# UMF EXAMENE

# YEAR 1 SEMESTER II BIOCHEMISTRY

1. About the process of fatty acid (FA) degradation it can be said:

    1. The product of beta-oxidation is malonyl-CoA
    2. The rate-limiting step is catalyzed by carnitine-acyl transferase I
    3. FA are activated by phosphorylation in order to be degraded
    4. Beta-oxidation involves 4 reactions in a sequence, of which 2 are reductions
    5. The rate-limiting step is catalyzed by carnitine-acyl transferase II
    6. The transport of FA in the mitochondria requires citrate as a carrier
    7. The immediate product of beta-oxidation is acetyl-CoA
    8. The activation of FA takes place in the cytosol
    9. The transport of FA in the mitochondria requires carnitine as a carrier
    10. Beta-oxidation takes place in the mitochondria and involves 4 reactions in a sequence, of which 2 are dehydrogenations

2. What can be said about the process of fatty acid (FA) activation?

    1. FA are activated with coenzyme A
    2. It takes place in the cytoplasm
    3. It takes place in the mitochondria
    4. FA activation is catalyzed by FA kinases
    5. The reaction of FA activation requires ATP participation, which is cleaved into AMP + PPi
    6. Similarly to glucose, FA are activated by phosphorylation
    7. The reaction of FA activation requires ATP participation, which is cleaved into ADP + Pi
    8. The active form of FA is fatty acyl-phosphate
    9. FA activation is catalyzed by acyl-CoA synthetase
    10. The active form of a FA is fatty acyl-CoA

3. What can be said about the transfer of the fatty acyl (FA) residue through the inner mitochondrial membrane (IMM)?

    1. Inside the mitochondrial matrix, the products of carnitine acyl transferase II (CAT II) are carnitine and acyl-CoA
    2. Outside the IMM, the fatty acyl residue is transferred from coenzyme A to carnitine by carnitine acyl transferase I (CAT I)
    3. Acyl-carnitine represents the form in which the FA is transported through the IMM
    4. Inside the mitochondrial matrix, citrate acyl transferase II transfers the fatty acyl residue from citrate to coenzyme A
    5. The transfer requires citrate as a carrier
    6. The transfer requires carnitine as a carrier
    7. It represents the rate-limiting step of the process of FA degradation
    8. It represents the first stage of FA degradation
    9. Outside the IMM, the fatty acyl residue is transferred from coenzyme A to citrate by citrate acyl transferase I
    10. Acyl-citrate represents the form in which the FA is transported through the IMM

4. About the process of beta-oxidation it can be said:

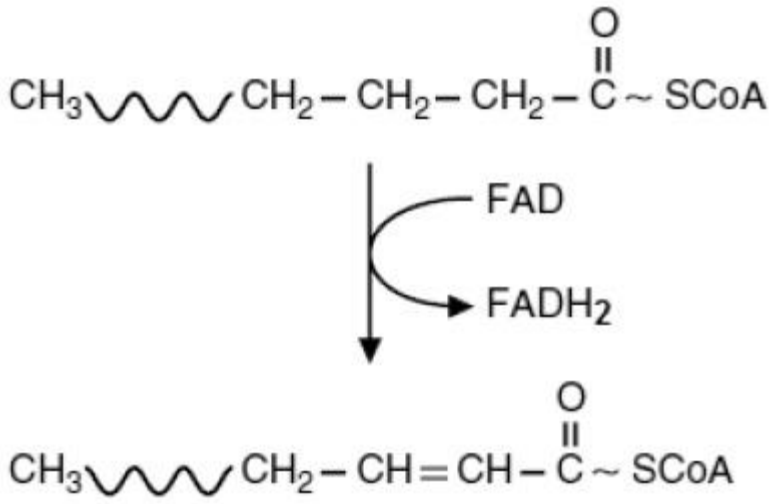
    1. It comprises 4 reactions that are repeated several times
    2. After a sequence of four reactions of beta-oxidation, the first 2 C atoms of the FA are removed as acetyl-CoA
    3. It takes place in the mitochondria
    4. It represents the third stage of fatty acid (FA) degradation
    5. The first dehydrogenation reaction in a sequence of beta-oxidation uses FAD as a coenzyme
    6. The second dehydrogenation reaction in a sequence of beta-oxidation uses NAD+ as a coenzyme
    7. It comprises 3 reactions, of which two are dehydrogenations
    8. It comprises 2 dehydrogenation reactions, the first one with NAD+ and the second one with FAD

9. After a sequence of four reactions of beta-oxidation, the first 2 C atoms of the FA are removed as CO2
10. It takes place in the cytoplasm, because the FA cannot enter the mitochondria

5. About the process of fatty acid (FA) degradation it can be said:

    1. The rate-limiting step of FA degradation is catalyzed by acyl-CoA dehydrogenase
    2. During the process of beta-oxidation, part of the ATP is produced by oxidative phosphorylation and part by substrate-level phosphorylation
    3. FA degradation takes place by beta-oxidation and does not involve the participation of Krebs cycle
    4. All the 3 stages take place in the mitochondria
    5. The 3 stages takes place in the cytoplasm and mitochondria
    6. The acetyl-CoA that results from beta-oxidation is immediately used for the synthesis of other FA
    7. The mitochondrial beta-oxidation is strictly coupled with the respiratory chain
    8. The purpose of FA degradation is to provide ATP
    9. Acetyl-CoA that results from beta-oxidation is further degraded in the Krebs cycle
    10. FA can be degraded only in aerobiosis

6. Regarding the image below it can be stated:



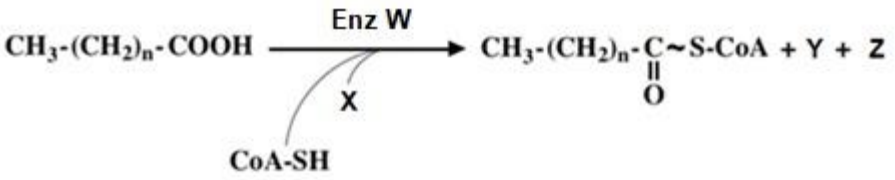

1. It is an oxidation reaction
2. It is a reduction reaction
3. FADH2 will be re-oxidized in the respiratory chain and will provide 2 ATPs by oxidative phosphorylation
4. FADH2 will be re-oxidized in the respiratory chain and will provide 3 ATPs by oxidative phosphorylation
5. It belongs to the process of fatty acid synthesis
6. It is the third of the four reactions of beta-oxidation
7. It is catalyzed by acyl-CoA reductase
8. It belongs to the process of fatty acid beta-oxidation
9. It is catalyzed by acyl-CoA dehydrogenase
10. It is the first of the four reactions of beta-oxidation

7. What can be stated about the regulation of fatty acid degradation?

1. Acyl-CoA dehydrogenase, the regulatory enzyme, is activated by high levels of citrate
2. Carnitine acyl transferase I (CAT-I) is allosterically inhibited by high levels of malonyl-CoA, which derive from fatty acid synthesis
3. The regulatory enzyme is acyl-CoA synthetase
4. Acyl-CoA synthetase, the regulatory enzyme, is inhibited by malonyl-CoA

5. Carnitine acyl transferase I (CAT-I) leads to the formation of acyl-carnitine
6. The rate limiting step is the activation of fatty acid with coenzyme A
7. Carnitine acyl transferase I (CAT-I) is located outside the inner mitochondrial membrane
8. The regulatory enzyme is acyl-CoA dehydrogenase
9. The rate limiting step is the transfer of the fatty acyl residue from the cytosol to the mitochondria
10. The regulatory enzyme is carnitine acyl transferase I (CAT-I)

8. Regarding the reaction presented below it can be stated:



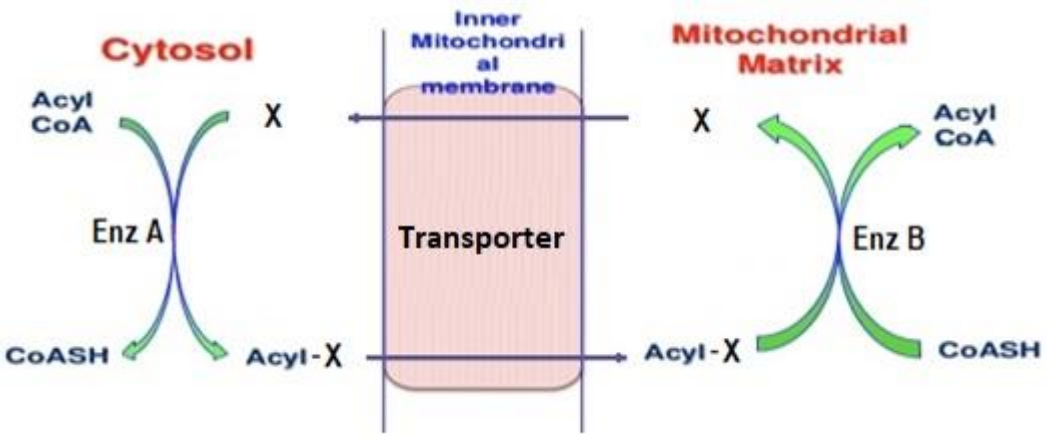

1. Y and Z are ADP and Pi
2. It represents the activation of fatty acids
3. It takes place in the mitochondria
4. Y and Z are AMP and PPi
5. The reaction takes place in the cytoplasm
6. X is ATP
7. It is the rate-limiting step of the process of fatty acid degradation
8. Enzyme W is acyl-CoA synthetase
9. Enzyme W is fatty acid synthase
10. X is GTP

9. About the mitochondrial beta-oxidation of fatty acids it can be stated:

1. Beta-oxidation is usually followed by the degradation of acetyl-CoA in the Krebs cycle
2. FADH2 and NADH formed during beta-oxidation are re-oxidized in the respiratory chain

3. The sequence of four reactions of beta-oxidation are repeated n/2-1 times
4. It comprises 2 dehydrogenation reactions that use the coenzymes FAD and NAD+, respectively
5. Beta-oxidation can take place in anaerobic conditions because oxygen is not a direct participant in the four reactions of the process
6. In the last reaction of beta-oxidation, malonyl-CoA is released
7. Fatty acids are a good source of energy for the brain
8. FAD and NADP+ are used in the dehydrogenation reactions of beta-oxidation
9. To be engaged in beta-oxidation, fatty acids must be previously activated with coenzyme Q
10. In the last reaction of beta-oxidation, the first 2 carbon atoms of the fatty acid are released as acetyl-CoA

10. The following is/are true about the image below:




1. Enzyme A is carnitine acyl transferase I (CAT-I)
2. Enzyme B is carnitine acyl transferase II (CAT-II)
3. Enzyme B is the regulatory enzyme of the process of fatty acid degradation
4. It represents the second stage in the process of fatty acid degradation
5. It represents the first stage in the process of fatty acid degradation

6. X is citrate
7. Enzyme A is the regulatory enzyme of the process of fatty acid degradation
8. X is carnitine
9. Enzyme B is fatty acid synthase
10. Enzyme A is acyl-citrate synthetase

11. What can be stated about the synthesis of ketone bodies (KB)?

1. KB synthesis has an increased rate in starvation and uncontrolled diabetes mellitus
2. KB synthesis is significantly increased after carbohydrate meals
3. The precursor of KB synthesis is acetyl-CoA derived from glucose degradation
4. KB synthesis takes place in the adipose tissue
5. Acetoacetate is obtained from acetoacetyl-CoA by removal of coenzyme A
6. Acetoacetate is formed from β-hydroxy-β-methyl-glutaryl-CoA (HMG-CoA) by removal of acetyl-CoA
7. Acetoacetate can give rise to the other two KB, β-hydroxybutyrate and acetone
8. KB synthesis takes place in the mitochondria of liver cells
9. The precursor of KB synthesis is acetyl-CoA derived from fatty acid oxidation
10. Acetone is obtained by the decarboxylation of β-hydroxybutyrate

12. What can be stated about ketone body (KB) degradation?

1. In order to be degraded, KB must be first activated with coenzyme A and ATP (similarly to fatty acids)
2. KB represent an energy source for several extrahepatic tissues, including the brain
3. The liver cannot use KB because it doesn’t contain the enzyme necessary for their activation
4. KB are degraded in the cytoplasm of cells from various tissues

5. In order to be degraded, KB must be first activated with coenzyme A that is transferred from succinyl-CoA
6. Acetoacetate and β-hydroxybutyrate are degraded to 2 acetyl-CoA molecules, that will be oxidized in the Krebs cycle
7. Out of the three KB, acetoacetate and acetone can be used as an energy source
8. Acetone (one of the three KB) cannot be utilized as an energy source
9. KB represent a good metabolic fuel for the liver
10. The brain cannot obtain energy from KB because it doesn't contain the enzymes necessary for their degradation

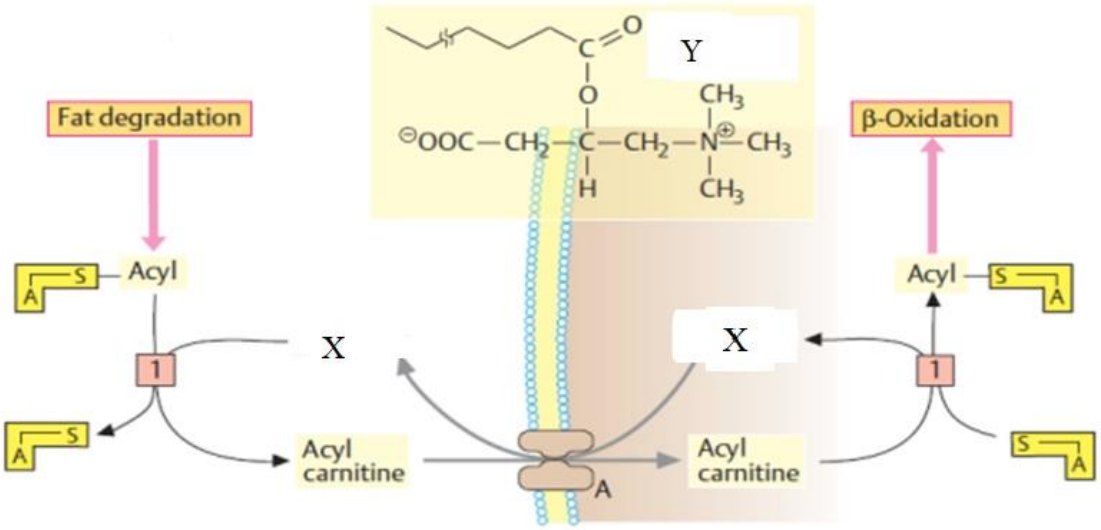
13. Select the correct statement(s) about ketone bodies (KB):

1. Excess KB from the blood are eliminated in the urine, and acetone is partially eliminated through the exhaled air
2. The increased concentration of KB in the blood leads to ketoacidosis
3. KB contribute to the energy metabolism of the liver
4. Ketogenesis is increased following the degradation of triglycerides in the liver, which provides fatty acids for KB synthesis
5. A decreased insulin to glucagon ratio leads to the increase of ketogenesis
6. Increased levels of KB in the blood lead to the increase in blood pH
7. After prolonged fasting, KB have a significant contribution to the energy metabolism of the brain
8. Ketonuria occurs after carbohydrate-reach meals
9. Ketogenesis is increased following the release of high amounts of free fatty acids from the process of lipolysis in the adipose tissue
10. An increased insulin to glucagon ratio leads to an increase of ketogenesis

14. About the process of fatty acid (FA) degradation it can be said:

1. FA degradation, also known as beta-oxidation, occurs in the mitochondria of the cell
2. Fatty acyl-CoA molecules cannot pass through the inner mitochondrial membrane directly, and so must be transported across the membrane as an acyl-carnitine
3. Once inside the mitochondrial matrix, the acyl-CoA molecule undergoes a series of four enzymatic reactions, collectively called the beta-oxidation cycle, which sequentially removes two carbon units from the fatty acid chain in the form of acetyl-CoA
4. The end products of FA degradation are acetyl-CoA, which enters the TCA cycle for energy production, and reduced cofactors (NADH and FADH2), which enter the electron transport chain for ATP synthesis
5. Once inside the mitochondrial matrix, the acyl-CoA molecule undergoes a series of six enzymatic reactions, collectively called the beta-oxidation cycle
6. FA degradation, also known as beta-oxidation, occurs in the cytosol of the cell
7. The first step in FA degradation involves the conversion of the fatty acid to its corresponding acyl-CoA derivative by the action of the enzyme fatty acyl-CoA dehydrogenase
8. The second step in FA degradation is the transport of the acyl-CoA molecule from the cytosol to the mitochondrial matrix by the action of the enzyme carnitine acyltransferase II (CAT-II)
9. The first step in FA degradation involves the conversion of the fatty acid to its corresponding acyl-CoA derivative by the action of the enzyme fatty acyl-CoA synthetase
10. The end products of FA degradation are pyruvate, which enters the TCA cycle for energy production

15. Regarding the following image it can be stated:




1. The acyl-CoA molecule is transported across the inner mitochondrial membrane via a shuttle system that involves the carnitine acyl-transferase enzymes
2. X - represents Acetyl - carnitine
3. Fatty acids are transported to the mitochondria for beta-oxidation by simple diffusion across the inner mitochondrial membrane
4. The image represents the transfer of activated fatty acids from the cytosol to the mitochondria.
5. The activation of fatty acids to their corresponding acyl-CoA derivatives occurs in the mitochondrial matrix by the enzyme fatty acyl-CoA synthetase
6. Y – represents Acetyl- CoA
7. Y – represents Acyl-carnitine
8. The acyl-carnitine molecule is transported across the inner mitochondrial membrane via a carnitine/acylcarnitine translocase
9. The image represents the transfer of activated fatty acids from the mitochondria to the cytosol.
10. X - represents carnitine

16. About the process of beta-oxidation, it can be said:

1. The first step of beta-oxidation is the addition of two carbon atoms to the acyl-CoA molecule in the form of acetyl-CoA

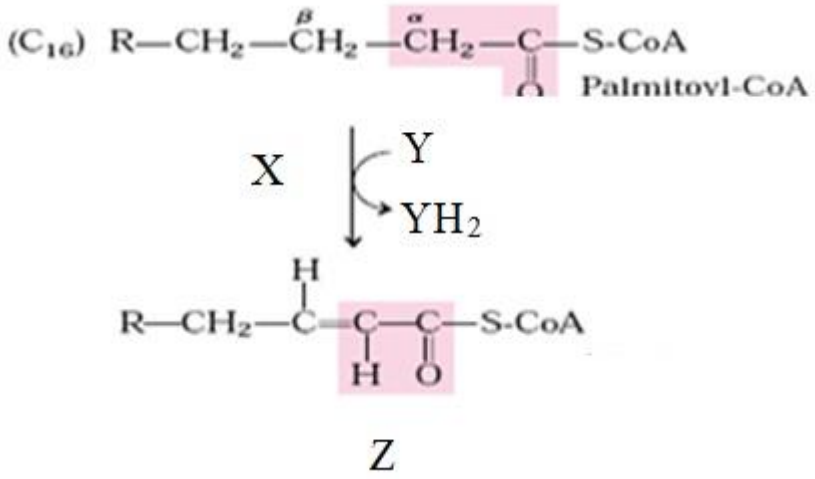
2. This process continues with the removal of successive pairs of carbon atoms until the entire fatty acid molecule is converted to acetyl-CoA
3. Each round of beta-oxidation generates two molecules of ATP, one molecule of FADH2, and one molecule of NADH
4. The first step of beta-oxidation is the removal of two carbon atoms from the acyl-CoA molecule in the form of acetyl-CoA, which enters the TCA cycle for energy production
5. Beta-oxidation takes place in the mitochondrial matrix
6. The final step of beta-oxidation is the conversion of the last acyl-CoA molecule to succinyl-CoA by the enzyme succinyl-CoA synthetase
7. The final step of beta-oxidation is the conversion of the last acyl-CoA molecule to acetyl-CoA by the enzyme thiolase
8. Each round of beta-oxidation generates one molecule of FADH2 and one molecule of NADH, which are used in the electron transport chain to generate ATP
9. Beta-oxidation only occurs with saturated fatty acids and not with unsaturated fatty acids
10. Beta-oxidation takes place in the cytosol

17. About the process of beta-oxidation, it can be said:

1. The main enzyme involved in fatty acid beta-oxidation is fatty acid synthase
2. The acetyl-CoA produced by beta-oxidation enters the citric acid cycle, which produces ATP
3. The activated fatty acid undergoes a series of four reactions that remove two-carbon units as acetyl-CoA molecules
4. Beta-oxidation is a metabolic process that occurs in the cytoplasm of the cell
5. This process requires the input of energy in the form of ATP and NADPH
6. The fatty acid is activated by attaching CoA to it to form acyl-CoA, which is transported into the mitochondria by carnitine

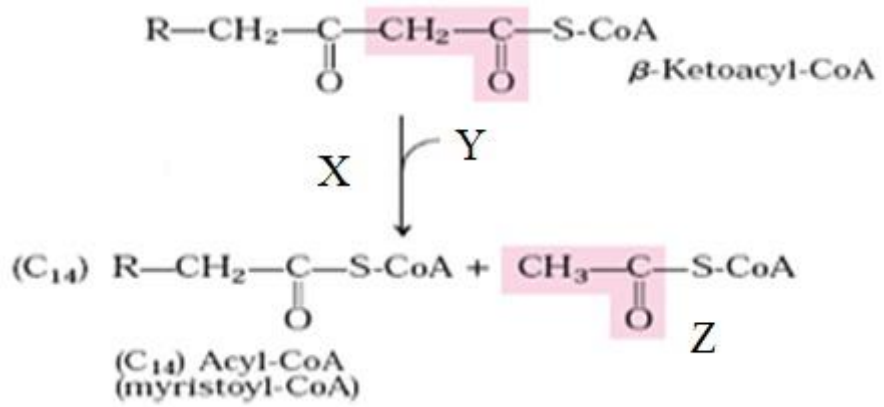
7. Beta-oxidation is a process that involves the breakdown of fatty acids to produce energy
8. During fatty acid beta-oxidation, acetyl-CoA molecules are combined to form a long chain fatty acid molecule
9. The malonyl-CoA molecule is used as a substrate in beta-oxidation
10. Beta-oxidation is a metabolic process that occurs in the mitochondria of cells

18. Regarding the image below it can be stated:




1. Y is FAD
2. The enzyme X is acyl-CoA dehydrogenase
3. Y is NAD
4. Z is trans - Enoyl-CoA
5. The enzyme X is thiolase
6. The picture represents the first reaction in the beta - oxidation of fatty acids
7. The image represents the activation of fatty acids
8. Z is Ketoacyl-CoA
9. The reaction takes place in the mitochondria
10. Z is Acetyl-CoA

19. Regarding the image below it can be stated:




1. Y is CoA-SH
2. Compound Z is acetyl-CoA
3. Compound Y is FAD
4. The enzyme X is acyl-CoA dehydrogenase
5. The reaction is part of fatty acids beta-oxidation
6. It is an oxidation reaction
7. The image represents the last of the four reactions of beta-oxidation
8. The enzyme X is thiolase (acyl-CoA acetyltransferase)
9. The reaction is part of fatty acids biosynthesis
10. Compound Z is acyl-CoA

20. Regarding the fatty acid activation, it can be stated:

1. The process of fatty acid activation occurs in the cytoplasm of the cell
2. Fatty acid activation does not require the input of ATP
3. Once activated, fatty acids can be transported into the mitochondria for beta-oxidation
4. This reaction is catalyzed by the enzyme fatty acyl-CoA synthetase
5. The attachment of the fatty acid to CoA requires the input of two high-energy phosphate bonds from ATP, which is hydrolyzed to AMP and pyrophosphate in the process
6. Fatty acid activation in the cytoplasm requires biotin and FAD

7. Fatty acid activation occurs in the mitochondria
8. Fatty acid activation involves the attachment of the fatty acid to a molecule of coenzyme A (CoA)
9. The end product of fatty acid activation process is acetyl-CoA
10. Activated fatty acids can only be used for energy production

21. About regulation of fatty acids degradation, we can state:

1. Insulin stimulates the fatty acids degradation process
2. Malonyl-CoA inhibits CAT I (carnitine-acyl-transferase I) preventing the entry of activated acid into mitochondria
3. Genetic deficiency of CAT I (carnitine acyl transferase I) does not affect the fatty acids degradation
4. The regulatory enzyme is thiolase
5. Propionyl-CoA inhibits CAT I (carnitine-acyl-transferase I) preventing the entry of activated acid into mitochondria
6. The genetic deficiency of CAT I (carnitine-acyl-transferase I) affects the liver, causing the liver to decline to synthesize glucose during a post-period (severe hypoglycemia, coma)
7. High levels of ATP, NADH, and acetyl-CoA can inhibit the activity of enzymes involved in beta-oxidation
8. Hormones such as glucagon, epinephrine, stimulate the breakdown of fatty acids, while insulin inhibits it
9. The rate-limiting step in the fatty acids degradation is catalyzed by CAT I (carnitine-acyl-transferase I)
10. Hormones such as glucagon, epinephrine, inhibit the breakdown of fatty acids

