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Self-evaluation guide for medical students

400 Biochemistry MCQ's



Professor Maria R. Sanchez

A guide to self-evaluation

400 Biochemistry MCQ's

Professor Maria R. Sanchez



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Table of contents

Foreword	6
Section One	7
Reflections on undergraduate Biochemistry	7
Teaching, learning and evaluation	7
The illusion of understanding	8
Suggestions	10
The teacher's side	10
The student's side	15
Section Two	
Reflections on evaluating biochemical knowledge	20
Evaluation as a tool to achieve objectives	21
Section Three	
Multiple choice questions	26
On Structure of amino acids and proteins	27
On Enzymes	
On Cell Biology	41
On Membranes	58
On Introduction to Metabolism and Signal Transduction	63
On Krebs cycle and oxidative phosphorylation	68
Metabolism of Carbohydrates	
Metabolism of Lipids	94
Metabolism of Amino acids	
Hemoproteins and Nucleotide Metabolism	111
Nucleotide Metabolism	117
Metabolic Integration and Regulation	119
Nutrition and Digestion	138
Alcoholism	
Cutaneous Porphyria in alcoholic patient	149
Obesity	151
Metabolic Syndrome	154
Hypercholesterolemia and overweight	155

Hypercholesterolemia IIa	.156
Myocardial infarction	.158
Type 1 Diabetes	.160
Type 2 Diabetes	.162
Iron deficiency anemia	.165
Anemia associated to infectious processes	.166
Sickle Cell Anemia	.167
Glucose 6–P dehydrogenase deficiency	.168
Hyaline Membrane	.169
Gilbert's Syndrome	.170
Biochemistry is also useful in understanding other kinds of	
medical problems	.171
References	

Foreword

This book is intended to be a tool for medical students and their teachers. It is inspired fundamentally by my experience of over thirty years dedicated to teaching Biochemistry at the Razetti School of Medicine (Escuela de Medicina Luis Razetti. Facultad de Medicina. Universidad Central de Venezuela). The student readers will find in it a set of recommendations I hope will be useful for their approach to the study of Biochemistry. There is also a second part with a large series of questions designed for self-evaluation. In that part, younger teachers will find some guidelines for the making of questions, something that may lighten the burden this task poses to them.

The book is based on my experience as a teacher, something that over the years has been food for reflection, study and research on the obstacles that hinder student performance. I hope this work will suggest ways for teachers to help students to overcome such obstacles and students to practice self-help on this pressing matter. (1,2,3,4,5).

Section One

Reflections on undergraduate Biochemistry

Teaching, learning and evaluation

The illusion of understanding

It is a common experience to find that some students do not understand why they get low marks when evaluated on a subject they have the illusory feeling they understand fully and on which they have worked hard. We, teachers, have often met students overwhelmed by that sad situation, something that drives us to feel there is, unfortunately, very little we can do to get them out of error. It is indeed a hard problem and a difficult one to solve; in my view, for students this problem represents a very difficult hurdle and for teachers an even greater challenge than guiding them to grasp and comprehend a difficult concept that they consciously acknowledge not to follow.

The causes of this illusion are varied. Some of them have been identified by experts on the subject and lie in a bewildering wide range of factors that span from 'misconceptions' to the effect of internet on that illusion, the latter instance clearly shown by recent studies on the theme (6,7,8). It is not my intention to address that subject here. However, experience has allowed me to identify some frequent sources of error that can be treated here, albeit superficially.

One of them is the tendency frequently shown by students to satisfy themselves with teleological or functional explanations of what they are studying; this may lead them to believe this is what is expected not realizing that they have to demonstrate their knowledge of the biochemical events involved. An example: To the question 'Why does the synthesis of ketone bodies increase when fasting?' The answer is 'Because there is need to save glucose in order to ensure the function of tissues that need it' or something of that sort; this answer plainly disregards a series of hormonal and metabolic changes that result in ketone body synthesis.

In my opinion, this tendency to try explaining phenomena functionally or by teleology is a reasonable and natural mental process. Such a course of thought may even help us when doing research work in reasoning and understanding (I am sure many scientists will agree on this point). But, and this is a big but, in the student's case it may actually be an obstacle to learning biochemical phenomena.

In the example above, we can help overcome this type of obstacle if, while acquiescing to the 'functional' explanation, at the same time point out that cells (let alone molecules) do not 'think', and for that reason cannot know there is 'need to save glucose'; i.e. that there is an essential difference between a series of chemical events and a series of mental events.

It is also common among students the noxious idea that transforms biochemical reactions into something akin to alchemy. In this line of thinking molecules change 'magically'; it goes something like this: 'oxaloacetate becomes malate' or 'glucose becomes glucose 6-phosphate', and so on.

The enzyme concerned is missing!

In statements like those just mentioned, there is not a clear notion that when a substrate changes into product a chemical reaction has taken place. This image of a 'magical transformation' is probably the reason why students forget to mention the role of enzymes catalyzing intracellular biochemical processes, an allimportant point in the biochemical learning process, since regulation of metabolic pathways depends on activation/deactivation of certain enzymes. And that is a key concept to understand and explain metabolic changes happening in the body as an effect of the action of hormones or neurotransmitters.

Suggestions

The teacher's side

Ways to help

Part of the erroneous conceptions many students show may be due to an inability to construct an adequate mental representation of phenomena. In their view, Biochemistry, in contrast to Anatomy or Histology, is an abstract and alien subject and abstraction is not easy at this stage. This inability may have its origin in the way teachers and many textbooks approach some matters. For example, the naïve impression that a cell is a sort of big swimming pool where organelles and molecules move freely; thus we hear statements like "Glut 4 transporter 'translocates' from cytoplasm to membrane" that may actually impair a correct interpretation of this phenomenon. In this example, in order to facilitate a mental image of what the students are trying to learn and consequently its comprehension, it would be advisable to mention the role of the cytoskeleton, membrane adhesion molecules, membrane interconnection, etc. in order to facilitate a mental image of what the students are trying to learn and consequently its comprehension This mention to structure need not be detailed or extensive: a brief mention will suffice in most cases.

Another important point is that too often we, the teachers, fail to mention what to us but not for the student, is obvious; this simple lapse in explaining may be the reason for a lack of understanding a biochemical process. An outstanding example of this is found when biochemical knowledge is presented like alchemy and the need for molecular contact is not explained, when it should be stressed that 'molecules must come into contact' for a chemical change to happen; e.g. typically, transference of cholesterol and apoprotein between two lipoproteins is described in a majority of textbooks with expressions like 'HDL transfers...' etc. When explaining, we fail to emphasize the fact that for a chemical reaction to take place there must be a physical interaction between reactants. There must be an efficient interaction between molecules for a transformation to happen. It may seem obvious to us, but if it is not given due emphasis, for some students the result may be their inability to relate adequately a concatenated series of events. They do not grasp the idea that this contact is necessary and we do not realize we are using a biochemical language familiar to us but not to them. As Miguel de Unamuno (Spanish philosopher, 1864-1936) used to say 'There is great need daily to repeat what, being so well known, is forgotten'

1 The way students approach studying

Medical students are for the most part studious and disciplined; from this statement it follows that if they have problems with academic performance in Biochemistry, it is seldom due to lack of work; their problems are far more likely to arise from an inadequate way of tackling the subject matter.

A superlative example is the use of flash cards. This may induce the person that makes them to feel they know their contents, when probably all it does, at most, is ring a bell. Some students spend much time making impeccable cards or summaries, something that probably soothes their anguish because they get the impression that all contents is organized. It is sad to think that in fact they have wasted precious time that would have been better used in trying to understand what they are writing down in the card. Wrongly, they feel that summaries are abbreviated copies of the program's contents instead of what they should be: a brief of what they have comprehended and understood. Alas! Making summaries makes sense when it is a personal synthesis of what has been learned.

In my university there exists the figure of Advisory Teacher: students attaining poor results are assigned to a lecturer for

guidance. As part of my work I played that role for many years. I always started by asking students to show me their notes, summaries and any other relevant material of study they made and used for studying. In many cases there were beautifully color-illustrated copybooks and to my surprise, some of them had been handed down by students that had previously passed the Biochemistry course. They were baffled when told 'Throw away the lot, for it is of no use to you'. Surprisingly (or not?) very few took my advice!

Another fact of relatively new occurrence is the use of internet. It may goad the student into the belief that this information in the web is akin to having it 'inside their head' (8).

Although not intending to reinforce memorization, I firmly believe it is indispensable to memorize some ideas and facts. In teaching, it is generally taken as pejorative dogma that to memorize is sinful. Nevertheless, it is virtually impossible, for example, to deduct what happens in metabolism when there is lack of insulin if a few facts are not taken into account. And evidently those facts are used in the deducting process because they are in our memory. What is certainly inconvenient is to memorize mere facts and/or useless (or nearly so) knowledge. For instance, it is of little use, if any, to memorize all the reactions of glycolysis if there is no comprehension of glycolytic activity within the frame of particular metabolism of the diverse tissues. and the functioning of the organism as a whole. In ultimate terms, this is what really matters from a physiological point of view. The importance of insisting on the regulation of enzyme activity in the various metabolic pathways and the complex interactions among them cannot be overestimated. Teachers have a significant role here in the guidance of the student on what should be memorized and what should not.

2 The importance of Biochemistry in medical studies

Most medical schools feel the need to stress the important relationship between Biochemistry (and other so-called 'basic

sciences') and sound medical practice. This has brought the incidence of new ways of teaching, the restructuring of programs and the use of vignettes referring to clinical conditions. I contend that it is not good practice to obviate some biochemical knowledge just because it cannot be evidently related to disease and clinical practice. In my view some instances of basic biochemical knowledge are necessary to understand medical publications or certain clinical conditions. For example, the enzymic concept of the constant K_M is an essential one to understand the activity of enzymes dealing with glucose in hepatocytes.

There is another pitfall on this subject, which is to make a forced relationship with a clinical subject, something that may easily upend Biochemistry teachers. An outstanding example: using a case of acute porphyria to highlight the importance of biochemical knowledge. Normally, a case is presented of a patient that comes to hospital with abdominal pain of unknown etiology, a typically challenging case for doctors. But then biochemists make up questions about biochemical aspects of porphyria that have nothing to do with the pain that brought the patient to hospital or the biochemical changes that contribute to this pain appearing... simply because, to this day, nobody knows for certain the reason for such pain! This is a clear [wrong] message to students: the relationship of Biochemistry and clinical studies is something esoteric.

Would it not be better to use a case of hematopoietic porphyria? This disease, besides being more frequent, does allow relating the effect of sunlight on molecules with the patient's symptoms. And also, using it as model would allow the connection to the use of δ aminolevulinic acid in phototherapy. This porphyria may be less interesting from the intellectual clinical point of view, but it shows a clear link between biochemical knowledge and understanding disease from the scientific angle. Yes, it is an agreed dogma that a scientific training is an obvious necessity for a good doctor.

On the other hand, it is of the utmost importance to realize that the practice of Medicine is a complex endeavor: without the ability to communicate with patients and an understanding of their individuality, clinical Medicine loses a sizeable part of its sense and efficacy. This poses a dilemma: arguably, scientific knowledge is not the most important part of the skills a medical student must acquire. And, yes again, at the other end of the spectrum is the emphasis on irrelevant biochemical detail for the comprehension of molecular basis of disease. An amalgam of these two factors, the individuality of patients and irrelevant detail might make the student believe that there is no need for Biochemical knowledge to be a doctor.

Following this line of argument, let us think about the idea of highlighting the relationship between Biochemistry and Medicine by means of the use of rare diseases as examples. This may be very illustrative of the importance of a particular enzyme, like the deficiency of muscle glycogen phosphorylase (McArdle's disease). This may be very interesting for biochemists, but for medical students it might suggest that Biochemistry is related only to strange diseases of little frequency. If this type of example is to be used, there must be caution and emphasis in that these diseases are examples and the students are unlikely to see a case of any of them in their whole life.

Actually, there are frequent pathologies, like diabetes, obesity, hyperlipidemias, iron deficiency anemia, liver diseases and cancer among others, that offer good opportunities to illustrate the important relationship of Biochemistry and Medicine. I do not apologize for my insistence on this point, it is wholly intentional.

Furthermore, making the case for Biochemistry in Medicine and in parallel with the above warning, there is no doubt that biochemical knowledge is essential to confront some misleading offers of drug advertising, a tendency that in present day world is no small matter. Nowadays and with increasing frequency, a doctor who has the discipline or the need or just plain and simple curiosity of reading scientific articles, faces (in any field) a very strange language, fit for experts, full of terms and abbreviations that seem to make up a secret code. In this area teachers of Biochemistry have a task as important as linking Biochemistry and Medicine: capacitate the student for self-learning, for continuous education. This has become a very important matter and, in fact, it is a problem that tends to get more complicated with time.

The student's side

Suggestions for students: How to study.

The following recommendations are complementary to those on hygiene of study habits (not dealt with here) and are specific for studying Biochemistry.

Question the book. Do not read it as a novel. Work on the concepts, and since it is known that attention to study diminishes after about 45 minutes, it is advisable to take a small break after this lapse of work. This also means that if you spent that time reading generalities you will have 'wasted' time reading not-very-important issues. It is indeed surprising how little students use the analytical index to look for what they want to know. Try to get familiar with its use, it will save a lot of time.

Try to make a study plan encompassing all you want to revise. Try to be specific on what you want to know, which requires assessing with precision what you know or think you know. Previous evaluations, if accessible, may be very useful for this.

I consider it excellent habit that teachers foster analysis of student's mistakes in evaluations.

Don't be afraid of chemical formulae: they may be useful to understand processes.

Searching through Internet

I recommend finding and using criteria of confidence for information found in Internet. There are quite a few universities'

pages on the subject. Here I mention some of them:

http://guides.lib.berkeley.edu/evaluating-resources

http://www.library.georgetown.edu/tutorials/researchguides/evaluating-internet-content

It is also recommended learning to make your search more efficient:

http://www.ed.ac.uk/information-services/library-museumgallery/finding-resources/library-databases/databases-overview/ databases-search

http://libraryguides.mta.ca/research_help/research_tips/ academic_research

While it is important to become an expert searcher it may be harmful to become <u>a compulsive searcher</u>. Keep in mind that finding relevant and trustworthy information does not mean it is understood. In just the same way it is unnecessary to read three (or many) textbooks to learn the basics, it is unnecessary to spend hours searching internet for something you can easily find in a textbook. Actually, the internet features that may be of great help in understanding and gaining insight and knowledge are animations and simulations. Here again, it is vital to make sure the source is valid.

Now, a few helpful tips that may be used to study metabolism.

Bear in mind that an immense majority of biochemical transformations are catalyzed by enzymes and that they will not happen at any detectable speed in the absence of a specific enzyme; this is a basic reason why every time you refer to a biochemical transformation, **the enzyme involved in catalysis must be mentioned**.

Draw a scheme on a sheet of paper for every metabolic pathway, especially pointing:

Tissue where it takes place and subcellular location Irreversible reactions

Reactions where ATP is formed or spent (Also GTP)

Reactions where oxidation-reduction coenzymes are at work (NAD, NADP, FAD)

Reactions where branching of a metabolic pathway occurs or where there is connection to another pathway.

Regulatory enzymes.

Keep these sheets handy all the time you are studying, for you will have to refer to them constantly for specific reactions. Don't try to memorize them at the first approach. As you analyze each metabolic pathway, its regulation and relationships to other pathways, you will memorize them effortlessly, if that should turn out to be necessary.

You must make it clear in your mind whether an enzyme is a regulatory one, and if so:

To what kind of regulation is it subject?

Does a hormone trigger its activation of inhibition ('signal transduction')?

Identify the enzymes that interconnect pathways that happen

+

simultaneously. For example, reactions that use NADH + H from beta oxidation of fatty acids for gluconeogenesis.

Identify biochemical facts that allow these interactions. Keep in mind that neither molecules nor cells 'think' and therefore a biochemical transformation only happens if the enzymes involved are not only present but in thei0r active form, besides other necessary chemical conditions. Separate pathways according to the physiological situation in which they occur. Enumerate those in fasting condition (glucagon and adrenalin prevalence) and those in post absorptive state (insulin is prevalent)

Identify in each physiological situation which ways are oxidative or catabolic and which anabolic. Notice that in each physiological situation oxidative ways provide the reducing power and energy for the synthetic ways happening simultaneously.

Identify hormonal changes that bring about changes in each situation as well as the way in which hormones influence enzymatic activity:

Receptors, G proteins, transcription factors

Production of 'second messengers'. (Which?)

Activation and or inhibition of enzymes (Which?), via what mechanism? Allosteric? Covalent modification?

Regulation of gene expression

Following these guidelines may help you to understand better what you read and minimize the need for memorizing facts.

As for the themes dealing with storage and transmission of genetic information, once again: Enzymes, enzymes, enzymes. Particular features of each one of those processes depend on enzyme characteristics. For example, the impossibility of DNA polymerase to initiate synthesis starting from two mononucleotides explains the need for a primer.

Make sure you understand what 5' and 3' mean.

Keep in mind that all relationships among nucleic acids are

given by complementarity anti parallelism.

It may be obvious for many, but even as generally explained: first replication and then transcription and translation, only the latter two are sequential. At which point in cell life do replication, transcription and translation occur? How are they regulated?

If you understand the basics, you will find little trouble in understanding the rest of it.

Section Two

Reflections on evaluating biochemical knowledge

Evaluation as a tool to achieve objectives

Any conception teachers have on the role of Biochemistry in medical studies will be inevitably found in the way they evaluate students, something that to my mind would lead to the desirable practice of self-questioning on this subject.

In spite of the teacher insisting on the importance of a certain point, or the emphasis on that point made in the textbook, the student will not realize his understanding (or lack of) until the point turns up in evaluation. And this is one of the functions of evaluation, be it formative or additive: to make the student conscious of what has been understood and what has not. But this is a valid point only if evaluation is rightly done. The way we plan and deploy evaluation can contribute to overcoming obstacles to understanding...or foster them!

For example, if when evaluating we insist on molecular details of little relevance for a clear understanding of the theme, then students will focus their attention on those details when studying. We humans have a knack for interpreting signals that point to what is expected from us.

The teacher must ask himself what he/she wants the students to learn. Sometimes it is good practice to write the answer we would like to receive before writing the question to which 'that' is the answer. When we assemble multiple selection questions, we frequently think of erroneous (false answer) items because we remember mistakes made by students.

A good question can be a stimulus to curiosity or direct attention to aspects not taken into the picture previously. The importance that questions considered easy may attain cannot be underestimated: they may be clarifying and integrating, thus becoming organizers of thought.

If the main interest is to underline relationships between tissues, processes and/or metabolic pathways, some questions may

be very easy. For example:

Apo B48 and Apo B100 have in common:

- a) The kind of lipoprotein that transports them
- b) The receptor to which they attach
- c) The gene that codifies for them
- d) Being freely transferable between lipoproteins
- e) The tissue where they are synthesized

Just intends to bring attention to RNA processing and lipoprotein metabolism.

Multiple choice questions

There is much bibliography pointing to features a good multiple-choice question should have. I especially recommend: Item Writing Manual from National Board of Medical Examiners (NBME)

http://www.nbme.org/publications/item-writing-manual.html

- Test Construction: Some Practical Ideas for Marilla D. Svinicki, available in:

http://www.rubrics4assessment.net/test_construction.pdf

Presently I include a summary:

- 1. The leading statement must be meaningful itself. It may or may not be a question. One word statements are not recommended
- 2. It should not contain non-pertinent material
- 3. As far as possible, avoid negative statements. Like 'The following are NOT properties...'
- 4. All items must be grammatically congruent with the leading statement
- 5. There must be only one correct answer. Avoid the use of 'the most correct...' or 'the best explanation...'
- 6. Item length should not be a clue i.e. the correct answer should not be the longest

- 7. All items should be plausible. Avoid items too obviously wrong.
- 8. Reduce to a minimum or 0 the use of special alternatives like 'All of the above', None of the above', A and C are correct, etc.
- 9. Avoid the use of interdependent answers, that is: only if one answer is correct it is possible to answer another correctly

Questions with 'all of the above' should not be abused of, but they may be useful to drawing the student's attention towards some aspects they may overlook or that we know are frequent sources of error. In this case they become good questions for self-evaluation sessions.

In this book it is avoided to show questions that in the leading statement have expressions like 'except', 'it is not correct', etc. There will always be one correct answer item. To my mind it is counter to good sense making the correct answer a false statement and I think it may even add to confusion as a component of the question.

Making questions in Biochemistry for evaluation of medical students

The following suggestions may help teachers to make questions to evaluate medical students of Biochemistry. Obviously, they do not exclude others more elaborate and complex. Nevertheless, their usefulness may derive from the fact that in most instances, university teachers of the subject are in the job mainly by being experts in Biochemistry rather than experts on education.

Evaluation in Biochemical themes often involves questions on the following aspects of the subject:

1. MOLECULES: Characteristics that make some molecules stand out due to some aspects of their structure or function or in some cases the relationship between the two. Metabolic intermediates, energy-reserve molecules, enzymes, transporters, storage-association, genetic expression, etc.

