
  clinical implications 125
  common themes among theories 123–4
  future directions 124–5
  personality 112
  personality theories
    five-factor theory (FFT) 116–18
    interpersonal circumplex theory 119–21
    self-regulation theory 121–3
    trait–affect theory 118–19
  personality, theory of 112–16
sociometer theory 12, 26, 159
SOFIE program 573
  effects 575–6
  therapist 579–80
Standards for Educational and Psychological Testing (SEPT) 298
  reliability 299
    internal consistency 299
    test–retest reliability 299
  validity 299–300
    beyond nomological net 302–3
    common method variance (CMV) 303
    convergent and discriminant validity 302
    definition of validity 300–1
    factorial discriminant validity 302
startle reflex 36, 94
state anxiety 444–5
state-level positive and negative affect 443
status evaluation 35–6
Stopa model of SAD 11–12

stress response 454–5
  endogenous glucocorticoids 455–6
  exogenous glucocorticoid administration 456–7
striatum 70
structural equation modeling 168
Structured Clinical Interview for DSM-IV (SCID-IV) 283
Structured Clinical Interview for Social Anxiety Spectrum (SCI-SHY) 284
submissive behaviors 34
  objectively assessed 446–7
  self-reported 446
submissive caring 44
subordinate living 39–40
subordination 33–6
  low rank and positive evaluation 38–40
  paranoia and SAD 37–8
  self-blame and self-criticism 36–7
substance abuse and SAD 549, 564
  future directions 563
    cognitive restructuring techniques 564
    individual treatment 563
    learning skills 563
  mechanisms of poorer treatment outcomes 554
    coping-motivated use 554
    performance fears 554
    socially motivated use 554–5
  public health implications 552
  rates 549–50
  recommendations 559
    assessment 559–60
    diagnostic feedback 560–1
    integrated treatment 561–3
  temporal relations 551–2
  treatment outcomes 552–3
    anxiety and relapse 553
    anxiety remains following treatment 553
    poor treatment adherence 553
  treatment populations 551
  treatment strategies 555–6
    integrated treatments 558–9
    parallel treatments 556–8
subtypes of SAD 99–101
  CBT outcomes 486
  comorbid avoidant personality disorder (APD) 249–50
  generalized social anxiety disorder (GSAD) 247–9
sympathetic activation 94–6
sympathetic–adrenal meduallary (SAM) axis 91, 454–5

taijin kyofusho (TKS) 102, 229–30, 253, 281
taxometric analyses 114
testosterone 462, 465
  endogenous release 462–3
  exogenous administration 463–4
thought record 502–3
threat displays 345
threat-detection systems 346
tiagabine 533
tobacco use 213, 550
topiramate 533
trait–affect theory 118–19
trait-level positive and negative affect 442–3
transactional model 183–4
translational research 391, 402–3
  behavioral genetics and endophenotypes of SAD 400
    brain-derived neurotrophic factor (BDNF) 401
    oxytocin receptor (OXTR) gene 401
    serotonin transporter (5-HTT) gene 400–2
  neuroimaging techniques 394–5
    amygdala hyperreactivity 395–7
    amygdala response before and after SAD treatment 398–9
    frontal involvement in emotion regulation 397–8
    treatment selection 399–400
  role of cognitive enhancers 392
    cortisol 393
    D-cycloserine (DCS) 392–3
    oxytocin 393–4
tranylcypromine 530

treatment for SAD
  see also cognitive behavioral therapy (CBT) for SAD
  implications from evolutionary models 43–4
  pharmacotherapy 237
  preventive intervention 142–7
  single case studies 237–8
  substance abuse and SAD 555–6
    integrated treatments 558–9, 561–3
    parallel treatments 556–8

unconditioned stimulus (US) 456
uncontrollable processing 333

valproic acid 533
vasopressin 458
  administration 461
  withdrawal behaviors 459
venlafaxine 526, 527, 528, 529
ventrolateral prefrontal cortex (VLPFC) 397
video feedback 478–9, 488, 510
virtual reality exposure therapy (VRET) 515

wellbeing 160–1
William's syndrome (WS) 79
word–sentence association paradigm (WSAP) 421