22. About the synthesis of ketone bodies (KB), it can be stated:

1. Ketone bodies are synthesized in the muscles and transported to the liver for storage
2. Ketone body production can be elevated in conditions such as uncontrolled diabetes, leading to the development of diabetic ketoacidosis (DKA)

3. Ketone bodies are synthesized from acetyl-CoA in the mitochondrial matrix of liver cells
4. Ketone bodies are synthesized from acetyl-CoA molecules that are generated from the breakdown of fatty acids in the liver
5. The production of ketone bodies increases in response to low insulin levels and high glucagon levels, which promote the breakdown of stored fat
6. Ketone bodies are not transported to the brain for use as an energy source
7. The main ketone body produced in the liver is acetyl-carnitine
8. Ketone bodies can only be used as an energy source in the liver
9. The three ketone bodies synthesized by the liver are acetoacetate, beta-hydroxybutyrate, and acetone
10. The synthesis of ketone bodies occurs mainly during periods of high carbohydrate intake

23. About the ketone bodies (KB), it can be stated:

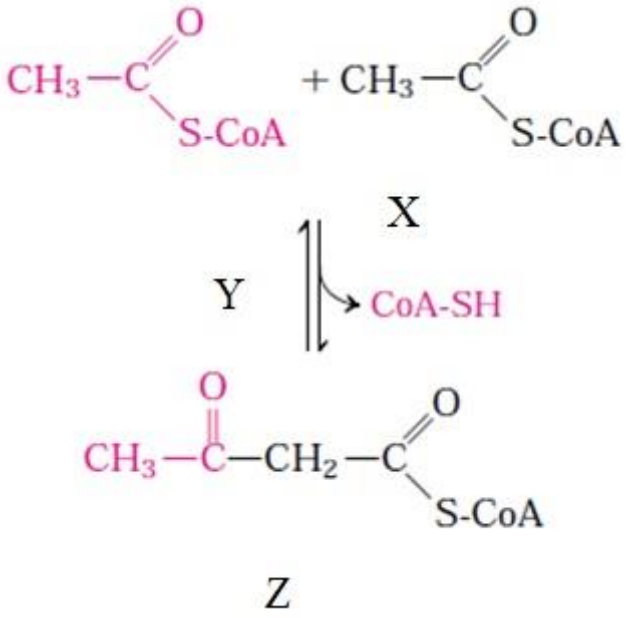
1. When insulin levels are low, such as during periods of fasting, the liver increases its production of ketone bodies
2. Ketone bodies are transported from the liver to other tissues, such as the brain, heart, and skeletal muscles, where they are used as an energy source
3. The liver cannot use ketone bodies, because it lacks the enzyme necessary for their activation (the transferase)
4. The three ketone bodies produced in the liver are acetyl-CoA, malonyl-CoA, and citrate
5. Ketone bodies can only be used as an energy source by the liver and cannot be utilized by other tissues in the body
6. Ketone bodies are synthesized in muscle tissue through a process called ketogenesis
7. The ketone bodies degradation involves previous activation of acetoacetate with coenzyme A, which is transferred from succinyl-CoA under the action of a transferase
8. The degradation of ketone bodies yields acetyl-CoA molecules, which enter the TCA cycle for energy production

9. Ketone bodies are transported from the liver to the adipose tissue, where they are stored for later use as an energy source
10. The synthesis of ketone bodies occurs mainly during periods of high carbohydrate intake, when the body needs an alternative source of energy to glucose

24. Select the correct statement(s) about ketone bodies (KBs):

1. KBs are produced in the liver from acetyl-CoA, which is generated during the breakdown of fatty acids
2. KBs are primarily used as a source of energy by the liver
3. KBs are used as a source of energy during periods of high-carbohydrate diets
4. A high-fat, low-carbohydrate diet can decrease the production of ketone bodies, as well as prolonged fasting
5. KBs are acidic and can cause metabolic acidosis if their levels in the blood become too high
6. Elevated levels of KBs in the blood can occur in conditions such as diabetic ketoacidosis (DKA) or during prolonged fasting
7. KBs can cross the blood-brain barrier and provide energy to the brain when glucose availability is low, such as during fasting
8. The production of KBs is regulated by the hormones insulin and glucagon
9. KBs are produced in the brain through the process of ketogenesis
10. KBs are stored in adipose tissue

25. The following is/are true about the image below:




1. Enzyme Y is thiolase
2. The image represents the first step in the triglyceride's synthesis
3. The process can take place only in the mitochondria
4. Enzyme Y is HMG-CoA synthase
5. It represents the first reaction in the synthesis of ketone bodies (ketogenesis)
6. Z - is acetoacetyl-CoA
7. Z - is acetoacetate
8. The process can take place both in the cytosol and in the mitochondria
9. X - is acetoacetyl-CoA
10. X - is Acetyl-CoA

26. Which of the following statements about fatty acid (FA) synthesis is/are correct?

1. FA synthesis is activated by a low insulin/glucagon ratio
2. During FA synthesis, the intermediates are bound to ACP (acyl carrier protein), which is part of FA synthase complex
3. Citrate allosterically activates acetyl-CoA carboxylase
4. During FA synthesis, the intermediates are bound to coenzyme A

5. FA are synthesized from excess glucose in the diet
6. High levels of citrate in the cytosol inhibit FA synthesis
7. FA synthesis is activated by a high insulin/glucagon ratio
8. It comprises dehydrogenation reactions
9. Malonyl-CoA formed during FA synthesis inhibits the process of FA degradation
10. The C atoms are provided by acetyl-CoA derived from FA beta-oxidation

27. What can be said about the process of fatty acid (FA) synthesis?

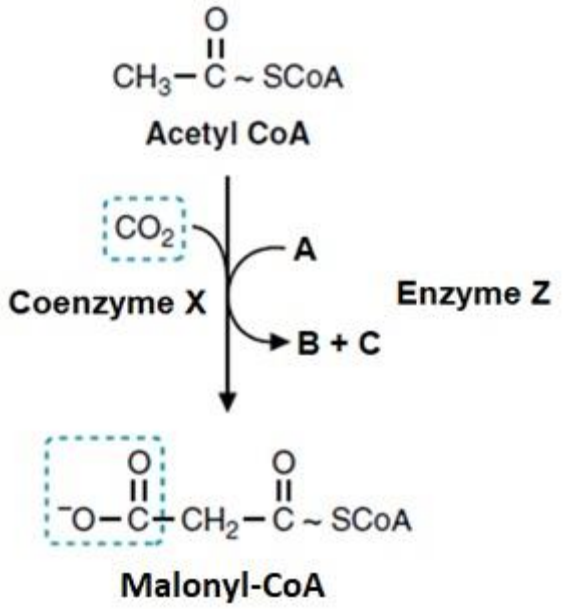
1. Fatty acid synthase is an enzyme complex located in the cytoplasm that specifically synthesizes palmitic acid
2. It takes place in the mitochondria
3. The acetyl residue is transferred from the mitochondria to the cytoplasm incorporated into citrate
4. The fatty acid synthase complex can produce any saturated FA having more than 14 C atoms
5. It requires biotin as a coenzyme for the FA synthase complex
6. It comprises reduction reactions that use NADPH as a H donor
7. It uses acetyl-CoA resulted from glucose degradation
8. The acetyl residue (the source of C atoms for FA synthesis) is transferred through the inner mitochondrial membrane by carnitine
9. It comprises reduction reactions that use NADH as a H donor
10. An important step in FA synthesis is the carboxylation of acetyl-CoA to malonyl-CoA

28. Choose the correct answer(s) about fatty acid synthase:

1. It is a multienzyme complex containing 4 enzymes
2. It uses both acetyl-CoA and malonyl-CoA as substrates
3. It is subject to allosteric regulation
4. It produces stearic acid (18 C)
5. It contains two reductases that use NADPH as a H donor
6. It is located in the mitochondria
7. It is located in the cytoplasm

8. It produces palmitic acid
9. It is a multienzyme complex composed of seven enzymes and a molecule of ACP (acyl carrier protein)
10. It contains two dehydrogenases that use NAD+ and FAD as coenzymes

29. Regarding the reaction presented in the figure, the following is/are true:




1. Coenzyme X is TPP (thiamine pyrophosphate)
2. It takes place in the mitochondria
3. Enzyme Z is named malonyl-CoA synthase
4. Enzyme Z is allosterically inhibited by citrate
5. Coenzyme X is biotin
6. A, B and C are ATP, ADP and Pi
7. It is part of the process of fatty acid synthesis
8. It represents the rate-limiting step of the process of fatty acid synthesis
9. A, B and C are ATP, AMP and PPi
10. Enzyme Z is named acetyl-CoA carboxylase

30. The following can be said about the rate-limiting step and the regulatory enzyme of the process of fatty acid synthesis:

    1. Citrate is an allosteric activator of acetyl-CoA carboxylase
    2. The rate-limiting step is represented by the carboxylation of acetyl-CoA to malonyl-CoA
    3. The regulatory enzyme is activated when the level of cytoplasmic citrate is low
    4. Insulin activates the regulatory enzyme by promoting its dephosphorylated form
    5. Acetyl-CoA carboxylase is inhibited when it is phosphorylated by a protein kinase
    6. The regulatory enzyme is covalently regulated and the active form is the phosphorylated one
    7. The regulatory enzyme is activated by long chain fatty acyl-CoA
    8. The rate-limiting step is catalyzed by fatty acid synthase
    9. Long chain fatty acyl-CoA are negative modulators of the regulatory enzyme
    10. Insulin activates the regulatory enzyme by promoting its phosphorylated form

31. Select the correct answer(s) about the process of fatty acid synthesis:

    1. It does not consume ATP
    2. It is active after carbohydrate meals
    3. It uses NADH (formed in glycolysis) for the reductive steps
    4. It is an ATP-consuming process
    5. It takes place mainly in the muscle
    6. The fatty acid synthase complex requires one molecule of acetyl-CoA and seven molecules of malonyl-CoA for the synthesis of palmitic acid
    7. It is active during fasting
    8. The reductive steps use NADPH formed mainly in the pentose-phosphate pathway
    9. It takes place mainly in the liver
    10. The rate-limiting step is catalyzed by fatty acid synthase

32. Select the correct answer(s) about unsaturated fatty acids:

    1. Linoleic acid (C18:2, Δ9,12) and linolenic acid (C18:3, Δ9,12,15) are essential fatty acids
    2. Arachidonic acid may be obtained from membrane phospholipids under the action of phospholipase C
    3. Arachidonic acid is a mono-unsaturated fatty acid
    4. Arachidonic acid may be obtained from membrane phospholipids under the action of phospholipase A2
    5. Humans can synthesize mono-unsaturated fatty acids containing a double bond between C9-C10
    6. Arachidonic acid (C20:4, Δ5,8,11,14) is a poly-unsaturated fatty acid that can be synthesized from linoleic acid (C18:2, Δ9,12)
    7. Arachidonic acid is the precursor of eicosanoids (prostaglandins, thromboxanes and leukotrienes)
    8. Humans synthesize mono-unsaturated fatty acids containing a double bond at C12
    9. Arachidonic acid can be synthesized by the fatty acid synthase complex
    10. Linoleic acid (C18:2, Δ9,12) can be synthesized in the human body from stearic acid (C18) by introducing two double bonds

33. Select the corrects statement(s) about fatty acids (FA):

    1. The precursor for FA synthesis is acetyl-CoA derived from excess glucose in the diet
    2. Fatty acids can be synthesized in the body, mainly in the liver
    3. FA are synthesized in the liver during fasting periods
    4. FA synthesis does not consume ATP
    5. The precursor for FA synthesis is acetyl-CoA derived from FA beta-oxidation
    6. FA synthesis in the liver is inhibited by citrate
    7. During fasting, the liver receives FA from the degradation of TGs in the adipose tissue, and these FA activated with CoA inhibit the endogenous synthesis of FA in liver cells

8. After synthesis, FA may be used for the synthesis of triglycerides or membrane lipids
9. FA produced by the liver are released in the blood, where they circulate bound to albumin
10. FA synthase produces palmitic acid, which can be elongated and desaturated to give rise to other FA

34. The acetyl-CoA is produced in the mitochondria and must be transported into the cytosol for the synthesis of fatty acids. Which of the following is true regarding the transport of Acetyl CoA?

    1. Acetyl-CoA cannot cross the inner mitochondrial membrane - so to reach the cytoplasm, it combines with oxaloacetate to form citrate
    2. Acetyl-CoA is transported by its specific transporter protein
    3. Acetyl-CoA is transferred into the cytosol in the form of acyl-carnitine
    4. Acetyl-CoA is converted to carnitine, enters the cytosol, and acetyl-CoA is regenerated
    5. Acetyl-CoA is converted into pyruvate, enters into the cytosol and acetyl-CoA is regenerated
    6. In the cytoplasm, an enzyme called citrate lyase, cleaves citrate back into acetyl-CoA and oxaloacetate
    7. Acetyl-CoA diffuses through the inner mitochondrial membrane
    8. Acetyl-CoA is converted into citrate, enters into the cytosol and acetyl-CoA is regenerated
    9. Acetyl-CoA transfer from mitochondria to cytosol is done through citrate
    10. The citrate carrier (citrate transporter) is involved in this process

35. Which of the following are required for the synthesis of fatty acids?

    1. Acyl-CoA
    2. Carnitine
    3. Carbon dioxide ($CO_2$)
    4. Palmitate

5. Acetyl-CoA
6. Oxygen
7. ATP
8. Malonyl-CoA
9. FAD
10. NADPH

36. Which of the following enzymes are involved in the synthesis of fatty acids?

    1. Malonyl-CoA decarboxylase
    2. Ketoacyl reductase
    3. Enoyl reductase
    4. Malonyl-CoA synthase
    5. Acetyl-CoA carboxylase
    6. Carnitine-acyl-transferase II (CAT-II)
    7. Acyl-CoA dehydrogenase
    8. Thioesterase
    9. Carnitine acyl transferase I (CAT I)
    10. Fatty acid synthase

37. Regarding fatty acid synthesis, we can state the following:

    1. Fatty acid synthesis is catalyzed by a multienzyme complex called fatty acid synthase
    2. Fatty acid synthase uses NADH as a reducing agent to add two carbon units to the growing fatty acid chain
    3. Fatty acid synthesis occurs in the mitochondria of the cell
    4. Fatty acid synthesis occurs in the cytoplasm of cells
    5. The starting molecule for fatty acid synthesis is pyruvate, which is derived from the breakdown of glucose
    6. The starting molecule for fatty acid synthesis is acetyl-CoA
    7. Fatty acid synthesis involves a series of enzymatic reactions that are catalyzed by the enzyme fatty acid oxidase
    8. The final product of fatty acid synthesis is linoleic acid, an 18-carbon saturated fatty acid

9. Fatty acid synthase uses NADPH as a reducing agent in two reactions of fatty acid synthase complex
10. The final product of fatty acid synthesis is palmitic acid, a 16-carbon saturated fatty acid

38. What is the role of insulin in the regulation of fatty acid synthesis?

1. It activates hormone-sensitive lipase
2. It activates AMP-activated protein kinase (AMPK)
3. It promotes the synthesis of fatty acids
4. It inhibits acetyl-CoA carboxylase
5. It stimulates a phosphatase that removes the phosphate from acetyl-CoA carboxylase
6. It increases the level of malonyl-CoA
7. It stimulates beta-oxidation of fatty acids
8. It activates acetyl-CoA carboxylase
9. Insulin promotes the dephosphorylated form of the key enzyme of fatty acid synthesis
10. It inhibits fatty acid synthase

39. Acetyl-CoA carboxylase (ACC) is a key enzyme involved in fatty acid synthesis in the body. About this enzyme we can state:

1. Insulin stimulates acetyl-CoA carboxylase activity by dephosphorylating and activating it
2. Glucagon stimulates acetyl-CoA carboxylase activity by phosphorylating and activating it
3. One of the primary regulators of acetyl-CoA carboxylase activity is carnitine
4. Citrate is an allosteric activator for acetyl-CoA carboxylase
5. Dietary intake of excess calories increases the synthesis of acetyl-CoA carboxylase
6. It catalyzes the conversion of malonyl-CoA to acetyl-CoA
7. High levels of citrate inhibit acetyl-CoA carboxylase activity
8. Insulin inhibits acetyl-CoA carboxylase activity
9. It requires biotin, ATP and CO2

10. It catalyzes the conversion of acetyl-CoA to malonyl-CoA, which is a key step in the biosynthesis of fatty acids

40. Regarding acetyl-CoA carboxylase (ACC), we can state the following:

    1. It requires biotin and AMP
    2. ACC is regulated by several mechanisms, including allosteric regulation, covalent regulation and induction/repression
    3. The allosteric regulation of ACC involves the binding of citrate, which activates the enzyme, and palmitoyl-CoA, which inhibits the enzyme
    4. The allosteric regulation of ACC involves the binding of palmitoyl-CoA, which activates the enzyme, and citrate, which inhibits the enzyme
    5. ACC is not regulated by any mechanisms, and its activity remains constant in all physiological conditions
    6. ACC is a biotin-dependent enzyme that catalyzes the carboxylation of acetyl-CoA to form malonyl-CoA
    7. ACC is activated by insulin
    8. ACC is an enzyme that catalyzes the decarboxylation of acetyl-CoA to form malonyl-CoA
    9. ACC is a key enzyme involved in fatty acid degradation in the body
    10. The covalent regulation of ACC involves the phosphorylation of the enzyme by AMP-activated protein kinase (AMPK), which inactivates the enzyme

41. Regarding the biosynthesis of unsaturated fatty acids, we can state:

    1. For the synthesis of other fatty acids, palmitic acid undergoes elongation and desaturation processes
    2. Palmitic acid is a poly-unsaturated fatty acid
    3. Once palmitic acid is formed, it can be further modified to produce unsaturated fatty acids
    4. Unsaturated fatty acids can only be obtained from the diet and cannot be synthesized de novo by the body

5. Carboxylase is the enzyme responsible for introducing double bonds into fatty acids
6. Palmitic acid is an essential fatty acid
7. The most common site of unsaturation in fatty acids is at the C-9 position, where Δ9 desaturase introduces a double bond between C-9 and C-10
8. The first step in the biosynthesis of unsaturated fatty acids is the desaturation of a saturated fatty acid by a desaturase enzyme
9. Linoleic acid and linolenic acid are essential fatty acids
10. Linolenic acid is a mono-unsaturated fatty acid

42. Regarding arachidonic acid, we can state the following:

1. Arachidonic acid is a 16-carbon saturated fatty acid
2. The dietary intake of essential fatty acids, such as linoleic acid, is necessary for the endogenous biosynthesis of arachidonic acid
3. The dietary intake of essential fatty acids is not necessary for the endogenous biosynthesis of arachidonic acid
4. Arachidonic acid-derived eicosanoids are associated with a variety of physiological processes, including inflammation and blood clotting
5. The release of arachidonic acid from cell membrane phospholipids is catalyzed by the action of phospholipase A2
6. The release of arachidonic acid from cell membrane phospholipids is catalyzed by the action of lipoprotein lipase
7. Arachidonic acid is a precursor for the biosynthesis of eicosanoids, including prostaglandins, thromboxanes, and leukotrienes
8. Arachidonic acid is not involved in the biosynthesis of eicosanoids
9. Arachidonic acid is a polyunsaturated fatty acid with 20 carbon atoms
10. Arachidonic acid is not present in the cell membrane phospholipids

43. Regarding the biosynthesis of eicosanoids, we can state the following:

1. Eicosanoids are derived from arachidonic acid, a 20-carbon poly-unsaturated fatty acid
2. Eicosanoids are not involved in physiological processes such as inflammation and blood clotting
3. Arachidonic acid is released from cell membrane phospholipids by the action of phospholipase A2
4. Eicosanoids are synthesized from a polyunsaturated fatty acid - C20:4, Δ5,8,11,14
5. Inhibition of eicosanoid biosynthesis by drugs, such as nonsteroidal anti-inflammatory drugs (Aspirin - acetylsalicylic acid), is a common strategy for the treatment of inflammatory conditions
6. The conversion of arachidonic acid to prostaglandins is catalyzed by Acetyl-CoA carboxylase
7. The biosynthesis of eicosanoids does not involve any enzymatic reactions
8. Eicosanoids are derived from palmitic acid, a 16-carbon poly-unsaturated fatty acid
9. The conversion of arachidonic acid to prostaglandins is catalyzed by cyclooxygenase (COX) enzyme
10. The release of arachidonic acid from cell membrane phospholipids is catalyzed by the action of acyl-transferase enzymes

44. What can be stated about triglycerides (TGs)?

1. TGs circulate in a free form in the blood plasma
2. The pathway of TG synthesis contains phosphatidic acid as an intermediate
3. TGs are composed of glycerol and 3 fatty acids
4. TGs belong to the category of glycerophospholipids
5. TGs are synthesized mainly in the muscle tissue and deposited here as an energy reservoir
6. TG stored in the adipose tissue represent an important energy reserve
7. TGs are synthesized mainly in the liver and adipose tissue
8. TGs are components of cell membranes

9. TGs are obtained from the reaction between glycerol and 3 fatty acids
10. TGs are hydrophobic compounds

45. Select the correct answer(s) about the process of triglyceride (TG) synthesis:

    1. It takes place mainly in the adipose and hepatic cells
    2. Fatty acids are transported to the site of TG synthesis by ACP (acyl carrier protein)
    3. It is active in the fasting periods
    4. Glycerol-phosphate may be obtained from an intermediate of glycolysis
    5. Glycerol phosphate may be obtained through glycerol phosphorylation both in the liver and adipose tissue
    6. Glycerol-phosphate may be obtained from glycerol in the liver, but not in the adipose tissue
    7. For participating in TG synthesis, glycerol must be first phosphorylated
    8. For participating in TG synthesis, fatty acids must be activated with coenzyme A
    9. After synthesis, TGs are incorporated in the plasma membrane of cells
    10. It is activated by glucagon

46. What can be said about triglycerides (TGs)?

    1. The TGs produced by the adipose tissue are stored for future use
    2. The TGs produced by adipose cells are released into the blood incorporated into VLDL
    3. TG synthesis in the adipose tissue utilize glycerol-phosphate obtained from glycerol under the action of glycerol kinase
    4. TGs are produced to various degrees in all the tissues of the human body
    5. The TGs produced by hepatic cells are normally stored in these cells

6. The TGs produced by the liver are released in the blood incorporated in VLDL
7. In both the liver and adipose tissue, the pathway of TG synthesis contains phosphatidic acid as an intermediate
8. Insulin stimulates the TG synthesis
9. The process of TG synthesis is active after carbohydrate meals
10. Insulin decreases the process of TG synthesis

47. What can you say about the process of triglyceride (TG) degradation in the adipose tissue?

1. The release of each of the 3 fatty acids from a TG molecule is catalyzed by the same lipase
2. The fatty acids that result from lipolysis are used as an energy source by the adipose cells
3. The glycerol molecule that is released by lipolysis will be used by the liver in gluconeogenesis
4. The glycerol that results from lipolysis will be reused by the adipose tissue for subsequent TG synthesis
5. The majority of fatty acids resulted from lipolysis are released into the blood and will be used as an energy source by several tissues
6. It is active after meals
7. TG lipase is not influenced by hormones
8. The rate-limiting step is catalyzed by TG lipase, also called hormone-sensitive lipase
9. Fatty acids released by lipolysis are transported in the blood by chylomicrons
10. It is active during fasting

48. Select the correct statement(s) about triglyceride lipase:

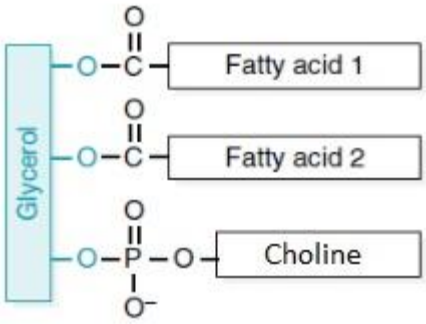
1. It is covalently regulated
2. Insulin inactivates it by dephosphorylation catalyzed by a phosphatase
3. It is covalently regulated and the active form is the dephosphorylated one

4. Its active form is the phosphorylated one
5. It catalyzes the rate-limiting step in the process of triglyceride synthesis
6. It is regulated by allosteric mechanism
7. It catalyzes the rate-limiting step in the process of lipolysis
8. Its phosphorylated form is inactive and is promoted by insulin
9. Glucagon promotes its phosphorylation by means of protein kinase A
10. It is inhibited by glucagon and epinephrine

49. Choose the true statement(s) about glycerol-3-phosphate:

1. It is a hydrophobic compound
2. It is used for triglyceride synthesis
3. It is released from the process of lipolysis in the adipose tissue
4. In the adipose tissue it can be formed from glycerol by the action of glycerol kinase
5. It can be formed from DHAP (dihydroxyacetone phosphate, an intermediate of glycolysis) by a reduction reaction
6. In the liver it can be obtained from glycerol by an ATP-dependent phosphorylation
7. It is the precursor of phosphatidic acid
8. It cannot be used for triglyceride (TG) synthesis because TGs do not contain phosphate
9. It is an intermediate in glycerophospholipid synthesis
10. The liver cannot obtain it from glycerol because it doesn’t contain glycerol kinase

50. What can be stated about the compound in the image?

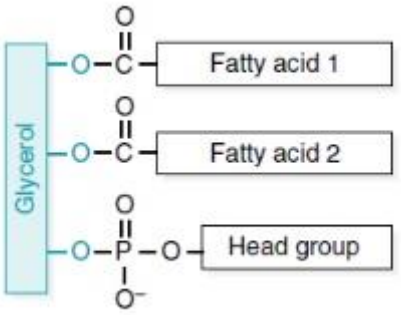



1. It is degraded by triglyceride lipase
2. Its synthesis pathway contains phosphatidic acid as an intermediate
3. Diacylglycerol is not an intermediate in its synthesis pathway
4. It is sphingomyelin
5. It is found in cell membranes
6. Phospho-choline is donated by CDP-choline during the synthesis of this compound
7. It is stored in the cytosol of cells from various tissues
8. It is phosphatidyl choline
9. It may be obtained from triglycerides by the replacement of the third fatty acid with phospho-choline
10. It can be obtained from phosphatidyl-ethanolamine by 3 methylations

51. Which of the following compounds can be found in cell membranes?

1. Sphingolipids
2. Esterified cholesterol
3. Diacylglycerol
4. Monoglycerides
5. Glycolipids
6. Tripalmitoyl-glycerol
7. Free cholesterol
8. Phosphatidyl-ethanolamine
9. Triglycerides
10. Phosphatidyl-choline

52. What can be stated about the degradation of phospholipids (PL), whose general structure is presented in the image below?