- 2. PROCESSES: Digestion and absorption of nutrients, transport mechanisms through membranes, storage, genetic expression, signal transduction, etc.
- 3. METHODS: Techniques and procedures used to achieve biochemical knowledge.

On all these it is possible to inquire about their:

- 1. REGULATION: Ways to regulate enzyme activity and processes. Role of hormone-generated signals.
- 2. STRUCTURE: Outstanding aspects of molecular structure, mainly those related to their function.
- 3. LOCALIZATION: Subcellular, cell or tissue location of enzymes and/or processes.
- 4. INTER RELATION of processes among them, according to the physiological situation, location, etc.
- 5. ENERGY AND EQUILIBRIUM: Thermodynamic aspects related to reactions
- 6. FUNCTION: Role of a molecule or enzyme, a reaction or process in cellular function.

It may also be useful to be guided in what is to be asked by what is expected in the answer. It may be:

1. DEFINE

Group by categories and subcategories Identify essential properties

2. COMPARE:

Establish similarities and differences

3. DESCRIBE

Enumerate qualities, properties, characteristics of the object or phenomenon being described

4. EXPLAIN

Establish causal relationships

Establish conditional judgement (of the sort If...then...)

Of course, the kind of question depends on the expected achievement, of objectives or competences to be evaluated. Some associations are easier than others to establish. In multiple-choice evaluation this organization of the aspects to be evaluated will help in the choice of items, drawing attention to certain points.

Examples

Example A: <u>DEFINE</u> THE STRUCTURE OF A MOLECULE Identify the following functional group:

- a) Aldehyde
- b) Carboxyl
- c) Hydroxyl
- d) Ester
- e) Ketone

Example B: EXPLAIN WHEN A REACTION IS AT EQUILIBRIUM

A chemical reaction is at equilibrium when:

- a) initial speed is the same as final speed
- b) velocities of formation of reactants and products are equal
- c) all reactants have become products
- d) concentrations of reactants and products are equal
- e) the speed of product formation is greater than that of products

Example C: A combination

TO <u>DEFINE</u> THE STRUCTURE AND <u>FUNCTION</u> OF A MACROMOLECULE

Bacterial structures known as plasmids are:

- a) two-chain DNA molecules
- b) extrachromosomal DNA molecules
- c) copied many times in each cell division
- d) molecules carrying genes (that confer antibiotic resistance to bac
- e) all of the above

Section Three

Multiple choice questions

On Structure of amino acids and proteins

1) Alanine's lateral chain (-CH3) is classified as:

- a) aromatic
- b) polar
- c) non polar
- d) acid
- e) basic

A1

2) In the following ionization reactions of functional groups of amino acids:

```
\begin{array}{l} \text{R-COOH} \rightarrow \text{R-COO}^- + \text{H}^+ \\ \text{R-NH}_2 + \text{H}^+ \rightarrow \text{R-NH}_3^+ \end{array}
```

- a) R-COO- is the conjugate base of R-COOH
- b) R-COOH and R-NH2 are non dissociated acids
- c) R-COO- y R-NH3+ are conjugated bases
- d) R-NH3+ is the conjugate base of R-NH2
- e) R-COOH y R-NH2 are conjugated bases

A2

3) What is the net charge of glycine at pH = 6?

```
pK COOH= 2,3 ; pK NH<sub>3</sub><sup>+</sup> =9,7

a) -1

b) 0

c) -2

d) +1

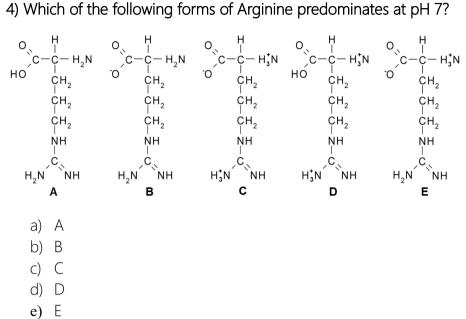
e) 2

A3

A1 \underline{C} \leftarrow

A2 \underline{A} \leftarrow
```

A3 <u>**B**</u>←



A4

5) Peptide: ala-glu-ser-lys-gly will bind to an anion exchange resin at pH:

- a) 13
- b) 7
- c) 5
- d) 1
- e) equal to the peptide's pH

A5

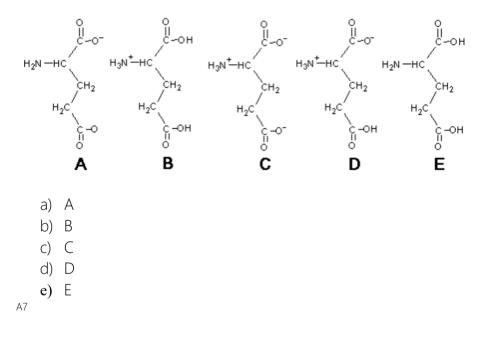
A4	<u>C</u>	<u>←</u>
A5	A	<u> </u>

6) In electrophoresis, which of the following peptides will migrate to the cathode at pH 7?

- a) ala-glu-ser-phe-gly (A-E-S-F-G)
- b) asp-gly-glu-ser-asp (D-G-E-S-D)
- c) tyr-ser-tyr-thr-ser (Y-S-Y-T-S)
- d) ala-leu-ile-gly-val (A-L-I-G-V)
- e) his-arg-tyr-lys-val(H-R-Y-K-V)

A6

7) At pH 7, which of the following forms of glutamate prevails?



A6_	E	←
A7	<u>C</u>	←

8) The binding of oxygen to Hb:

- a) Is favored by a decrease in pH
- b) displaces the proximal histidine residue to the plane of the heme ring
- c) brings about the oxidation of heme from ferrous to ferric
- d) Is favored by 2,3 biphosphoglycerate
- e) brings about the formation of inter and intramolecular salt bridges

A8

9) In proteins, primary structure refers to the:

- a) amino acid sequence
- b) subunit contents
- c) tridimensional conformation
- d) alfa helix or beta pleated sheet contents
- e) salt bridges and disulfide bonds contents

A9

10) Tertiary structure of proteins is stabilized by means of

- a) hydrogen bonds
- b) salt bridges
- c) van der Waals' forces
- d) disulfide bonds
- e) all of the above

A10

A8 <u>B</u> ←
A9 <u>A</u> ←
A10 <u>E</u> ←

11) The unfolded protein response (UPR) may include:

- a) decrease in protein synthesis
- b) stimulation of the synthesis of more chaperon proteins
- c) bring about the degradation of badly folded proteins
- d) induce cellular apoptosis
- e) all of the above

A11

12) Chaperonins are proteins that recognize badly folded proteins by means of regions that are:

- a) hydrophobic and exposed to the aqueous environment
- b) rich in positively charged amino acids that bind to ATP
- c) randomly folded
- d) folded as beta when they should be alpha helical

A12

13) An Unfolded Protein Response (UPR) may arise in:

- a) viral infections
- b) parasite infections
- c) fever
- d) obesity
- e) all of the above

A13

A	1	1	E	(—	
			٨		

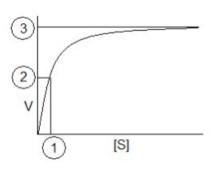
A12<u>A</u>←

A13 <u>E</u> ←

14) The graph below represents the relationship between concentration of glucose and the velocity of the reaction catalized by hexokinase:

Glucose +ATP \rightarrow glucose 6 P + ADP

Identify what is represented by each number



- a) 1= Vmax; 2= K_M; 3= ½ Vmax
- b) 1= Vmax; 2 = ½ Vmax; 3= 1/2 [S]
- c) 1= Vmax; 2= ½ Vmax; 3= K_M
- d) 1= K_M; 2= ½ Vmax; 3= Vmax
- e) 1= Vmax; 2= ½ Km; 3= Km

A14

15) Enzymes catalyze reactions because they decrease the:

- a) velocity of formation of the ES complex
- b) reaction's energy of activation
- c) velocity of dissociation of the EP complex
- d) difference in energy existing between reactans and products
- e) reaction's Keq

A15

A14 $\underline{D} \leftarrow$ A15 $\underline{B} \leftarrow$

16) An international unit of an enzyme is the amount of:

- a) enzyme that transforms 1 µmol de substrate per minute per mg de protein
- b) substrate that can be transformed per molecule of enzyme per second
- c) enzyme that transforms 1 mol of substrate per second
- d) enzyme that transforms 1 μmol of substrate per minute
- e) enzyme total present in a sample

A16

17) Trypsin is secreted as a precursor of the type known as zymogens, characterized by being:

- a) multiple forms of the same enzyme
- b) activated by covalent irreversible modification
- c) denatured enzymes
- d) activated when they join their apoenzyme
- e) enzymes that have no prosthetic group

A17

18) The following reaction is catalized by a transaminase, which belongs to the group of:

- a) hydrolases
- b) transferases
- c) lyases
- d) ligases
- e) isomerases

A18

A16 $\underline{D} \leftarrow$ A17 $\underline{B} \leftarrow$ A18 $\underline{B} \leftarrow$ 19) In a metabolic pathway, the enzyme proportionally least limiting the velocity is that which:

- a) is at the beginning of the pathway
- b) catalyzes an irreversible reaction
- c) catalyzes reactions that are near equilibrium
- d) shows low activity due to a high K or low Vmax

Μ

e) is present in low concentration

A19

20) In an enzymatic reaction, a non-competitive inhibitor:

- a) diminishes Km
- b) diminishes Vmax
- c) diminishes K_M and Vmax
- d) does not affect Vmax
- e) augments KM

A20

21) Relating to kinetic characteristics of an enzymatic reaction, it is correct to assert that:

- a) KM is a measure of the velocity of reaction
- b) Vmax is reached when enzyme and substrate concentrations become equal
- c) $^{1\!\!/}_{2}$ de Vmax is reached when S is equal to Km
- d) Vmax is independent from enzyme concentration
- e) KM is equal to 1/S which is the point when ½ Vmax is reached

A21

- A19 <u>C</u> ←
- A20 <u>**B**</u>←
- A21 <u>C</u>←

22) Phosphofructokinase I is an allosteric enzyme, so it follows that its active site:

- a) may suffer modifications in amino acid sequence due to regulating metabolites
- b) has little specificity for the substrate, which gives the enzyme its regulatory character
- c) besides the substrate, it can recognize some regulating metabolites
- d) can be spatially reorganized when a regulator molecule binds to the enzyme

A22

23) The conversion of trypsin zymogen into the active enzyme is achieved through:

- a) covalent irreversible changes
- b) covalent reversible changes
- c) non-covalent modifications
- d) allosteric modulation

A23

24) Regulation of enzymatic activity through irreversible covalent modification implies:

- a) hydrolysis of specific peptide bonds
- b) binding of components at points different from the active site
- c) phosphorylation
- d) breaking of disulphur bridges
- e) polymerization of multienzymatic complexes

A24

A22 <u>E</u> ←

A23 **A** ←

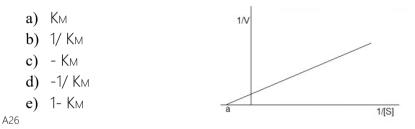
A24 <u>D</u> ←

25) When glucose concentration is equal to K_M of hexokinase, the velocity of reaction is:

- a) Vmax
- b) 0,5 Vmax
- c) 2 Vmax
- d) numerically equal to K_M
- e) the initial velocity

A25

26) In the following representation of Lineweaver-Burk's equation, what does point a represent?



27) KM of glucokinase is ~ 7 mM. When the concentration of glucose is much lower than that value, the reaction's velocity is:

- a) equal to maximum velocity
- b) proportional to substrate concentration
- c) half maximum velocity
- d) very slow
- e) cannot be measured

- A25 <u>**B**</u>←
- A26 **A** ←
- A27 <u>B</u>←

28) Which one of the following is an indispensable condition for the reaction to proceed?

Glucose + ATP \rightarrow glucose 6P + ADP + Pi ΔG° = -4,0 kJ mol⁻¹

- a) increase the concentration of ATP
- b) increase the temperature
- c) presence of hexokinase
- d) hydrolysis of ATP
- e) increase the concentration of glucose

A28

29) The velocity of the immense majority of reactions inside cells will depend on the:

- a) ΔG value
- b) Keq value
- c) presence of an enzyme
- d) concentration of products
- e) concentration of reactants

A29

30) The maximum velocity of an enzymatic reaction depends on the:

- a) proportion of molecules in transition state
- b) affinity of the enzyme for its substrate
- c) value of the equilibrium constant
- d) concentration of substrate
- e) ΔG° of the reaction

A30

A28 $\underline{C} \leftarrow$ Despite the reaction having a negative ΔG , it will not proceed at any detectable speed unless an enzyme is present. Whence the importance of always making sure an enzyme is present when depicting intracellular reactions.

A29 <u>C</u> ←

A30 <u>A</u> ←

31) Coenzymes supply the enzymatic reactions in which they participate with:

- a) a regulatory site for the enzyme activity
- b) stability for the enzyme-substrate complex
- c) a functional group participating in catalysis
- d) the necessary energy to reach the transition state

A31

32) A substrate and an allosteric regulator of a given enzyme have in common that they:

- a) bind covalently to their respective sites in the enzyme
- b) determine conformational changes on the enzyme
- c) are found in equimolar concentrations
- d) are modified during the reaction

A32

33) An inhibitor is more specific

- a) the more it resembles the substrate
- b) If it binds covalently to the enzyme
- c) It binds covalently to the functional groups of the active site of the enzyme
- d) If it impairs efficiently the binding of the substrate to the enzyme

A31	C	←	

- A32 <u>B</u>←
- A33 <u>A</u>←

34) In an enzymatic reaction the transition state is characterized by:

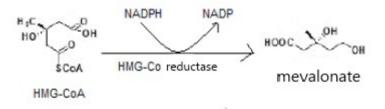
- a) the substrate is its maximum molecular distortion state
- b) both enzyme and substrate are in a distorted electronic configuration
- c) having an unstable electronic configuration
- d) having a high energetic level
- e) all of the above

A34

35) Statins share in their structure the following chemical



They are drugs used to inhibit hydroxymethyglutaryl coenzyme A reductase (HMG-CoA reductase) an enzyme participating in cholesterol synthesis by catalysis of this reaction:



What kind of inhibition is that of statins over HMG-CoA reductase?:

- a) allosteric
- b) acompetitive
- c) non-competitive
- d) competitive

A35

A34 <u>E</u> ← A35 <u>D</u> ←

36) pH changes affect reactions catalized by enzymes because they produce changes in the ionization of:

- a) amino acid residues involved in the binding of the substrate
- b) amino acid residues involved in catalysis
- c) substrate groups
- d) amino acid residues involved in enzyme stability
- e) all of the above

A36

37) The Michaelis-Menten constant (Km) is:

- a) low for enzymes having a greater Vmax
- b) numerically equal to the equilibrium constant for the reaction of dissociation of the complex (E-P) to E + P
- c) the substrate concentration at which $v = \frac{1}{2}$ Vmax
- d) higher the higher the affinity of the enzyme for the substrate
- e) all of the above

A37

38) Generally speaking, regulatory enzymes catalyze reactions:

- a) at the beginning of metabolic pathways
- b) at branching points of metabolic pathways
- c) far from chemical equilibrium
- d) whose velocity is modified by small changes in substrate concentration
- e) all of the above

A36	<u>E</u>	<u> </u>

- A37 <u>C</u> ←
- A38 <u>E</u> ←

39) In clinical lab results enzyme activity is generally reported as:

- a) Specific activity
- b) International units
- c) Enzymatic activity
- d) Catalytic activity
- e) katals

A39

40) The specificity of the reaction catalized by an enzyme is determined by:

- a) the chemical structure of the substrate
- b) the amino acid residues in the active site
- c) its KM value

d) its sensitivity to regulatory mechanisms

A40

On Cell Biology

41) DNA and RNA polymerases have in common:

- a) the need for a DNA template
- b) direction of synthesis $(5' \rightarrow 3')$
- c) liberation of pyrophosphate as a product
- d) catalysis of ester bond formation
- e) all of the above

A41

A39<u>C</u>←

A40 <u>**B**</u>←

A41 <u>E</u> ←

42) Sterol accumulation in the plasma membranes is a characteristic of cells that are:

- a) apoptotic
- b) malignant
- c) in phase S of the cell cycle
- d) foam cells
- e) stem cells

A42

43) In order to incorporate the genetic information contained in an mRNA to a bacterial genome it is indispensable to use:

- a) the enzyme reverse transcriptase
- b) a plasmid
- c) the enzyme DNA ligase
- d) a restriction endonuclease
- e) all of the above

A43

44) Restriction enzymes:

- a) hydrolyze phosphodiester bonds in all types of nucleic acids
- b) require a free 3' –OH for their activity
- c) pull apart the two chains of DNA in specific regions
- d) have editing function
- e) are specific for pulling apart DNA chains in regions separating genes

A42 <u>D</u> ←	
A43 <u>E</u> ←	
A44 C ←	

45) The correct sequence in the transference process called 'southern blot' is:

- a) alkaline dissociation of DNA to separate the strands, electrophoresis in agarose gel, digestion by a restriction enzyme, transfer to a nitrocellulose membrane
- b) digestion of DNA by a restriction enzyme, alkaline dissociation to separate the strands, electrophoresis in agarose gel, transfer to a nitrocellulose membrane
- c) digestion of DNA by a restriction enzyme, electrophoresis in agarose gel, alkaline dissociation to separate the strands, transfer to a nitrocellulose membrane
- d) alkaline digestion to separate strands, digestion with a restriction enzyme, electrophoresis in agarose gel, transfer to a cellulose membrane
- e) digestion of DNA by a restriction enzyme, electrophoresis in a nitrocellulose membrane, alkaline dissociation to separate strands, transfer to an agarose gel

A45

46) Bacterial structures called plasmids are:

- a) double stranded DNA molecules
- b) extrachromosomal DNA molecules
- c) copied many times in each cell division.
- d) gene carriers that confer antibiotic resistance to bacteria.
- e) all of the above

A46

A45 $\underline{\mathbf{C}} \leftarrow$

A46 <u>E</u> ←

47) To perform a polymerase chain reaction (PCR) it is indispensable to have:

- a) a DNA template
- b) deoxyribonucleotides
- c) RNA primers
- d) tag polymerase
- e) all of the above

A47

48) The PCR reaction in real time is different from conventional or standard PCR because it:

- a) needs recently extracted DNA
- b) does not amplify DNA but RNA
- c) is more efficient, but slower than conventional
- d) allows quantification of the amplified product
- e) may be used to determine the sequence of the amplified product

A48

49) The following reaction:

....C-C-U-A-U-C-OH- \rightarrow ..C-U-A-U-OH + C

Is catalized by:

- a) DNA polymerase I
- b) helicase
- c) gyrase
- d) DNA ligase
- e) 3'-5' exonuclease

A49

A47<u>E</u>←

A48 <u>D</u> ←

A49 <u>E</u> ←

50) If transcription of the DNA fragment depicted below were from left to right

.... ATTCAG..... 5'TAAGTC.....

The sequence of the resulting RNA fragment would be:

- a) 5'....GACUUA...
- b) 5'....ATTCAG....
- c) 5'....AUUCAG....
- d) 5'....UAAGUC....
- e) 5′....CTGAAT....

A50

51) Analysis of a nucleic acid fragment results in adenine concentration different from that of thymine and guanine different from cytosine, the conclusion would be:

- a) double helix DNA
- b) double helix RNA
- c) single stranded RNA
- d) single stranded DNA
- e) messenger RNA

A50	<u>C</u>	<u>←</u>
A51	D	←

52) In the process of protein synthesis:

- a) each amino acid recognizes its place on mRNA owing to its specific structure
- b) the binding of ribosomal RNA to mRNA is possible due to their bases homology
- c) each amino acid binds selectively to the anticodon of a specific tRNA
- d) the place of each amino acid in the chain is determined by the mRNA sequence
- e) all of the above

A52

53) Given the following sequence of mRNA, choose which tRNA will be next to incorporate:

....CGCUAAUGGUAUGCAGC...

a) 1

- b) 2
- c) 3
- d) 4
- **e**) 5

A53

A52 $\underline{D} \leftarrow$

A53 <u>B</u>←

54) Replication is said to be semiconservative because:

- a) DNA polymerase III does not make mistakes
- b) each new resulting double helix is made up of one original strand and a new one
- c) only one of the strands is conserved by replication
- d) DNA polymerase I makes a mistake every 100 million bases
- e) each of the four resulting chains is made by 50 per cent DNA of original chains and fifty percent of new DNA

A54

55) In order to obtain mRNA from a given cell type affinity chromatography may be used joining the matrix to a polynucleotide with a base sequence:

- a) poly U
- b) complementary to the introns
- c) repetitive o initiation codons
- d) repetitive of initiation anticodons
- e) in which phosphate is replaced by another polyvalent cation ${\rm }^{\rm A55}$

56) Highly repetitive DNA is made up of sequences that go from hundreds to millions of copies that:

- a) are the regions recognized by transcription factors
- b) guarantee there are enough copies of the constituting genes
- c) codify only for rRNA and tRNA
- d) codify for histones
- e) are not transcribed

A56

A54 <u>D</u> ←

A55 <u>A</u>←

A56 <u>E</u> ←

57) Tetracyclines are antibiotics capable of inhibiting:

- a) peptidyl transferase
- b) the start of translation
- c) the cell cycle
- d) the activation of amino acids

A57

58) If a sample of double stranded DNA has 20% moles of guanine, what would be the percentage of moles of thymine?