1. It is catalyzed by enzymes from the lyases class
2. There are four types of phospholipases: A, B, C, and D
3. The action of phospholipase C on phosphatidyl choline generates diacylglycerol and phosphocholine
4. The action of phospholipase C on phosphatidyl choline generates phosphatidic acid and choline
5. It is catalyzed by phospholipases
6. The action of phospholipase C on phosphatidyl inositol generates diacylglycerol and inositol phosphate
7. Phospholipase A2 removes the fatty acid at the first position of the glycerol residue in PL
8. It is catalyzed by hormone-sensitive lipase
9. Phospholipase A2 removes the fatty acid at the second position of the glycerol residue in PL
10. Four types of phospholipases are involved in PL degradation: A1, A2, C, and D

53. Similarities and differences between phosphatidyl choline (PC) and sphingomyelin (SM):

1. Both are amphipathic compounds
2. PC can be transformed into SM
3. The alcohol in PC is glycerol, while in SM it is sphingosine

4. In both PC and SM, the attachment of one or two fatty acids to the alcohol leads to the formation of a ceramide
5. Both contain phopsho-choline
6. Both PC and SM are found deposited in the cytoplasm of cells
7. PC contains 2 fatty acids, while SM contains 1 fatty acid
8. PC is a triglyceride, SM is a membrane lipid
9. Only PC contain phospho-choline, SM does not contain phosphate because choline is directly linked to sphingosine
10. Both can be found in cell membrane

54. Which of the following participate in the synthesis of phosphatidyl choline?

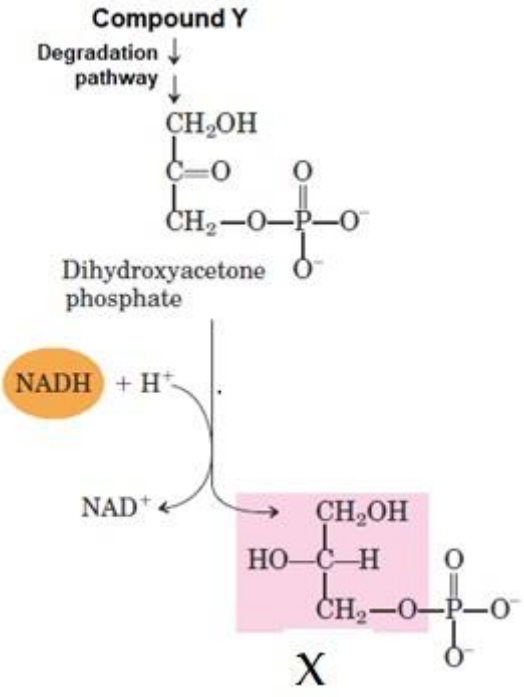
1. Diacylglycerol
2. Two fatty acids activated with ACP (acyl carrier protein)
3. Inositol
4. Monoacylglycerol
5. ADP-choline
6. The alcohol sphingosine
7. Two fatty acids activated with CoA-SH: one saturated and one unsaturated
8. Glycerol phosphate
9. CDP-choline
10. Phosphatidic acid

55. What can be stated about triglycerides (TGs)?

1. They are the main storage form of fat in the body and can be found in adipose tissue
2. TGs are an important source of energy for the body
3. The final step in TG synthesis involves the addition of a phosphate group to a glycerol molecule
4. TG synthesis occurs mainly in muscle tissue
5. TG synthesis occurs in the mitochondria of cells
6. TGs are present in circulating lipoproteins such as chylomicrons and VLDL

7. Excess dietary intake of TGs can lead to obesity and other metabolic disorders
8. The first step in TG synthesis is the conversion of glucose to glycerol
9. TGs are a type of lipids that are composed of a glycerol backbone and three fatty acid chains
10. The glycerol backbone for TG synthesis is derived from fatty acids

56. Regarding the following image it can be stated:



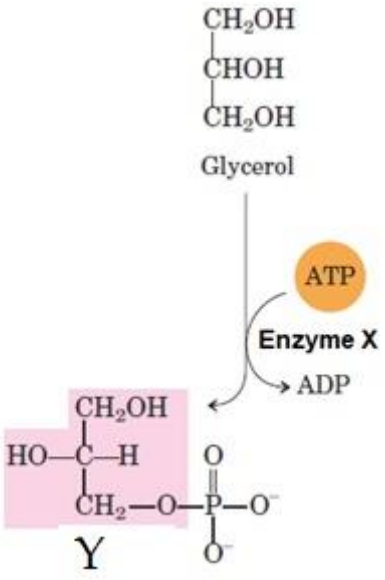

1. Glycerol phosphate dehydrogenase converts dihydroxyacetone phosphate (DHAP) to glycerol-3-phosphate, which can then be used for TG synthesis
2. Y is named triglyceride
3. Compound X is named phosphatidic acid
4. Glycerol kinase converts dihydroxyacetone phosphate (DHAP) to glycerol-3-phosphate in adipose tissue
5. Compound X is named glycerol-3-phosphate
6. The reaction it is catalyzed by glycerol kinase

7. It is the only pathway by which the liver can obtain glycerol-3-phosphate
8. The reaction is catalyzed by glycerol phosphate dehydrogenase
9. It is the initial step of TG synthesis in adipose tissue
10. Y is named glucose

57. What can be said about triglycerides (TGs)?

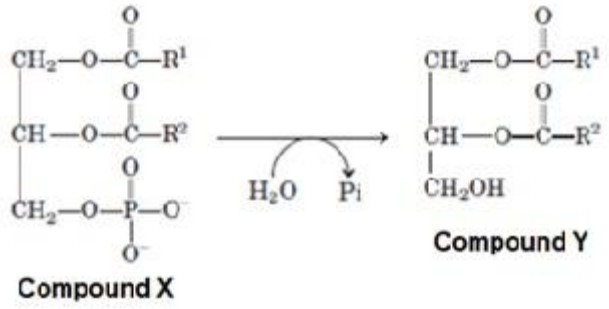
1. TG synthesis does not involve the esterification of glycerol-3-P with fatty acids
2. The TG synthesis pathway in the adipose tissue does not involve phosphatidic acid as an intermediate
3. Elevated levels of TGs in the blood are known as hypertriglyceridemia
4. The initial step of TG synthesis is the conversion of glycerol-3-phosphate to glucose
5. TGs are a type of steroid molecule that are synthesized in the liver from cholesterol
6. TGs can be broken down into fatty acids and glycerol through a process called lipolysis
7. The fatty acids used for TG synthesis are always obtained from dietary sources
8. Lipolysis in the adipose tissue is stimulated by hormones like glucagon and adrenaline
9. TGs are synthesized in the liver and intestine, as well as in adipose tissue
10. Lipolysis in the adipose tissue is inhibited by insulin

58. Regarding the following image it can be stated:




1. It is the initial step of TG synthesis in the adipose tissue
2. X is an enzyme that is absent from the adipose tissue
3. Enzyme X convert dihydroxyacetone phosphate (DHAP) to glycerol-3-phosphate in adipose tissue
4. Enzyme X is named pyruvate kinase
5. The reaction is catalyzed by glycerol phosphate dehydrogenase
6. Y is involved in triglyceride synthesis
7. It is the initial step of TG synthesis in the liver
8. Compound Y is named dihydroxyacetone phosphate
9. Enzyme X is named glycerol kinase
10. Compound Y is named glycerol-3-phosphate

59. Regarding the following image it can be stated:

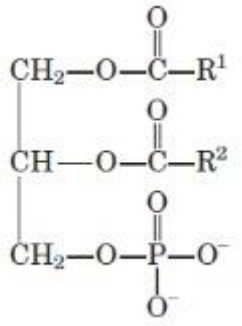


1. The reaction is catalyzed by glycerol-3-phosphate dehydrogenase
2. Compound X is named glycerol-3-phosphate
3. It represents the conversion of glycerol 3-phosphate to phosphatidic acid
4. Compound Y is named triacylglycerol
5. Compound Y is named diacylglycerol
6. Compounds X and Y are precursors for the synthesis of triacylglycerols (TAGs)
7. The reaction is catalyzed by phosphatidic acid phosphatase
8. Compound X is named phosphatidic acid
9. Compound X is a key intermediate in the biosynthesis of phosphatidylcholine, phosphatidylethanolamine, and phosphatidylserine
10. The enzyme that catalyzes the reaction requires pyridoxal phosphate (PLP) as a coenzyme

60. What can be said about the lipolysis process?

1. Lipolysis is stimulated by insulin
2. Fatty acids released by lipolysis are transported in the blood bound to albumin
3. Hormone-sensitive lipase (HSL) is an enzyme that plays a key role in the regulation of lipolysis
4. Insulin inhibits lipolysis in adipose tissue by increasing the activity of protein phosphatase
5. Lipoprotein lipase is an enzyme involved in the hydrolysis of triglycerides (TGs) stored in adipose tissue
6. Hormone-sensitive lipase (HSL) is activated by glucagon and epinephrine
7. The hormone-sensitive lipase is activated by protein kinase A-mediated phosphorylation
8. Hormone-sensitive lipase (HSL) is inhibited by glucagon
9. Hormone-sensitive lipase (HSL) hydrolyzes triacylglycerols (TAGs) into glycerol and activated fatty acids (acyl-CoA)
10. Lipolysis is the breakdown of triglycerides into glycerol 3-phosphate and free fatty acids

61. What can be stated about the compound in the image?



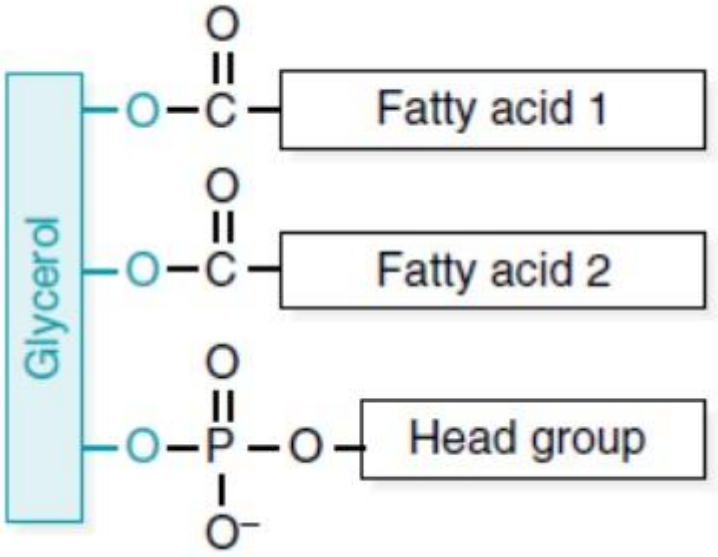
1. Can be converted to diacylglycerol
2. It is phosphatidic acid structure
3. It is synthesized just in the liver
4. It is synthesized by the acylation of glycerol-3-phosphate by acyltransferases
5. It is named diacylglycerol
6. It is triacylglycerol
7. Represents phosphatidyl choline structure
8. It is formed by the action of the enzyme phospholipase D on phosphatidylcholine or phosphatidylethanolamine
9. It is the common intermediate in the formation of lecithin (phosphatidyl choline) and triacylglycerols
10. It is sphingomyelin

62. Membrane lipids include the following compounds:

1. Glycerophospholipids
2. Glycolipids
3. Taurine
4. Glycine
5. Sphingophospholipids
6. Triglycerides
7. Diacylglycerol
8. Sphingolipids
9. Phospholipids

10. Monoacylglycerol

63. What can be stated about the structure presented in the image below?




1. If the head group is choline, the compound it is named phosphatidyl-choline (lecithin)
2. It is the structure of sphingomyelin
3. Phospholipase C (PLC) cleaves the bond between the glycerol backbone and the phospho-head group
4. If the head group is choline, the compound it is named phosphatidyl-ethanolamine
5. It presents a long chain fatty acid connected through an amide linkage with the amino group of sphingosine
6. It is the structure of glycerophospholipid
7. Two fatty acids are attached through ester bonds to the first and second position of glycerol
8. Glycerophospholipids contain a saturated fatty acid at C-1 and an unsaturated fatty acid at C-2
9. Head group can be a fatty acid
10. It is the structure of glycolipid

64. Regarding the synthesis of phosphatidyl-ethanolamine, phosphatidyl-choline (lecithin) and phosphatidyl-serine, we can state:

    1. Phosphatidyl-ethanolamine, phosphatidyl-choline (lecithin), and phosphatidyl-serine are all sphingolipids
    2. The initial steps in the synthesis of glycerophospholipids are similar to those of triacylglycerol synthesis
    3. Glycerol 3-phosphate reacts with fatty acyl-CoA to form phosphatidic acid
    4. The first step in their synthesis is the formation of ceramide
    5. Ceramide is used as a precursor for the synthesis of phosphatidyl-ethanolamine, phosphatidyl-choline (lecithin) and phosphatidyl-serine
    6. Phosphatidyl-serine can be synthesized from CDP-diacylglycerol and glycine
    7. The head group (ethanolamine, choline, serine) is activated with CTP, to form CDP-head group
    8. Phosphatidic acid is cleaved by a phosphatase to form diacylglycerol
    9. DAG reacts with the activated head group
    10. Phosphatidyl-ethanolamine is synthesized from CDP-ethanolamine and choline

65. What can be stated about the degradation of phospholipids?

    1. Phosphatidylserine is degraded by the decarboxylation of phosphatidylserine to phosphatidylinositol
    2. The degradation of phospholipids involves the hydrolysis of the ester bonds between the glycerol backbone and the fatty acid chains or the head groups of the phospholipids
    3. Phospholipids are degraded by a complex process that involves the activation of fatty acids
    4. Phospholipase A1 (PLA1) cleaves the fatty acid at the C-1 position of phosphatidylcholine and phosphatidylethanolamine
    5. The enzymes responsible for the hydrolysis of these bonds are called phospholipases

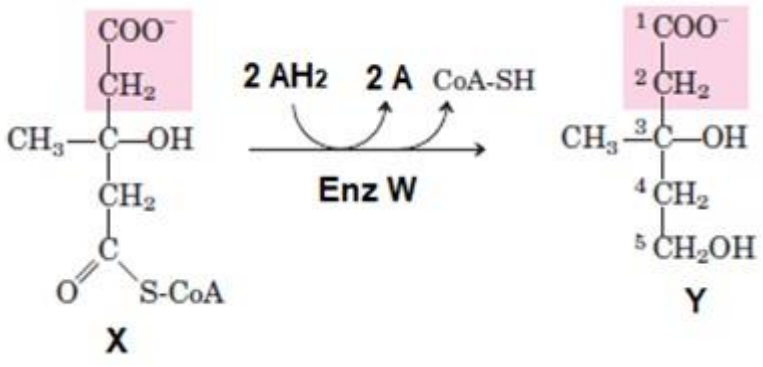
6. Phospholipase D (PLD) is an enzyme involved in phospholipids synthesis
7. Phospholipase C (PLC) cleaves the phosphodiester bond between the glycerol backbone and the phospho-head group of phosphatidylinositol-diphosphate, generating inositol triphosphate (IP3) and diacylglycerol (DAG)
8. Phospholipase A2 (PLA2) cleaves the fatty acid at the C-2 position of phosphatidylcholine and phosphatidylethanolamine
9. The action of phospholipase C on phosphatidyl-inositol forms phosphatidic acid and inositol phosphate
10. The action of phospholipase C on phosphatidyl-inositol leads todiacylglycerol and inositol triphosphate

66. Regarding the metabolism of glycerophospholipids it can be stated:

1. Glycerophospholipids are a class of phospholipids that are important components of cellular membranes
2. Glycerophospholipids play no role in membrane structure and function
3. The only enzyme involved in the degradation of glycerophospholipids is phospholipase A2
4. De novo synthesis of glycerophospholipids involves the condensation of glycerol-3-phosphate and two acyl-CoA molecules to form phosphatidic acid (PA)
5. Glycerophospholipids are synthesized exclusively in the liver
6. Glycerophospholipids consist of a glycerol backbone that is esterified with two fatty acid chains and a phosphate group, which is usually linked to another polar head group such as choline, ethanolamine, or serine
7. The synthesis of glycerophospholipids occurs exclusively through the CDP-choline pathway
8. One important enzyme involved in the metabolism of glycerophospholipids is phospholipase A2 (PLA2), which hydrolyzes a fatty acid chain from the glycerol backbone
9. Phospholipase D (PLD) is involved in the synthesis of phosphatidic acid (PA)

10. The cleavage of glycerophospholipids by phospholipase C always results in the generation of diacylglycerol and inositol phosphates

67. The following reaction represents the rat-limiting step in the pathway of cholesterol synthesis. What can be said about it?




1. W is covalently regulated, the active form being the phosphorylated one
2. X is aceto-acetyl-CoA
3. Y is mevalonic acid (mevalonate)
4. X is HMG-CoA (β-hydroxy- β methyl- glutaryl-CoA)
5. A is NAD+
6. W is HMG-CoA (β-hydroxy- β methyl- glutaryl-CoA) reductase
7. A is NADP+
8. High levels of cholesterol inside cells decrease the amount of enzyme W
9. Y is HMG-CoA (β-hydroxy- β methyl- glutaryl-CoA)
10. W is HMG-CoA synthase

68. Choose the correct statement(s) about cholesterol:

1. Esterified cholesterol is found in cell membranes
2. The regulatory enzyme of cholesterol synthesis pathway is allosterically inhibited by cholesterol
3. Cholesterol is synthesized in the mitochondria of cells
4. Malonyl-CoA is an intermediate in the pathway of cholesterol synthesis

5. Free cholesterol is an amphipathic lipid and is a component of cell membranes
6. Cholesterol is the precursor of steroid hormones
7. Cholesterol synthesis takes place in the cytosol
8. The process of cholesterol synthesis does not consume ATP
9. The precursor for cholesterol synthesis is acetyl-CoA
10. Cholesterol can be synthesized in the body, mainly in the liver

69. About the pathway of cholesterol synthesis it can be stated:

1. Cholesterol produced by the liver may be delivered to other tissues after incorporation into chylomicron lipoproteins
2. It takes place in the majority of tissues, but mainly in the liver
3. The first steps of the pathway are common to those involved in ketone body synthesis, which however takes place in a different cell compartment
4. It requires ATP consumption
5. It takes place mostly in the adipose tissue
6. It requires NADPH for reduction reactions
7. It consumes GTP as a source of energy
8. Squalene is an intermediate in cholesterol synthesis
9. The precursor for cholesterol synthesis in the liver is acetoacetate
10. It requires NADH for reduction reactions

70. The following is(are) correct about the regulatory (key) enzyme of the process of cholesterol synthesis:

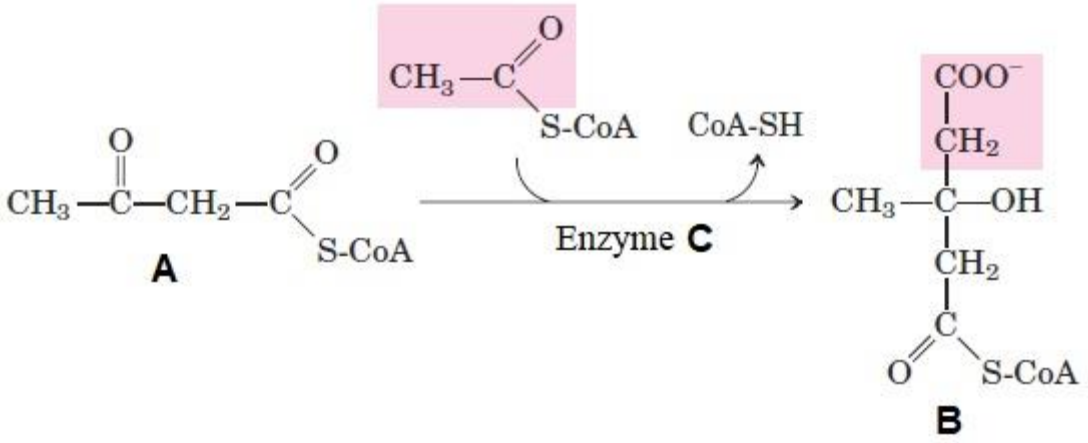
1. The enzyme is activated when it is phosphorylated by a protein kinase
2. The key enzyme is allosterically inhibited by cholesterol
3. It catalyzes the conversion of HMG-CoA (β-hydroxy- β methyl-glutaryl-CoA) to mevalonate
4. It catalyzes the conversion of aceto-acetyl-CoA to HMG-CoA (β-hydroxy- β methyl- glutaryl-CoA)
5. It is called HMG-CoA (β-hydroxy- β methyl- glutaryl-CoA) synthase

6. It uses NADPH (formed in the pentose-phosphate pathway) as a reducing agent
7. It is called HMG-CoA (β-hydroxy- β methyl- glutaryl-CoA) reductase
8. The activity of the key enzyme is regulated in a covalent manner and the active form is the dephosphorylated one
9. It uses FADH2 as a coenzyme
10. Cholesterol inhibits its own synthesis by causing a decrease in the synthesis of the key enzyme

71. Which of the following compounds can be found as intermediates in the pathway of cholesterol synthesis?

    1. Acetoacetyl-CoA
    2. Glutaryl-CoA
    3. Lanosterol
    4. Phosphatidic acid
    5. Palmitic acid
    6. Choline
    7. HMG-CoA (β-hydroxy- β methyl- glutaryl-CoA)
    8. Acetoacetate
    9. Mevalonic acid
    10. Squalene

72. What can be said about the reaction in the image?




1. In the cholesterol synthesis pathway, compound B will be reduced to mevalonate with 2 NADPH molecules
2. Enzyme C is HMG-CoA reductase
3. It is part of cholesterol synthesis and also of fatty acid synthesis
4. Enzyme C is the rate limiting step in the process of cholesterol synthesis
5. To be ultimately converted to cholesterol, compound B be undergoes the action of HMG-CoA lyase
6. Compound A may be give rise to acetoacetate by removal of CoA-SH
7. It is part of the cholesterol synthesis pathway
8. It is part or the ketone body synthesis pathway
9. Enzyme C is HMG-CoA synthase
10. Compound A is acetoacetyl-CoA and compound B is HMG-CoA (β-hydroxy- β methyl- glutaryl-CoA)

73. In the synthesis of cholesterol in the liver:

1. Sqalene is a non-cyclic compound formed from the condensation of 6 activated isoprene units
2. Acetyl-CoA is used as a precursor
3. Part of the synthesized cholesterol is sent to other tissues as part of HDL lipoproteins
4. The formation of mevalonate requires 2 NADPH molecules

5. Acetoacetate can be used as a precursor
6. ATP is required for the formation of the activated isoprene units
7. ATP is used for the activation of squalene
8. HMG-CoA reductase is allosterically inhibited by cholesterol
9. The HMG-CoA is reduced with 2 NADH molecules
10. HMG-CoA reductase can be inactivated by phosphorylation catalyzed by AMP-activated protein kinase

74. Which statement(s) about the biosynthesis and the metabolism of cholesterol is/are correct?

1. Cholesterol is degraded to acetyl-CoA
2. The main route for eliminating excess cholesterol from the body is in the form of bile acids
3. HMG-CoA reductase can be activated bt dephosphorylation catalyzed by a phosphatase
4. Cholesterol can be esterified with a long-chain fatty acid and stored in this form in the cytoplasm
5. The rate-limiting step in the synthesis of cholesterol is the reduction of HMG-CoA to mevalonate
6. GTP is necessary for the synthesis of activated isoprene units from mevalonate
7. For the synthesis of activated isoprenes from mevalonate, ATP is consumed
8. HMG synthase is the regulatory enzyme in the process of cholesterol synthesis
9. The synthesis of HMG-CoA reductase is decreased by high levels of intracellular cholesterol
10. Cholesterol can be esterified with acetyl-CoA and stored in this form in the cell

75. What can be stated about bile acids?

1. Primary bile acids are conjugated with glycine or taurine
2. Bile acids decrease the formation of additional bile acids by inhibiting 7-alpha-hydroxylase

3. Bile acids are excreted mainly by the kidney
4. Conjugated bile acids are secreted into the small intestine as components of bile
5. They are formed in the liver from cholesterol
6. The first reaction in the pathway of bile acids is the hydroxylation of cholesterol at position 7
7. Glycocholic acid is a bile acid conjugated with glucose
8. Conjugation of bile acids takes place in the small intestine
9. Bile acids are degradation products of bilirubin
10. Bile acids stimulate 7-alpha-hydroxylase

76. Choose the correct statement(s) about bile acids:

1. Cholic acid is a primary bile acid
2. Conjugated bile acids are reabsorbed from the intestine, and transported to the liver
3. Primary bile acids are conjugated with glucuronic acid
4. The first step in bile acid synthesis is the introduction of a hydroxyl group at C-3 of cholesterol
5. Secondary bile acids are formed by the deconjugation of primary bile acids and the loss of the hydroxyl group at C-7
6. Bile acids are degraded in the intestine to urobilinogen
7. Bile acids are hydrophobic compounds
8. 7-alpha-hydroxylase catalyzes the first step in the process of bile acid synthesis
9. Bile salts have an important role in the digestion and absorption of dietary lipids
10. Only a small proportion (<5%) of the bile acids undergo entero-hepatic circulation

77. About bile acids it can be stated:

1. 95% of the bile acids entering the gut undergo entero-hepatic circulation, that allows their reutilization
2. The difference between primary and secondary bile acids it the absence of the -OH group at C-3 in the secondary bile acids

3. They are formed from cholesterol
4. Compared to primary bile acids, secondary bile acids (formed in the intestine) do not contain a hydroxyl group at C-7
5. 7-alpha-hydroxylase catalyzes the rate-limiting step an is inhibited by cholic acid
6. They are formed from fatty acids, in the bile
7. Bile acids inhibit HMG-CoA reductase, the key enzyme of the pathway that produces them
8. All bile acids that reach the intestine are eliminated in the feces
9. Taurocholic acid is a secondary bile acid
10. Bile acids are amphipathic compounds that facilitate the digestion and absorption of dietary lipids

78. About the biosynthesis of cholesterol it can be stated:

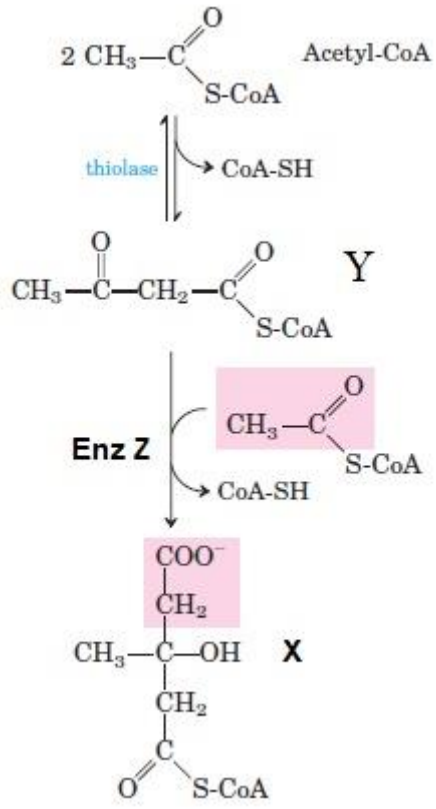
1. The first cyclic compound in the pathway is squalene
2. It takes place with ATP generation
3. Occurs in mitochondria
4. The first cyclic compound in the pathway is lanosterol
5. It is achieved with ATP consumption
6. Involves the participation of NADPH
7. Occurs in the cytoplasm
8. The precursor is acetyl-CoA
9. Involves the participation of NADH
10. The precursor is malonyl-CoA

79. What can be stated about cholesterol?

1. It is a precursor for the synthesis of important molecules such as steroid hormones, bile acids, and vitamin D
2. High levels of LDL cholesterol are associated with an increased risk of atherosclerosis and cardiovascular disease
3. Cholesterol is involved in the production of hormones of the adrenal medulla
4. HDL cholesterol is known as "bad cholesterol"

5. The rate-limiting step in cholesterol synthesis is the conversion of lanosterol to cholesterol
6. Cholesterol is a type of lipid that is essential for the structure and function of cell membranes
7. Cholesterol is mainly synthesized in the liver, but can also be obtained from dietary sources
8. It is transported in the blood by lipoproteins, mainly LDL and HDL
9. Cholesterol is an essential nutrient that can be obtained just from the diet
10. LDL cholesterol is known as "good cholesterol"

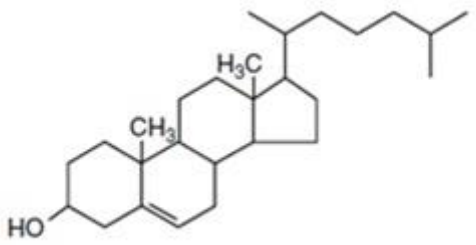
80. Select the correct answer(s) about the process in the picture:




1. The reaction catalyzed by enzyme Z is the rate limiting step in cholesterol synthesis
2. Enzyme Z is HMG-CoA synthase
3. Compound X is named mevalonic acid

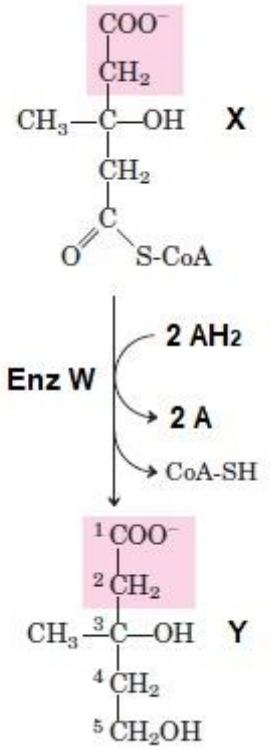
4. Three acetyl-CoA molecules condense in two steps to form HMG-CoA
5. Enzyme Z is HMG-CoA reductase
6. The image represents the triglyceride synthesis
7. Compound Y is named acetoacetyl-CoA
8. The process can take place both in the cytosol and in the mitochondria
9. Compound Y is reduced to mevalonate with NADPH
10. Compound X is named HMG-CoA

81. Regarding the image below it can be stated:




1. Esterification of this compound in the cells is catalyzed by the enzyme acyl-CoA: cholesterol acyltransferase (ACAT)
2. It is named cholic acid
3. It is cholesterol structure
4. It is cortisol structure
5. Its synthesis is activated by glucagon
6. It is a precursor of vitamin D
7. It is cholesterol ester
8. It is a precursor for the synthesis of steroid hormones
9. It is a lipid molecule with a complex structure that contains four rings of carbon atoms known as a steroid structure
10. Represents a conjugated bile acid

82. What can you say about the following image?




1. Compound X is named HMG-CoA
2. Enzyme W is HMG-CoA reductase
3. It is part of the cholesterol synthesis process
4. Compound X is named acetoacetyl-CoA
5. Enzyme W is HMG-CoA synthase
6. It is the rate-limiting step in the biosynthesis of cholesterol
7. Compound A is NAD+
8. Compound A is FAD
9. Compound Y is mevalonate
10. Compound Y is HMG-CoA

83. Select the correct statement(s) about cholesterol synthesis:

1. Lecithin, cholic acid, squalene are intermediates in cholesterol synthesis
2. Squalene is a cyclic compound that arises as an intermediate in cholesterol synthesis

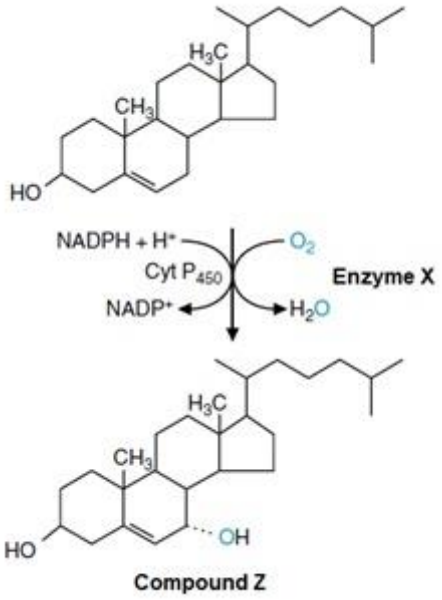
3. The linear squalene is converted into a cyclic structure named lanosterol
4. The rate-limiting step in cholesterol biosynthesis is the reduction of mevalonate to lanosterol
5. The synthesis of cholesterol requires a variety of enzymes and cofactors, including NADPH and ATP
6. HMG-CoA reductase converts HMG-CoA to geranyl pyrophosphate
7. For the synthesis of activated isoprene units, ATP is consumed
8. For the synthesis of activated isoprene units from mevalonate, GTP is consumed
9. Geranyl-pyrophosphate, farnesyl-pyrophosphate, squalene are intermediates in cholesterol synthesis
10. Two molecules of farnesyl-pyrophosphate undergo a head-to-head fusion to form squalene

84. Choose the true statement(s) about regulation of cholesterol biosynthesis:

1. Cholesterol decreases the synthesis of HMG-CoA reductase
2. Familial hypercholesterolemia is a disorder in which a defective LDL receptor is formed, such that it cannot bind the LDL particle
3. The HMG-CoA reductase is activated by ATP-dependent phosphorylation
4. Cholesterol induces the formation of HMG-CoA reductase
5. Specific drugs for lowering hypercholesterolemia competitively inhibit an enzyme which catalyzes the formation of mevalonate within the cholesterol biosynthesis pathway
6. Statins are competitive inhibitors of HMG-CoA reductase
7. HMG-CoA reductase is phosphorylated and inactivated by AMP-activated protein kinase
8. The statin drugs are structural analogs of HMG-CoA → they competitively activate HMG-CoA reductase
9. Glucagon and glucocorticoids increase the expression of HMG-CoA reductase gene

10. Insulin and thyroid hormones decrease the expression (the rate of transcription) of HMG-CoA reductase gene

85. Regarding the image below it can be stated:




1. Represents the first reaction in biosynthesis of bile acids
2. Enzyme X is 7α-hydroxylase
3. Compound Z is named 7-alpha-hydroxycholesterol
4. Enzyme X adds a hydroxyl group to C-7
5. Compound Z is named chenodeoxycholic acid
6. Enzyme X is 7-alpha-cholesterol reductase
7. Represents the first reaction in biosynthesis of cholesterol
8. Cholesterol is converted to 7-alpha-hydroxycholesterol by the enzyme cholesterol 7-alpha-dehidrogenase
9. Compound Z is intermediate in cholic acid synthesis
10. Conjugated bile acids are deconjugated in the small intestine by a pancreatic hydrolase

86. Which of the following can be stated about the bile acids?

1. Primary bile acids are cholic acid and chenodeoxycholic acid
2. Their synthesis takes place in liver cells
3. Cholesterol is the precursor of bile acids
4. Are degraded to bile pigments
5. Primary bile acids are activated with coenzyme A and then conjugated with glycine or taurine
6. Glycocholic acid is a bile acid conjugated with glucose
7. Bile acids are formed from cholesterol in the pancreas
8. 7 α-hydroxylase catalyzes the rate-limiting step
9. Are degradation products of bilirubin
10. Bile acids are formed from cholesterol in the intestine

87. Which of the following can be stated about the cholesterol 7α-hydroxylase?

1. It is inhibited by cholesterol
2. Catalyzes the rate-limiting step in the conversion of cholesterol to bile acids in the liver
3. It is a rate-limiting enzyme in the cholesterol synthesis pathway
4. The enzyme is inhibited by bile acids
5. The enzyme is activated by statin drugs
6. It catalyzes the deamination of cholesterol at the 7α position
7. Cholesterol 7α-hydroxylase is a cytochrome P450 enzyme
8. The enzyme is activated by cholesterol
9. It converts cholesterol to 7α-hydroxycholesterol
10. It catalyzes the conversion of 7α-hydroxycholesterol to cholesterol

88. Which of the following compounds are bile acids/bile salts?

1. Cholesterol
2. Taurocholic acid
3. Chenodeoxycholic acid
4. Cholic acid
5. Cysteine
6. Glycocholic acid
7. Malonyl-CoA

8. Glycine
9. Taurine
10. Lithocholic acid

89. Regarding the bile acids it can be stated:

1. Cholic acid has two -OH groups in addition to cholesterol at C-7 and C-12
2. Bile acids through deconjugation and 7-α-dehydroxylation form cholesterol
3. Cholic acid has one -OH group in addition to cholesterol at C-7
4. Chenodeoxycholic acid has two -OH groups in addition to cholesterol at C-7 and C-12
5. Lithocholic acid is synthesized in the liver
6. Lithocholic acid is a secondary bile acid
7. Primary bile acids are cholic acid and chenodeoxycholic acid
8. Chenodeoxycholic acid has -OH group in addition to cholesterol at C-7
9. Primary bile acids are formed from cholesterol in the intestine
10. In contrast to secondary bile acids, primary bile acids are hydroxylated at carbon 7

90. Which of the following can be stated about the bile acids?

1. Bile acids are synthesized from phospholipids in the liver
2. Bile acids are not recycled in the enterohepatic circulation
3. The primary (conjugated) and secondary bile salts are absorbed in the ileum and return to the liver via the portal circulation → this is known as the entero-hepatic circulation of bile salts
4. Intestinal bacteria produce enzymes that act upon conjugated bile acids, causing deconjugation and 7-α-dehydroxylation
5. Bile acids emulsify and solubilize dietary fats
6. Primary bile acids are formed from the oxidation of secondary bile acids
7. Bile acids are synthesized from cholesterol, and their secretion into bile helps to eliminate excess cholesterol from the body

8. Bile acids are converted to stercobilin in the intestine
9. Conjugated bile acids are secreted into the bile, where they are present in an ionized form, forming bile salts
10. Bile acids are important for the digestion and absorption of proteins

91. Select the correct statement(s) about chylomicrons:

1. They are formed in the small intestine and contain dietary lipids
2. They contain apoprotein B-100
3. They transport the dietary cholesterol from the intestine to the extrahepatic tissues
4. They transport dietary cholesterol to the liver
5. They are subjected to the action of LCAT (lecithin cholesterol acyl transferase)
6. They are formed in the liver
7. ApoE on their surface activates lipoprotein lipase
8. They are subjected to the action of lipoprotein lipase in the capillaries of extrahepatic tissues
9. Chylomicron remnants are taken up by the liver through receptors that recognize apoE on their surface
10. They contain apoprotein B-48

92. Choose the correct statement(s) about apolipoproteins:

1. ApoA-I activates lipoprotein lipase
2. ApoB-48 is found in the composition of chylomicrons
3. ApoB-100 is a ligand for the LDL receptor
4. ApoA-I activates LCAT (lecithin cholesterol acyl transferase)
5. ApoB-48 is found on the surface of VLDL
6. They are located in the interior of the plasma lipoprotein particles
7. ApoC-II activates LCAT (lecithin cholesterol acyl transferase)
8. They are located on the surface of plasma lipoproteins
9. ApoC-II activates lipoprotein lipase
10. ApoB-100 is a structural component of chylomicrons

93. What can be said about lipoprotein lipase (LPL)?

    1. It is located in the capillaries of muscle and adipose tissue
    2. It is activated by apoC-II
    3. It is activated by apoB-48 located on the surface of chylomicrons and VLDL
    4. It is located in the capillaries of the liver
    5. It degrades the cholesterol esters from HDL
    6. Its action causes a decrease in the triglyceride content of chylomicrons and VLDL
    7. It degrades the phospholipids from chylomicrons and VLDL
    8. A genetic deficiency of LPL leads to an increased level of triglycerides in plasma
    9. It hydrolyzes the triglycerides from chylomicrons and VLDL
    10. It degrades the triglycerides stored in the adipose cells

94. What can be stated about the VLDL lipoproteins?

    1. After being secreted into the blood, they receive apoB-100 from HDL
    2. They contain apoB-100
    3. The triglycerides in their composition are degraded by the hormone-sensitive lipase
    4. They are rich in cholesterol, which represents more than 50% of their composition
    5. They contain apoB-48
    6. Part of VLDL remnants are taken up by the liver
    7. They are taken up in extrahepatic tissues through the LDL receptor
    8. VLDL, after conversion in IDL, are the precursors of LDL
    9. In the capillaries of some extrahepatic tissues they are subjected to the action of lipoprotein lipase
    10. They are synthesized by the liver

95. Choose the correct statement(s) about the LDL lipoproteins:

1. They transport triglycerides from the liver to the extrahepatic tissues
2. The only apoprotein that they contain is apoB-100
3. They transport cholesterol synthesized by the liver, to the extrahepatic tissues
4. They contain apoB-100, apo C and apoE
5. They are taken up in tissues by the LDL receptor, which recognizes apoE on their surface as a ligand
6. They are taken up in tissues by means of receptor-mediated endocytosis
7. The cholesterol that they contain is esterified by LCAT (lecithin cholesterol acyl transferase)
8. Increased LDL levels in plasma are associated with the development of atherosclerosis
9. They are synthesized in the liver
10. They derive in the plasma from IDL, which derive from VLDL

96. Which of the following events occur(s) after the uptake of LDL particles inside cells?

1. Increase in the number of LDL receptors in the plasma membrane
2. Increased degradation of LDL receptors
3. Activation of ACAT (acyl cholesterol acyl transferase)
4. Decreased synthesis of the LDL receptor
5. Esterification of intracellular cholesterol by LCAT (lecithin cholesterol acyl transferase)
6. Increased esterification of cholesterol, followed by its deposition in the cytosol
7. Increased synthesis of cholesterol
8. Decreased synthesis of HMG-CoA reductase
9. Intracellular deposition of the triglycerides contained in LDL particles
10. Decreased receptor-mediated endocytosis of additional LDL particles

97. Which statement(s) about the HDL lipoproteins is/are correct?

1. They remove excess triglycerides from extrahepatic tissues and transport them to the liver
2. They are subjected the action of lipoprotein lipase
3. They are subjected to the action of LCAT (lecithin cholesterol acyl transferase)
4. An increased HDL level in plasma is associated with an increased risk of atherosclerosis
5. They take up excess cholesterol from the extrahepatic tissues and transport it to the liver
6. ApoC-II on their surface stimulates LCAT (lecithin cholesterol acyl transferase)
7. They contain apolipoproteins A, C, D, E, but do not contain apoB
8. The major apoprotein that they contain is apoB-100
9. They have an important contribution to the decrease of the risk of atherosclerosis
10. They are formed mostly in the liver

98. What can be stated about LCAT (lecithin cholesterol acyl transferase)?

1. It is an enzyme synthesized and secreted by the adipose tissue
2. It causes an increase of the cholesterol ester content in HDL
3. It catalyzes cholesterol esterification with the fatty acid transferred from C-2 of lecithin
4. It is activated by apoB-100
5. It catalyzes the intracellular esterification of cholesterol
6. It catalyzes the transfer of the fatty acid from esterified cholesterol to a lecithin molecule
7. It is activated by apoA-I
8. It is an enzyme produced by the liver and then secreted into the blood
9. It catalyzes the esterification of cholesterol present in HDL
10. It is involved in the metabolism of LDL lipoproteins

99. Select the correct statement(s) about plasma lipoproteins:

1. VLDL deliver the fatty acids synthesized by the liver to the extrahepatic tissues
2. LDL are taken up in tissues by receptor-mediated endocytosis
3. LDL transport the cholesterol synthesized by the liver to the extrahepatic tissues
4. VLDL transport triglycerides from the adipose tissue to other tissues
5. Chylomicrons transport the dietary cholesterol to the extrahepatic tissues
6. Chylomicrons transport the dietary cholesterol to the liver
7. HDL remove excess triglycerides from extrahepatic tissues and transport them to the liver
8. HDL are involved in the elimination of excess cholesterol from extrahepatic tissues, as bile acids in the gut
9. LDL are taken up inside cells by the LDL receptor, which recognizes apoE on the surface of LDL
10. LDL are synthesized by liver cells

100. What can be said about the plasma lipoproteins and their metabolism?

1. The triglycerides present in VLDL are degraded by the hormone-sensitive lipase
2. Chylomicrons are the only type of lipoproteins that contain apoB-48
3. The lipids found in the surface layer of plasma lipoproteins are triglycerides and esterified cholesterol
4. ApoB-100 is the major apoprotein present in chylomicrons
5. The interior of plasma lipoproteins contains cholesterol esters and triglycerides
6. ApoB-100 is the only apoprotein present in LDL
7. Lipoprotein lipase hydrolyzes the triglycerides from VLDL and chylomicrons
8. LDL particles are taken up inside cells by diffusion through the plasma membrane
9. LCAT (lecithin cholesterol acyl transferase) is activated by apoA-I present on the surface of HDL

10. HDL provides the extrahepatic tissues with the necessary amount of cholesterol

101. What can be stated about apolipoprotein B-100?

1. It activates lipoprotein lipase
2. It is present in chylomicrons
3. It is present in VLDL particles
4. It is synthesized by the intestinal cells
5. It is not found in HDL particles
6. It is the ligand for the LDL receptor
7. It is one of the apoproteins present in HDL particles
8. It activates the enzyme that esterifies the cholesterol present in HDL
9. It is present in LDL particles
10. It is synthesized by the liver

102. The following can be said about plasma lipoproteins:

1. They transport dietary lipids and lipids synthesized in the body
2. The proteins, being hydrophobic, are mainly located in the core of the lipoprotein particles
3. They are water-soluble particles
4. Lipids carried by lipoprotein particles derive exclusively from food
5. Esterified cholesterol is located on the surface of lipoprotein particles
6. The surface layer of lipoprotein particles contains phospholipids and free cholesterol
7. Triglycerides are located on the surface of lipoprotein particles
8. Triglycerides are located in the core of lipoprotein particles
9. Esterified cholesterol is located in the core of lipoprotein particles
10. The main role of lipoprotein particles is to transport glycoproteins to tissues

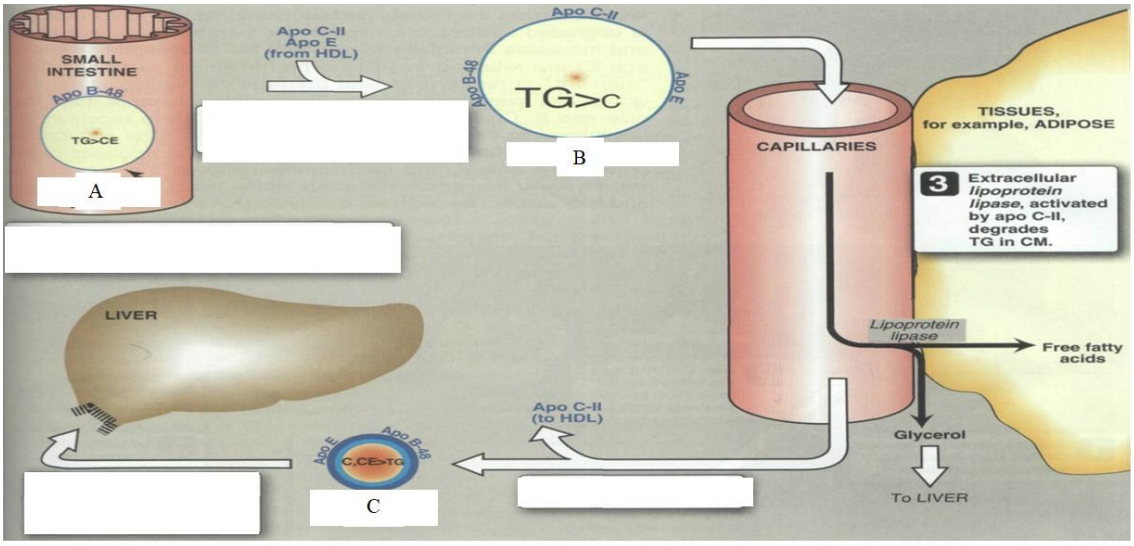
103. Regarding the lipoproteins, it can be stated:

1. The major classes of plasma lipoproteins include chylomicrons, very low-density lipoproteins (VLDL), low-density lipoproteins (LDL), and high-density lipoproteins (HDL)
2. Plasma lipoproteins are complex particles that transport lipids, including cholesterol and triglycerides, throughout the bloodstream
3. Chylomicrons are the smallest lipoproteins
4. Chylomicrons are synthesized in the liver
5. Lipoproteins are complex molecules that transport proteins through the bloodstream
6. Are composed of a combination of lipids (such as cholesterol and triglycerides) and enzymes
7. Chylomicrons are lipoprotein particles that are produced in the intestine and transport dietary triglycerides
8. Lipoproteins are composed of a core of lipids, including cholesterol esters and triglycerides, surrounded by a shell of proteins, phospholipids, and free cholesterol
9. HDL are rich in triglycerides
10. Chylomicrons are the largest lipoproteins