- a) 20%
- b) 30%
- c) 40%
- d) 80%
- e) not known. Insufficient data

A58

59) In prokaryotes, as translation proceeds the growing polypeptide chain remains bound to the ribosome through:

- a) an amino acid-bearing protein
- b) ribosomal 55 RNA
- c) a segment of mRNA
- d) an aminoacyl-tRNA
- e) peptidyl transferase

A57	B	<u>←</u>

- A58 <u>**B**</u>←
- A59 <u>D</u> ←

60) Which of the following antibiotics inhibits peptidyl transferase?

- a) streptomycin
- b) rifamycin
- c) actinomycin D
- d) tetracycline
- e) chloramphenicol

A60

61) In eukaryotes one of the characteristics of mRNA that differentiates it from ribosomal and tRNA is:

- a) to have a poly A tail
- b) being synthesized in the nucleus
- c) be modified after transcription
- d) to have methylated bases
- e) to have double-helix regions

A61

62) How many amino acids will have the peptide product of the expression of the following polydeoxyribonucleotide?

5'-CCTACCGCGGAATCATTAACAT-3' Start codon = AUG

a) 4

- b) 5
- c) 6
- d) 7
- e) 8

A62

A60 <u>E</u> ←

A61 <u>A</u>←

A62 <u>A</u>←

63) Transcription of an eukaryotic gene may give rise to:

- a) an oligomeric protein
- b) a polypeptide
- c) several polypeptides
- d) a ribonucleic acid
- e) a double stranded chain of DNA

A63

64) During replication, helicase acts:

- a) stabilizing the replication bubble
- b) avoiding DNA supercoiling
- c) hydrolyzing phosphodiester bonds in the direction $5' \rightarrow 3'$
- d) breaking hydrogen bonds between base-pairs of DNA
- e) recognizing the promoter site

A64

65) It is said that the genetic code is partially degenerate because:

- a) there are amino acids not codified by any codon
- b) some codons do not codify for any amino acid
- c) an amino acid has the same codon in all species
- d) a codon may codify for more than one amino acid
- e) an amino acid may have several codons

A63	D	<u>←</u>	

- A64 <u>D</u> ←
- A65 <u>E</u> ←

66) The high fidelity of the process of protein synthesis is due mainly to:

- a) the activity of peptidyl transferase
- b) coupling between the ribosome and transfer RNA
- c) the correction activity of aminoacyl-tRNA synthetase
- d) the presence of initiation and elongation factors la for each aminoacyl-tRNA
- e) all of the above

A66

67) Bearing in mind that according to the genetic code, each one of the following codons determines the incorporation of the amino acid at its right:

> UUU phenylalanine (F) AAA lysine (K) AAU asparagine (N) AUA isoleucine (I) UUA leucine (L) UAU tyrosine (Y)

Then, if in an in vitro system for protein synthesis artificial poly (UA) is used as messenger RNA it would be expected that peptides formed will contain:

- a) phenylalanine (F) and lysine (K)
- b) asparagine (N) and lysine (K)
- c) phenylalanine (F) and leucine (L)
- d) polyleucine (L)
- e) tyrosine (Y) and isoleucine (I)

A67

A66 <u>C</u> ←

A67 <u>**B**</u>←

68) DNA ligase catalyzes the:

- a) incorporation of deoxyribonucleotides in order to join DNA fragments
- b) formation of hydrogen bonds to join both chains of DNA after replication
- c) formation of phosphodiester links that close DNA interruptions
- d) joining of primer and DNA to start replication

A68

69) In the process of protein synthesis:

- a) each amino acid recognizes its place in mRNA thanks to its specific structure
- b) each codon anticodon pair must have identical sequence to avoid reading errors
- c) binding of mRNA to ribosomal RNA is possible due to the similarity of bases
- d) each amino acid binds selectively to the anticodon of its specific tRNA
- e) the placement of each amino acid depends on the codons of mRNA

A69

70) Genomic imprinting is:

- a) expression of genes that cause diseases
- b) presence of certain genes in the genome
- c) differential expression of one of the alleles
- d) absence of certain genes from the genome
- e) presence in the genome of genes that allow individual identification

A70

A68 <u>C</u> ←

A69 <u>E</u> ←

A70 <u>C</u> ←

71) Polymorphisms are certain DNA sequences that:

- a) codify for proteins that the human species shares with other species
- b) vary among individuals
- c) are repetitive
- d) are palindromes

A71

72) Proto-oncogenes codify for proteins that:

- a) repair DNA
- b) suppress tumor growth
- c) deliver a signal for apoptosis
- d) regulate the proliferation and growth of cells

A72

73) Among transcription factors motifs may be found:

- a) helix-turn-helix
- b) helix-loop-helix
- c) zinc finger
- d) leucine zippers
- e) all of the above

A73

74) DNA replication in cell is carried out:

- a) to guarantee there is protein synthesis
- b) when there is stimulus for cell division
- c) as a step previous to transcription
- d) as a consequence of mitosis
- e) when cells grow old

A74

- A71 <u>B</u>←
- A72 <u>D</u> ←
- A73 <u>E</u> ←

A74 <u>B</u>←

75) The function of DNA replication is

- a) to guarantee the supply of enough copies of regulatory genes for transcription
- b) the supply of enough sites for the start of transcription
- c) mitosis to take effect
- d) all of the above

A75

76) Epigenetic modifications include:

- a) changes in the structure of nucleosomes
- b) synthesis of interference RNAs
- c) methylation of DNA bases
- d) all of the above

A76

77) Alleles are:

- a) different forms of the same gene
- b) copies of the same gene within a chromosome
- c) each of the complementary chains of a gene
- d) all the genes given by one of the parents

A77

78) Transgenic organisms:

- a) those in which a gene is deleted
- b) express genes from a different organism
- c) present mutations induced in their own genes
- d) are induced to increase the transcription of a gene $_{\rm A78}$
- A75 <u>C</u> ←
- A76 <u>E</u> ←
- A77 **A** ←
- A78 <u>B</u> ←

79) Deacetylation of histones by situins (enzymes with deacylase activity) brings about:

- a) favored binding to DNA
- b) impaired transcription of some genes
- c) favored DNA organization in nucleosomes
- d) all of the above

A79

80) Shortening of chromosomes as a result of repeated cell divisions is a consequence of the

- a) progressive cell aging
- b) increased expression of telomerase in aged cells
- c) need of a primer for DNA polymerases activity
- d) progressive loss of the repair ability of DNA polymerase I

A80

81) Telomerase is a:

- a) primase
- b) exonuclease
- c) endonuclease
- d) reverse transcriptase

A81

82) Interference RNA is a double helix RNA that:

- a) contains sequences complementary to some mRNAs
- b) contains information for the synthesis of transcription factors
- c) impairs transcription of some genes
- d) participates in the removal of introns in RNA
- e) mimics the structure of tRNAs impairing their binding to the ribosome

A82

- A79 <u>C</u> ←
- A80 <u>C</u> ←
- A81 <u>D</u> ←

A82 <u>A</u> ←

83) DNA methylation is different from histones acetylation in that the former:

- a) impairs the formation of nucleosomes
- b) is a signal for the corrective activity of DNA polymerase I
- c) contributes to recognition of sensitive regions of restriction endonucleases
- d) does not change the DNA sequence but is inheritable
- e) impairs the initiation of transcription

A83

84) The process of transcription:

- a) happens after DNA replication
- b) depends on the type of cell
- c) does not depend on supply of ATP
- d) allows a single RNA to give rise to different proteins

A84

85) In paternity tests, any cell from the alleged child may be used because:

- a) the selection of DNA segment to study depends on the type of primer used
- b) in all cells there are enzymes capable of synthesizing DNA
- c) the DNA polymerase used in the PCR test is not very specific
- d) all cells have the same genetic charge

A85

A83	<u>E</u> ←	
-----	------------	--

A84 <u>D</u>←

A85 <u>D</u> ←

86) Microsatellites are:

- a) small DNA fragments repeated in consecutive fashion
- b) DNA fragments found at the chromosomes' extremes
- c) DNA fragments that join the chromosomes' arms
- d) DNA fragments able to go from one point to another in the chromosome

A86

87) Microsatellites are useful in individual identification studies because they are:

- a) easily accessible from a technical point of view
- b) small fragments
- c) polymorphic
- d) inheritable
- e) more abundant in sexual chromosomes

A87

88) According to the WHO, the principal cause of proliferation of resistant bacterial strains to antibiotics is the

- a) inadequate use of antibiotics
- b) high frequency of DNA mutations in bacteria
- c) high frequency of transmission of plasmids among bacteria
- d) high mobility of human populations
- e) loss of vaccination effectiveness

A86	A	<u>←</u>
A87	<u>C</u>	<u>←</u>
A88	A	←

89)A point mutation is:

- a) the change of a nitrogen base by another in the base sequence of DNA
- b) the elimination of a single nitrogen base in the sequence of bases of DNA
- c) the addition of a single nitrogen base to the sequence of bases of DNA
- d) the substitution of a single amino acid by another in the amino acid sequence of a chain of protein

A89

On Membranes

90) Kt for the erythrocyte glucose transporter (GLUT 1) is approximately 2mM and that of the beta cell transporter (GLUT 2) is approximately 10mM. If the glycemia value is 5mM, the transport speed:

- a) is greater in the erythrocyte than in the beta cell
- b) is half of the maximum for GLUT 2
- c) by GLUT 1 decreases because it is saturated
- d) of GLUT 2 may increase but not that of GLUT 1

A90

91) Simple diffusion and facilitated diffusion have in common that they:

- a) possess rectangular hyperbolic kinetics
- b) are done in a favorable gradient
- c) may reach saturation
- d) are specific
- e) all of the above

A91

- A89 <u>A</u> ←
- A90 <u>D</u> ←

A91 <u>**B ←**</u>

92) The fluidity of the cell membrane:

- a) decreases with increasing length of the chain of the fatty acids present
- b) depends on its protein content
- c) is inversely proportional to the permeability of the membrane
- d) increases with the amount of transporters present in it
- e) increases with the degree of unsaturation of the fatty acids present

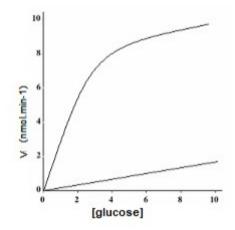
A92

93) The carbohydrates present in the plasma membrane determine the function of proteins that act as:

- a) enzymes
- b) receptors
- c) transporters
- d) recognition proteins
- e) signal transducing proteins

- A92 <u>E</u> ←
- A93 <u>D</u> ←

94) The following is a representation of the absorption of glucose in the intestine. His analysis allows us to conclude that it is done by:



- a) two types of transporters
- b) two different types of transport
- c) a cotransporter
- d) its coupling to a sodium gradient

A94

95) A solution is isotonic with respect to the plasma when it has the same:

- a) glucose concentration
- b) sodium chloride concentration
- c) ion concentration plus glucose
- d) osmolarity
- e) amount of solutes

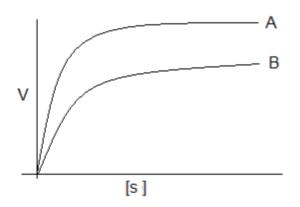
A95

A94 $\underline{\mathbf{B}} \leftarrow$ A95 $\mathbf{D} \leftarrow$ 96) The passage of substances through channels in the plasma membrane differs from the passage through transporters in that:

- a) it is less specific
- b) it is not saturable
- c) can be affected by voltage changes
- d) always occurs in favor of a gradient
- e) the channels are not always protein structure

A96

97) The following graph represents the kinetics of the transport of two substances through the membrane, its analysis leads to the conclusion that:



- a) the transport of A is more specific than that of B
- b) the transport of A is active and that of B passive
- c) the transport of B is active and that of A passive
- d) both transports can be saturated
- e) both A and B are transported by a favorable gradient ${\rm \tiny A97}$

A96 $\underline{C} \leftarrow$ A97 $\underline{D} \leftarrow$ 98) Which of the following goes across the membrane in the process of osmosis:

- a) water
- b) gases
- c) glucose
- d) small solutes
- e) all of the above

A98

99) Cholesterol is an essential component of membranes because:

- a) participates in some systems of cotransport
- b) prevents the formation of bumps in the lipid bilayer
- c) protects its integrity
- d) facilitates the incorporation of some proteins
- e) acts regulating its fluidity

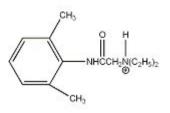
A99

100) Lipid rafts are structures of the plasma membrane that:

- a) do not have cholesterol
- b) are rich in unsaturated fatty acids
- c) are microdomains related to signal transduction
- d) have more fluidity than the rest of the membrane
- e) all of the above

A98 <u>A</u> ←	
A99 <u>E ←</u>	
A100 <u>D</u> ←	

101) Lidocaine hydrochloride is a local anesthetic that, in addition to its anesthetic effect, can cause other changes in cells, depending on dose and the route of administration. Based on the analysis of its formula, which statements can be true regarding lidocaine:



- a) it has an amphipathic nature
- b) it can be incorporated into cell membranes
- c) it can interfere with signal transduction
- d) it can cause dysfunction of the electronic transport chain
- e) all of the above

A101

On Introduction to Metabolism and Signal Transduction

102) The mechanisms regulating enzymatic activity both allosteric and covalent, reversible and irreversible have as a common effect that they

- a) alter the enzyme's charge
- b) change the active site conformation
- c) require the participation of other enzymes
- d) change the primary structure of the enzyme
- e) bring about changes in quaternary structure of the enzyme $_{\rm A102}$

A101 **E** ← A102 **B** \leftarrow

103) Anabolic and catabolic pathways share this characteristic:

- a) they happen until equilibrium is reached
- b) their ΔG values are negative
- c) acetyl-coA is a common intermediary
- d) each one has specific regulatory mechanisms

A103

104) Regulation of metabolism is advantageous because:

- a) allows hormones to exert their action
- b) limits the number of reactions necessary for each metabolic pathway
- c) determines energy saving
- d) prevents all reactions from reaching equilibrium

A104

105) Hormones may influence metabolism in effector cells by producing:

- a) changes in the speed of protein synthesis
- b) activation or inhibition of some enzymes
- c) changes in permeability of the plasmatic membrane
- d) epigenetic modifications
- e) all of the above

A105

106) Hormones involved in cellular energetic metabolism:

- a) promote its reaching equilibrium
- b) allow the body to stay away from equilibrium
- c) are liberated in response to equilibrium alterations
- d) are the only way of affecting the equilibrium constants of reactions catalized by regulatory enzymes

- A103 $\underline{\mathbf{B}} \leftarrow$
- A104 **D** ←
- A105 $E \leftarrow$
- A106 $\underline{\mathbf{B}} \leftarrow$

107) One of the mechanisms trough which insulin affects cAMP concentration in effector tissues is:

- a) inhibition of protein GEF (guanine nucleotide exchange factor)
- b) stimulation of protein GDI (guanosine nucleotide dissociation inhibitor)
- c) stimulation of phosphodiesterase
- d) increase in the concentration of metabolites inhibiting adenyl cyclase

A107

108) The result of the stimulation G proteins may be the intracellular liberation of:

- a) cAMP
- b) cGMP
- c) IP3
- d) ionic calcium
- e) all of the above

A108

109) Effector systems of the action of hormones include:

- a) stimulation of an enzymatic activity of the receptor
- b) activation of G proteins
- c) opening of the ionic canals
- d) regulation of gene transcription
- e) all of the above

A109

A107 $\underline{C} \leftarrow$ A108 $\underline{E} \leftarrow$ A109 $\underline{E} \leftarrow$

110) Cessation of G proteins activation happens when:

- a) the hormone separates from the receptor
- b) an G-inhibiting protein is activated
- c) cAMP is hydrolyzed
- d) alpha subunits separate
- e) GTP bound to the alfa subunit is hydrolyzed

A110

111) As a consequence of the action of cholera toxin:

- a) the concentration of intracellular cAMP decreases
- b) the dissociation of the subunits of G protein is inhibited
- c) protein kinase A becomes inactive
- d) phosphodiesterase becomes inactive
- e) the alpha subunit of G protein loses its GTPase activity

A111

112) Steroid hormones act at the cellular level:

- a) inhibiting pre existing enzymes
- b) through a second messenger
- c) by regulating gene expression
- d) through G proteins
- e) modifying the concentration of intracellular ionic calcium

A112

113) G proteins:

- a) act as receptors for some hormones
- b) are signal transductors through the membranes
- c) synthesize intracellular second messengers
- d) catalyze la synthesis of cAMP from ATP
- e) act as intracellular messengers for some hormones
- A110 <u>E</u> ←
- A111 <u>E</u> ←
- A112 **C** ←
- A113 **B** ←

114) Steroid hormones are characterized because

- a) they bind to specific genes in DNA modifying their transcription
- b) when binding to their receptor they increase its affinity to DNA
- c) affect transcription by regulating the activity of RNA polymerase
- d) bind to the mRNAs that codify for some proteins inhibiting its degradation

A114

115) A common characteristic to all oncogenes is that they codify for proteins that:

- a) are viral
- b) are tumor markers
- c) impair the emergence of tumors
- d) intervene in signal transduction
- e) have abnormal folding due to mutations

A115

116) Protein kinases A and C have in common that they:

- a) are membrane proteins
- b) are allosterically activated
- c) depend on ionic calcium for their activation
- d) are activated by alpha subunits of G proteins
- e) all of the above

A116

A114 $\underline{B} \leftarrow$ A115 $\underline{D} \leftarrow$ A116 $\underline{B} \leftarrow$

117) Kinases dependent on AMP and cAMP have in common that:

- a) their activity depends on the 'energetic charge' of the cell
- b) they are activated as a response to glucagon
- c) they can be phosphorylated
- d) they are serine kinases

A117

On Krebs cycle and oxidative phosphorylation

Is spite of widespread opinion among doctors and students that it is useless to know the reactions of the Krebs cycle, many alterations seen in diabetes mellitus are related to the cycle; the same is true for metabolic adaptations during fasting or following food ingestion.

118) With the exception of citrate synthase, enzymes regulating the speed of the Krebs cycle have in common that they:

- a) catalize negative ΔG° reactions
- b) catalize oxidation-reduction reactions
- c) function with catalytic levels of substrate
- d) catalize oxidative decarboxylation reactions

e) are regulated allosterically by the concentration of NAD $_{\rm A118}$

+



119) During a fast, in spite of increased activity of pyruvate carboxylase, the concentration of oxaloacetate inside liver mitochondria stays low because:

- a) it passes through the mitochondrial membrane into the cytoplasm
- b) the speed of the Krebs cycle is increased
- c) it is used to make aspartate, compensating for the amino acid deficit provoked by fasting
- d) most of it serves as a substrate for citrate synthase to form citrate that constantly exits the mitochondria in that situation
- e) the Keq of the reaction catalized by malate dehydrogenase favors accumulation of malate

A119

120) A patient of chronic alcoholism may present a thiamine deficiency (B1 vitamin), because alcohol inhibits its intestinal absorption. In this case the functioning of which enzyme would be impaired?

- a) glutamate dehydrogenase
- b) succinate dehydrogenase
- c) isocitrate dehydrogenase
- d) pyruvate dehydrogenase
- e) malate dehydrogenase



121) Which of the following Krebs cycle enzymes is located in the internal mitochondrial membrane?