104. Regarding the apolipoproteins it can be stated:

1. ApoB-100 is synthesized in the intestine
2. ApoB-48 is synthesized in the liver
3. Apo A-I activates cholesterol acyl transferase (LCAT)
4. Apolipoprotein B (apoB) is the primary protein component of HDL
5. Apo C-II activates lipoprotein lipase
6. Apo C-II inhibits lipoprotein lipase
7. ApoB-48 is synthesized in the intestine
8. ApoA is the major protein component of low-density lipoprotein (LDL)
9. ApoB-100 binds to LDL receptor
10. ApoB-100 is synthesized in the liver

105. In the following image:




1. C is named chylomicron remnant
2. B is mature chylomicron
3. Chylomicrons transport endogenous triglycerides to peripheral tissues
4. The image represents the metabolism of chylomicrons
5. A is named nascent chylomicron
6. The process represents VLDL metabolism
7. Chylomicrons are composed of triglycerides (exogenous), cholesterol, phospholipids, and apo B-100
8. Chylomicrons are composed of triglycerides (exogenous), cholesterol, phospholipids, and apo B-48
9. B is nascent chylomicron
10. A is named VLDL

106. Regarding the chylomicrons it can be stated:

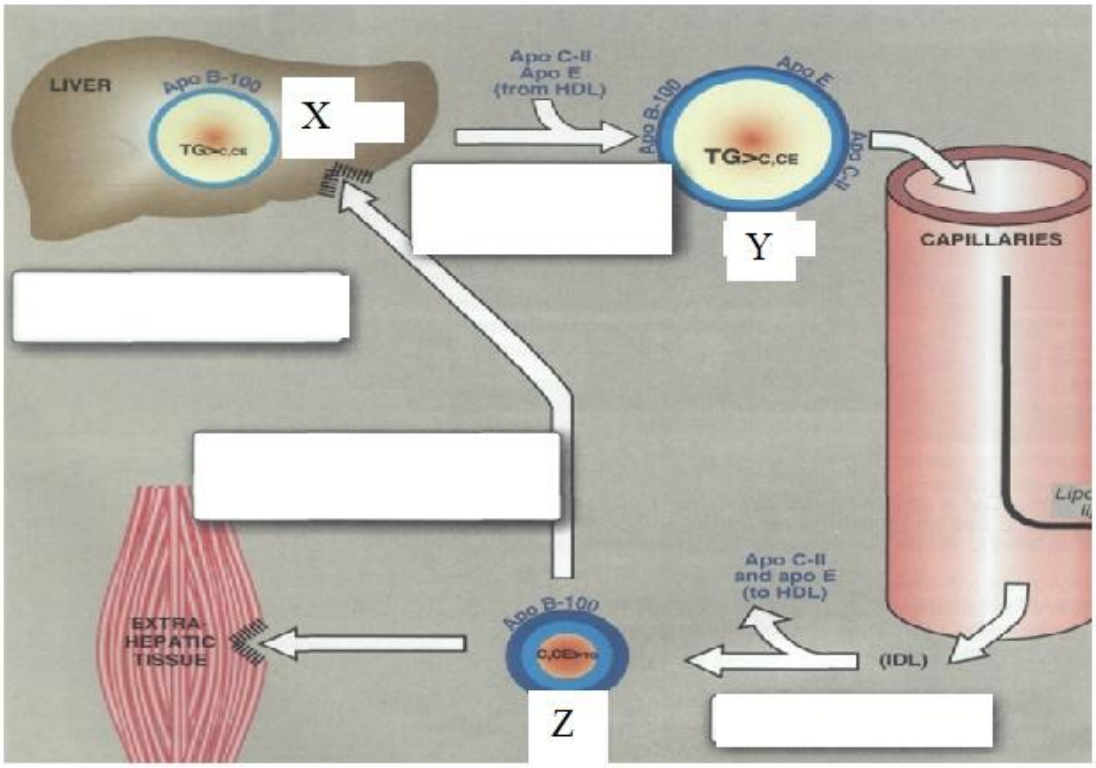
1. Chylomicrons confer a lactescent aspect to the serum or plasma
2. After their triglycerides have been hydrolyzed, chylomicron remnants are taken up by the liver for further processing and metabolism
3. Apo B-100 is required for chylomicron formation
4. Chylomicrons are metabolized to LDL particles
5. Nascent chylomicrons are synthesized in the liver

6. Apo B-48 is unique to chylomicrons and is required for their assembly and secretion from intestinal cells
7. Chylomicrons are called the "bad cholesterol" because they can contribute to the development of atherosclerosis
8. Nascent chylomicrons are synthesized in the intestine
9. Chylomicrons are composed of cholesterol and apo B-100
10. Chylomicrons are the largest lipoprotein particles

107. Which statement about apolipoproteins is true?

1. Apo C is a ligand of the LDL receptor
2. Apolipoprotein A-I is a component of LDL particles
3. Apolipoproteins are necessary for the structural integrity and function of lipoproteins
4. Apo B-100 is the primary protein component of chylomicrons
5. Apo A-I is the main protein component of LDL particles
6. Apo C-II activates the lipoprotein lipase
7. Apo B-48 is a characteristic structure element of chylomicrons
8. Apo B-100 is a ligand of the LDL receptor
9. Apolipoproteins are proteins that bind to lipids to form lipoproteins
10. Apolipoprotein B-100 is synthesized in the liver and is the major protein component of HDL particles

108. Regarding the image below it can be stated:




1. Y is mature VLDL
2. X is nascent VLDL
3. The primary function of VLDL is to transport cholesterol from the liver to extrahepatic tissues
4. Z is LDL
5. Chylomicrons are formed in liver cells
6. Lipoprotein lipase cleaves triacylglycerols of the VLDL
7. The image represents the metabolism of VLDL
8. Y is chylomicron
9. Z is HDL
10. The image represents the metabolism of chylomicrons

109. Which of the following statements about the metabolism of VLDL are correct?

1. VLDL particles are synthesized and secreted by the liver and small intestine

2. The primary function of VLDL is to transport excess cholesterol from peripheral tissues back to the liver
3. In addition to triacylglycerols, nascent VLDL contain some cholesterol and cholesteryl esters, as well as apo B-100
4. Intermediate-density lipoprotein (IDL) can be metabolized into LDL
5. Nascent VLDL receive apo C and apo E from HDL particles and become mature VLDL
6. Activation of lipoprotein lipase by apo C-II causes the release of free fatty acids from the VLDL triacylglycerols
7. VLDL is converted to intermediate-density lipoprotein (IDL) by the action of lipoprotein lipase (LPL)
8. The primary apolipoprotein found on VLDL is apo A-I
9. During the conversion of VLDL to IDL, the core of IDL becomes enriched in triacylglycerols
10. Nascent VLDL contain apo B-48

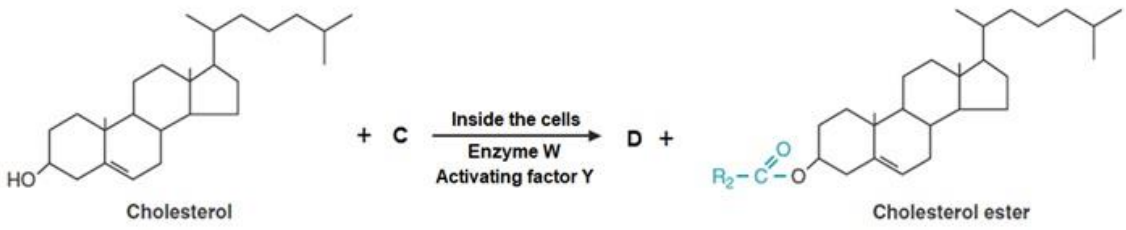
110. About LDL, we can state:

1. In LDL, cholesterol is converted to cholesterol esters by LCAT
2. The only apolipoprotein associated with LDL is apoB-100
3. High levels of LDL in the circulation can contribute to the formation of atherosclerotic plaques in the arterial walls
4. LDL removes cholesterol from cell membranes in the extrahepatic tissues
5. The major apoprotein in LDL is apo B-48
6. The major source of triacylglycerols for human tissues is LDL
7. LDL is formed from the metabolism of VLDL in the circulation
8. LDL is the carrier of free fatty acids to tissues for energy during the fasting state
9. LDL stands for low-density lipoprotein and is often referred to as "bad cholesterol"
10. LDL contains a high proportion of cholesterol esters

111. Which of the following statements about the metabolism of LDL are correct?

1. LDL is taken up by cells in the body through the interaction between the LDL receptor and apoB-48, a protein component of LDL
2. The function of LDL particles is to provide cholesterol to the peripheral tissues
3. LDL contains mostly cholesterol esters and a smaller amount of triglycerides
4. The internalized LDL particles are subsequently degraded in lysosomes, releasing the triglycerides
5. LDL is produced from VLDL after it has released its triglycerides to peripheral tissues
6. The LDL receptor binds with highest affinity to Apo C-II
7. LDL are internalized through receptor-mediated endocytosis
8. LDL and HDL are the major carriers of triacylglycerols in the blood
9. Excess LDL can accumulate in the arterial walls, leading to the formation of atherosclerotic plaques
10. Low density lipoproteins (LDLs) are converted to intermediate density lipoproteins (IDLs)

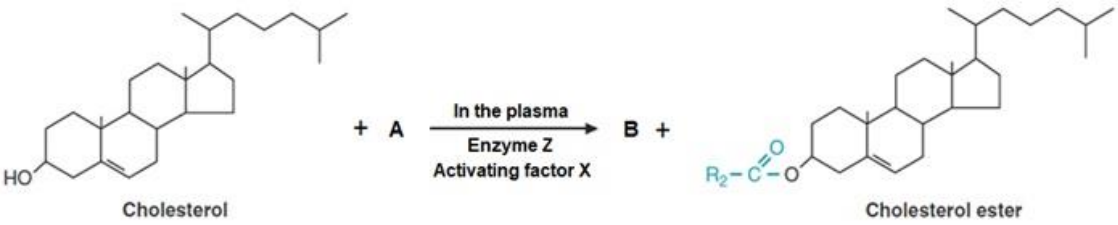
112. Regarding the following image it can be stated:




1. C is phosphatidylcholine (lecithin)
2. Activating factor Y is cholesterol
3. D is lysophosphatidylcholine (lysolecithin)
4. The enzyme W is LCAT (Lecithin-cholesterol acyltransferase)
5. The reaction is involved in the de novo synthesis of cholesterol
6. The enzyme W is ACAT (Acyl-CoA: Cholesterol Acyltransferase)
7. ACAT (acyl-CoA: cholesterol acyltransferase) is an enzyme involved in the metabolism of LDL

8. C is Acyl-CoA
9. D is CoA-SH
10. Product D is Acyl-CoA

113. Regarding the image below it can be stated:




1. Activating factor X is apolipoprotein A-I (apo A-I)
2. A is phosphatidylcholine (lecithin)
3. It represents the cholesterol catabolism
4. Activating factor X is cholesterol
5. B is lysophosphatidylcholine (lysolecithin)
6. A is a fatty acid
7. LDLs have enzyme Z attached to their surface
8. Enzyme Z catalyzes the transfer of a fatty acid from lecithin to the free cholesterol molecule
9. The enzyme Z is ACAT (Acyl-CoA: Cholesterol Acyltransferase)
10. The enzyme Z is LCAT (Lecithin-cholesterol acyltransferase)

114. Which of the following statements about the metabolism of HDL are correct?

1. HDL is formed from VLDL as it loses triglycerides through the action of lipoprotein lipase
2. In the liver, the cholesterol esters brought by HDL are hydrolyzed and cholesterol is converted to bile acids
3. Because of the inverse relationship between plasma HDL concentration and atherosclerosis, HDL is designated as the "good" cholesterol carrier

4. The molecular defect most responsible for familial hypercholesterolemia is a lack or deficiency of receptors for HDL
5. HDL binds to HDL receptors on the surface of cells and is taken up by endocytosis
6. Nascent HDL are disk-shaped particles composed of phospholipids, free cholesterol and apoproteins A, C and E
7. Elevated HDL cholesterol levels are a major risk factor for the development of atherosclerosis
8. HDL particles can be oxidized by reactive oxygen species, and oxidized HDL is taken up by scavenger receptors on macrophages, leading to the formation of foam cells
9. In the reverse cholesterol transport pathway, cholesterol is transferred to the liver from HDL2 by a scavenger receptor of the type B family (SR-B1)
10. Nascent HDLs have an enzyme attached to their surface: LCAT (lecithin cholesterol acyl transferase

115. Cholesterol synthesis involves:

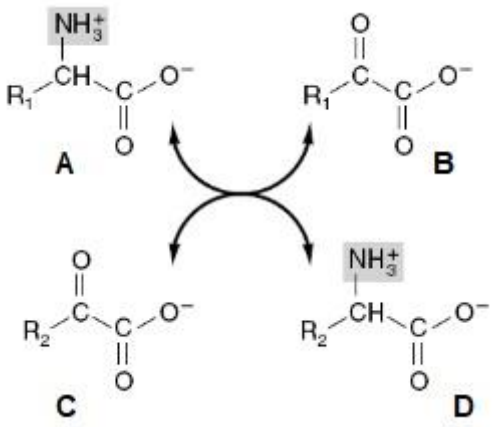
1. Condensation of three acetyl-CoA molecules to form HMG-CoA
2. GTP consumption
3. Condensation of three isoprene units, with the formation of mevalonic acid
4. Conversion of lanosterol to active isoprene units
5. The participation of NADH
6. Conversion of mevalonic acid to active isoprene units
7. Conversion of HMG-CoA to mevalonic acid
8. The participation of NADPH
9. Conversion of squalene to lanosterol
10. Conversion of mevalonic acid to HMG-CoA

116. The following can be said about the plasma lipoproteins:

1. Increased LDL concentration is considered a risk factor for atherosclerosis
2. The four classes of lipoproteins differ with respect to their density

3. The blood collected after the overnight fast is normally rich in chylomicrons
4. The main classes of apoproteins are chylomicrons, LDL, VLDL and HDL
5. LDL particles are synthesized in the circulation, from HDL
6. VLDL particles are synthesized in hepatocytes
7. The uptake of LDL from the blood by cells is achieved by a mechanism of receptor-mediated endocytosis
8. HDL particles are rich in triglycerides, which they transport to extrahepatic tissues
9. VLDL particles are synthesized at the intestinal level
10. Chylomicrons transport dietary lipids

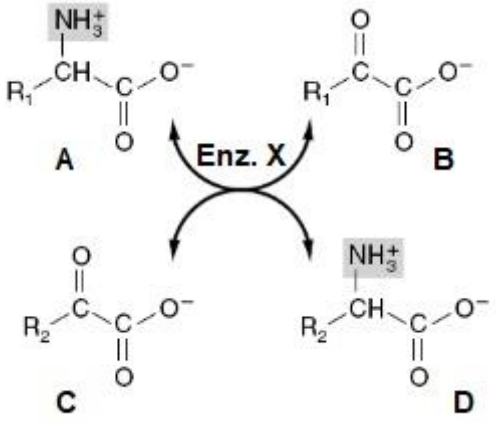
117. What can be said about the reaction presented in the image?




1. It represents the first step in the catabolism of most amino acids
2. It is a deamination reaction
3. If A is alanine, then B is lactate
4. It is a transamination reaction
5. C is α-ketoglutarate, in most cases
6. The enzymes that catalyze this type of reaction are called amino acid deaminases
7. If A is aspartate, then B is pyruvate
8. If A is aspartate, then B is oxaloacetate
9. It requires vitamin B12 as a coenzyme

10. If A is alanine, then B is pyruvate

118. The following is/are true about the reaction presented in the image:



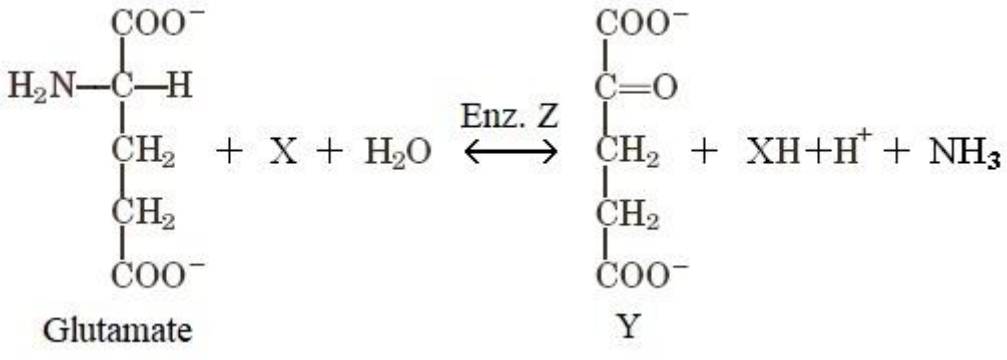

1. X is generically called transaminase
2. It requires pyridoxal phosphate (active form of vitamin B6) as a coenzyme
3. If A is aspartate, then X is GOT
4. The reaction requires folic acid as a cofactor
5. If B is oxaloacetate, then X is GOT
6. If A is alanine, then X is GPT
7. The reaction can be written A + B ↔ C + D
8. If B is pyruvate, then X is GPT
9. The reaction takes place only in the liver
10. The reaction can be written A + C ↔ B + D

119. Select the correct statement(s) about the transamination process:

1. It requires the participation of vitamin B6, in the form of pyridoxal phosphate
2. It requires vitamin B2 as a coenzyme
3. It is catalyzed by aminotransferases
4. It involves the transfer of the amino group from an amino acid to a keto acid

5. The transfer of the amino group from an amino acid to a keto acid requires ATP as an energy donor
6. In transamination reactions, the amino group of amino acids is released as free ammonia
7. It represents the first step in the catabolism of most amino acids
8. The transaminase GPT is also called AST
9. Transamination reactions are irreversible
10. Transamination of most amino acids lead to the synthesis of glutamate from α-ketoglutarate

120. What can be stated about the reaction presented in the image?




1. Y is oxaloacetate
2. X may be NAD+ or NADP+
3. It takes place in most tissues
4. Z is glutamate dehydrogenase
5. X is FAD
6. Z is glutamate aminotransferase
7. It is a reaction that is specific for the liver
8. It represents the oxidative deamination of glutamate
9. The ammonia that results from the reaction will be eliminated from the body as uric acid
10. It follows after the transamination of amino acids

121. Choose the correct answer(s) about glutamate dehydrogenase:

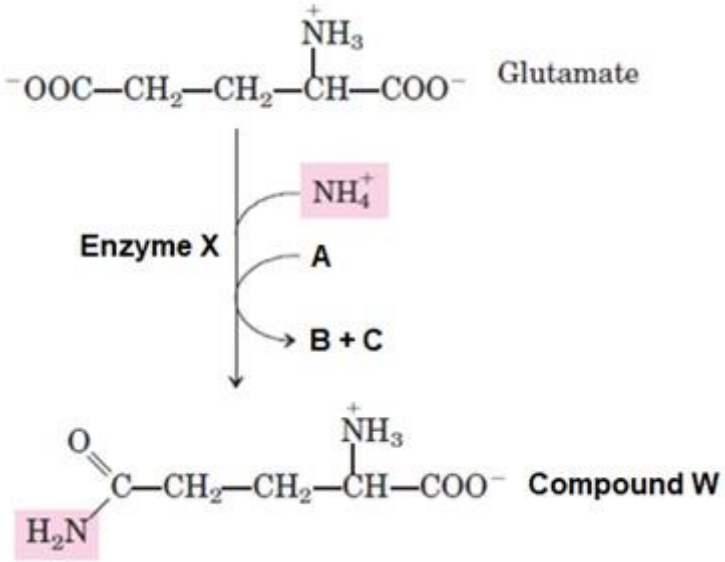
1. It uses pyridoxal phosphate as a coenzyme

2. It catalyzes a reaction that represents the second step in the catabolism of most amino acids, after the transamination process
3. It catalyzes the release of the amino group of glutamate as free ammonia
4. It catalyzes a reversible reaction
5. It catalyzes a reaction in which glutamate is converted to α-ketoglutarate
6. It uses either NAD+ or NADP+ as coenzyme
7. It is an enzyme belonging to the urea cycle
8. It catalyzes the oxidative deamination of glutamate to oxaloacetate
9. It is involved in amino acid synthesis, but not in amino acid catabolism
10. It catalyzes the transfer of the amino group of most amino acids to α-ketoglutarate

122. Select the correct statement(s) about ammonia (NH3) transport from extrahepatic tissues to the liver:

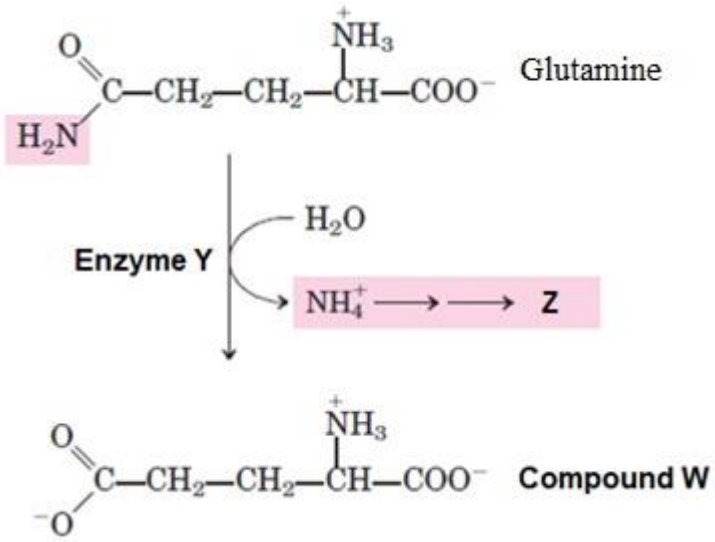
1. Ammonia is transported in the blood in the form of glutamine
2. Glutamine synthetase is involved in glutamine formation
3. Glutamine synthetase does not require ATP participation
4. Glutaminase is involved in glutamine formation in extrahepatic tissues
5. Glutaminase catalyzes the release of ammonia from glutamine in the liver
6. Glutamine synthetase catalyzes glutamine formation from α-ketoglutarate and NH3
7. Glutaminase reaction is a simple reversal of glutamine synthetase reaction
8. Ammonia is transported in the blood as the α-amino group of glutamine
9. Glutaminase is a hydrolase
10. Glutamine is synthesized from glutamate and ammonia, with ATP consumption

123. What can be stated about the reaction presented in the image?




1. B and C are ADP and inorganic phosphate
2. X is glutamate dehydrogenase
3. W is glutamine
4. B and C are AMP and inorganic pyrophosphate
5. A is ATP
6. A is NAD+
7. W is α-ketoglutarate
8. The reaction is important for the process of ammonia transport in the blood
9. X is glutamate transaminase
10. X is glutamine synthetase

124. Which statements are true regarding the reaction presented in the image?




1. W is α-ketoglutarate
2. Glutamine is the main non-toxic transport form of ammonia into the blood
3. Enzyme Y is present in the liver and kidney
4. If the reaction takes place in the liver, Z is urea
5. Y is glutaminase
6. If the reaction takes place in the kidney, Z is urea
7. Y is glutamine synthetase
8. W is glutamate
9. In this reaction the α-amino group of glutamine is released as ammonia
10. Y is glutamine lyase

125. Which of the following statements about the urea cycle is/are correct?

1. The two nitrogen atoms of the urea molecule are provided by ammonia and aspartate
2. The reaction catalyzed by carbamoyl-phosphate synthetase I uses 2 ATPs
3. Ornithine is the direct precursor of urea
4. Carbamoyl-phosphate synthetase I catalyzes the covalent binding of ammonia to carbon dioxide
5. Urea is produced by the hydrolysis of arginine

6. The urea cycle is important because it leads to ATP synthesis
7. The reactions of urea cycle take place only in the cytosol of liver cells
8. The reactions of urea cycle take place both in the mitochondria and in the cytosol of liver cells
9. One of the two nitrogen atoms of urea is directly provided by glutamate
10. Ornithine, a participant in the urea cycle, is an amino acid that is also found in proteins

126. What can be said about the urea cycle?

1. It takes place only in the mitochondria of liver cells
2. After synthesis, urea is transported to the kidney where it is hydrolyzed by urease
3. The rate-limiting step of the process is catalyzed by carbamoyl-phosphate synthetase I
4. It takes place both in the liver and kidney
5. After synthesis in the liver, urea is eliminated through the bile
6. It comprises 5 reactions that take place in the liver
7. The synthesis of one urea molecule consumes 4 high-energy bonds
8. The urea cycle has a low rate in case of starvation
9. Aspartate that enters the third reaction can be regenerated from fumarate, which is released in the fourth reaction
10. It has the purpose of detoxifying ammonia

127. What can be stated about the urea cycle and its regulation?

1. The rate of urea cycle is increased in case of high-protein diets
2. Starvation leads to a decrease of the rate of urea cycle
3. Arginine has a positive influence on urea cycle by increasing the formation of N-acetyl-glutamate, the allosteric activator of the rate-limiting enzyme
4. Low activity of urea cycle enzymes leads to decreased plasma levels of ammonia

5. The rate-limiting enzyme is covalently regulated
6. Carbamoyl-phosphate synthetase I is allosterically activated by N-acetyl-glutamate
7. The rate of urea cycle is increased in case of starvation
8. The rate-limiting step is catalyzed by arginase
9. N-acetyl-glutamate is an allosteric activator of ornithine-carbamoyl-transferase
10. The rate-limiting step is catalyzed by carbamoyl-phosphate synthetase I

128. Select the correct statement(s) about the process of urea synthesis:

1. An increased catabolism of tissue proteins leads to an increased level of urea in the blood
2. Four of the five reactions involved in urea synthesis form a cyclic process
3. Urea represents the main form in which ammonia is eliminated from the body
4. Ammonia provides both nitrogen atoms of the urea molecule
5. Defects of enzymes of the urea cycle lead to an increase of ammonia concentration in the blood
6. Aspartate enters the process by condensing with ornithine
7. Citrulline is an intermediate of the process
8. Urea is synthesized in the kidney and then eliminated in the urine
9. Arginine is an intermediate of the process
10. Urea represents the form in which the α-carboxyl group of amino acids is eliminated from the body

129. What can be said about the following reaction?$NH_3 + CO_2 + 2ATP \rightarrow H_2N{-}CO{-}O{-}PO_3^{2-} + 2ADP + Pi$:

1. It takes place in the mitochondria of liver cells
2. It takes place in the cytosol of liver cells
3. It is involved in the synthesis of amino acids
4. It represents the rate-limiting step of the urea synthesis process
5. It is catalyzed by a kinase

6. The enzyme that catalyzes this reaction is activated by glutamate
7. It belongs to urea cycle
8. It is catalyzed by carbamoyl-phosphate synthetase I
9. The enzyme that catalyzes this reaction is activated by N-acetyl glutamate
10. It requires vitamin B6 as a coenzyme

130. Choose the correct statement(s) about the metabolic fate of the α-amino group of amino acids:

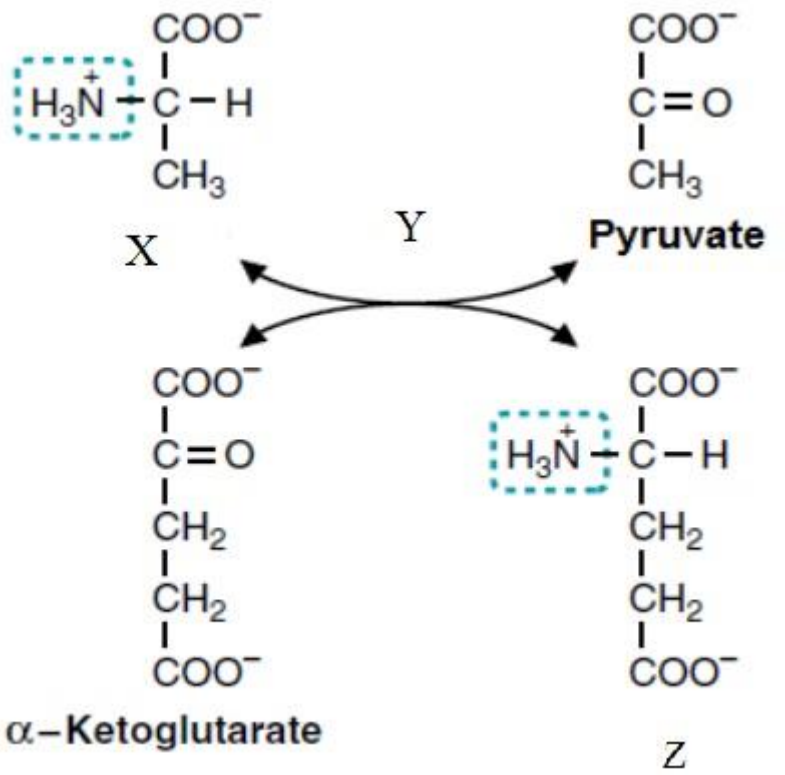
1. For most amino acids, the amino group is first transferred to α-ketoglutarate in transamination reactions
2. The majority of ammonia that results from the amino group is eliminated by the kidney as the NH4+ ion
3. Ammonia that results from the amino group is transported to the liver incorporated into the glutamine molecule
4. The ammonia that results from the amino group circulates in the blood in a free form
5. Following the processes of transamination and oxidative deamination of glutamate, the amino group is eliminated as ammonia
6. Glutamate that is formed in transamination reactions releases the amino group in a reaction catalyzed by glutamate dehydrogenase
7. All amino acids can easily release their amino group as ammonia
8. The amino group is ultimately eliminated from the body as urea
9. The amino group is ultimately eliminated from the body as uric acid
10. In the first reaction of the urea cycle, the amino group of amino acids is transferred on carbon dioxide

131. Regarding the decarboxylation of amino acids, it can be stated:

1. It is an irreversible reaction
2. It represents the removal of the carboxyl group under the action of biotin

3. The enzyme that catalyzes the reaction uses a coenzyme that comes from vitamin B12
4. The enzyme that catalyzes the reaction uses pyridoxal phosphate (PLP) as a coenzyme
5. The enzyme that catalyzes the reaction uses a coenzyme that comes from vitamin B2
6. It represents the removal of the amino group
7. It generates compounds called biogenic (biological) amines
8. The decarboxylation of amino acids is an important step in the biosynthesis of certain neurotransmitters, such as dopamine and serotonin
9. The enzyme responsible for catalyzing the decarboxylation of amino acids is called amino acid decarboxylase
10. It is a reversible reaction

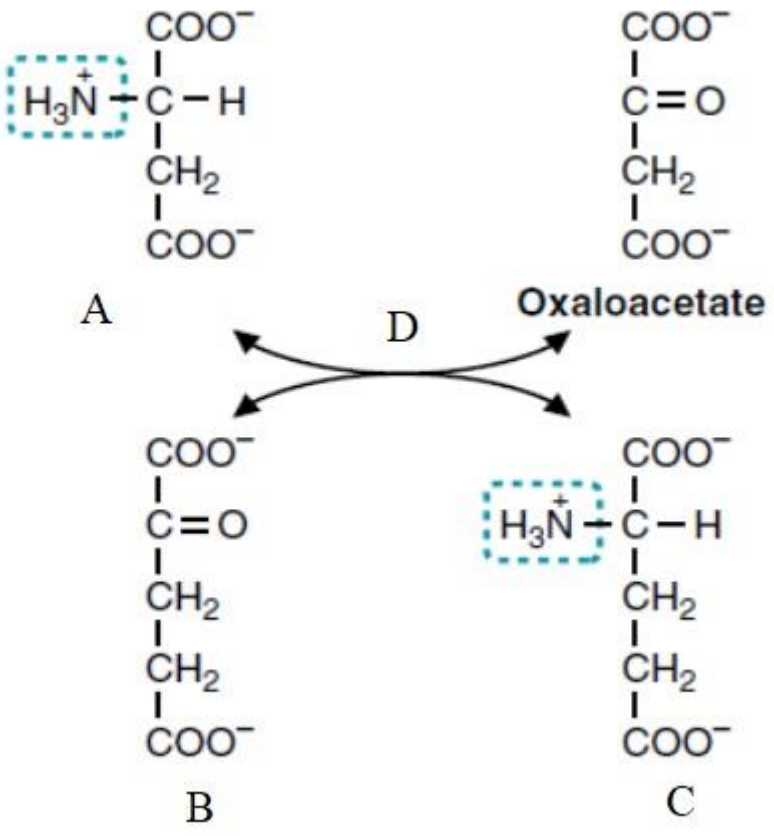
132. Regarding the following image it can be stated:




1. Enzyme Y is named aspartate aminotransferase (GOT)

2. Compound Z is an alpha-keto acid
3. Compound X is named alanine
4. The process shown in the image is part of the particular (individual) degradation pathways of amino acids
5. Compound Z is named aspartate
6. The process shown in the image is part of the common degradation pathways of amino acids
7. Compound Z is named glutamate
8. The enzyme that catalyzes the reaction requires pyridoxal phosphate (PLP) as a coenzyme
9. Compound X is named glutamate
10. Enzyme Y is named alanine aminotransferase (GPT)

133. In the following image:



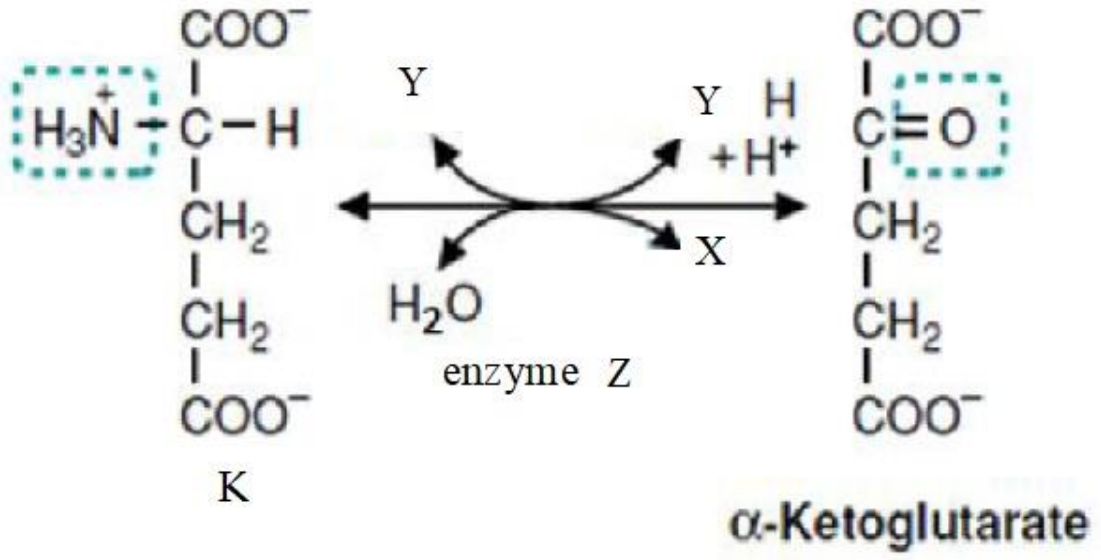

1. Compound A is named aspartate

2. The process represents the decarboxylation of amino acids
3. Enzyme D uses pyridoxal phosphate (PLP) as a coenzyme
4. Compound C is named glutamate
5. Compound C is named aspartate
6. Compound B is named α-ketoglutarate
7. Compound A is an alpha-keto acid, named aspartate
8. Enzyme D is named aspartate aminotransferase (GOT)
9. Compound A is named alanine
10. Enzyme D is alanine aminotransferase (GPT)

134. Regarding glutamate dehydrogenase (GDH) we can state:

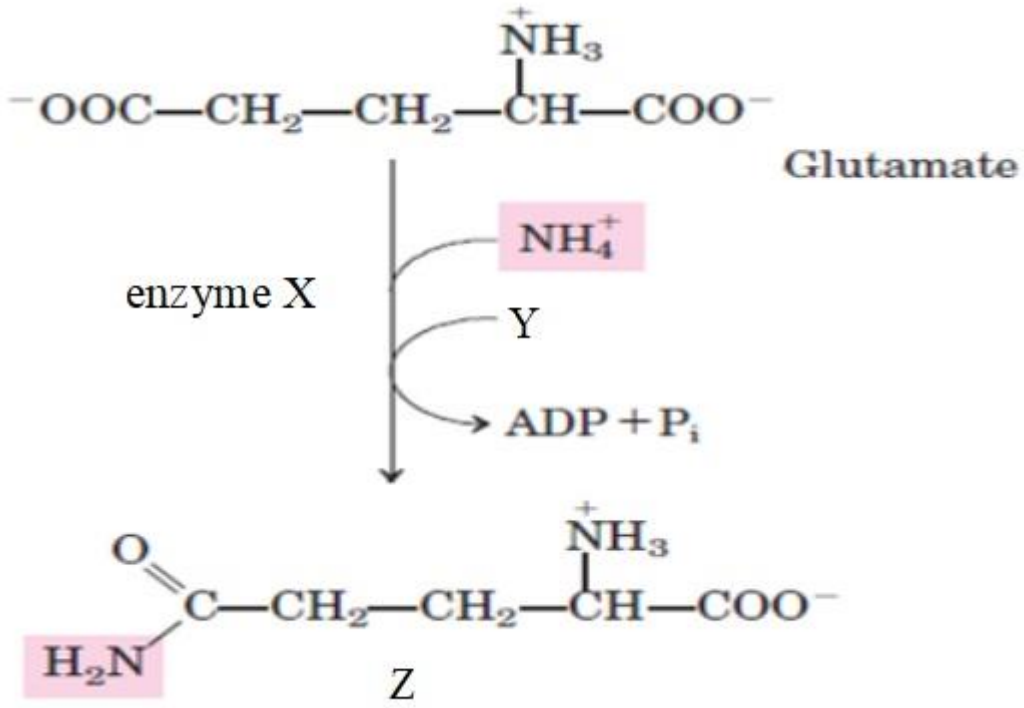
1. It catalyzes the second step in the catabolism of amino acids, following the transamination process
2. GDH requires biotin as a cofactor
3. GDH catalyzes a reversible reaction
4. GDH activity is not regulated by any factors
5. Glutamate dehydrogenase is an enzyme that catalyzes the conversion of glutamate to α-ketoglutarate and ammonia
6. It catalyzes an irreversible reaction
7. GDH catalyzes the reversible conversion of aspartate to alpha-ketoglutarate
8. It catalyzes the removal of alpha amino group in the form of ammonia in the process of biogenic (biological) amines synthesis
9. GDH requires NAD+ or NADP+ as a cofactor
10. GDH functions in both the degradation and the synthesis of amino acids

135. Regarding the image below it can be stated:




1. Y is NAD(P)
2. The enzyme Z is glutaminase
3. Compound X is ammonia
4. Compound Y is ammonia
5. It represents the removal of the alpha amino group in the form of ammonia in the decarboxylation process
6. The enzyme Z is glutamate dehydrogenase (GDH)
7. It represents a transamination process
8. Compound K is named glutamate
9. It represents the removal of the alpha amino group in the form of ammonia in the deamination process
10. Compound X is urea

136. Regarding the image below it can be stated:




1. Enzyme X incorporates toxic free ammonia into a non-toxic compound, thus protecting the tissues
2. Compound Z represents the non-toxic transport form of ammonia
3. Compound Z is glutamine
4. Enzyme X generates free ammonia in the liver and kidney
5. Compound Z is α-ketoglutarate
6. The enzyme X catalyzes the transformation of glutamate into α-ketoglutarate
7. The enzyme X is glutamine synthetase
8. Compound Y is ATP
9. Compound Y is ammonia
10. The enzyme X is glutamate dehydrogenase (GDH)

137. Which of the following statements about the urea cycle are correct?

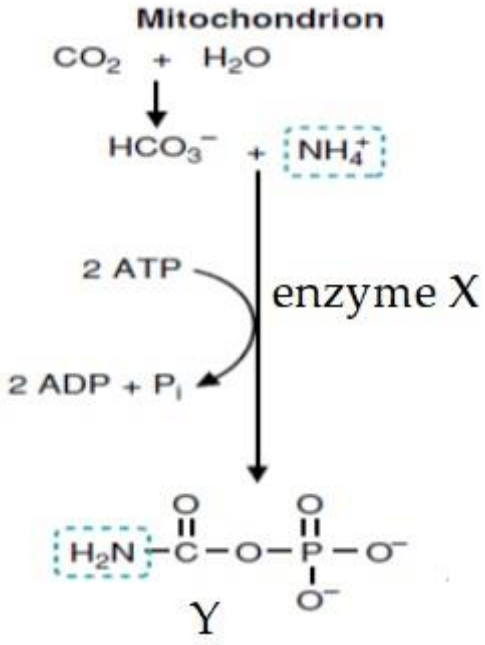
1. Ammonia is first incorporated into carbamoyl phosphate
2. The first reaction in the urea cycle involves the synthesis of arginine
3. It takes place in the liver

4. Urea arises as a product of arginase action upon arginine
5. The urea cycle generates energy in the form of ATP
6. The two nitrogen atoms that are incorporated into urea enter the urea cycle in the form of ammonia and aspartate
7. The urea cycle takes place just in mitochondria
8. The enzymes of the urea cycle are located both in the mitochondria and cytosol
9. In the cleavage reaction of arginino-succinate with the synthesis of arginine one molecule of ATP is required
10. Urea is produced directly by hydrolysis of ornithine

138. About the regulation of urea cycle, we can state:

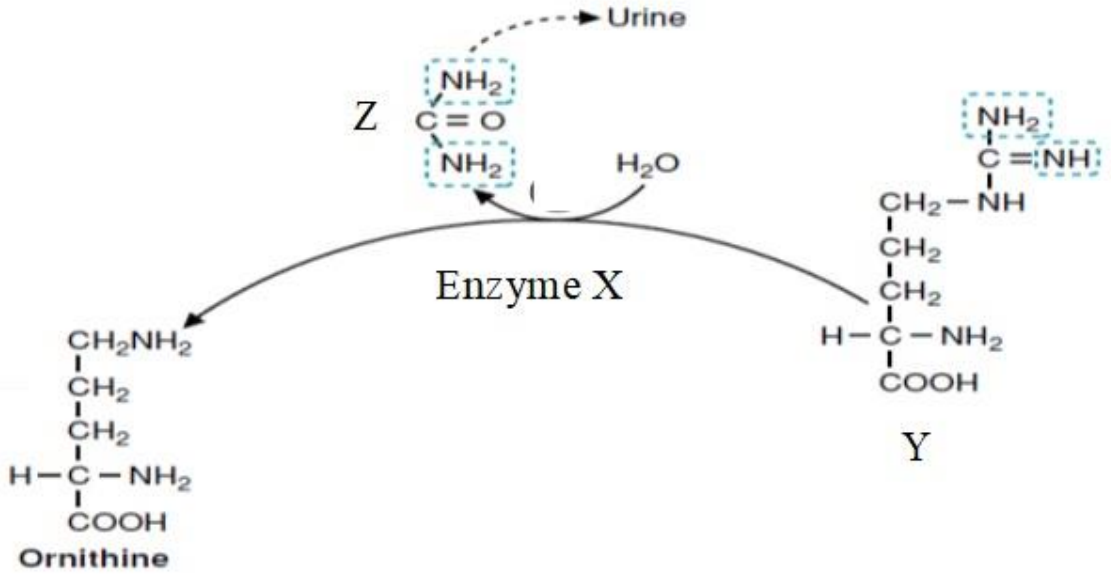
1. The enzyme ornithine transcarbamoylase (OTC) catalyzes the rate limiting step in the urea cycle
2. The enzyme arginase is inhibited by biotin
3. Argininosuccinate lyase, the enzyme that cleaves argininosuccinate to produce arginine and fumarate, is activated by N-acetyl-glutamate (NAG)
4. The rate of urea synthesis in liver is correlated with the concentration of N-acetyl-glutamate
5. Carbamoyl phosphate synthetase (CPS-I) is activated, indirectly, by the accumulation of arginine
6. Carbamoyl phosphate synthetase (CPS-I) is inhibited by arginine
7. The activity of argininosuccinate synthetase (ASS), the enzyme that catalyzes the fourth step of the urea cycle, is inhibited by citrulline
8. Carbamoyl phosphate synthetase (CPS-I) is allosterically activated by N-acetyl-glutamate (NAG)
9. Carbamoyl phosphate synthetase (CPS-I) is the rate limiting enzyme in urea cycle
10. The consumption of a protein-rich meal increases the level of N-acetyl-glutamate (NAG) in the liver, leading to enhanced urea synthesis

139. Regarding the image below it can be stated:




1. It represents the incorporation of ammonia into carbamoyl phosphate
2. It represents the incorporation of ammonia into argininosuccinate
3. It represents the first reaction in the urea cycle
4. The enzyme X catalyzes the transformation of ammonia into urea
5. The enzyme X is carbamoyl phosphate synthetase (CPS-I)
6. The enzyme X is carbamoyl phosphate lyase
7. X is the rate limiting enzyme in urea cycle
8. Compound Y is arginine
9. The enzyme X is ornithine transcarbamoylase (OTC)
10. Compound Y is named carbamoyl phosphate

140. Regarding the following image it can be stated:




1. It is the first reaction in the urea cycle
2. The enzyme X is carbamoyl phosphate synthetase (CPS-I)
3. Compound Z is urea, a very toxic compound because it contains two amino groups
4. Z is urea
5. It represents the last reaction in the urea cycle
6. The process takes place in the cytosol
7. The process takes place in the mitochondria
8. The enzyme X is arginase
9. It represents the transamination process
10. Y is arginine

141. Regarding the reaction below it can be stated:Arginino-succinate → A + B (catalyzed by enzyme X):

1. The enzyme X is named argininosuccinate synthetase
2. Compound B is named urea
3. The reaction takes place in the cytosol
4. The enzyme X is arginase
5. The reaction requires ATP consumption

6. It represents the incorporation of ammonia into carbamoyl phosphate
7. The enzyme X is named argininosuccinate lyase
8. It represents a reaction in the urea cycle
9. A and B are arginine and fumarate
10. Arginine-succinate is formed in the reaction between citrulline and aspartate

142. Regarding citrulline it can be stated:

1. Citrulline is synthesized by the enzyme ornithine transcarbamoylase
2. The enzyme argininosuccinate lyase cleaves arginosuccinate to arginine and citrulline
3. It is transported from the cytosol to the mitochondria to be converted into argininosuccinate
4. It is an intermediate in the urea cycle
5. Citrulline condenses with aspartate to form arginine
6. Citrulline is synthesized from ornithine and carbamoyl phosphate
7. It generates ornithine under the action of carbamoyl phosphate synthetase I (CPS I)
8. Citrulline is transported from mitochondria to cytosol
9. It generates argininosuccinate under the action of ornithine transcarbamoylase
10. Citrulline is converted into argininosuccinate by the enzyme arginosuccinate synthetase

143. Select the correct statement(s) about the ammonia:

1. Ammonia is primarily metabolized in the liver and converted to urea
2. Ammonia is formed by transamination and oxidative deamination
3. The primary fate of ammonia in the liver is its conversion to glutamate
4. The two nitrogen atoms of urea are derived just from ammonia
5. Toxic urea is converted into non-toxic ammonia

6. Increased levels of ammonia can be toxic and cause damage to various organs and tissues, especially to the brain
7. Ammonia that is formed in the extrahepatic tissues is transported to the liver incorporated in glutamine
8. In the urea cycle ammonia is first incorporated into carbamoyl phosphate
9. Ammonia is the final product of urea catabolism
10. Ammonia is synthesized in the kidney and liver with the participation of three amino acids: arginine, glycine and methionine

144. Which of the following are necessary for the synthesis of alanine starting from ammonia and α-ketoglutarate?

1. Oxaloacetate
2. Glutamate dehydrogenase
3. NAD+
4. Pyruvate
5. GOT
6. Glutamine synthetase
7. NADPH
8. Vitamin B6
9. GPT
10. Vitamin B12

145. Which of the following are necessary for the synthesis of aspartate starting from ammonia and α-ketoglutarate?

1. Oxaloacetate
2. Vitamin B6
3. FAD
4. α-ketoglutarate reductase
5. Tetrahydrofolate
6. Pyruvate
7. GPT
8. Glutamate dehydrogenase

9. GOT
10. NADPH

146. Select the correct answer(s) about tetrahydrofolate (FH4):

1. Its role is to transfer one-carbon groups
2. N5,N10-methylene-FH4 is used for the synthesis of purine nucleotides
3. N5-methyl FH4 is the methyl group donor in the synthesis of TMP from dUMP
4. Serine is involved in the conversion of FH4 to N5,N10-methylene-FH4
5. It is formed from folate by two reduction reactions with FADH2
6. N5-methyl FH4 is used in the conversion of homocysteine to methionine
7. N5-methyl FH4 is the methyl group donor in the synthesis of epinephrine from norepinephrine
8. It is required as a coenzyme in transamination reactions
9. N5,N10-methylene-FH4 is involved in the synthesis of TMP
10. It is the coenzyme form of folic acid

147. Choose the correct answer(s) about the amino acid methionine (Met):

1. Methionine can be regenerated from homocysteine by receiving a methyl group from N5-CH3 tetrahydrofolate
2. S-adenosyl-methionine (SAM) is used as a methyl group donor in the synthesis of TMP from dUMP
3. The role of methionine is to transfer a sulfur-containing chemical group to various acceptors
4. Methionine synthase catalyzes the formation of methionine from cysteine
5. During its metabolism, methionine is converted to homocysteine
6. S-adenosyl-methionine (SAM) can donate a methyl group to tetrahydrofolate, leading to the formation of N5-CH3 tetrahydrofolate

7. Its active form, S-adenosyl-methionine (SAM), is formed in a reaction between Met and AMP
8. The conversion of homocysteine to methionine requires a tetrahydrofolate derivative and vitamin B12
9. The role of S-adenosyl-methionine (SAM) is to donate a methyl group in several synthesis reactions
10. Its active form is S-adenosyl-methionine (SAM)

148. S-adenosyl-methionine (SAM) is used as a methyl group donor in:

1. Synthesis of melatonin from serotonin
2. Synthesis of purine nucleotides
3. Synthesis of serine from glycine
4. Synthesis of TMP from dUMP
5. Synthesis of creatine from guanidinoacetate
6. Synthesis of epinephrine from norepinephrine
7. Reactions catalyzed by methyl transferases
8. Synthesis of threonine from serine
9. Synthesis of thymine from uracil
10. Synthesis of phosphatidyl-choline from phosphatidyl-ethanolamine