- a) malate dehydrogenase
- b) isocitrate dehydrogenase
- c) α -ketoglutarate dehydrogenase
- d) succinate dehydrogenase
- e) citrate synthase

A121

122) The fact that ΔG° of the reaction catalized by malate dehydrogenase is +7,0 kcl.mol⁻¹ implies that:

- a) it is a regulatory enzyme of the Krebs cycle
- b) it allows the interrelation of the Krebs cycle with other metabolic pathways
- c) the reaction is exergonic
- d) its Keq is larger than 1
- e) at equilibrium, malate production predominates

A122

123) Which of the following Krebs cycle reactions is associated to the production of NADH + H^+ ?:

- a) succinyl-CoAà succinate
- b) acetyl-CoA + oxaloacetate à citrate
- c) malate à oxaloacetate
- d) citrate à isocitrate
- e) succinate à fumarate

A123

A121 $\underline{D} \leftarrow$ A122 $\underline{E} \leftarrow$ A123 $\underline{C} \leftarrow$

124) The Krebs cycle is considered an amphibolic pathway because:

- a) its intermediaries are precursors for metabolic pathways
- b) it functions as final way of cell catabolism
- c) its metabolites participate in the cell's anabolism as well as catabolism
- d) it is present in all cell of the body
- e) all its reactions are reversible

A124

125) In the Krebs cycle, anaplerotic reactions are those catalized by the enzymes:

- a) citrate synthase and glutamate dehydrogenase
- b) pyruvate dehydrogenase and malate dehydrogenase
- c) pyruvate dehydrogenase and pyruvate carboxylase
- d) glutamate dehydrogenase and pyruvate carboxylase

e) glutamate dehydrogenase and pyruvate dehydrogenase

126) When oxidative phosphorylation is uncoupled oxygen consumption:

- a) accelerates but phosphorylation of ADP is unaffected
- b) increases and phosphorylation of ADP ceases
- c) ceases but phosphorylation of ADP is unaffected
- d) ceases and phosphorylation of ADP decreases
- e) and phosphorylation of ADP increase

A126

A124 $\underline{C} \leftarrow$ A125 $\underline{D} \leftarrow$ A126 $\underline{B} \leftarrow$

127) The Chemiosmotic theory of oxidative phosphorylation contends that:

- a) the energy of a proton gradient is utilized for the synthesis of ATP
- b) an electron gradient is generated at the internal mitochondrial membrane
- c) hydrolysis of ATP is used for the formation of an energy rich proton gradient
- d) protons accumulate in the mitochondrial matrix and are then pumped out through the complex Fo-F1
- e) gradients of electrons as well as protons are generated on both sides of the internal mitochondrial membrane

A127

128) Substrate-level phosphorylation requires:

- a) the oxidation of NADPH instead of NADH
- b) the formation of an electron gradient
- c) inhibition of the respiratory chain.
- d) a high energy charge inside the cell
- e) the formation of a high energy intermediary

A128

129) ATP synthesized in mitochondria:

- a) diffuses freely to the outside of mitochondria
- b) exits from them through the Fo-F1 ATPase complex
- c) is transported to the cytosol by a protein in exchange for ADP
- d) is actively expelled to cytosol by an energy-requiring pump
- e) is transported to the cytosol by a protein coupled to the Na⁺/K⁺ pump

A129

A127 <u>A</u>←

A128 <u>E</u> ←

A129 <u>C</u> ←

130) Identify the oxidizing agent in the following reaction (E= enzyme):

NADH + H⁺ + E-FMN \leftrightarrow NAD⁺ + E-FMNH₂

- a) NADH + H^+
- b) E-FMN
- c) NAD $^+$
- d) E-FMNH₂

A130

131) Given the following reaction and its ΔG^{o} value

$A + C \rightarrow B + D$ $\Delta G^{\circ} = -15 \text{ kcal/mol}$

It can be inferred that:

- a) its equilibrium constant is greater than 1
- b) it will only happen if coupled to an exergonic reaction
- c) the products have more free energy than the reactants
- d) it does not require a catalyst
- e) it has a high velocity of reaction

A131

132) ATP is a high energy compound because:

- a) in its structure there are easily transferable phosphate groups
- b) its ionic character confers to the molecule an electronic distribution favoring hydrolysis
- c) Its energy of hydrolysis is -7 kcal/mol
- d) Its hydrolysis products are more stable
- e) all of the above

A132

A130 $\underline{B} \leftarrow$ A131 $\underline{A} \leftarrow$ A132 $\underline{E} \leftarrow$

133) Cytochromes are:

- a) hydrogen atoms transporters
- b) proton acceptors
- c) hydrure ions acceptors
- d) electron acceptors

A133

134) Phosphorylation of complex IV in the mitochondrial electron transport chain by protein kinase A:

- a) allows its inhibition by ATP
- b) stimulates electron transport
- c) facilitates its activation by AMP
- d) triggers uncoupling of oxidative phosphorylation

A134

135) An uncoupling molecule might be used to bring about weight loss because:

- a) heat would be liberated, accelerating metabolism
- b) there would be heat liberation, favoring fatty acid mobilization through the mitochondrial membrane
- c) part of the energy coming from food oxidation would be dissipated as heat
- d) all of the above

A135

A133 $\underline{D} \leftarrow$ A134 $\underline{A} \leftarrow$ A135 $\underline{C} \leftarrow$

136) Atractyloside interferes with the synthesis of ATP in the mitochondrion because it:

- a) inhibits the transference of electrons between cytochromes b and c1
- b) inhibits the ADP/ATP transport system
- c) blocks the transference of electrons at the complex IV level of the respiratory chain is a protonophore and dissipates the proton gradient

d) impairs the formation of high-energy intermediates

A136

137) Antimycin and rotenone act as follows:

- a) inhibiting transport of H⁺ through the Fo-F1-ATPase complex
- b) destroying the H⁺ gradient through the membrane
- c) blocking ATP/ADP transport
- d) increasing oxygen consumption
- e) inhibiting electron transport

A137

138) When the standard free energy change of a chemical reaction is0:

- a) its Keq > 1
- b) it is endergonic
- c) it proceeds at very low speed
- d) it continues until running out of substrates

- A136 $\underline{\mathbf{B}} \leftarrow$
- A137 <u>E</u>←
- A138 <u>**B</u>←</u></u>**

139) The energetic charge of the cell is defined by the:

- a) energy liberated by hydrolysis of ATP and other high-energy molecules
- b) state of oxidation / reduction of coenzymes
- c) ratio ATP/ ADP + AMP
- d) total of high-energy compounds
- e) availability of oxidable substrates

A139

140) Some of the reactive oxygen species (ROS):

- a) are active in the regulation of metabolism
- b) participate in immune response
- c) cause oxidative damage to biomolecules
- d) participate in the synthesis of essential molecules
- e) all of the above

A140

141) Among ROS the most reactive is:

- a) superoxide anion radical (O_2 .⁻)
- b) hydroperoxide radical (HOO[.])
- c) hydroxyl radical (OH·)
- d) hydroxide ion (OH⁻)
- e) hydrogen peroxide (H₂O₂)

A139	<u>C ←</u>
A140	<u>E</u> ←
A141	<u>C</u> ←

142) It has been postulated that the damage by re perfusion after a period of ischemia is probably due to:

- a) increase in anaerobic oxidation of glucose during ischemia
- b) accumulation of NADH + H⁺ during ischemia
- c) saturation by electrons of the respiratory chain components during ischemia
- d) overproduction of ROS when blood flow is reestablished leading to insufficient antioxidant defense
- e) all of the above

A142

143) α -ketoglutarate dehydrogenase and pyruvate dehydrogenase are dissimilar because:

- a) they catalyze irreversible reactions
- b) of the type of reaction that catalyze
- c) they use a different coenzyme
- d) their regulation mechanisms are different
- e) of their subcellular location

A143

144) Ketone bodies synthesis is a way:

- a) of reoxidizing mitochondrial NADH
- b) to export reducing equivalents to peripheral tissues
- c) of regenerating CoA
- d) all of the above

A144

A142 $E \leftarrow$ A143 $D \leftarrow$ A144 $E \leftarrow$

145) Oxaloacetate does not pass through the internal mitochondrial membrane because:

- a) in its absence, all acetyl-CoA would be concerted to ketone bodies
- b) it is necessary to guarantee the function of the Krebs cycle
- c) its half- life inside the mitochondrion is too short
- d) there is no transporter for it in the membrane

A145

146) Some reactions of the Krebs cycle:

- a) allow the extraction of reducing equivalents from cell fuels
- b) constitute the main source of CO₂ in the body
- c) are indispensable for the conversion of carbohydrates into fat
- d) participate in the conversion of amino acids and lactate into glucose
- e) all of the above

A146

147) The cyclic character of the Krebs cycle implies that:

- a) coenzymes can be re used
- b) all reactions are reversible
- c) requires a constant supply of acetyl-CoA
- d) citrate is regenerated at each round of the cycle
- e) malate in catalytic concentrations accelerates oxygen consumption

A145	<u>D</u> ←
A146	<u>E</u> ←
A147	<u>E</u> ←

148) Coupling between electron transport and ATP synthesis in mitochondria is due to the:

- a) appearance of a proton gradient
- b) vector organization of the components of the respiratory chain
- c) existence of an inter membrane space
- d) structure of the FoF1 complex
- e) redox potential of the components of the respiratory chain ${\rm A}^{\rm 148}$

149) A decrease in mitochondrial ADP concentration can inhibit electron transport because:

- a) the energetic charge of the cell is high
- b) the concomitant ATP increase inhibits complex I
- c) the flow of protons through the FoF1 complex
- d) the Krebs cycle slows down

A149

150) The malate shuttle allows the net transport from cytosol to mitochondria of:

- a) ATP
- b) NADH
- c) ketoacids
- d) amino acids
- e) reducing equivalents

A150

A148 $\underline{A} \leftarrow$ A149 $\underline{C} \leftarrow$ A150 $\underline{E} \leftarrow$

151) α -ketoglutarate dehydrogenase and pyruvate dehydrogenase:

- a) show a negative delta G. that makes them catalyze irreversible reactions
- b) catalyze reactions of oxidative decarboxylation
- c) are regulated by the concentration of NAD⁺
- d) are multi-enzyme complexes
- e) all of the above

A151

152) Hydrolysis of pyrophosphate produced in a reaction may shift the equilibrium because:

- a) it modifies delta G^o of the reaction
- b) it does not require an enzyme to participate
- c) increases the speed of the reaction
- d) decreases the concentration of the reaction products
- e) hydrolysis pyrophosphate shows a negative ΔG^{o}

A152

153) The activity of citrate synthase is regulated by:

- a) phosphorylation / dephosphorylation
- b) allosteric inhibition by ATP
- c) allosteric activation by NAD⁺
- d) retro inhibition by citrate
- e) inhibition by acetyl-CoA

A153

A151 $\underline{\mathbf{E}} \leftarrow$ A152 $\underline{\mathbf{D}} \leftarrow$ A153 $\mathbf{D} \leftarrow$ 154) A decrease in ATP/ADP + AMP ratio may increase the Krebs cycle speed because allosterically it activates:

- a) α -ketoglutarate dehydrogenase
- b) isocitrate dehydrogenase
- c) succinate dehydrogenase
- d) malate dehydrogenase
- e) citrate synthase

A154

Metabolism of Carbohydrates

155) Which of these two names expresses best the structure of the following molecule inside the body? Lactate ($C_3H_5O_3^-$) or lactic acid ($C_3H_6O_3$):

- a) the two names represent the same molecule
- b) lactate, because at physiological pH it exists as its conjugate base
- c) lactate, because at physiological pH the lactate isomer predominates
- d) lactic acid because it has all the atoms derived from half a molecule of glucose

A155

156) Glucose enters the erythrocyte via a transport type characterized by being:

- a) simple diffusion
- b) stimulated by insulin
- c) facilitated by GLUT 1
- d) dependent on the supply of ATP
- e) facilitated by a sodium ion co-transporter

- A154 <u>**B**</u>←
- A155 <u>**B**</u>←
- A156 <u>C</u> ←

157) Glucose transport into the pancreatic beta cells is mediated by:

- a) GLUT1
- b) GLUT2
- c) GLUT3
- d) GLUT4
- e) GLUT5

A157

158) Which of the following glucose transporters is activated as a consequence of insulin effect?

- a) GLUT1
- b) GLUT2
- c) GLUT3
- d) GLUT4
- e) GLUT5

A158

159) Compared to other GLUT transporters, GLUT2:

- a) shows the highest kt for glucose
- b) is activated by insulin
- c) transports glucose against a concentration gradient
- d) co-transports sodium ions together with glucose
- e) is highly specific for glucose

A157	<u>B</u> ←
A158	<u>D</u> ←
A159	<u>A</u> ←

160) Hexokinase and glucokinase have in common:

- a) their tissue location
- b) their substrates and products
- c) being inhibited by their products
- d) being inducible enzymes
- e) all of the above

A160

161) Which of the following mechanisms is involved in the regulation of glucokinase activity?

- a) allosteric inhibition by citrate
- b) phosphorylation by protein kinase A
- c) translocation to the nucleus
- d) inhibition by its product
- e) allosteric inhibition by ATP

A161

162) The activity of glucokinase is involved in the regulation of glycaemia because:

- a) its activity increases in proportion to the increase in glycaemia
- b) favors the utilization of glucose for hepatic glycogen synthesis
- c) acts as a glycaemia-sensor for the beta cells in the pancreas
- d) restricts the increase in glycaemia after ingestion of food
- e) all of the above

A162

A160 $\underline{B} \leftarrow$ A161 $\underline{C} \leftarrow$ A162 $\underline{E} \leftarrow$ 163) In anaerobic conditions, NADH produced in glycolysis is reoxidized through the following reaction:

- a) fructose 1,6-bisphosphateà glyceraldehyde-3P + dihydroxyacetone-P
- b) glyceraldehyde-3P à 1,3-DPG
- c) pyruvate à lactate
- d) dihydroxyacetone-P à glyceraldehyde-3P
- e) malate à oxaloacetate

A163

164) Adenylate kinase catalyzes the following reaction, 2ADP \leftrightarrow ATP + AMP

that in erythrocytes is tightly linked to the activity of glycolysis, because:

- a) it produces AMP that reverts inhibition of PFK by ATP
- b) supplies AMP necessary for pyruvate kinase activity
- c) supplies ATP necessary for phosphorylation of glucose by hexokinase
- d) it is the main source of ATP for the erythrocyte

A164

165) The activity of phosphofructokinase in erythrocytes is inhibited by:

- a) ADP
- b) ATP
- c) AMP
- d) citrate
- e) fructose 2,6-bisphosphate

A165

A163 $\underline{C} \leftarrow$ A164 $\underline{A} \leftarrow$ A165 $\underline{B} \leftarrow$ 166) The speed of hepatic glycolysis is different from that in skeletal muscle in that it:

- a) increases when cAMP increases
- b) decreases when ATP increases
- c) is regulated by insulin
- d) depends on the ratio NADH/NAD
- e) increases when AMP increases

A166

167) The regulation of pyruvate kinase activity in liver is different from that in skeletal muscle because in the former it is:

- a) activated by fructose 1,6 bisphosphate
- b) allosterically inhibited by ATP
- c) inhibited by protein kinase A
- d) retro-inhibited by its product
- e) activated by acetyl CoA

A167

168) Lactate liberated to the blood is metabolized preferentially by cardiac tissue because:

- a) it is easily metabolizable in high energy demands condition
- b) heart cells have a membrane transporter for lactate
- c) it protects cardiac cells against the pH change due to lactate
- d) it represents glucose saving for the cell
- e) this is favored by the kinetic characteristics of the enzyme LDH-1 (H4)

A168

A166 $\underline{C} \leftarrow$ A167 $\underline{C} \leftarrow$ A168 $\underline{E} \leftarrow$

169) Glycolysis and gluconeogenesis have in common that:

- a) require input of reduced coenzymes
- b) their speed depends mainly on substrate availability
- c) they are exergonic processes
- d) they are inhibited by their respective products

A169

170) Which of the following glycolytic reactions is inhibited in the liver through phosphorylation by protein kinase A?

- a) glucokinase
- b) pyruvate kinase
- c) phosphofructokinase
- d) lactate dehydrogenase
- e) glyceraldehyde 3 p dehydrogenase

A170

171) Muscular glycolysis accelerates following the increase in intracellular concentration of:

- a) NADH +H+
- b) glucose
- c) AMP
- d) oxygen
- e) H+

A171

Glycogen metabolism is regulated in slightly different ways in muscle and liver. In each of these tissues glycogen fulfills different functions. When you study, be sure to notice the way glycogen phosphorylase and glycogen synthase are regulated in each of these tissues.

A169 \subseteq \leftarrow Within the cell reactions that require energy are coupled to those that supply it. Therefore, processes will only happen if the net result is energy liberation.

A170 <u>B</u> ← A171 <u>C</u> ←

172) Glycogen synthase catalyzes the:

- a) transference of glucose from UDP-glucose to a glycogen chain residue
- b) joining of free glucose residues to UDP
- c) joining of free glucose molecules to a glycogen chain residue
- d) formation of UDP-glucose + PPi
- e) branching of the glycogen chain with alfa 1-6 bonds

A172

173) Hepatic glycogen phosphorylase, as opposed to that in muscle:

- a) is phosphorylated by a phosphorylase kinase
- b) is activated when it is phosphorylated
- c) is inhibited by glucose
- d) attaches Pi as such and not from ATP to form glucose-1-P
- e) is active when the intracellular concentration of cAMP increases

A173

174) The increase in calcium ion concentration that stimulates glycogenolysis in muscle is due to:

- a) increase in AMP concentration
- b) membrane depolarization
- c) stimulation of alfa adrenergic receptors
- d) stimulation of beta adrenergic receptors

A172	<u>A</u> ←
A173	<u>C</u> ←
A174	<u>B</u> ←

175) The increase in calcium ion concentration that stimulates glycogenolysis in liver is due to:

- a) increase in AMP concentration
- b) the response to membrane depolarization
- c) stimulation of beta adrenergic receptors
- d) stimulation of alfa adrenergic receptors

A175

176) AMP can stimulate glycogenolysis in muscle because it:

- a) activates AMP kinase
- b) it is a signal of decrease in ATP concentration
- c) stimulates glycogen phosphorylase b activity
- d) promotes the entry of calcium ions to the cells

A176

177) An increase in glycaemia favors glycogen synthesis in liver independently of insulin because glucose:

- a) enters hepatic cells in exchange for calcium ions that inactivate the calcium/calmodulin-dependent protein kinases
- b) activates phosphodiesterase diminishing cAMP levels
- c) can activate protein phosphatase 1
- d) activates allosterically glycogen synthase
- e) increases the activity of glucokinase thus providing substrate to glycogen synthase

A177

A175 $\underline{D} \leftarrow$ A176 $\underline{C} \leftarrow$ A177 $\underline{C} \leftarrow$ 178) In the liver the reducing equivalents for synthesis of glucose are provided fundamentally by:

- a) oxidation of ketone bodies
- b) oxidation of pyruvate by pyruvate dehydrogenase
- c) β -oxidation
- d) glycolysis
- e) oxidation of glutamate

A178

179) In kidney the reducing equivalents for synthesis of glucose are provided fundamentally by:

- a) oxidation of ketone bodies
- b) oxidation of pyruvate by pyruvate dehydrogenase
- c) β -oxidation
- d) glycolysis
- e) oxidation of glutamate

A179

180) Kidney and liver gluconeogenesis have in common that they:

- a) depend on the input of NADH from β -oxidation
- b) are linked to excretion of amino nitrogen
- c) are regulated by pyruvate carboxylase
- d) are stimulated by glucagon
- e) stimulated by adrenalin

A178	<u>C</u>	<u> </u>
A179	E	<u> </u>
A180	B	←

181) Malate dehydrogenase activity in mitochondria and cytosol is important for gluconeogenesis because this:

- a) allows the transport of reducing equivalents out of mitochondria
- b) allows the utilization of amino acids' carbon atoms
- c) allows the exit of oxaloacetate formed inside mitochondria
- d) impedes oxaloacetate to be used for citrate synthesis
- e) all of the above

A181

182) Pyruvate dehydrogenase and pyruvate carboxylase have as a common feature that they are regulated by:

- a) phosphorylation/dephosphorylation
- b) the concentration of pyruvate
- c) the concentration of acetyl CoA
- d) the ratio ATP/ADP
- e) the ratio NADH/NAD

A182

183) The activity of the enzyme pyruvate carboxylase can be allosterically modified by:

- a) pyruvate
- b) ATP
- c) citrate
- d) acetyl-CoA
- e) oxaloacetate

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A183
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A181 $\underline{E} \leftarrow$ A182 $\underline{C} \leftarrow$ A183 $\underline{D} \leftarrow$

184) Fructose 2,6-bisphosphate:

- a) is synthesized by the bifunctional enzyme
- b) is degraded by the bifunctional enzyme
- c) stimulates the activity of phosphofructokinase 1
- d) inhibits the activity of fructose 1,6 bisphosphate phosphatase
- e) all of the above

A184

185) Gluconeogenesis from pyruvate is favored by high concentrations of:

- a) citrate
- b) malonyl-CoA
- c) acetyl-CoA
- d) NAD+
- e) fructose 2,6-bisphosphate

A185

186) The activity of the enzyme pyruvate carboxylase can be modified allosterically by:

- a) pyruvate
- b) ATP
- c) isocitrate
- d) oxaloacetate
- e) acetyl-CoA

A184	<u>→ U</u>
A185	<u>C</u> ←
A186	<u>E</u> ←

187) Lactate dehydrogenase is an enzyme that may exist in five different forms (isoenzymes), that are different in:

- а) Км
- b) regulation
- c) mechanism of action
- d) subcellular location
- e) all of the above

A187

188) In human gluconeogenesis cannot be performed from acetyl CoA because:

- a) thermodynamically it is too unfavorable
- b) the reaction catalized by pyruvate dehydrogenase is irreversible
- c) it is not possible to convert a hydrophobic compound into a hydrophilic one
- d) the oxaloacetate formed in the Krebs cycle does not contain net carbon atoms from acetyl CoA
- e) glucose is a more oxidized molecule than fatty acids

A188

189) A deficiency of glucose 6P dehydrogenase in erythrocytes can affect the linking of oxygen to Hb because:

- a) it decreases the formation of ATP
- b) it decreases CO₂ formation, preventing the Bohr effect
- c) glycolysis is predominant and this increases 2,3 diphosphoglycerate formation
- d) it decreases the capacity to keep the Hb iron in its reduced state
- e) it alters the structure of membrane proteins with hemolysis ${\mbox{\tiny A189}}$
- A187 $\underline{A} \leftarrow$ A188 $\underline{B} \leftarrow$ A189 $\underline{D} \leftarrow$

190) Referring to the oxidative phase of the pentose phosphate pathway it is correct to assert that:

- a) for every mol of glucose oxidized two moles of ATPs are consumed
- b) ribose -5-P is oxidized to CO₂ and H2O
- c) all its reactions are reversible
- d) for every mol of pentose completely oxidized 2 moles of ATP are formed

e) it produces NADPH + CO₂ + ribose-5P

A190

191) Substrate cycles of futile cycles:

- a) only happen in catabolic pathways
- b) constitute a useless expenditure of energy
- c) decrease the minimum flow of a metabolic pathway
- d) decrease the speed of a metabolic pathway
- e) contribute to keep a constant concentration of ATP inside the cell

A191

192) During a fast gluconeogenesis is stimulated because:

- a) it is necessary that glucose be available to tissues that depend on it
- b) the effect of glucagon predominates over that of insulin
- c) the input of fatty acids to the liver increases
- d) the input of amino acids to the liver increases
- e) all of the above

A192

A190 $\underline{E} \leftarrow$ A191 $\underline{C} \leftarrow$ A192 $\underline{B} \leftarrow$

193) What is the advantage of increasing gluconeogenesis during a fast?