149. Choose the vitamin(s), coenzyme(s) and enzyme(s) that participate in amino acid metabolism:

1. Glutamate pyruvate transaminase
2. Vitamin D
3. Hormone-sensitive lipase
4. HMG-CoA reductase
5. Tetrahydrofolate
6. Pyridoxal phosphate
7. Fatty acid synthase
8. Vitamin B12
9. Acetyl-CoA carboxylase
10. Glutamate dehydrogenase

150. Select the correct statement(s) about the metabolism of amino acids:

1. The carbon skeleton of some amino acids may be completely degraded to provide ATP
2. The carbon skeleton of some amino acids may be converted to glucose
3. Non-essential amino acids are not important for protein synthesis
4. All 20 amino acids may be synthesized from ammonia and various carbon skeletons
5. Aspartate is synthesized in a reaction between oxaloacetate and ammonia
6. The carbon skeleton of some amino acids may be converted to ketone bodies
7. Some amino acids are converted to nitrogenous compounds having specific roles
8. Alanine is synthesized in a reaction between pyruvate and ammonia
9. Amino acid degradation involves the removal of the amino group as ammonia
10. During amino acid catabolism, their carbon skeleton is ultimately converted to urea

151. What can be said about vitamin B12?

1. It is involved in amino acid metabolism
2. Its deficiency leads to decreased homocysteine levels
3. It is used as a coenzyme in transamination reactions
4. Its deficiency leads to a decreased regeneration of tetrahydrofolate from N5-methyl tetrahydrofolate
5. It participates in the formation of methionine from homocysteine and N5-methyl tetrahydrofolate
6. It is used in the conversion of phenylalanine to tyrosine
7. Its deficiency leads to increased homocysteine levels
8. Its deficiency leads to increased methionine levels
9. It participates in lipid metabolism
10. It is used as a coenzyme in the conversion of methyl-malonyl-CoA to succinyl-CoA

152. What can be said about the amino acid phenylalanine (Phe)?

1. The normal metabolic route that it takes is transamination to phenyl-pyruvate
2. It can be synthesized in the human body from tyrosine
3. It is the precursor of serotonin
4. Its conversion to tyrosine requires tetrahydrofolate as a coenzyme
5. The genetic deficiency of Phe hydroxylase leads to mental retardation if left untreated
6. Conversion of Phe to tyrosine is catalyzed by Phe hydroxylase
7. It is a sulfur-containing amino acid
8. Conversion of Phe to tyrosine requires the cofactor tetrahydrobiopterin (BH4)
9. It is an essential amino acid
10. It is the precursor of tyrosine

153. Select the correct answer(s) about the metabolism of phenylalanine (Phe):

1. The excessive accumulation of Phe in individuals suffering from phenylketonuria leads to toxic effects exerted on the brain
2. It is used in the synthesis of proteins
3. The genetic deficiency of Phe hydroxylase are associated with low Phe levels
4. One of the metabolic paths that it takes is conversion to tryptophan
5. It is converted to tyrosine in a hydroxylation reaction that uses tetrahydrofolate (FH4) as a cofactor
6. The genetic deficiency of phenylalanine hydroxylase leads to a disease called phenylketonuria
7. The genetic deficiency of Phe hydroxylase leads to a disease called albinism
8. It is converted to tyrosine in a hydroxylation reaction that uses tetrahydrobiopterin (BH4) as a cofactor
9. The conversion of Phe to tyrosine requires pyridoxal phosphate as a cofactor

10. In individuals suffering from phenylketonuria, Phe is converted to phenylpyruvate, which is eliminated in the urine

154. What can be stated about the amino acid tyrosine (Tyr)?

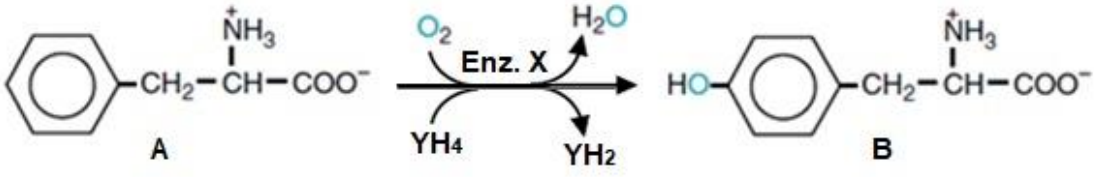
1. It is the precursor of epinephrine (adrenaline)
2. It is the precursor of melanin
3. The first reaction in the conversion of Tyr to catecholamines is catalyzed by tyrosinase
4. The first reaction in the conversion of Tyr to melanin is catalyzed by tyrosine hydroxylase
5. It can be converted to DOPA in a reaction that uses tetrahydrobiopterin (BH4) as a cofactor
6. It can be synthesized in the body from phenylalanine
7. The genetic deficiency of tyrosinase leads to a disease called albinism
8. The genetic deficiency of Tyr hydroxylase leads to a disease called phenylketonuria
9. The deficiency of phenylalanine hydroxylase leads to the impossibility of synthesizing tyrosine in the body
10. It is the precursor of melatonin

155. Select the correct answers about the amino acid tryptophan (Trp):

1. It can be used for the synthesis of the coenzymes NAD+ and NADP+
2. Trp hydroxylase uses vitamin B6 as a cofactor
3. The genetic deficiency of Trp hydroxylase leads to a disease called albinism
4. Trp can be produced in our body from phenylalanine
5. It is the precursor of melatonin in the pineal gland
6. It is the precursor of melanin
7. Trp hydroxylase is involved in the conversion of Trp to serotonin and uses tetrahydrobiopterin (BH4) as a cofactor
8. The conversion of Trp to serotonin involves a hydroxylation and a decarboxylation

9. It is the precursor of serotonin
10. It is the precursor of dopamine and epinephrine (adrenaline)

156. What can be said about the reaction presented in the image?



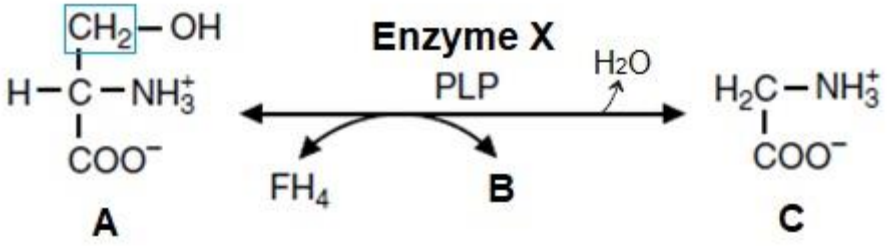

1. The genetic deficiency of enzyme X leads to a severe disease called phenylketonuria
2. Enzyme X is tryptophan hydroxylase
3. Enzyme X is phenylalanine hydroxylase
4. Cofactor YH4 is tetrahydrofolate
5. A is phenylalanine
6. B is the precursor of serotonin
7. B is tyrosine
8. A is tryptophan
9. B is hydroxy-tryptophan
10. Cofactor YH4 is tetrahydrobiopterin

157. What can be stated about folic acid?

1. Tetrahydrofolate fulfills the role of electron carrier to NADP in synthesis processes
2. Lack of folic acid can cause megaloblastic anemia
3. As a result of the reduction process in the organism, folic acid becomes tetrahydrofolate (FH4), or the active form of folic acid that has a coenzymatic role
4. The carbon units that can be transferred by tetrahydrofolate (FH4) include: methyl, methylene, methenyl, formyl
5. In the body, the active form of folic acid is dihydrofolate (FH2)

6. In the organism, folic acid brought through dietary intake undergoes two successive reduction reactions in the presence of dihydrofolate reductase with the participation of NADPH+H+
7. Tetrahydrofolate fulfills the role of H transporter to NADP in the process of reducing oxidized glutathione
8. Folic acid is a dipeptide because it contains two amino acids: glutamic acid and p-aminobenzoic acid (PABA)
9. Tetrahydrofolate serves as a transporter of one-carbon units that are to be transferred to different substrates
10. Folic acid is also called cobalamin

158. Regarding the following image it can be stated:




1. Compound B will lead to the formation of other FH4 derivatives
2. The reaction catalyzed by X requires the presence of PLP, which comes from vitamin PP
3. Compound C can be used for the synthesis of creatine and glutathione
4. Compound B is N5,N10-methylene FH4
5. Compound A is Glycine
6. Compound C is glycine
7. Compound A is serine
8. X is serine hydroxymethyl transferase
9. Compound B is dihydrofolate (FH2)
10. An important role of compound B is to be used in the conversion of norepinephrine (noradrenaline) to epinehrine (adrenaline)

159. What can be said about methionine metabolism?

1. Methionine is a non-essential amino acid
2. Methionine metabolism is not involved in the synthesis of cysteine
3. Methionine participates in the synthesis of cysteine, which is a component of glutathione
4. Methionine synthase is an enzyme involved in the breakdown of methionine
5. Methionine synthase catalyzes the conversion of homocysteine to methionine
6. Methionine can donate a methyl group to various acceptors without needing any previous transformation
7. Methionine is the source of metabolite homocysteine
8. Methionine can be synthesized from cysteine by the addition of a methyl group
9. Methionine is an amino acid containing the group -S-CH3 in its side chain
10. Methionine is a precursor for the synthesis of S-adenosylmethionine (SAM)

160. What can be said about S-adenosylmethionine (SAM)?

1. SAM is not involved in the methylation processes
2. SAM is a molecule that can donate a phosphate group (-PO4) to other molecules
3. SAM is synthesized from methionine and ATP
4. SAM is primarily used as a fuel source for the body
5. SAM is a methyl donor, meaning that it can donate a methyl group (-CH3) to other molecules
6. S-adenosylmethionine (SAM) is a nucleic acid
7. SAM is involved in the synthesis of epinephrine and creatine
8. N5-methyl tetrahydrofolate donates a methyl group to homocysteine to form methionine, which can then be used for protein synthesis or to regenerate SAM
9. SAM metabolism leads to the formation of homocysteine
10. SAM is a carbohydrate that is found in the human body

161. Select the correct statement(s) about phenylalanine (Phe):

1. It can be synthesized from tyrosine by removing the OH group through a dehydration reaction
2. It is an exclusively ketogenic amino acid
3. When there is a deficiency of Phe hydroxylase, phenylalanine is converted to phenylpyruvate following a transamination reaction
4. When there is a deficiency of Phe hydroxylase, tyrosine becomes an essential amino acid and must be supplied through food intake
5. The normal metabolic route that it takes is transamination to phenyl-pyruvate
6. Phenylketonuria is caused by the genetic deficiency of phenylalanine transaminase
7. It is an essential amino acid
8. It can be synthesized from tyrosine by removing the OH group with the consumption of ATP
9. The genetic deficiency of Phe hydroxylase leads to a severe disease called phenylketonuria
10. When there is a deficiency of tetrahydrobiopterin (BH4), Phe cannot be converted to tyrosine

162. Choose the true statement(s) about tyrosine:

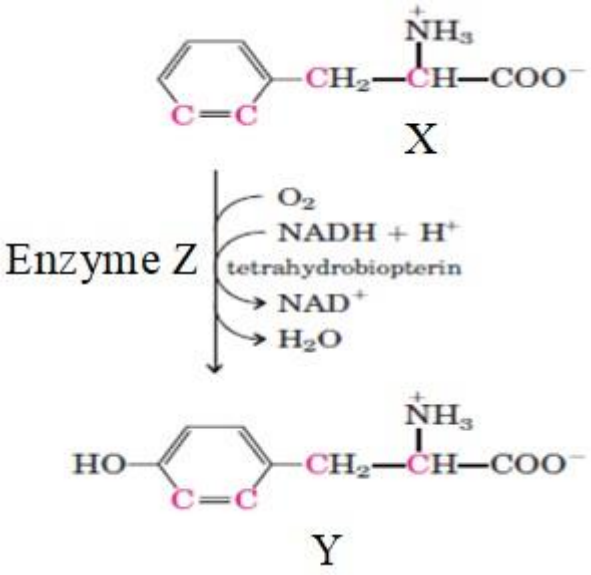
1. It generates a compound called dopamine from which both adrenaline and melatonin can be synthesized
2. Noradrenaline is synthesized from adrenaline under the action of a transferase that uses SAM as cosubstrate
3. Adrenaline is synthesized from noradrenaline through a transamination reaction
4. Adrenaline is synthesized directly from dopamine under the action of a dopa-decarboxylase
5. Its synthesis from phenylalanine requires the cofactor tetrahydrobiopterin BH4)
6. It is converted to dopamine by a hydroxylation reaction followed by a decarboxylation
7. The deficiency of tyrosine hydroxylase does not affect melanin synthesis from tyrosine

8. Genetic deficiency of tyrosinase blocks the synthesis of melanin, causing the appearance of albinism, the disease prone to skin burns and skin cancers
9. It is an essential amino acid in case of a phenylalanine hydroxylase deficiency
10. Noradrenaline is synthesized under the action of the dopamine beta-hydroxylase enzyme, which uses vitamin B6 as a coenzyme

163. What can be stated about the tryptophan?

1. The tryptophan hydroxylase enzyme uses tetrahydrobiopterin as a cofactor
2. The tryptophan hydroxylase enzyme uses tetrahydrofolate as coenzyme
3. 5-hydroxy tryptamine is also called melatonin or the sleep hormone
4. 5-hydroxy tryptamine is also called serotonin
5. Under the action of tryptophan hydroxylase, it generates tyrosine
6. Under the action of tryptophan hydroxylase, it generates 5-hydroxy tryptophan
7. Under the action of a PLP(pyridoxal phosphate)-dependent decarboxylase, 5-hydroxy tryptophan generates 5-hydroxy tryptamine
8. Under the action of a PLP(pyridoxal phosphate)-dependent transaminase, 5-hydroxy tryptophan generates 5-hydroxy tryptamine
9. Tryptophan can be obtained from phenylalanine
10. It is an essential glucogenic and ketogenic amino acid

164. Regarding the following image it can be stated:



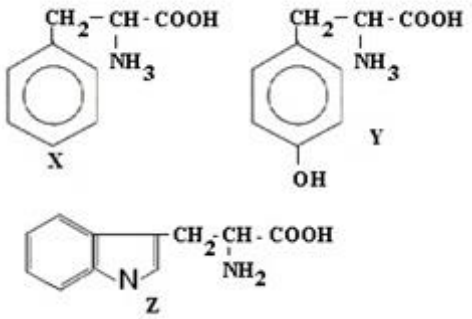

1. In enzyme Z deficiency, tyrosine becomes an essential amino acid
2. Compound Y is phenylalanine, an amino acid
3. Enzyme Z is tryptophan hydroxylase
4. Albinism is a genetic disease caused by enzyme Z deficiency
5. Enzyme Z is named phenylalanine hydroxylase
6. Y is tyrosine
7. Compound Y is a precursor for the synthesis of the hormone melatonin
8. Compound X is an essential amino acid
9. Compound X is named tyrosine
10. Compound X is phenylalanine

165. What can be stated about tyrosine?

1. Phenylalanine hydroxylase catalyzes a reversible reaction and requires tetrahydrobiopterin
2. Tyrosine is involved in the production of thyroid hormones
3. Tyrosine can be converted to serotonin in the body through a series of chemical reactions
4. Genetic disorders such as phenylketonuria (PKU) can lead to a deficiency in tyrosine and other important molecules
5. Tyrosine is precursor for melatonin

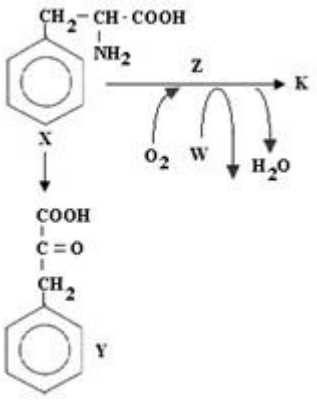
6. Tyrosine can be converted to L-DOPA, which is then converted to dopamine
7. Hydroxylation of phenylalanine to tyrosine is catalyzed by phenylalanine hydroxylase which requires dihydrofolate (FH2)
8. Tyrosine is a non-essential amino acid, precursor for melanin
9. Tyrosine is a precursor for the synthesis of several important molecules, including catecholamines
10. Tyrosine is involved in the production of collagen

166. Regarding the following image it can be stated:




1. All three amino acids are glucogenic and ketogenic
2. X and Z are essential amino acids
3. All three amino acids contain an aromatic ring
4. All three amino acids are processed by hydroxylases that use coenzyme BH4 (tetrahydrobiopterin)
5. X, Y, Z represent Phe, Tyr and Trp respectively
6. Compound Z generates dopamine and adrenaline
7. Compound X is reversibly transformed into Y under the action of a hydratase
8. Following a transamination reaction, compound Y generates phenylpyruvate, a toxic compound
9. Compound Y generates 2 hormones: serotonin and melatonin
10. Compound Z generates both melatonin and melanin

167. Regarding the following image it can be stated:




1. W represents CO2 from the removal of the alpha carboxyl group, in the process of forming compound K
2. Z is a hydratase that introduces the -OH group into compound X, resulting in tyrosine
3. X turns into Y under the action of a deaminase
4. Enzyme Z is phenylalanine hydroxylase, which requires BH4 (tetrahydrobiopterin) as a cofactor
5. Z is phenylalanine hydroxylase, which uses BH2 (dihydrobiopterin) as a coenzyme
6. Y represents phenyl pyruvic acid
7. Compound K is named tyrosine
8. W is tetrahydrobiopterin that converts to dihydrobiopterin
9. X is phenylalanine
10. X is a non-essential amino acid

168. Glycine is involved in the synthesis of the following compounds:

1. Creatine
2. Cysteine
3. N5,N10-methylene-tetrahydrofolate
4. GABA
5. Glutathione
6. Thymine
7. Serine
8. Adenine
9. Heme

10. Cytosine

169. What can be stated about serotonin?

1. It is a neurotransmitter
2. It is synthesized from tryptophan
3. Its synthesis from 5-hydroxy-tryptophan uses the cofactor tetrahydrobiopterin (BH4)
4. It is synthesized from tyrosine
5. Its synthesis involves the participation of the coenzyme form of vitamin B6 (pyridoxal phosphate)
6. Its synthesis from tryptophan involves a hydroxylation and a decarboxylation
7. It is an amino acid
8. It is obtained from tryptophan by deamination
9. It is secreted by the pineal gland
10. It is obtained from 5-hydroxy-tryptophan through decarboxylation

170. Choose the correct statement(s) about melatonin:

1. It is produced in the pineal gland
2. It is a pigment produced in specialized cells of the skin
3. It is obtained from phenylalanine
4. It is involved in inducing the sleep
5. It is synthesized from tryptophan
6. Its synthesis involves the participation of SAM (S-adenosyl-methionine)
7. Its synthesis pathway involves the participation of vitamin B12
8. It is synthesized from tyrosine
9. Its synthesis pathway contains serotonin as an intermediate
10. DOPA is an intermediate in its synthesis pathway

171. What can be said about dopamine?

1. It is obtained from tyrosine by a hydroxylation followed by a decarboxylation

2. It is obtained from tyrosine by a single reaction (decarboxylation)
3. It is obtained by the decarboxylation of 5-hydroxy-tryptophan
4. Its synthesis involves the participation of pyridoxal-phosphate (active vitamin B6)
5. DOPA is an intermediate in its synthesis pathway
6. Its synthesis involves the participation of tetrahydrofolate as a coenzyme
7. It is obtained from DOPA by deamination
8. It may be converted to norepinephrine and epinephrine (adrenaline)
9. It is an amino acid
10. Its synthesis involves the participation of tyrosine hydroxylase and DOPA decarboxylase

172. Select the correct statement(s) about biological amines:

1. Histamine is a biological amine obtained from histidine
2. GABA is obtained by the removal of the α-carboxyl group of glutamic acid
3. DOPA is a biological amine synthesized by tyrosine decarboxylation
4. Melanin is a biological amine obtained from tyrosine
5. GABA is obtained by the removal of the γ-carboxyl group of glutamic acid
6. Their synthesis requires vitamin B12 as a cofactor in the amino acid decarboxylation reactions
7. Serotonin is an example of biological amine
8. They are obtained by the decarboxylation of certain amino acids
9. Their synthesis involves the participation of pyridoxal-phosphate (active vitamin B6)
10. They are synthesized by the deamination of certain amino acids

173. What can be said about phosphocreatine?

1. It is formed in the muscle
2. It is an energy storage form in the muscle

3. It is formed in the liver in a reaction catalyzed by creatine kinase
4. It irreversibly transfers a phosphate group to ADP and forms creatinine
5. It is a high-energy compound formed by oxidative phosphorylation
6. It can be converted to creatinine
7. It is synthesized from creatine and ATP
8. It can be converted to creatinine in a reaction catalyzed by creatine kinase
9. It is obtained in a reversible reaction catalyzed by creatine kinase
10. It can donate a phosphate group to GDP to give rise to GTP

174. What are the correct answer(s) about creatine?

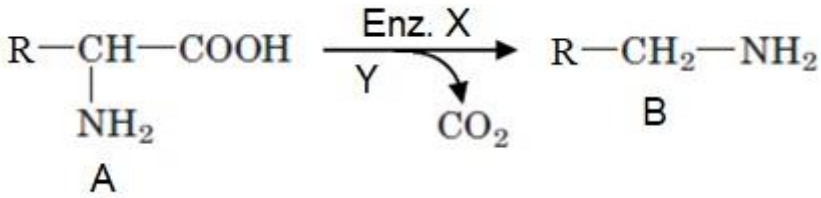
1. It is the substrate of creatine kinase, which converts it to creatinine
2. It receives a phosphate from ATP to form the high-energy compound phosphocreatine
3. Phosphocreatine is a ready source of ATP for muscle contraction
4. The reaction “creatine + ATP → creatine-phosphate + ADP” is irreversible
5. Most of the creatine found in the blood is taken up in the muscle
6. It is obtained from the amino acids arginine, glycine and cysteine
7. Creatine circulating in the blood is taken up in the liver to be converted to phosphocreatine
8. It is a nitrogenous compound formed with the participation of arginine, glycine and S-adenosyl-methionine
9. It can be converted to creatinine by a spontaneous reaction
10. It is a biological amine

175. Select the correct answer(s) about glutathione (GSH):

1. It has an important role in the decomposition of $H_2O_2$
2. Glutamine, cysteine and alanine are found in glutathione structure
3. It is composed of glutamate, cysteine and glycine
4. Glutathione reductase regenerates reduced glutathione from its oxidized form by using NADPH

5. It decomposes hydrogen peroxide in a reaction catalyzed by glutathione reductase
6. Its anti-oxidant role is accomplished in the reaction catalyzed by glutathione peroxidase
7. It is a tetrapeptide
8. Reduced glutathione can be formed from oxidized glutathione by using NADH as a reducing agent
9. It is an amino acid
10. It is a tripeptide

176. Select the correct answer(s) regarding the reaction presented in the image:



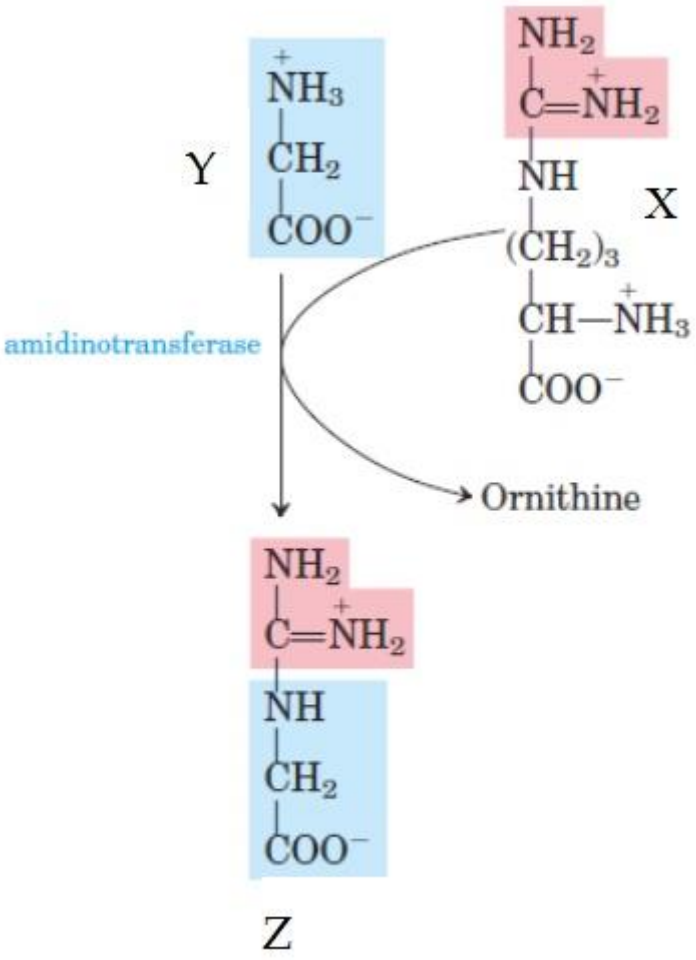

1. Enzyme X is generically called “amino acid decarboxylase”
2. If A is glutamic acid, B is glutamine
3. Coenzyme Y derives from folic acid
4. It is a carboxylation reaction
5. If A is histidine, B is histamine
6. Because B is an amine, enzyme X is called “amine synthase”
7. If A is 5-hydroxy-tryptophan, B is melatonin
8. If A is glutamic acid, B is GABA
9. Coenzyme Y is pyridoxal phosphate (derived from vitamin B6)
10. If A is 5-OH-tryptophan, B is serotonin