- a) to guarantee glucose availability to tissues depending on it
- b) to allow the use of energy liberated by fat oxidation
- c) to allow the use of amino acids carbon atoms liberated from muscle
- d) all of the above

A193

Bear in mind that an 'advantage' for the cell or organism is one thing and the biochemical reason for the phenomenon is another matter.

Metabolism of Lipids

194) Only a very small proportion of fatty acids is actually free in the body. The immense majority of them are linked to proteins, CoA or ACP, depending on their location. This is a major advantage because it prevents:

- a) disorganize membranes on account of their detergent power
- b) being metabolized out of control
- c) leak out from cells and not be available for use
- d) inactivation of glycolysis by inhibition of phosphofructokinase
- e) unleash inflammatory reactions

A194

195) Cholesterol esters:

- a) are part of cell membranes
- b) may be formed both inside and out of cells
- c) have amphipathic character
- d) are more soluble in water that free cholesterol ${\scriptstyle {\rm A195}}$

A193 $\underline{D} \leftarrow$ A194 $\underline{A} \leftarrow$ A195 $\underline{B} \leftarrow$ 196) Domestic cooking oil is made up mainly of:

- a) phospholipids
- b) free fatty acids (FFA)
- c) triacylglycerols (TAG)
- d) a mixture of TAG and free fatty acids
- e) a mixture of phospholipids y FFA

A196

197) Oils are more fluid than other fats because they contain:

- a) more unsaturated fatty acids
- b) less triglycerides
- c) more phospholipids
- d) less cholesterol
- e) lipids that can from micelles

A197

198) Bilirubin is:

- a) synthesized from cholesterol
- b) a product of degradation of the heme group
- c) essential for intestinal digestion of fatty acids
- d) reabsorbed at the ileum

A198

199) Fatty acids circulate in blood:

- a) linked to apo B
- b) free in salt form
- c) in chylomicrons
- d) in HDL
- e) bound to albumin

A199

A196 <u>C</u> ←

- A197 <u>A</u> ←
- A198 **B** ←
- A199 <u>E</u> ←

200) Among human serum lipoproteins the fraction with the highest cholesterol contents is:

- a) α -lipoproteins or HDL
- b) β -lipoproteins or LDL
- c) pre- β -lipoproteins or VLDL
- d) chylomicrons

A200

201) Lipoprotein lipase (LPL) plays a role in the degradation of triacylglycerols located in:

- a) primary micelles
- b) secondary micelles
- c) extracellular space
- d) adipocytes
- e) the liver

A201

202) The apo B48 and the apo B100 have in common:

- a) the gene that codifies for them
- b) the type of lipoprotein that transports them
- c) the receptor that recognizes them
- d) being freely transferable among lipoproteins
- e) the tissue that synthesizes them

A200	B	<u>←</u>
A201	<u>C</u>	<u>←</u>
A202	A	←

203) Chylomicrons may show one of the following attributes

- a) high contents of Apo-B and Apo-C
- b) being rich in exogenous triacylglycerols
- c) no esterified cholesterol in their structure
- d) high velocity of electrophoretic migration
- e) higher floating density than all lipoproteins

A203

204) Which of the LDL components interacts with receptors?

- a) free cholesterol
- b) apoprotein B 100
- c) apoprotein CII
- d) cholesterol esters
- e) apoprotein A

A204

205) In a patient with a genetic condition altering synthesis of Apo CH, there would be a decrease in plasma levels of:

- a) chylomicrons and VLDL
- b) HDL and LDL
- c) VLDL and HDL
- d) chylomicrons and LDL

A205

A203	B	←

A204 <u>**B**</u>←

A205 <u>**B**</u>←

206) Recent studies show that cholesterol in ingested food has little effect on the plasma concentration of LDL and the ratio LDL/HDL. This may be due to an inhibitory effect on the

- a) activity of LPL
- b) synthesis of apo E
- c) synthesis of Apo B
- d) bile salts absorption
- e) activity of HMG CoA reductase

A206

207) Acyl-CoA-cholesterol acyltransferase (ACAT) and lecithincholesterol acyltransferase (LCAT) are similar in:

- a) the type of reaction they cathalize
- b) their regulation mechanism
- c) their substrates
- d) their products
- e) their location

A207

208) In reverse cholesterol transport HDL's pickup cholesterol from tissues membranes by means of:

- a) ATP-binding cassette transporter (ABCA1)
- b) Cholesteryl-ester transfer protein
- c) scavenger receptor B1
- d) LCAT
- e) apo A1

A208

A206 $\underline{\mathbf{E}} \leftarrow$ A207 $\underline{\mathbf{A}} \leftarrow$ A208 $\underline{\mathbf{A}} \leftarrow$

209) Receptors SR-A and SR-A2 present in macrophages bind lipoproteins that:

- a) have lost part of their apoprotein
- b) are cholesterol rich
- c) are incompletely degraded
- d) are in excess
- e) have been oxidized

A209

210) HDL may be formed from VLDL and QM:

- a) because some remnants become HDL joining to Apo A-1
- b) by the action of hepatic lipase over remnants
- c) gemmation during the action of LPL
- d) HDL cannot originate from these lipoproteins

A210

211) Sterol regulatory element-binding protein (SREBP) is activated:

- a) through phosphorylation
- b) when joining cholesterol
- c) by proteolytic cleavage
- d) when it joins the sterol regulatory element of DNA
- e) through dephosphorylation

A209	<u>E ←</u>
A210	<u>C</u> ←
A211	<u>C</u> ←

212) The entry of cholesterol into cells may regulate transcription of HMG-CoA reductase because it is capable of joining:

- a) SREBP
- b) SREBP cleavage-activating protein (SCAP)
- c) sterol regulatory element (SER)
- d) site-2 protease (S2P)
- e) site-1 protease (S1P)

A212

213) The activity of HMG-CoA reductase may be affected by:

- a) phosphorylation/dephosphorylation
- b) the level of transcription of its mRNA
- c) competitive inhibition
- d) allosteric regulation
- e) all of the above

A213

214) Cholesterol may be excreted from the body by:

- a) daily loss of bile salts
- b) skin defoliation
- c) pulmonary epithelium defoliation
- d) intestinal epithelium defoliation
- e) all of the above

A212	<u>B</u> ←
A213	<u>E</u> ←
A214	<u>E</u> ←

215) Synthesis of HDL happens because:

- a) cholesterol and phospholipids are joined in the liver and intestine as nascent HDL
- b) the Gemmation from VLDL and chylomicrons as a consequence of LPL action over these lipoproteins
- c) transference in blood of cholesterol and phospholipids to free apoprotein A1
- a) all of the above

A215

216) The function of carnitine in lipid metabolism is related to the:

- a) activation of fatty acids for TAG synthesis
- b) transport of long chain fatty acids in blood
- c) biosynthesis of fatty acids and their activation
- d) intercellular transport of activated fatty acids
- e) transference of fatty acids through mitochondrial membranes $_{\mbox{\scriptsize A216}}$

217) A carnitine deficit may bring about a low blood sugar crisis in fasting, partly because this affects the function of:

- a) citrate synthase
- b) glucose 6-phosphatase
- c) pyruvate carboxylase
- d) acetyl-CoA carboxylase

A217

A215 $\underline{\mathbf{E}} \leftarrow$ A216 $\underline{\mathbf{E}} \leftarrow$ A217 $\underline{\mathbf{C}} \leftarrow$ 218) The hormone-sensitive lipase intervenes in degradation of triacylglycerols:

- a) of micelles
- b) in adipocytes
- c) outside cells
- d) in liver

A218

219) The activity of carnitine palmitoyltransferase I is regulated allosterically by:

- a) ATP
- b) citrate
- c) acetyl-CoA
- d) malonyl-CoA
- e) long chain acyl CoA's

A219

220) Formation of ketone bodies is a process that takes place in:

- a) hepatocytes' cytosol
- b) adipocytes mitochondria
- c) skeletal muscle mitochondria
- d) hepatocytes mitochondria
- e) adipocytes' cytosol

A220

A218 $\underline{B} \leftarrow$ A219 $\underline{D} \leftarrow$ A220 $\underline{B} \leftarrow$

221) Ketone bodies:

- a) may be used as fuel by extra hepatic tissues
- b) are synthesized only in pathological conditions
- c) bring about irreversible damage to nervous tissue, even in small concentrations
- d) may bring about an increase in blood pH

A221

222) One of the factors favoring ketone body synthesis in hepatic mitochondria is a increase in:

- a) velocity of oxidative phosphorylation
- b) synthesis of oxaloacetate
- c) ratio NADH/NAD
- d) ratio ATP/ADP

A222

223) On which of the following enzymes depends the increase in ketone body synthesis by the liver:

- a) malate dehydrogenase
- b) pyruvate dehydrogenase
- c) citrate synthase
- d) acyl CoA dehydrogenase
- e) carnitine palmitoyl transferase l

A223

A221 <u>A</u>←

A222 <u>C</u> ←

A223 $\underline{A} \leftarrow$ Although the increase in acetyl CoA stimulates the activity of pyruvate carboxylase, there is also an increase in the concentration of NADH + H + that displaces the reaction:

Malate + NAD⁺ \rightarrow oxaloacetate + NADH + H⁺

Towards the formation of malate, the use of oxaloacetate for citrate synthesis decreases and acetyl CoA accumulates, with the consequent increase in the synthesis of ketone bodies.

224) For the catabolism of ketone bod, ies a requirement is:

- a) ATP for activating acetoacetate in the presence of HSCoA
- b) cAMP
- c) NADH + H+
- d) a Krebs cycle intermediary

A224

225) The cholesterol transporter ABCA1 allows:

- a) intestinal absorption of cholesterol
- b) cholesterol incorporation into cell membranes
- c) cholesterol passing from cells to nascent HDL
- d) exchange of cholesterol among plasma lipoproteins

A225

226) Scavenger receptors are different from LDL receptors in that they

- a) are not subject to regulation
- b) join lipoproteins that have apo E
- c) when deficient predispose to atherosclerosis
- d) allow incorporation of cholesterol by cells

A226

227) Which of the following fatty acids is omega 3?

- a) CH₃CH=CHCH₂CH₂CH=CHCH₂CH=CH(CH₂)₇COOH
- b) CH₃(CH₂)₇CH=CHCH₂CH₂CH=CHCH₂CH=CHCOOH
- c) CH₃CH₂CH=CHCH₂CH=CHCH₂CH=CH(CH₂)₇COOH
- d) CH₃(CH2)₇CH=CHCH₂CH=CHCH₂CH=CH CH₂COOH
- _____
- A224 <u>D</u> ←
- A225 <u>C</u> ←
- A226 <u>A</u> ←
- A227 <u>C</u> ←

228) The main regulatory enzyme in fatty acid synthesis is:

- a) malic enzyme
- b) carnitine palmitoyl transferase I
- c) acyl CoA synthetase (thiokinase)
- d) pyruvate dehydrogenase
- e) acetyl-CoA carboxylase

A228

229) In adipocytes glycerol 3-phosphate necessary for triacylglycerol synthesis comes from:

- a) dephosphorylation of 1,3 bisphosphoglycerate
- b) phosphorylation for glycerol by glycerol kinase
- c) oxidation of glyceraldehyde 3-phosphate
- d) dihydroxyacetone phosphate reduction
- e) decarboxylation of oxaloacetate

A229

230) For 'de novo' synthesis of fatty acids, acetyl CoA is produced by decarboxylation of:

- a) pyruvate
- b) malonyl CoA
- c) some amino acids
- d) glycerol 3-phosphate

A228	<u>E</u> ←
A229	<u>→ U</u>
A230	<u>A</u> ←

231) In humans the largest portion of amino nitrogen is excreted as:

- a) uric acid
- b) ammonium chloride
- c) urea
- d) glutamine
- e) creatinine

A231

232) Of which enzyme is urea a product?

- a) uricase
- b) aspartase
- c) glutaminase
- d) arginase
- e) urease

A232

233) Glutamine:

- a) transports ammonia from nervous tissue to kidney and liver
- b) is for the most part synthesized in liver
- c) is synthesized by glutaminase
- d) is deaminated a α -ketoglutarate

A233

A231 $\underline{C} \leftarrow$ A232 $\underline{D} \leftarrow$ A233 $\underline{A} \leftarrow$ 234) The enzyme regulating urea synthesis is:

- a) arginase
- b) carbamoyl phosphate synthetase I
- c) argininosuccinate lyase
- d) ornithine transcarbamylase
- e) argininosuccinate synthetase

A234

235) About essential amino acids it may be stated that:

- a) their deficiency brings about negative nitrogen balance
- b) branched ones may regulate protein synthesis
- c) they cannot be synthesized by the human body
- d) they confer biological value to proteins
- e) all of the above

A235

236) The absence of which of the following amino acids in a protein diminishes its biological value?

- a) glutamate
- b) tryptophan
- c) alanine
- d) ornithine
- e) glycine

A236

A234	B	<u>←</u>
A235	E	<u>←</u>
	D	

A236 <u>**B**</u>←

237) A diet lacking one essential amino acid can give rise to a decrease in:

- a) urea synthesis
- b) glutamine synthesis
- c) muscle protein degradation
- d) the ratio of ingested nitrogen/excreted nitrogen
- e) urinary excretion of ammonia nitrogen

A237

238) The biological value of a protein is determined by:

- a) the amount of calories it contributes to the body
- b) its molecular weight
- c) its amino acid sequence
- d) its cost of purchase
- e) its proportion of essential amino acids

A238

239) The reaction catalized by glutamate dehydrogenase may be considered as:

- a) anaplerotic
- b) anabolic
- c) catabolic
- d) amphibolic
- e) all of the above

A239

A237 $\underline{D} \leftarrow$ A238 $\underline{E} \leftarrow$ A239 $\underline{E} \leftarrow$ 240) Adrenalin, noradrenalin and dopamine are synthesized from:

- a) tryptophan
- b) tyrosine
- c) methionine
- d) arginine
- e) lysine

A240

241) Which of the following amino acids may be found phosphorylated in a protein?

- a) methionine and lysine
- b) serine and tyrosine
- c) aspartate and glycine
- d) histidine and serine
- e) isoleucine and threonine

A241

242) Defenders of the harmlessness of monosodium glutamate as an industrial food additive argue that it is not dangerous because when ingested as free glutamate:

- a) does not enter blood because it is used by enterocytes
- b) is the same as that abundantly found in natural proteins
- c) is used by intestinal bacteria and not absorbed
- d) is eliminated in urine
- e) is eliminated in feces

A242

A240 $\underline{\mathbf{B}} \leftarrow$ A241 $\underline{\mathbf{B}} \leftarrow$ A242 $\underline{\mathbf{A}} \leftarrow$

243) The combined activity of aminotransferases and glutamate dehydrogenase allows the:

- a) utilization of amino acids for gluconeogenesis
- b) utilization of amino acids for lipogenesis during the post prandial period
- c) input of amino nitrogen for urea synthesis
- d) synthesis of non-essential amino acids
- e) all of the above

A243

244) Elimination of amino nitrogen during a prolonged fast is achieved thanks to the workings of:

- a) glutaminase
- b) aminotransferases
- c) glutamate dehydrogenase
- d) carbamoyl-P-synthetase
- e) all of the above

A244

245) Ubiquitin is a protein:

- a) with protease activity
- b) marker of proteins to be degraded
- c) that points to proteins that will incorporate into de Golgi apparatus
- d) precursor of ubiquinone or coenzyme Q
- e) enhances the anchoring of ribosomes to endoplasmic reticulum

A245

A243 $\underline{\mathbf{E}} \leftarrow$ A244 $\underline{\mathbf{E}} \leftarrow$ A245 $\mathbf{C} \leftarrow$ 246) Intake of glutamine by the kidney depends fundamentally on:

- a) local rate of protein synthesis
- b) glycaemia
- c) plasma concentration of acid
- d) ingestion of proteins
- e) rate of exchange of renal cells

A246

247) In a state of sepsis (infection) muscle protein degradation and exit of glutamine from this tissue increase. In this state, glutamine is accomplishing the function of:

- a) contributing to excrete acid which may be augmented in this situation
- b) serving as fuel for cells of the immune system
- c) feed nitrogen for the synthesis of nitrogen bases to cells of the immune system
- d) feed nitrogen for NAD+ synthesis
- e) all of the above

A247

Hemoproteins and Nucleotide Metabolism

248) Which of the following metabolites can act as a precursor in the synthesis of heme group and glucose?:

- a) acetyl-CoA
- b) succinyl-CoA
- c) glycerol-3-phosphate
- d) ribose -5- phosphate
- e) pyruvate

A248

A246 $\underline{C} \leftarrow$ A247 $\underline{E} \leftarrow$ A248 $\underline{B} \leftarrow$ 249) A protecting role against gastrointestinal infections in the newborn has been attributed to lactoferrin in breast milk. It is believed that this is due to lactoferrin:

- a) supplies the iron necessary for the immune cells attacking pathogens
- b) promotes the synthesis of antibodies in the newborn
- c) can bind directly to the toxins secreted by gastrointestinal pathogens
- d) sequesters the iron that impairs bacterial growth
- e) supplies the iron for generation of free radicals that damage bacteria

A249

250) Most of the iron in the body is bound to proteins, which is advantageous, because in its free state iron:

- a) catalizes the formation of free radicals
- b) competes with Ca⁺⁺ for the transport canals
- c) stimulates liberation of proinflammatory lymphokines
- d) promotes development of tumors
- e) all of the above

A250

251) Cells can take iron from circulating blood from:

- a) transferrin
- b) heme group
- c) Hb
- d) iron not bound to transferrin
- e) all of the above

A251

A249 $\underline{D} \leftarrow$ A250 $\underline{E} \leftarrow$ A251 $\underline{E} \leftarrow$ 252) In one of the following conditions there may be a deficiency of iron in blood plasma with an increase in intracellular deposits:

- a) hemochromatosis
- b) iron deficiency anemia
- c) hemolytic anemia
- d) an inflammation processes
- e) strict vegetarianism

A252

253) Hepcidin regulates the expression of:

- a) ferroportin
- b) the mRNA of ferritin
- c) the mRNA of transferrin receptor
- d) of iron-receptor proteins
- e) erythropoietin

A253

254) The iron responsive elements (IRE) that regulate the synthesis of apoferritin and the ferritin receptor are:

- a) intron sequences of DNA from the codifying genes of these proteins
- b) proteins that bind iron and act as transcription factors
- c) proteins that can bind to mRNA regions
- d) membrane receptors
- e) sequences of mRNA

A254

A252 $\underline{D} \leftarrow$ A253 $\underline{A} \leftarrow$ A254 $\underline{E} \leftarrow$

255) The binding of iron regulatory proteins (IRP) to the iron responsive elements (IRE) determines an increase in the synthesis of:

- a) transferrin receptor
- b) apoferritin
- c) ferroportin
- d) delta amino levulinate synthase 2 (ALAS2)
- e) hepcidin

A255

256) CO₂ transport in blood is carried out mainly under the form of:

- a) H₂CO₃
- **b)** CO₂
- c) HCO3⁻
- d) carbaminohemoglobin
- e) carboxyhemoglobin

A256

257) Which of the following factors affect the quantity of O_2 bound to Hb?

- a) pO₂
- b) pH
- c) pCO₂
- d) the concentration of 2,3-bisphosphoglycerate
- e) all of the above

A255	<u>A</u> ←
A256	<u>C</u> ←
A257	<u>E</u> ←

258) The percentage saturation of hemoglobin by oxygen is increased when one of the following is increased:

- a) concentration of 2,3 bisphosphoglycerate
- b) concentration of hydrogen ions
- c) concentration of Hb
- d) partial pressure of CO₂
- e) partial pressure of O₂

A258

259) The 'Bohr effect' describes the decrease in Hb affinity for oxygen when:

- a) pO₂ increases
- b) pH is decreases
- c) pCO₂ decreases
- d) the concentration of 2,3 bisphosphoglycerate increases $_{\mbox{\tiny A259}}$

260) In Hb, CO₂ is bound to:

- a) the imidazole groups of histidine residues
- b) amino terminal groups of peptide chains
- c) the iron of the heme group
- d) the OH groups of serine residues

A260

A258	E	←	

A259 <u>**B**</u>←

A260 <u>**B</u> ←</u></u>**

261) 2,3 bisphosphoglycerate decreases the affinity of Hb for oxygen because:

- a) it interferes with the binding of O₂ to the heme group by joining to the proximal histidine residue
- b) competes with O₂ for the heme group due to its negative charges
- c) admits protons from some amino acid residues, making ionized groups where there were none
- d) gives phosphate groups for reversible phosphorylation of Hb
- e) forms ionic bonds with some amino acid residues of the beta chains

A261

262) The binding of hydrogen ions (H⁺) to Hb

- a) does not happen under physiological conditions
- b) determines the formation of saline bridges that stabilize the T form
- c) neutralizes negative charges of COO⁻ of some amino acid residues
- d) adds a positive charge to the iron atom in heme
- e) alters the structure of the heme group

A262

263) CO₂ expired by the lungs comes mainly from:

- a) the urea cycle and other pathways producing metabolic waste
- b) decarboxylation reactions of metabolites
- c) toxin-elimination reactions
- d) oxidative metabolism

A263

A261 <u>E</u>←

A262 $\underline{\mathbf{B}} \leftarrow$

A263 <u>E</u>←

264) The larger part of O₂ inspired is used for:

- a) metabolite oxidation
- b) final acceptance of electrons in the respiratory chain
- c) oxygenating tissues
- d) exchange

A264

Nucleotide Metabolism

265) Among the precursors for the synthesis of purines we find:

- a) ribose, ATP, methionine y glycine
- b) ribose, ATP, glutamine y glycine
- c) ribose, glutamine, CO₂, ATP, H₂O y aspartate
- d) ribose, methionine, y ATP
- e) ribose, glutamine, aspartate, ATP y methionine

A265

266) As a product of pyrimidines catabolism, one can find:

- a) urea
- b) xanthine
- c) beta-alanine
- d) hypoxanthine
- e) uric acid

A266

A264 $\underline{\mathbf{B}} \leftarrow$ A265 $\underline{\mathbf{D}} \leftarrow$

A266 <u>C</u> ←

267) The activity of the enzyme glutamine phosphoribosyl amidotransferase is activated by:

- a) AMP
- b) GMP
- c) IMP
- d) PRPP
- e) glutamine

A267

268) Allopurinol inhibits the enzyme:

- a) carbamoyl phosphate synthetase II
- b) xanthine oxidase
- c) glutamine phosphoribosyl amidotransferase
- d) aspartate transcarbamylase
- e) arginase

A268

269) On a patient showing hyperuricemia it is advisable to lower consumption of:

- a) dairy products
- b) fats
- c) beef
- d) eggs
- e) all of the above

A267	<u>→ U</u>
A268	<u>B</u> ←
A269	<u>C</u> ←

270) The enzyme carbamoyl phosphate synthetase II is inhibited by:

- a) CTP
- b) UTP
- c) ATP
- d) carbamoyl phosphate
- e) PRPP

A270

271) The enzymes carbamoyl phosphate synthetase I and II have in common their:

- a) codifying gene
- b) substrates
- c) product
- d) regulation
- e) location

A271

Metabolic Integration and Regulation

272) Acetyl-CoA is an allosteric activator of:

- a) acetyl CoA carboxylase
- b) carnitine palmitoyltransferase I
- c) pyruvate carboxylase
- d) pyruvate dehydrogenase
- e) ATP citratre lyase

A272

A270 <u>A</u> ←

A271 <u>C</u>←

A272 <u>C</u> ←

273) Acetyl-CoA carboxylase is activated allosterically by:

- a) citrate
- b) acyl-CoA
- c) malonyl-CoA
- d) acetyl-CoA
- e) biotin

A273

274) Hepatic gluconeogenesis only happens if there is a simultaneous increase in:

- a) glycolysis
- b) glycogenolysis
- c) lipolysis
- d) β-oxidation
- e) ketogenesis

A274

275) Pyruvate dehydrogenase is activated when:

- a) there is an increase in the concentration of acetyl-CoA
- b) it is dephosphorylated
- c) there is an increase in the concentration of pyruvate
- d) there is an increase in liver concentration of AMP
- e) the Krebs cycle is activated

A275

A273 $\underline{C} \leftarrow$ A274 $\underline{D} \leftarrow$ A275 $\underline{B} \leftarrow$

276) The destination of pyruvate within mitochondria is determined by

- a) the cell's needs
- b) its concentration
- c) the velocity of its use
- d) the activation state of the enzymes of which it is a substrate
- e) the concentration of the products of the reactions of which it is a substrate

A276

277) Insulin is a hormone that favors the uptake of glucose by:

- a) adipocyte
- b) erythrocyte
- c) hepatocyte
- d) pancreas beta cells
- e) enterocyte

A277

278) Insulin inhibits the secretion of glucagon because:

- a) when glycemia increases there is less glucagon secretion
- b) it allows saving gluconeogenic substrates
- c) it is no longer needed to liberate glucose from the liver
- d) alpha cells have receptors for insulin

A278

A276 $\underline{D} \leftarrow$ A277 $\underline{A} \leftarrow$ A278 $\underline{D} \leftarrow$

279) From a physiological point of view, inhibition of glucagon secretion as a consequence of insulin action is important because:

- a) it is no longer needed to liberate glucose from the liver
- b) alpha cells have receptors for insulin
- c) it allows saving gluconeogenic substrates
- d) when glycemia increases there is less glucagon secretion ${\mbox{\tiny A279}}$

280) Some regulatory enzymes exist in two forms: active and inactive enzyme that may interconvert through:

- a) competitive inhibition
- b) peptide bond hydrolysis
- c) making and breaking of thioester bonds
- d) making and breaking of phosphate bonds
- e) making and breaking of disulphide bonds

A280

281) Malonyl-CoA plays a regulatory role of metabolism because it affects the activity of:

- a) acyl-CoA dehydrogenase
- b) malic enzyme
- c) acetyl-CoA carboxylase
- d) carnitine palmitoyltransferase I
- e) pyruvate dehydrogenase

A281

282) Insulin promotes the synthesis of fatty acids in the liver because it promotes dephosphorylation of the enzyme:

- a) carnitine palmitoyltransferase I
- A279 <u>C</u> ←
- A280 <u>D</u> ←
- A281 <u>D</u> ←

- b) acetyl CoA carboxylase
- c) fatty acid synthase
- d) malic enzyme
- e) cytosolic isocitrate dehydrogenase

A282

283) Insulin promotes glycolysis in hepatocytes because it promotes dephosphorylation of:

- a) glucokinase
- b) phosphofructokinase I
- c) phosphofructokinase II
- d) glyceraldehyde-3-phosphate dehydrogenase

A283

284) Coordinated regulation of fatty acid metabolism implies:

- a) increase in fatty acid synthesis when acetyl CoA carboxylase is activated
- b) increase in oxidation of fatty acids when carnitine palmitoyl transferase is activated
- c) decrease in fatty acid synthesis when there is an increase in the concentration of long chain acyl CoA
- d) increase in oxidation of fatty acids when there is an increase in their release from adipose tissue
- e) decrease in oxidation of fatty acids when acetyl CoA carboxylase is activated

A282	<u>B</u> ←
A283	<u>C</u> ←
A284	<u>E</u> ←

285) Which of the following enzymes catalyzes a reaction where pyruvate is one of the products?

- a) pyruvate kinase
- b) pyruvate dehydrogenase
- c) pyruvate carboxylase
- d) phosphoenolpyruvate carboxykinase (PEPCK)

A285

Note: These same distractor ítems may also be used to ask:

Which of the following enzymes is activated (or inhibited) by acetyl CoA?

Which of the following enzymes is present both in cytosol and mitochondria?

This would serve the purpose of

-Drawing attention to ubiquity of pyruvate in metabolism

-Highlighting the regulatory role of acetyl CoA

-Evidence the existence of phosphoenolpyruvate carboxykinase in cytosol

286) Protein kinase A phosphorylates and inactivates one of the following enzymes in hepatocytes:

- a) glycogen phosphorylase
- b) pyruvate carboxylase
- c) phosphofructokinase I
- d) pyruvate kinase
- e) glucokinase



287) There is an increase in output of pyruvate in both fasting and post absorptive states in hepatic mitochondria. However, in fasting pyruvate is converted into oxaloacetate and not into acetyl-CoA because:

- a) any necessary acetyl CoA is formed by beta oxidation
- b) it is necessary to synthesize glucose for cells that depend on it
- c) pyruvate dehydrogenase is inactive and pyruvate carboxylase is active
- d) this allows the utilization of amino acid carbon atoms for the synthesis of glucose

A287

288) The increase in intracellular concentration of acetyl CoA defines the destiny of pyruvate during fasting because it coordinately regulates these enzymes:

- a) pyruvate dehydrogenase y pyruvate carboxylase
- b) pyruvate kinase y phosphoenolpyruvate carboxykinase
- c) pyruvate carboxylase y phosphoenolpyruvate carboxykinase
- d) pyruvate kinase y pyruvate dehydrogenase
- e) pyruvate carboxylase y pyruvate kinase

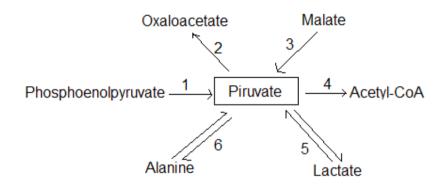
A288

289) Among the consequences of an increase in the concentration of acetyl CoA in liver mitochondria when fasting there may be:

- a) inhibition of acetyl CoA carboxylase
- b) activation of pyruvate carboxylase
- c) increase in ketone body synthesis
- d) all of the above

A289

A287 $\underline{C} \leftarrow$ A288 $\underline{C} \leftarrow$ A289 $\underline{E} \leftarrow$ The following image represents reactions in which pyruvate participates inside hepatocytes. Each number represents one enzyme. The next two questions are related to this graph.



290) The activity of which enzyme is favored by an increase in the congentration of acetyl CoA.

- b) 2
- c) 3
- d) 4
- **e**) 5

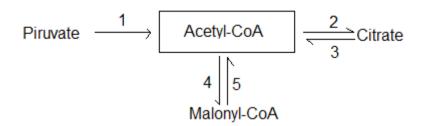
A290

291) The activity of which enzyme is activated when it is dephosphorylated

- a) 2
- b) 3
- c) 4
- d) 5
- e) 6

A291

A290 $\underline{\mathbf{B}} \leftarrow$ A291 $\underline{\mathbf{C}} \leftarrow$ The schematic reactions represent a number of pathways yo or from acetyl-CoA in cells. Each number represents an enzyme.



292) In muscle tissue, which enzyme is activated by phosphorylation of AMP-activated protein kinase (AMPK):

- a) 1
- b) 2
- c) 3
- d) 4
- **e**) 5

A292

293) Glucose is phosphorylated as it enters the hepatocyte because:

- a) allows its storing as glycogen
- b) it is necessary to obtain energy
- c) it is the substrate of glucokinase
- d) this impairs the outflow of glucose
- e) it contributes to maintain glycemia

A293

A292 $\underline{D} \leftarrow$ A293 $\underline{C} \leftarrow$ 294) What is the advantage of phosphorylation of glucose in liver from a biochemical point of view:

- a) allows its storing as glycogen
- b) contributes to the regulation of glycemia
- c) destabilizes the structure favoring ulterior transformation
- d) it impairs outflow of glucose

A294

295) What is the advantage of phosphorylation of glucose in liver from a biochemical point of view:

- a) contributes to regulation of glycemia
- b) impairs outflow of glucose from the cell
- c) allows its storage as glycogen
- d) all of the above

A295

296) Metabolic homeostasis is a state:

- a) that guarantees energetic contribution for cell functions
- b) characterized by a high ATP/ADP + AMP ratio
- c) that guarantees cell multiplication
- d) of balance between demand and supply of nutrients
- e) of equilibrium

A296

A294	D	<u>←</u>
1 205	D	←

A295 $\underline{D} \leftarrow$ A296 $\overline{D} \leftarrow$ 297) The liberation of hormones that intervene in metabolic homeostasis depends on the:

- a) body's needs
- b) proportion of lean mass
- c) proportion of fat mass
- d) diet's composition
- e) all of the above

A297

298) The ingestion of a hyper protein diet may simulate metabolically a state of fasting because:

- a) do not cover glucose needs of cells that depend on it
- b) produce an increase of the input of amino acids to the liver
- c) determine a negative nitrogen balance
- d) they bring about a low insulin/glucagon ratio

A298

299) The increase in lactate formation during protracted muscle contraction is accompanied by an increase in proton liberation. This has an inhibitory effect on their continued production (protons) because:

- a) uncontrolled increase of protons might alter the pH of blood
- b) phosphofructokinase is inhibited by a proton effect
- c) protons inhibit enzymes that liberate NADH + H⁺
- d) protons may damage muscle proteins

A299

A297 $\underline{D} \leftarrow$ A298 $\underline{E} \leftarrow$ A299 $\underline{B} \leftarrow$ 300) Malic enzyme:

- a) uses NADPH produced by the pentose pathway
- b) supplies NADPH for extramitochondrial synthesis of citrate
- c) allows the use or reducing equivalents coming from fatty acids to NADP
- d) can channel the transference of reducing equivalents from fatty acid allows the use of reducing equivalents of glycolysis in fatty acid synthesis
- e) allows the use or reducing equivalents coming from fatty acids for glucose synthesis

A300

301) The oxidation of fatty acids in muscle may be decreased by an increase in the concentration of:

- a) lactate
- b) citrate
- c) acetyl CoA
- d) NADH
- e) glucose

A301

302) Phosphocreatine:

- a) is synthesized from arginine, glycine and methionine
- b) is catabolized forming creatinine
- c) diffuses more quickly than ATP inside the cell
- d) in its synthesis kidney and liver play a part
- e) all of the above

A302

A300 $\underline{D} \leftarrow$ A301 $\underline{B} \leftarrow$ A302 $\underline{D} \leftarrow$ 303) The main source of energy in enterocytes is the oxidation of

- a) glucose
- b) glutamine
- c) ketone bodies
- d) short chain fatty acids
- e) long chain fatty acids

A303

In some instances, the same distractor items can be used with different leading phrases

304) The main source of energy in colonocytes is the oxidation of

- a) glucose
- b) glutamine
- c) ketone bodies
- d) short chain fatty acids
- e) long chain fatty acids

A304

305) Kidney gluconeogenesis from amino acids is different from hepatic one in that it:

- a) is concomitant to net production of bicarbonate
- b) uses predominantly glutamine as carbon source
- c) is more active in metabolic acidosis condition
- d) is not associated to urea synthesis
- e) all of the above

A305

A303 $\underline{B} \leftarrow$ A304 $\underline{D} \leftarrow$ A305 $\underline{E} \leftarrow$ 306) One of the metabolic changes accompanying the progressive decrease of respiratory quotient during aerobic exercise, is the activation of

- a) acetyl CoA carboxylase
- b) carnitine palmitoyl transferase
- c) phosphofructokinase
- d) glycogen phosphorylase
- e) pyruvate dehydrogenase

A306

307) Muscle glycolysis accelerates when there is an increase in the intracellular concentration of:

- a) phosphocreatine
- b) ATP
- c) citrate
- d) cAMP
- e) NADH

A307

308) The type of fuel used by muscle depends on:

- a) intensity of exertion
- b) duration of exercise
- c) availability of oxygen
- d) the type of muscle fiber involved
- e) all of the above

A308

A306	B	←	

A307 <u>D</u>←

A308 <u>E</u> ←

309) Muscle PFK is different from hepatic one in that it:

- a) is not regulated by fructose 2,6 bisphosphate
- b) is not inhibited by phosphocreatine
- c) is not inhibited by citrate
- d) is inhibited when pH decreases

A309

310) Glycolysis in heart muscle is different from that in liver because it:

- a) is not inhibited by $\mathsf{H}^{\scriptscriptstyle +}$ ions
- b) is not inhibited by citrate
- c) is activated when cAMP increases
- d) is inhibited by fructose 2,6 bisphosphate
- e) goes all the way to lactate formation

A310

311) Adiponectin increases the:

- a) synthesis of glucose by the liver
- b) oxidation of fatty acids in muscle tissue
- c) accumulation of TAG in adipose tissue
- d) inflow of glucose into adipose tissue
- e) synthesis of glycogen in the liver

A309	<u>B</u> ←
A310	<u>C</u> ←
A311	<u>B</u> ←

312) In heart muscle protein kinase A increases the intensity of contraction because it phosphorylates

- a) troponin l
- b) myosin
- c) actin
- d) creatine
- e) ^{A312}

313) In smooth muscle cells stimulation of beta adrenergic receptors determines muscle relaxation because it phosphorylates

- a) troponin l
- b) myosin
- c) actin
- d) creatine

A313

314) AMP kinase participates in the regulation of fuel use by muscle because it phosphorylates:

- a) phosphofructokinase 1
- b) phosphofructokinase 2
- c) creatine phosphokinase
- d) carnitine palmitoyl transferase I
- e) acetyl-CoA carboxylase II

A312	<u>A</u> ←
A313	<u>B</u> ←
A314	<u>E</u> ←

315) Creatinine can be used as an index of renal excretion function because:

- a) the amount excreted is proportional to its concentration in blood
- b) its concentration in urine does not change with urinary pH changes
- c) it is not modified by the kidney
- d) the daily amount excreted is constant
- e) it is a very stable compound

A315

316) Muscle tissue dependence on insulin is not absolute because it has

- a) AMP kinase
- b) glucose reserves as glycogen
- c) a small proportion of GLUT 1
- d) the capacity to synthesize small amounts of glucose in prolonged periods of fasting

A316

317) The activity of glucokinase is regulated by the concentration of:

- a) AMP
- b) glucose
- c) glucose 6 phosphate
- d) long chain acyl CoA's
- e) citrate

A317

A315 $\underline{D} \leftarrow$ A316 $\underline{A} \leftarrow$ A317 $\underline{B} \leftarrow$

318) The velocity of beta oxidation in liver depends on:

- a) availability of acetyl CoA
- b) activity of acetyl CoA carboxylase
- c) velocity of glycolysis
- d) input of fatty acids

A318

319) cAMP acts as a second messenger because:

- a) it has a short half life
- b) transmits to the inside of the cell a hormonal signal
- c) is a relatively small molecule and this favors its action

d) it indirectly mirrors the energetic charge of the cell

A319

320) In aerobic exercise:

- a) ATP is supplied by oxidative phosphorylation
- b) oxygen consumption is larger than in anaerobic exercise
- c) the Krebs cycle speed is lower than in anaerobic exercise
- d) muscle fiber does not contract to its maximum capacity
- e) phosphocreatine is not used as a source of energy

321) Besides hepatic tissue, which tissue contributes to metabolize lactate produced by skeletal muscle during anaerobic exercise?

- a) lung
- b) adipose
- c) heart muscle
- d) kidney

A321

A318 <u>**B**</u>←

- A319 **B** ←
- A320 <u>A</u>←
- A321 <u>C</u> ←

322) Lactate production by muscles increases when there is a low ratio of:

- a) ATP consumption to its synthesis in oxidative phosphorylation
- b) the intensity of exercise to oxygen consumption
- c) the use of glucose to that of fatty acids as a source of energy
- d) the contents of glycogen to its velocity of utilization
- e) the concentration of ATP to phosphocreatine

A322

323) Activation of PI3 kinase is the result of a conformational change when it interacts with:

- a) PIP3
- b) the SHR domain
- c) the phosphorylated receptor
- d) the phosphorylated IRS

A323

324) During physical exercise there is an increase in the production of lactate that follows an increase in the concentration of:

- a) ATP
- b) pyruvate
- c) NADH
- d) creatine
- e) AMP

A324

A322 $\underline{A} \leftarrow$ A323 $\underline{D} \leftarrow$ A324 $\underline{C} \leftarrow$ 325) Immediately after physical exercise there is an increase in:

- a) glycogen synthesis
- b) oxygen consumption
- c) ATP synthesis
- d) phosphocreatine synthesis
- e) all of the above

A325

Nutrition and Digestion

326) A meal composed by 20 grams de protein, 10 grams fat and 50 grams of carbohydrates means an intake of approximately:

- a) 80 kcal (334.72 kj)
- b) 290 kcal (1213.36 kj)
- c) 320 kcal (1338.88 kj)
- d) 370 kcal (1548.08 kj)
- e) 690 kcal (2886.96 kj)

A326

327) A decrease of linoleic acid in the diet can give rise to a deficiency in which fatty acid?

- a) stearic
- b) oleic
- c) palmitic
- d) palmitoleic
- e) arachidonic

A327

A325 <u>E</u> ←

A326 <u>D</u> ←

A327 <u>E</u> ←

328) Anemia can occur due to a diet lacking:

- a) pyridoxine
- b) folic acid
- c) linoleic acid
- d) carotenoids
- e) tocopherols

A328

329) Cooking foodstuffs makes them

- a) more palatable
- b) easier to chew
- c) more accessible to digestive enzymes
- d) more soluble
- e) all of the above

A329

330) Individual variations in plasma concentration of cholesterol due to ingestion in cholesterol-rich foods is due fundamentally to:

- a) the type of food ingested
- b) their ingestion associated to carbohydrates
- c) the Body Mass Index
- d) genotype of apoprotein E
- e) the glycemic index of the food

A330

331) Intestinal absorption of glucose is made mainly through:

- a) a glucose/calcium exchange system
- b) a symporter that works with a sodium gradient
- c) simple diffusion
- A328 <u>**B</u> ←</u></u>**
- A329 <u>E</u> ←
- A330 <u>D</u> ←

- d) transport systems requiring ATP
- e) an insulin dependent transporter

A331

332) Foodstuffs glycemic index is related to:

- a) their capacity to stimulate insulin liberation
- b) the total amount of carbohydrates they contain
- c) their effect on glycemia
- d) the amount of glucose they contain
- e) their caloric contents

A332

333) The energetic density of foods is larger the larger is their:

- a) glycemic index
- b) thermogenic effect
- c) proportion of oxygen
- d) reducing potential
- e) contents in essential nutrients

A333

334) Fiber present in foodstuffs:

- a) cannot be used by humans as a source of energy
- b) contribute to the elimination of colon bacteria
- c) may give rise to short-chain fatty acids that are absorbed
- d) facilitate absorption of certain nutrients

A331	B	←

- A332 <u>C</u> ←
- A333 **D** ←
- A334 $C \leftarrow$

335) Which of the following mechanisms may be invoked to explain the decrease in plasma cholesterol due to fiber in the diet?

- a) decrease in reabsorption of bile salts in the gut
- b) impairment the activity of lipid-digesting enzymes
- c) decrease in cholesterol absorption
- d) their degradation by bacteria may generate metabolites that inhibit HMG CoA reductase
- e) all of the above

A335

336) The concept 'functional foods' refers to:

- a) foods that besides their nutritive value give extra benefits to the body
- b) foods that improve the intestine function
- c) foods that are absorbed more efficiently

d) a term created by publicity to push sales of some products ${\mbox{\tiny A336}}$

337) The concept 'energy density' of foods refers to their input of calories:

- a) coming from essential nutrients per gram
- b) equivalents to one (1) gram of glucose
- c) equivalent to one (1) gram of fat
- d) per gram

A337

A335 $\underline{\mathbf{E}} \leftarrow$ A336 $\underline{\mathbf{A}} \leftarrow$ A337 $\underline{\mathbf{D}} \leftarrow$

338) The thermogenic effect of carbohydrates is approximately 7%; this implies that after ingestion of 100g of carbohydrates:

- a) is equivalent to ingest 107 grams
- b) only 93% will be used for their oxidation or storage
- c) 7% more energy is liberated when they are oxidized
- d) 7% of their energy is liberated as heat

A338

339) Bile salts contribute to digestion of lipids because they:

- a) stimulate pancreatic secretion
- b) increase pH of intestinal contents
- c) stimulate the activity of pancreatic lipase
- d) diminish surface tension of water

A339

340) Mixed micelles formation favors the absorption of digestion products because it creates a concentration gradient between the micellar phase and the enterocyte, thus contributing to overcome the obstacle posed by the:

- a) low affinity of transporters
- b) large size of products
- c) water layer adjacent to the intestinal lining
- d) absence of a transporter for them
- e) selective permeability of the membrane

A338	<u>B</u> ←
A339	<u>→ U</u>
A340	<u>C</u> ←

341) The interaction of bile salts with the transcription factor that recognizes them modifies hepatic metabolism because it inhibits synthesis of the enzyme:

- a) palmitoyl carnitine transferase I
- b) colesterol-7a-hydroxylase (CYP7A1)
- c) acetyl CoA carboxylase
- d) HMG-CoA reductase
- e) malic enzyme

A341

342) Bile salts can take part in the regulation of lipid metabolism because they interact with:

- a) hepatic X receptors (LXR)
- b) farnesoid X receptor (FXR)
- c) cAMP response element binding protein (CREB)
- d) peroxisome proliferating activated receptor (PPAR)
- e) steroid regulating element-binding protein (SREBP)

343) Fatty acids produced by digestion of fat:

- a) are incorporated into chylomicrons together with phospholipids and cholesterol
- b) enter the blood and are bound to albumin
- c) are reesterified to form TAG in the enterocyte
- d) pass into the thoracic duct and then to the blood
- e) get to the liver via the porta system

A343

A341 $\underline{B} \leftarrow$ A342 $\underline{D} \leftarrow$ A343 $\underline{C} \leftarrow$

344) Pancreatic lipase:

- a) exerts its action in the interface lipid-water
- b) is stimulated by bile salts
- c) cannot function on lipids that are not in micelles
- d) hydrolyzes fatty acids from the diet
- e) is activated by irreversible covalent modification

A344

345) A significant decrease in the cholesterol intake might affect its metabolism because of:

- a) a decrease in its excretion
- b) an increase in its endogenous synthesis
- c) an increase in its intestinal absorption
- d) a decrease in its utilization for synthesis of bile salts
- e) a decrease in its incorporation to cell membranes

A345

346) An expert in diabetes tuition advises patients to consume foods rich in fiber to improve metabolic control. This is considered correct because fiber in foodstuffs diminishes the:

- a) reabsorption of bile salts in the gut
- b) glycemic index of foodstuffs
- c) caloric intake due to its satiety power
- d) all of the above

A346

A344 $\underline{A} \leftarrow$ A345 $\underline{B} \leftarrow$ A346 $\underline{D} \leftarrow$

347) The glycemic index of foodstuffs may be modified by:

- a) the cooking method
- b) their fiber contents
- c) the state of ripening of fruits
- d) the degree of hydration
- e) all of the above

A347

348) All digestive enzymes have as a common feature that they:

- a) are secreted as precursors
- b) catalize hydrolysis reactions
- c) are activated by pH changes
- d) act in the digestive tract lumen
- e) are liberated by the pancreas

A348

349) Proponents of a carbohydrate-poor diet argue that unlike conventional diets with the same amount of calories, their varied methods:

- a) have a greater satiety power
- b) increases the offer of essential nutrients
- c) improves insulin secretion
- d) diminishes the risk of ketosis
- e) are better tolerated by those subject to it A349

A347	<u>E ←</u>
A348	<u>B</u> ←
A349	<u>A</u> ←

350) A dietetic regimen, despite being adequate in calories, can bring about ketosis due to insufficient:

- a) lipids
- b) carbohydrates
- c) proteins
- d) micronutrients
- e) lipid-soluble vitamins

A350

Biochemistry and clinical cases

A word of caution: These sketches with clinical data are used as a pretext to illustrate the relationship of biochemistry with clinical knowledge. However, from a medical point of view, only a partial vision and therefore necessarily incomplete is presented of a patient's clinical problems.

Besides, for the purposes of this book they have been simplified to a large extent with the intention of avoiding data that might distract the student's attention from the biochemical facts to be illustrated. For example, a diagnosis is not required here, for that is not the objective and students at this stage are not prepared to do it. It is necessary to insist on the fact that patients are <u>real</u> <u>people</u> with habits, problems, beliefs, specific socio-economic conditions, etc. that make each one unique. A large part of clinical medicine is precisely to adapt scientific knowledge to each patient and his or her particular problem. Biochemistry is not clinical medicine and, even less, Medicine. But without Biochemistry, Medicine loses much of its scientific character.

Clinical Cases

Alcoholism

We are dealing with a male patient, 56 years of age, chronic alcoholic that has ingested a large amount of alcohol in the last two days and has had very little to eat during that time. He arrives at the emergency room with nausea, dizziness, vomits, sweating, headache blurred vision and confusion. He is semiconscious and has alcoholic breath. Lab tests show alcoholaemia of 284 mg/dl, glycemia 2.1 mM and lactate 8.5 mM. He is subject to gastric lavage and glucose solution is administered intravenously.

351) From the biochemical point of view, individual alcohol tolerance is due to the activity of the enzyme:

- a) alcohol dehydrogenase
- b) aldehyde dehydrogenase
- c) acetyl-Co A synthetase
- d) CYP2E1
- e) all of the above

A351

352) Chronic alcoholism may produce fatty liver because of:

- a) decrease in beta oxidation
- b) increase in availability of glycerol P
- c) increase in the synthesis of TAG
- d) all of the above

A352

353) Among the metabolic changes brought about in the liver by alcohol ingestion, there is:

- a) decrease in fructose 2,6 bisphosphate formation
- b) decrease in synthesis of malonyl CoA
- c) increase in the ratio NADH/NAD
- d) decrease in the ratio ATP/ADP + AMP

A353

A351	<u>E</u> ←
A352	<u>→ U</u>
A353	C ←

354) A consequence of hepatic metabolism of ethanol may be hypoglycemia because the process produces:

- a) a decrease in beta oxidation
- b) an increase in lactate reduction
- c) a decrease in velocity of the Krebs cycle
- d) an increase in the synthesis of ketone bodies

A354

355) Chronic alcoholism may increase the plasma concentration of VLDL because it decreases the:

- a) hepatic synthesis of apo C
- b) activity of LPL
- c) hepatic oxidation of fatty acids
- d) degradation of VLDL

A355

Cutaneous Porphyria in alcoholic patient

Patient 68 years of age, with alcoholism history, consults skin lesions over the last year. Physical examination blisters were found and erosions covered by serohematic scabs and hypo pigmented scars located in forearms, back, hands and scalp. Laboratory tests show an increase in serum ferritin with a concentration of 1469.1 ng/ml porphyrin concentration in urine of 2,986 µg/24hours and an index of plasma porphyrins of 4.28. The histopathological study of lesions confirmed a diagnosis of Cutaneous Porphyria Type 1(CPT1).

A354 <u>**B**</u>←

A355 <u>C</u> ←

356) CPT1 is a consequence of the inhibition of the activity of:

- a) uroporphyrinogen decarboxylase
- b) porphobilinogen deaminase
- c) uroporphyrinogen III synthase
- d) ferrochelatase

A356

357) Cutaneous lesions appear in skin zones exposed to sunlight because porphyrins are excited chemically when they absorb ultraviolet light; this extra energy is transferred to oxygen molecules and this brings about:

- a) tissue hypoxia
- b) formation of ROS
- c) inhibition of the respiratory chain
- d) formation of water, contributing to blister formation ${\rm \tiny A357}$

358) Chronic ingestion of large amounts of alcohol may alter iron metabolism because it brings about a decrease in:

- a) expression of ferroportin
- b) synthesis of hepatic hepcidin
- c) the binding to iron response elements
- d) the expression of the divalent metals transporter $_{\scriptscriptstyle A358}$

A356	<u>A</u> ←
A357	<u>B</u> ←
A358	<u>B</u> ←

359) An increase of iron in the body can trigger the emergence of CPT because:

- a) it induces the synthesis of ALA synthase, increasing the formation of precursors of porphyrins
- b) decreases the activity of uroporphyrinogen decarboxylase, through the formation of an inhibitory compound
- c) stimulates the formation of ROS
- d) all of the above

A359

Obesity

Thirty-seven years old patient who wishes to lose weight, Physical examination finds a patient of 70 kg weight and 1,65 m height, abdominal circumference 88cm. laboratory tests show a concentration of TAG of 325mg/dl and glycemia of 102 mg/dl. An increase in physical exercise was prescribed, along with a diet with increase in the consumption of vegetables and fish on the diet.

360) Many of the beneficial effects of physical exercise are due to activation of AMP kinase. In striated muscle tissue this enzyme is activated as a consequence of:

- a) its phosphorylation by protein kinase A
- b) the increase in the ratio 5'-AMP/ATP
- c) increase in the concentration of phosphocreatine
- d) a decrease in cAMP
- e) its phosphorylation by PKB (akt)

A360

A359 $\underline{\mathbf{E}} \leftarrow$ A360 $\underline{\mathbf{B}} \leftarrow$ 361) La AMP kinase, among other effects, stimulates beta-oxidation of fatty acids in muscle because it phosphorylates and inactivates:

- a) 3-hydroxy-3-methylglutaryl-coa reductase
- b) malonyl-CoA decarboxylase
- c) palmitoyl-acyltransferase 1
- d) acetyl-CoA carboxylase

A361

362) The addition of polyunsaturated fatty acids in the diet has a beneficial effect on hypertriglyceridemia and it has been proposed that this happens because they diminish the expression of SREP1e, something that diminishes the synthesis of:

- a) glycerol phosphate acyl transferase
- b) acetyl CoA carboxylase
- c) fatty acid synthase
- d) ATP citrate lyase
- e) all of the above

A362

363) The SREPB are:

- a) DNA sequences codifying for proteins that regulate lipid metabolism
- b) nuclear receptors
- c) transcription factors
- d) a family of regulatory enzymes of lipid metabolism

A363

A361 $\underline{D} \leftarrow$ A362 $\underline{E} \leftarrow$ A363 $\mathbf{C} \leftarrow$ Male patient, 18 years of age, consults for moderate acne. Through interview the dermatologist finds out that the patient is a medical student that, by the way, considers biochemical knowledge as little ore than useless in medical studies. The doctor suggests looking up acne and diet in internet as a means to get information about his clinical problem and the importance of biochemistry to understand clinical findings and academic discussions about the influence of diet in the treatment of acne, in order to take informed decisions.

To introduce the discussion of acne as a metabolic disease can be strongly motivating for students

364) mTOR is a:

- a) component of the androgen receptor
- b) transcription factor
- c) kinase sensitive to nutrients
- d) sequence of DNA bases

A364

365) In skin oil glands mTOR is activated:

- a) as a consequence of hypoglycemia
- b) when AMP kinase is activated
- c) when PI3 kinase is inactivated
- d) when input of branched amino acids increases

A365

A364	C	<u>←</u>	
A365	E	<u>←</u>	

366) Which of the following foods may exacerbate acne?

- a) saturated fats
- b) high glycemic index items
- c) dairy products
- d) all of the above

A366

367) The activation of mTOR in skin oil glands will have as a consequence the stimulation of:

- a) the entrance of glucose into the cell
- b) the expression of genes regulated by SREBP-1c
- c) activity of AMP kinase
- d) incorporation of leucine and other branched amino acids

368) Which of the following may be a good therapeutic strategy for acne?

- a) increase the polyunsaturated fatty acids in the diet
- b) increase the activity of AMP kinase
- c) diminish dairy products consumption
- d) all of the above

A368

Metabolic Syndrome

Male patient,45, consults about losing weight. Physical examination reveals: height 1,82 m, weight 97 Kg for a Body Mass Index (BMI) of 29.3. Abdomen circumference 98 cm. Blood pressure 145/90, glycemia 98 mg7dl, TAG 400mg/dl. Treatment suggested is restricted calorie diet and physical exercise. After six months he has been

A366 <u>D</u> ←

A367 <u>B</u>←

A368 <u>D</u> ←

unable to change habits and clinical picture stays invariable. For this reason treatment with fibrates is indicated.

369) The most likely cause for hypertriglyceridemia in this patient is the:

- a) decrease in the activity of LPL
- b) increase in the synthesis of VLDL
- c) decrease in apo C
- d) decrease in clearance of VLDL and ChM remnants
- e) increase in chylomicrons

A369

370) Fibrates are capable of binding to activated receptors of the peroxisomes proliferator (PPAR), which are transcription factors regulating the synthesis of several molecules involved in lipid metabolism. One of the consequences of their action is to increase the synthesis of:

a) LPL

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b) apo Al
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- c) Acyl CoA synthetase
- d) ABCA1
- e) all of the above

A370

Hypercholesterolemia and overweight

Patient with overweight and cholesterol LDL of 170 mg/dl. This is a female patient, 30, coming for gynecological control. Physical examination: weight 65 Kg, height 1,58 m. Laboratory tests show LDL values of 170 mg/dl. A change of lifestyle is prescribed (physical exercise, and hypolipemic diet) plus statins.

A369 <u>B</u>←

A370 <u>E</u> ←

371) Statins are drugs used to lower cholesterol levels in patients. They act:

- a) inhibiting the synthesis of cholesterol by cells
- b) avoiding cholesterol deposits in blood vessels
- c) increasing the disposal of cholesterol in feces
- d) inhibiting the absorption of cholesterol from the gut
- e) decreasing the synthesis of cholesterol-making enzymes in cells

A371

372) Which of the following enzymes is affected by statins?

- a) site 1 protease
- b) 3-hydroxy-3-methylglutaryl-coa reductase
- c) intestinal cholesterol esterase
- d) lecithin-cholesterol acyl transferase (L-CAT)
- e) acyl cholesterol acyl transferase

A372

Hypercholesterolemia IIa

Male patient, 40, with cholesterol in blood values of 536 and TAG in normal range. His father died of a myocardial infarction (heart attack) at 45. Diagnosis: Hypercholesterolemia phenotype IIa, heterozygote. Treatment prescribed: diet, a physical exercise program and statins. After one year of treatment no significant change is found in cholesterol values and thus cholestyramine is added to the treatment.

A371 <u>A</u>←

A372 <u>**B**</u>←

373) Among the described effects of cholestyramine administration is the increase in:

- a) hepatic synthesis of cholesterol
- b) hepatic uptake of cholesterol
- c) bile salts synthesis
- d) bile salts excretion
- e) all of the above

A373

374) The effects of cholestyramine on hepatic synthesis of cholesterol are partly due to:

- a) increase in the synthesis of bile salts
- b) inhibition of HMG-CoA reductase
- c) activation of PPAR α
- d) activation of FXR
- e) all of the above

A374

375) A short time afterwards this patient started to suffer from flatulence and diarrhea; the treatment was changed from cholestyramine to ezetimibe, that produces inhibition of

- a) transformation of VLDL into LDL
- b) cholesterol synthesis
- c) cholesterol absorption
- d) bile salts synthesis
- e) VLDL synthesis

A375

A373 $\underline{\mathbf{E}} \leftarrow$ A374 $\underline{\mathbf{A}} \leftarrow$ A375 $\mathbf{C} \leftarrow$ 376) The target molecule for the action of ezetimibe is:

- a) LPL
- b) LCAT
- c) hepatic lipase
- d) HMG-CoA reductase
- e) the NPC1L1 transporter

A376

Myocardial infarction

This is a 68-year-old male patient, known to suffer from high blood pressure. A smoker of 20 cigarettes a day for 40 years until 6 months ago, refers feeling for the last three months retrosternal chest pain, oppressive, of moderate intensity that is triggered by walking approximately 100 m, irradiated to the neck, which lasts 3-5 minutes, with rest, for 3 months. He is brought to emergency, for presenting pain of equal location, of greater intensity, of two hours of evolution, which appeared at rest, accompanied by profuse sweating, dyspnea and great anxiety Physical examination: SV: Weight 110 Kg; PA: 180/120 mmHg; Pulse 120 PM; Respiratory frequency 22 PM; pale and sweaty skin.

377) During an Myocardial Infarction (MI) the body is under stress; this causes a series of hormonal and metabolic alterations. Among these changes is the release of catecholamines that can affect the metabolism producing:

- a) glycogenolysis
- b) lipolysis
- c) beta-oxidation
- d) all of the above

A377

A376 $\underline{\mathbf{E}} \leftarrow$ A377 $\underline{\mathbf{D}} \leftarrow$

378) The increase in FFA can damage cardiac function by:

- a) increase in the synthesis of ketone bodies leading to acidosis altering electrical conductivity
- b) inhibition of glucose utilization by cardiac tissue with consequential decrease in ATP synthesis
- c) triggering the inhibition of lactate dehydrogenase win concomitant increase in lactic acid concentration
- d) damage to the mitochondrial membrane and uncoupling of respiratory chain

A378

379) There is a finite number of binding sites for fatty acids in blood plasma, this means an increase in fatty acid concentration can give rise to an increase in Free Fatty Acids (FFA). This may happen if there is:

- a) stress, with catecholamine liberation
- b) increase in fat ingestion
- c) decrease in fatty acid oxidation
- d) hyperglycemia
- e) increase in insulin liberation

A379

380) The most specific test for Myocardial infarction is determining:

- a) lactate dehydrogenase 1
- b) lactate dehydrogenase 2
- c) creatine kinase II MB
- d) troponin
- e) myoglobin

A380

A378 $\underline{D} \leftarrow$ A379 $\underline{A} \leftarrow$ A380 $\underline{D} \leftarrow$ 381) Among the changes found in heart tissue as a consequence of myocardial infarction there is an increase in:

- a) intracellular pH
- b) lactate liberation
- c) acetyl CoA formation
- d) synthesis of malonyl CoA
- e) fatty acid utilization

A381

NB: Changes in lifestyle: dietetic habits (increase in consumption of fruits, cereals, fish, etc.) and increase in physical activity are a fundamental part of prevention and treatment of metabolic syndrome, diabetes, hyperlipemias and other diseases. However, a high percentage of patients find it very difficult to adopt new habits.

Type 1 Diabetes

Male patient, 28, no prior medical record of any importance is brought to ER with photophobia, cephalea, ketonic breath, nausea and vomits of alimentary contents on three occasions, with concomitant diffuse abdominal pain and general feeling of being unwell. He notices intense thirst, weight loss not quantified and marked polyuria for several days.

Physical examination. Vital signs. Temperature 38,50C, pulse 110 pm, breathing frequency 32 pm. Blood pressure 110/60 mmHg with no orthostatic change. General aspect: acute illness. Dry, dehydrated skin. Laboratory tests: Hb 11,5 g/dl; leucocytes 14,700/ul; platelets 220,000/ul; glucose 659 mg/dl; Urine: glycosuria +++ with ketonuria

A381 <u>**B**</u>←

382) Administration of insulin can revert the increase in ketogenesis because it stimulates, among other enzymes, hepatic activity of:

- a) carnitine palmitoyl transferase
- b) citrate synthase
- c) pyruvate dehydrogenase
- d) acetyl CoA carboxylase

A382

383) In an uncompensated diabetic patient there is inhibition of the Krebs Cycle in hepatic mitochondria because there is:

- a) an increase in the ratio NADH/NAD due to increased beta oxidation
- b) a decrease in oxaloacetate synthesis due to the inhibition of pyruvate carboxylase
- c) a decrease in the activity of pyruvate dehydrogenase
- d) an increase in utilization of Acetyl CoA for ketone bodies synthesis

A383

384) The mechanism responsible for the rapid descent of glycemia as an early effect of the injection of insulin is:

- a) activation of hepatic glycolysis
- b) translocation of glut to the sarcolemma
- c) inhibition of hepatic gluconeogenesis
- d) inhibition of muscle glycogenolysis

A384

A382 $\underline{D} \leftarrow$ A383 $\underline{A} \leftarrow$ A384 $\underline{B} \leftarrow$

Type 2 Diabetes

A 45-year-old female patient who attended a medical examination prior to undergoing cosmetic surgery. Physical examination revealed: height of 1.68 m and a weight of 72 kg for a BMI of 25.51. Laboratory tests showed fasting glycemia 132, HbA1c 6.8%. The diagnosis of type 2 diabetes was made and dietary treatment and a physical exercise program were indicated

385) At the pre-diabetic stages and in type 2 diabetes, there may be hyperinsulinemia jointly with a paradoxical increase in glucagonemia. This may be due to a:

- a) alfa cells resistance to insulin
- b) physiological response trying to compensate for the lack of insulin
- c) inhibition in enzymatic response to insulin action
- d) decrease in activity of enzymes that degrade glucagon's second messengers
- e) associated state of resistance to glucagon

386) Deleterious effects of chronic hyperglycemia are in large part due to the non-enzymatic binding of glucose (glycation) to cellular components. When this happens to proteins, irreversible changes occur affecting their structure and function. The factor which in the highest degree is involved in this protein modification is their:

- a) amino acid composition
- b) location
- c) half life
- d) function

A386

A385 $\underline{\mathbf{B}} \leftarrow$ A386 $\underline{\mathbf{C}} \leftarrow$

387) The binding of glucose to Hb to form HBA1c determines:

- a) an alteration in its primary structure
- b) formation of cross links between globin chains
- c) oxidation of the iron of heme group
- d) dissociation of the globin chains
- e) changes in its net charge

A387

388) Some of the beneficial effects for diabetes patients of the activation of AMP kinase may be due to phosphorylation of:

- a) components of the vesicles containing GLUT 4
- b) acetyl CoA carboxylase
- c) SREBP 1c
- d) 3-hydroxy-3-methylglutaryl reductase
- e) all of the above

A388

389) The activities of the kinases dependent on AMP and cAMP have in common the stimulation of

- a) glycolysis
- b) beta oxidation
- c) glycogenolysis
- d) translocation of GLUT 4 to the plasma membrane

A389

A387	E	←

- A388 <u>E</u> ←
- A389 <u>**B**←</u>

390) The benefits of physical exercise on the sensitivity to insulin are partly due to the increase of:

- a) secretion of adiponectin
- b) the stores of glycogen in liver
- c) an increase in insulin liberation from the pancreas
- d) the stores of glycogen in skeletal muscle
- e) muscle mass

A390

391) In its target cells metformin inhibits Complex I of the mitochondrial respiratory chain; this determines metabolic changes which include:

- a) inhibition of glycolysis
- b) stimulation of beta oxidation
- c) stimulation of glycogenolysis
- d) stimulation of the Krebs cycle

A391

392) Metabolic changes produced by metformin are mediated by the activation of:

- a) protein kinase A
- b) protein kinase C
- c) phospholipase C
- d) AMP kinase
- e) PI3 kinase

A392

A390 $\underline{A} \leftarrow$ A391 $\underline{B} \leftarrow$ A392 $\underline{D} \leftarrow$

Iron deficiency anemia

Boy, three years old, who consulted physician for extreme paleness. Physical examination shows weight 13.2 Kg height 91 cm Blood pressure 99/56. Very pale skin and mucous membranes. Laboratory findings: Hb 5.0 3.2 x10⁶ RBC/cmm, Hematocrit 14.6, VCM 46.8, HCM 13.5pg, CHCM 28.6g/dL. Presumptive diagnosis is iron deficiency anemia.

393) In this case it is likely to find a decrease in:

- a) haptoglobin
- b) erythropoietin
- c) transferrin
- d) serum ferritin
- e) total saturation capacity for iron

A393

394) Among the compensatory mechanisms found as a response to iron deficiency anemia there is a decrease in the synthesis of:

- a) hepcidin
- b) ferroporitin
- c) erythropoietin
- d) transferrin receptors
- e) responsive elements to iron

A394

395) The protein transporting iron among tissues is:

- a) ferritin
- b) hemopexin
- c) ferroportin
- d) transferrin
- e) ceruloplasmin (ferroxidase)

A395

A393 $\underline{D} \leftarrow$ A394 $\underline{A} \leftarrow$ A395 $\underline{D} \leftarrow$

Anemia associated to infectious processes

Female patient, 8 years-old girl brought to hospital by her mother, worried about paleness of little girl. Physical examination shows moderate paleness, hypertrophic tonsils and palpable sub maxillary lymph nodes. Laboratory finding: Hb 10g/dL Mother refers frequent tonsillitis episodes during last year. Diagnosis. Moderate anemia as a consequence of inflammatory bouts.

396) It has been postulated that anemia associated to chronic infectious and inflammatory processes:

- a) is a consequence of the utilization of iron by pathogens
- b) is a consequence of the utilization of iron by immune cells
- c) may be a defense mechanism by limiting iron to pathogens
- d) is due to an increase in red cell destruction

A396

397) In these situation, there is an increase in:

- a) transferrin
- b) transferrin saturation
- c) hepcidin
- d) haptoglobin
- e) degradation of Hb

A397

A396 <u>C</u> ←

A397 <u>C</u> ←

398) Correction of chronic infectious and inflammatory processes anemia can be achieved by:

- a) control of the inflammatory process
- b) parenteral administration of iron
- c) oral administration of iron, together with folic acid and vitamin b6
- d) administration of iron chelating agents
- e) all of the above

A398

Sickle Cell Anemia

Sickle cell anemia is not frequent. However, its prevalence increases in persons with sub-Saharan African ascent, as well as India, Saudi Arabia and Mediterranean countries. Migrations have increased its appearance in the American continent and in Europe.

Eight months old infant admitted to hospital with signs and symptoms of respiratory infection and marked anemia. Blood tests show drepanocytes. Diagnosis: Respiratory infection and sickle-cell anemia.

399) Frequently the first manifestation of sickle cell anemia appears after six months of life. This is so because:

- a) infants are frequently fed maternal milk up to that age
- b) infants at that age are more prone to infections because they no longer have the protection transferred by the mother
- c) fetal hemoglobin is replaced by adult Hb approximately at six months of age
- d) lesser age infants require less oxygen consumption to maintain their metabolism

A399



400) Hemoglobin S is the product of a point-mutation that results in the change or one amino acid residue:

- a) with negative charge for another with positive charge
- b) polar without charge for another with positive charge
- c) with positive charge for another polar without charge
- d) with negative charge for another non polar $_{\rm A400}$

Glucose 6–P dehydrogenase deficiency

Deficiency of glucose -6-phosphate dehydrogenase is the most common enzymopathy in human beings and one of the most frequent congenital defects.

A 4-year-old male child with a history of hyperbilirubinemia in the neonatal period brought to the emergency room by his mother when he presents marked jaundice without evident cause. He had been with a flu bout for which his mother had given him aspirin. Laboratory tests showed Hematocrit: 17.6% and Hb: 5.7 gr / dL, reticulocytes: 3.8%, Total bilirubin: 3.46 mg / dL, indirect: 2.96 mg / dL. He received treatment and underwent studies that led to the diagnosis of anemia due to glucose 6 P dehydrogenase deficiency.

401) To which metabolic pathway does the reaction catalized by glucose -6-P dehydrogenase belongs to?

- a) glycolysis
- b) neoglucogenesis
- c) pentoses pathway
- d) glycerogenesis

A401

A400 $\underline{D} \leftarrow$ A401 $\underline{C} \leftarrow$ 402) Hemolytic anemia associated to deficiency of glucose -6phosphate dehydrogenase can be explained because:

- a) there is a decrease in the intracellular concentration of reduced glutathione
- b) there is a reduction in lipid synthesis for the membrane of erythrocytes
- c) the supply of reducing equivalents for the respiratory chain fails
- d) less ATP is produced by the glycolytic pathway
- e) all of the above

A402

403) In the event of intravascular hemolysis, plasmatic haptoglobin:

- a) can bind to the erythrocyte membrane to minimize hemolysis
- b) increases in concentration in proportion to the degree of hemolysis
- c) binds to liberated hemoglobin, thus avoiding loss of iron
- d) binds to liberated iron neutralizing its toxicity

A403

Hyaline Membrane

A newborn girl has gone into the neonatal intensive care unit. 32 weeks of gestation, 2.150Kg weight and 44 cm length. She shows difficulty for breathing characterized by tachypnea, tiraje and general cyanosis. Diagnosis: hyaline membrane syndrome. Exogenous surfactant therapy started, via endotracheal instillation.

A402 <u>A</u> ←

A403 <u>C</u> ←

404) The Hyaline Membrane Syndrome is due to a decrease in the synthesis and secretion of pulmonary surfactant whose main phospholipid is.

- a) cholesterol
- b) cardiolipin
- c) sphingomyelin
- d) phosphatidyl inositol
- e) dipalmitoyl phosphatidylcholine

A404

405) Phospholipids are amphipathic molecules characterized by:

- a) having both a hydrophobic and a hydrophilic extreme
- b) reacting with two other molecules simultaneously
- c) having a positive and a negative pole
- d) having two poles of the same charge
- e) being able to react with proteins

A405

406) The pulmonary surfactant:

- a) prevents collapse of alveoli during expiration
- b) diminishes surface tension in alveoli
- c) makes monolayers adhering to the alveoli surface
- d) facilitates expansion of alveoli during inspiration
- e) all of the above

A406

Gilbert's Syndrome

Teenage girl taken to hospital by her mother due to yellowish

- A404 <u>E</u> ←
- A405 <u>A</u>←
- A406 <u>E</u> ←

coloring of skin and eyes that started two days ago. Mother refers that the girl had undertaken a hypocaloric diet for three days for weight reducing. Physical examination reveals icteric hue of skin and mucous membranes and no other clinical signs. Laboratory tests report: total bilirubin 2.53 mg/dL, direct 0.35 indirect 2.18 mg/dL All other values are normal, including hepatic function tests. Diagnosis. Gilbert's Syndrome.

407) Gilbert's syndrome is a benign and frequent hyperbilirubinemia due to a deficiency of the enzyme:

- a) ligandin
- b) heme oxygenase
- c) UDP glucuronyl transferase
- d) biliverdin reductase

A407

408) The enzymatic deficiency in Gilbert's syndrome affects:

- a) bilirubin conjugation
- b) bilirubin synthesis
- c) hemoglobin degradation
- d) bile formation

A408

Biochemistry is also useful in understanding other kinds of medical problems

Influenza is a serious public health problem, affecting millions of people each year. Vaccines against the disease, which is produced by an RNA virus, lose effectiveness after a time because of mutations in the genome of the virus that change its antigenicity

A407
$$\underline{C} \leftarrow$$

A408 $\underline{A} \leftarrow$

409) The high rate of mutations presented by the influenza virus is mainly due to the fact that:

- a) its RNA is unstable and undergoes spontaneous alterations of its structure
- b) viral proteins can adopt different conformations exposing different antigenic determinants
- c) viral genes are expressed randomly in each cycle of reproduction

d) RNA polymerases do not have an editing function ${\rm ^{A409}}$

410) The virus binds to the mucus of the respiratory tract, from where it is released into the body by the activity of neuraminidase. If you were to have resources to design drugs to treat the disease, which one would you choose:

- a) neuraminidase inhibitors
- b) those that prevent their binding to mucus
- c) better antibiotics
- d) more potent mucolytics

A410

Carolina and Sofia discuss the importance of biochemistry. Carolina is a lover of the natural, does not use allopathic medicines and is critical of any genetic manipulation of living beings, which to her mind makes the biochemists of the world responsible for possible disasters. Her friend tells her that she should not be so radical, that learning biochemistry can help her support her opinions. She argues that the use of natural medicine is efficient, cannot be intuitive, that not everything natural is better and that not everything that seems natural is so in fact. To reinforce her point, she challenges Carolina to

A409 <u>D</u> ←

A410 <u>A</u>←

answer the following questions.

411) How do you think the breed of your poodle toy originated (toy poodle)?

- a) by transgenic manipulation of another dog breed
- b) by evolution as all the species of creation
- c) by intentional crosses between breeds to produce a small animal

d) as a product of random crossing between different races ${\rm ^{A411}}$

412) Many plants contain acetylsalicylic acid, which explains the effects on pain or fever of some of them. This molecule present in plants, compared to the industrial synthetic acetylsalicylic acid:

- a) has the same structure and activity
- b) is safer for use in children
- c) has fewer side effects
- d) it's better because it's natural
- e) all of the above

A412

413) The use of medicinal plants:

- a) is less safe in children than the use of drugs manufactured in laboratories
- b) can cause concomitant unwanted effects due to compounds harmful to health
- c) involves the same risk, if any, that the consumption of the isolated drug
- d) it requires as much chemical knowledge about its effects as the isolated drugs
- e) all of the above

A413

A411 $\underline{C} \leftarrow$ A412 $\underline{A} \leftarrow$ A413 $\underline{E} \leftarrow$

414) The effect of medicinal plants on the organism:

- a) is due to the presence of molecules that interact chemically with cellular components
- b) can interfere with allopathic treatment
- c) can undesirablance alloy enhpathic treatment
- d) can be toxic
- e) all of the above

A414

Biochemistry will lift your self confidence. You can lift my spirits with a small donation.



References

- 1. Campbell, M. and Farrell, S. (2009). Biochemistry. 6th ed. Australia: Thomson/Brooks/Cole.
- 2. Chatterjea, M. and Shinde, R. (2012). Textbook of medical biochemistry. New Delhi: Jaypee Brothers Medical Publications (P) Ltd.
- Devlin T. M. (Ed.), Textbook of Biochemistry with Clinical Correlations. 5th Edition. Wiley-Liss, New York. 2002
- Fisher M, Goddu M, Keil F. Searching for explanations: How the Internet inflates estimates of internal knowledge. Journal of Experimental Psychology: General [Internet]. 2015;144(3):674-687. Available from: <u>http://dx.doi.org/10.1037/xge0000070</u>
- Fisher M, Keil F. The illusion of argument justification. Journal of Experimental Psychology: General [Internet]. 2014 [cited 29 March 2017];143(1):425-433. Available from: <u>https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3735824/</u>
- 6. Ganz T. Hepcidin and iron regulation, 10 years later. Blood [Internet]. 2011 [cited 16 October 2017];117(17):4425-4433. Available from: <u>https://dx.doi.org/10.1182%2Fblood-2011-01-258467</u>

https://link.springer.com/content/pdf/10.3758%2FBF03202442 .pdf

 Harrison-Findik D. Role of alcohol in the regulation of iron metabolism. World Journal of Gastroenterology [Internet]. 2007 [cited 18 April 2017];13(37):4925. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4434614/

- 9. Herrera, E. (2014) Bioquímica Básica. Elsevier España S. L., Barcelona.
- Lieberman M, Peet, A. (2013). Marks' Essential Medical Biochemistry, Second Edition Lippincott Williams & Wilkins
- Li T, Chiang J. Bile acids as metabolic regulators. Current Opinion in Gastroenterology [Internet]. 2015 [cited 15 April 2017];31(2):159-165. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4332523/
- Liu B, Newburg D. Human Milk Glycoproteins Protect Infants Against Human Pathogens. Breastfeeding Medicine [Internet].
 2013 [cited 17 April 2017];8(4):354-362. Available from: <u>https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3725943/</u>
- Mandl J, Mészáros T, Bánhegyi G, Csala M. Minireview: Endoplasmic Reticulum Stress: Control in Protein, Lipid, and Signal Homeostasis. Molecular Endocrinology [Internet]. 2013 [cited 16 November 2017];27(3):384-393. Available from: <u>https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5416932/</u>
- 14. Melnik, B.. Diet in Acne: Further Evidence for the Role of Nutrient Signalling in Acne Pathogenesis – A Commentary. Acta Dermato Venereologica, [online] 92(3), pp.228-231. 2012 [Cited 14 Aug. 2017].Available from: <u>https://www.medicaljournals.se/acta/content/html/10.2340/00</u> 015555-1358
- 15. Miguel V, Sánchez M. La investigación educativa en la Cátedra de Bioquímica de la Escuela "Luis Razetti" y su impacto sobre el diseño instruccional y el rendimiento estudiantil. Docencia Universitaria. 2007; 8(1): 131-146.
- 16. Newsholme, E.A., Leech, T.R. "Functional Biochemistry in Health and Disease". Ed. WileyBlackwell, 2010.
- 17. Rodwell, Victor W.,, et al. Harper's Illustrated Biochemistry. 30th ed. New York, N.Y.: McGraw-Hill Education LLC., 2015.
- 18. Rosenthal, M. Glew R. Medical Biochemistry: Human

Metabolism in Health and Disease. Hoboken, NJ.: John Wiley & Sons, 2009.

19. Shi L, Tu B. Acetyl-CoA and the regulation of metabolism: mechanisms and consequences. Current Opinion in Cell Biology [Internet]. 2015 [cited 7 April 2017];33:125-131. Available from:

http://www.sciencedirect.com/science/article/pii/S0955067415 000125

- 20. Sánchez M, Miguel V. Relación entre los conocimientos previos y el rendimiento en la asignatura Bioquímica en estudiantes de medicina. Revista de la Facultad de Medicina. 2006;29(2):114-120.
- Sánchez, M, Miguel, V, Díaz, K, Vílchez, G, Villasmil, S. López, M. Entorno virtual de enseñanza-aprendizaje para la construcción del conocimiento en Bioquímica médica. Revista de la Facultad de Medicina. 2009;32(1):31-37
- 22. Sánchez, M. Creencias epistemológicas de estudiantes de medicina. Archivos Venezolanos de Farmacología y Terapéutica. 2009; 28(1): 31-35.
- 23. Tarling E, Vallim T, Edwards P. Role of ABC transporters in lipid transport and human disease. Trends in Endocrinology & Metabolism [Internet]. 2013 [cited 22 April 2017];24(7):342-350. Available from:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3659191/

- 24. Unamuno, M. de. Obras completas de Miguel de Unamuno; edición y prólogo de Ricardo Senabre. Madrid: Biblioteca Castro. 2007.
- 25. Yang X, Zhang Y, Wang T, Liu Y. Sporadic Porphyria Cutanea Tarda Induced by Alcohol Abuse. Chinese Medical Journal [Internet]. 2017 [cited 16 November 2017];130(16):2011. Available from:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5555144/