177. Which of the following statements about creatinine are correct?

1. Serum creatinine is a sensitive indicator of kidney disease
2. Creatine is synthesized from glycine, arginine and cysteine

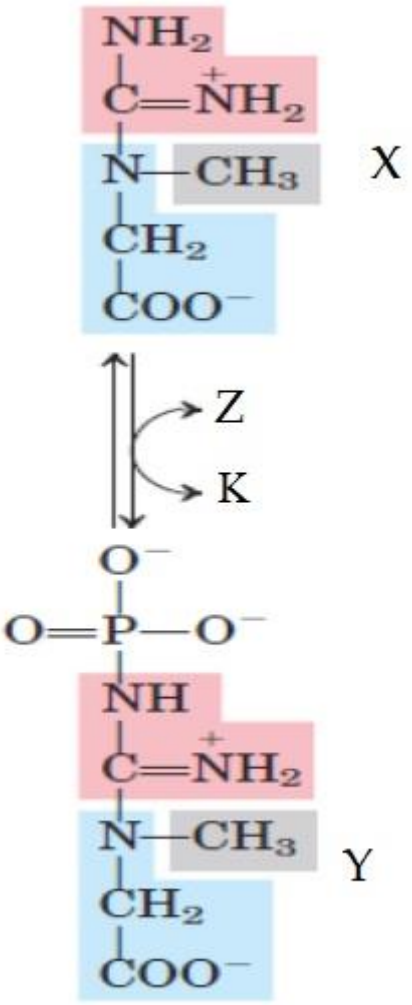
3. Biosynthesis of creatine involves two enzymes: amidino-transferase and hydrolase
4. It is formed from creatine phosphate or creatine
5. Creatine is excreted in the urine
6. Serum creatinine is an important indicator of liver disease
7. Creatinine is excreted in the urine
8. Conversion of creatine phosphate to creatinine is catalyzed by creatine kinase
9. Biosynthesis of creatine involves two enzymes: amidino-transferase and methyltransferase
10. In creatine synthesis are involved three amino acids: arginine, glycine and methionine

178. Regarding the following image it can be stated:



1. Compound Z is an amino acid
2. Compound X is named guanidinoacetate
3. The process shown in the image is the first reaction in creatine biosynthesis
4. Compound X is named arginine
5. Compound Y is named alanine
6. Z can undergo a methylation reaction in the presence of SAM, to generate creatine
7. Compound Z is named guanidinoacetate
8. Z can undergo a methylation reaction in the presence of SAH (S-adenosyl-homocysteine)
9. Compound Z is named arginine
10. Y is glycine

179. The reaction presented in the image is part of creatine metabolism. What can be said about it?




1. Compound Z is AMP
2. Compound K is ATP
3. Compound X is named creatinine
4. Compound Z is ATP
5. Compound X is named creatine phosphate
6. Compound X is named creatine
7. In muscles, creatine phosphate is converted to the high energy compound creatine
8. Compound Y is named creatine phosphate
9. Compound K is ADP
10. The reaction is catalyzed by creatine kinase

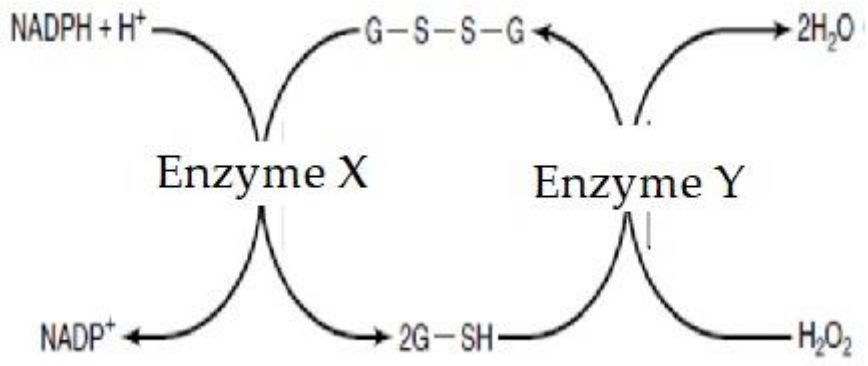
180. What can be said about the following reaction?Creatine + X ------- creatine phosphate + Y:

1. X represents ATP
2. The reaction takes place in the liver
3. Creatine is exclusively synthesized at the muscular level
4. Creatine is obtained by the hydrolytic removal of the phosphate group from creatine phosphate
5. Both creatine and creatine phosphate can be converted to creatinine by the enzyme creatine kinase
6. It is catalyzed by creatine kinase
7. The reaction is reversible
8. Creatine phosphate represents an energy storage form in the muscle
9. Y represents ADP
10. X is inorganic phosphate

181. Which of the following statements about the glutathione are correct?

1. The synthesis of glutathione consumes 2 ATP molecules
2. The first step of glutathione synthesis involves the combination of glutamate and glycine
3. Glutathione is a tripeptide composed of the amino acids glutamate, cysteine, and glycine
4. The second reaction in glutathione synthesis is catalyzed by the enzyme glutathione synthetase (GS)
5. Glutathione is an important antioxidant that helps to protect cells from oxidative damage
6. The second step of glutathione synthesis involves the addition of glutamate to gamma-glutamyl-cysteine
7. Glutathione is a tripeptide composed of the amino acids glutamate, cysteine, and glutamine
8. Glutathione is a high-energy compound
9. The first step of glutathione synthesis is catalyzed by the enzyme glutathione synthetase (GS)
10. The first step involves the synthesis of gamma-glutamyl-cysteine

182. Regarding the image below it can be stated:




1. Enzyme Y is named glutathione peroxidase
2. Enzyme Y is glutathione reductase
3. Enzyme X is named glutathione reductase
4. GSH is the reduced form of glutathione
5. Glutathione exists in cells in 2 states: oxidized (GSH) and reduced (GS-SG)
6. It represents the de novo synthesis of glutathione
7. When oxidative stress (accumulation of reactive oxygen species) occurs, GSH is oxidized to GS-SG
8. Through the hydrolysis of GS-SG, GSH is formed
9. Enzyme X is glutathione peroxidase
10. The oxidized form of glutathione is GS-SG

183. Which of the following statements about the glutathione (GSH) are correct?

1. GSH acts to reduce peptide bonds
2. Regeneration of oxidized GS-SG to reduced GSH by glutathione reductase requires NADPH
3. Glutathione is important as a cofactor for the enzyme glutathione peroxidase
4. Glutathione exists in two forms: the reduced form (GSH) and the oxidized form (GS-SG)
5. It is an important pro-oxidant

6. Glutathione is involved in the decomposition of hydrogen peroxide
7. Glutathione is a dipeptide
8. It is composed of glutamate, methionine and glycine
9. It is formed from glutamate, cysteine and glycine in two ATP-dependent reactions
10. Glutathione is involved in the transport of lipids across cell membranes

184. About the biological (biogenic) amines, we can state:

1. Histidine decarboxylase uses pyridoxal-5'-phosphate (PLP) as a cofactor
2. Biological amines are derived from lipids
3. Dopamine is derived from the amino acid tyrosine
4. Serotonin is derived from tryptophan
5. Examples of biological amines include dopamine, serotonin, histamine
6. Histamine is a biogenic amine synthesized from histidine by histidine carboxylase
7. Histamine is a mediator of the inflammatory response that is involved in allergic reactions
8. Dopamine is derived from the amino acid tryptophan
9. Serotonin is derived from tyrosine
10. Histamine and histidine are biological amines

185. Regarding the biological amines it can be stated:

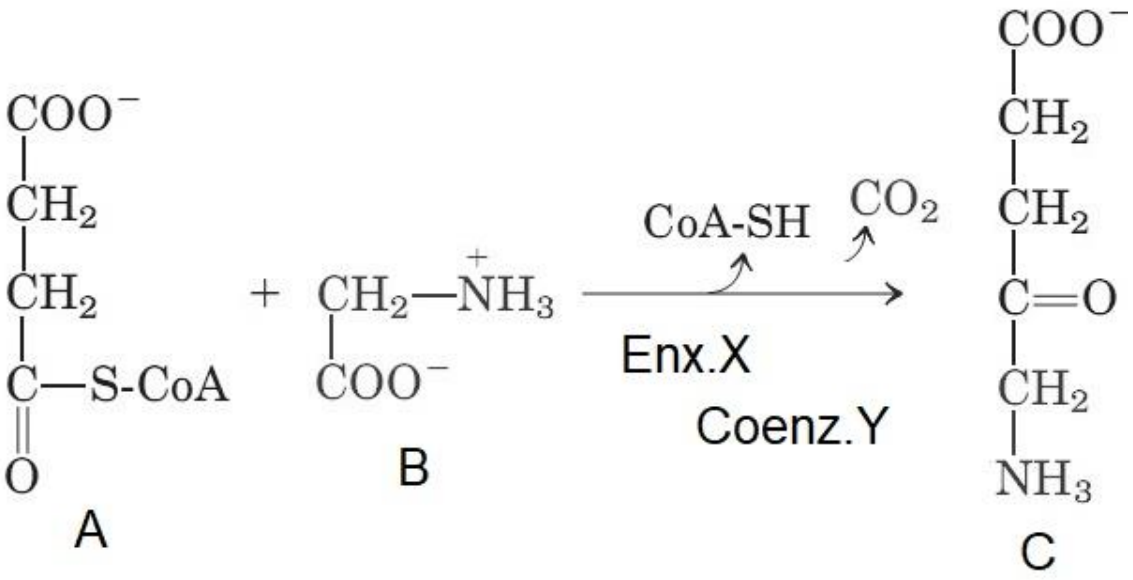
1. Phenylalanine is precursor for serotonin
2. Serotonin, dopamine, histamine are biological amines
3. Serotonin is a biogenic amine synthesized by the decarboxylation of 5-hydroxy-tryptophan
4. Some biological amines include neurotransmitters such as serotonin, dopamine, and norepinephrine
5. The amino acid histidine can be decarboxylated by histidine decarboxylase to form the biogenic amine dopamine

6. Amino acid decarboxylases are enzymes that catalyze the removal of a carboxyl group (-COOH) from an amino acid to form an amide
7. Tyrosine decarboxylase requires pyridoxal-5'-phosphate (PLP) as a cofactor
8. High levels of histamine can cause allergy symptoms
9. Histidine decarboxylase requires biotin as a cofactor
10. Biological amines are organic compounds that serve as the building blocks of proteins

186. What can be stated about heme?

1. The first reaction in the heme synthesis pathway is catalyzed by delta-aminolevulinate synthase
2. The rate limiting step of the heme synthesis pathway is catalyzed by heme synthase
3. Heme exerts a negative feedback effect on its own synthesis pathway
4. Heme contains four pyrrole rings condensed in a linear manner
5. It is synthesized starting from glycine and acetyl-CoA
6. It is synthesized mainly in the bone marrow and liver
7. It is the prostetic group of hemoglobin, myoglobin and cytochromes
8. It is found only in hemoglobin and myoglobin
9. Its precursors are glycine and succinyl-CoA
10. The first enzyme in the pathway, delta-aminolevulinate synthase, is inhibited by lead (Pb)

187. What can be said about the reaction presented in the image?




1. Y is tetrahydrofolate
2. B is glycine
3. X is heme synthase
4. It is the first reaction in the pathway of purine nucleotide synthesis
5. This condensation reaction requires ATP as an energy donor
6. C is delta-aminolevulinate
7. It is part of the urea cycle
8. Y is pyridoxal phosphate (active vitamin B6)
9. A is succinyl-CoA
10. It is the first reaction in the pathway of heme synthesis

188. Select the correct answers about the reaction presented below:A + B → delta-aminolevulinate + CoA-SH + CO2(A is a Krebs cycle intermediate, B is an amino acid):

1. B is alanine
2. This reaction belongs to the pathway of heme synthesis
3. The reaction belongs to the pyrimidine synthesis patway
4. The enzyme that catalyzes this reaction is repressed by heme
5. It is catalyzed by delta-aminolevulinate synthase, which is the regulatory enzyme of the pathway
6. A is acetyl-CoA

7. A is succinyl-CoA
8. B is glycine
9. The enzyme that catalyzes this reaction is regulated by phosphorylation and dephosphorylation
10. It takes place only in the bone marrow

189. Choose the correct statements about heme synthesis:

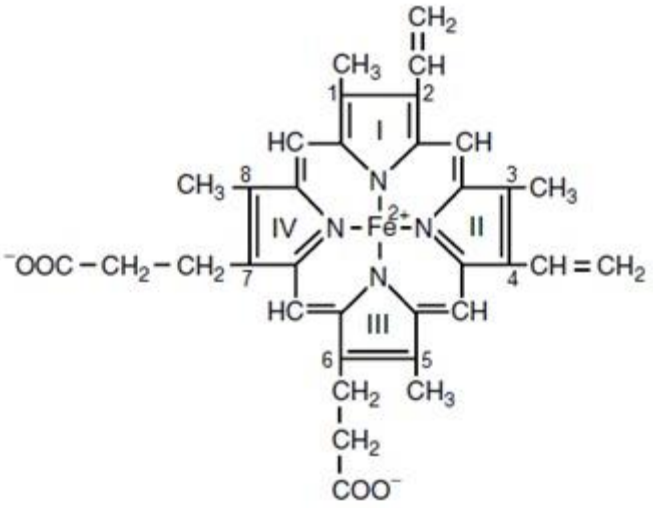
1. It takes place both in the mitochondria and cytosol of red blood cell precursors and liver cells
2. Heme synthase catalyzes the addition of Fe to the protoporphyrinogen molecule
3. Citrulline is an intermediate in the pathway
4. Heme activates the regulatory enzyme of the pathway
5. It takes place in the bone marrow and liver, in the cytosol exclusively
6. Three types of porphyrinogens are intermediates in the pathway
7. Porphobilinogen is an intermediate in the pathway
8. It requires the participation of vitamin B12
9. Heme synthase catalyzes the incorporation of iron into the protoporphyrin molecule
10. The rate-limiting step is catalyzed by delta-aminolevulinate synthase

190. What can be stated about the heme synthesis pathway?

1. Delta-aminolevulinate synthase is allosterically activated by heme
2. Heme synthase is the regulatory enzyme of the pathway
3. The reaction catalyzed by delta-aminolevulinate synthase requires tetrahydrofolate as a cofactor
4. It is inhibited by heme, which represses delta-aminolevulinate synthase
5. The genetic deficiencies of the enzyme in the pathway lead to a group of diseases called porphyrias

6. Porphyrias are diseases caused by enzyme deficiencies in the pathway and their symptoms are caused by the deficiency of intermediates located before the deficient enzyme
7. The first reaction and the last ones occur in the mitochondria
8. Alanine and succinate provide all the atoms of the porphyrin molecule
9. Protoporphyrinogen must be first oxidized to protoporphyrin in order to bind Fe2+
10. In the diseases called porphyrias, the manifestations are caused by the excessive accumulation of the intermediates located before the enzyme blockage

191. Select the correct answers about the compound presented in the image:




1. The regulatory enzyme in the synthesis pathway of this compound is catalyzed by heme synthase
2. It represents protoporphyrin
3. The regulatory enzyme of the synthesis pathway of this compound is covalently regulated and the active form is the dephosphorylated one
4. It is formed of protoporphyrin an iron
5. An intermediate of Krebs cycle and an amino acid provide all the atoms of the molecule, except iron

6. It represents heme
7. It consists of protoporphyrinogen and iron
8. The first reaction in the pathway that synthesizes this compound is catalyzed by delta-aminolevulinate synthase
9. Tetrahydrofolate is involved as a cofactor in the synthesis of this compound
10. The synthesis of this compound is inhibited by heme, through the repression of the regulatory enzyme of the pathway

192. What can be said about bilirubin?

1. Bilirubin is a linear tetrapyrrole
2. Bilirubin is further converted to biliverdin before being released into the blood
3. The formation of bilirubin from heme is accompanied by the release of carbon dioxide
4. It is formed in circulating red blood cells from heme
5. Bilirubin contains four pyrrole rings condensed in a cyclic manner
6. It is formed mainly in the spleen
7. The pathway of bilirubin formation from heme involves the conversion of protoporphyrin to protoporphyrinogen
8. Heme oxygenase catalyzes the first step of heme degradation to bilirubin
9. Unconjugated bilirubin is a hydrophobic molecule
10. It is the end product of heme catabolism

193. What can be said about the catabolism of heme?

1. The carbon atom of the α-methenyl bridge is eliminated as carbon dioxide
2. Most of the iron resulted from degraded heme is excreted to avoid toxic effects
3. The degradation of heme from hemoglobin normally takes place after the red blood cells have reached 60 days of life
4. The first step involves the release of iron, resulting in the formation of protoporphyrin

5. Heme oxygenase catalyzes the conversion of heme to a colorless compound called biliverdin
6. It leads to the release of carbon monoxide
7. It involves the oxidative cleavage of the porphyrin ring
8. Heme from hemoglobin is the only source of bilirubin
9. Biliverdin reductase converts biliverdin to bilirubin by using NADPH
10. Heme oxygenase converts heme to biliverdin

194. Select the correct answers about the heme degradation pathway:

1. Most of the iron that is released from heme will be reutilized
2. Biliverdin reductase completes the pathway transforming biliverdin to bilirubin
3. It takes place mainly in the liver
4. Heme oxygenase converts heme to biliverdin, which still contains the iron atom
5. NADPH is used in the pathway of heme degradation
6. Biliverdin is converted to bilirubin by an oxidation reaction
7. The end product of heme degradation is uric acid
8. The first step is catalyzed by heme oxygenase and its products are biliverdin, iron and CO
9. After synthesis from heme, biliverdin is mostly released into the blood
10. It takes place mainly in the spleen

195. What can be said about the metabolism of bilirubin?

1. Bilirubin is converted to urobilinogen in the liver
2. Conjugated bilirubin is excreted into the bile ducts
3. The bilirubin that is released into the blood by the spleen is transported by albumin
4. Unconjugated bilirubin circulates in a free form in the blood
5. The liver takes up bilirubin from the blood and conjugates it with UDP-glucuronic acid

6. The glucuronyl transferase from the liver catalyzes the reaction: “bilirubin + glucuronic acid → bilirubin glucuronide”
7. The purpose of bilirubin conjugation by the liver is to make it hydrophilic
8. Bilirubin is conjugated with glucuronic acid in the intestine
9. Being a degradation product of heme, bilirubin contains iron
10. In the intestine, bilirubin is transformed into urobilinogen and then stercobilinogen

196. What can be said about the following reaction?Bilirubin + UDP-glucuronic acid → bilirubin glucuronide + UDP:

1. It is catalyzed by bilirubin glucuronide synthase
2. The attachment of glucuronic acid to bilirubin makes it hydrophobic
3. Bilirubin glucuronide circulates into the bloodstream transported by albumin
4. It takes place in the spleen, where bilirubin is formed
5. Bilirubin glucuronide is also named conjugated bilirubin
6. Bilirubin glucuronide is released from the spleen and will be taken up by the liver
7. It is catalyzed by UDP-glucuronyl transferase
8. Bilirubin glucuronide is excreted into the bile ducts and thus reaches the intestine
9. It takes place in the liver
10. While bilirubin is hydrophobic, bilirubin glucuronide is hydrophilic

197. A patient with massive erythrocyte destruction will have the following biochemical disorders:

1. High levels of stercobilin in the feces
2. The absence of bilirubin in urine
3. High levels of both conjugated and unconjugated bilirubin
4. High levels of urobilinogen in urine
5. High levels of conjugated bilirubin in blood
6. The absence of urobilinogen in urine

7. High levels of unconjugated bilirubin in blood
8. The presence of bilirubin in urine
9. Low levels of iron in blood
10. High levels of iron in blood

198. A newborn with a deficiency of glucuronyl-transferase will have:

1. Increased conjugated bilirubin in blood
2. Yellow skin
3. Bilirubin absent in urine
4. Bilirubin present in urine
5. Decreased urobilinogen in urine
6. Increased unconjugated bilirubin in blood
7. Decreased conjugation of bilirubin in the liver
8. Increased formation of stercobilin in the intestine
9. Pale skin
10. Increased levels of both unconjugated and conjugated bilirubin

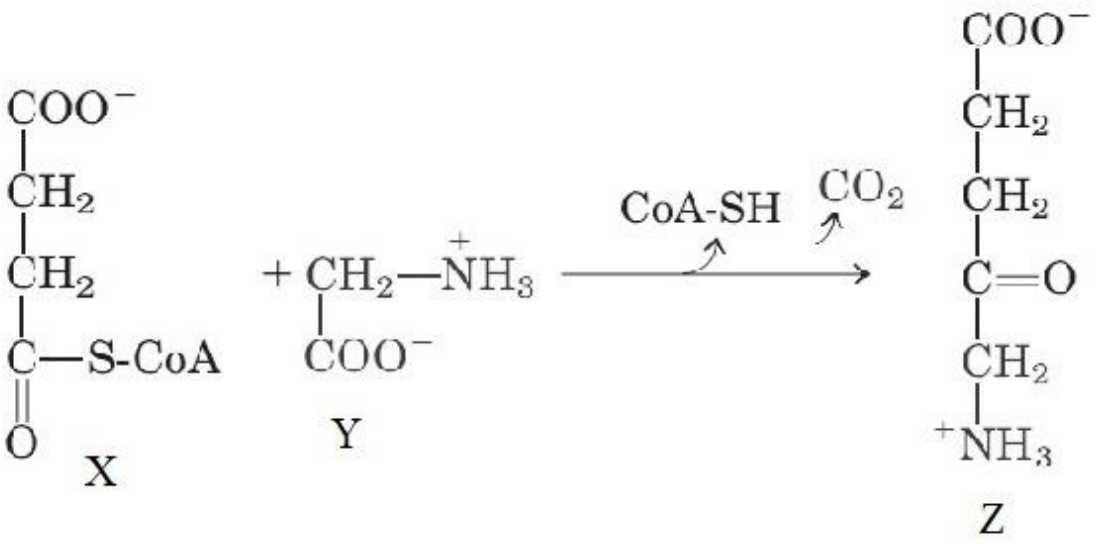
199. When the common bile duct is obstructed by a gallstone, the following changes may occur:

1. Pale stools
2. Urobilinogen absent from urine
3. Increased levels of unconjugated bilirubin in blood
4. Increased levels of urobilinogen in urine
5. The absence of bilirubin in urine
6. Bilirubin present in urine
7. Decreased conjugation of bilirubin by the liver
8. High levels of conjugated bilirubin in blood
9. Yellow skin
10. Increased levels of stercobilin in the feces

200. Which of the following statements about the synthesis of HEME are correct?

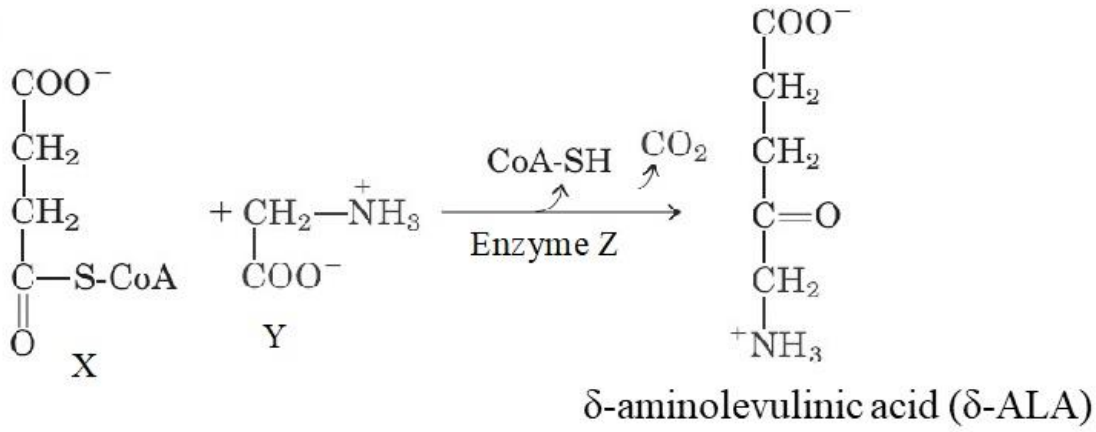
1. Delta-aminolevulinic acid (ALA) is transported to the cytoplasm, where it is converted to porphobilinogen (PBG) by the enzyme ALA dehydratase
2. Major site of heme synthesis regulation is at the level of heme synthase
3. The dehydration of two molecules of delta-aminolevulinic acid (ALA) to form porphobilinogen is catalyzed by the enzyme ALA synthase
4. Uroporphyrinogen III is converted to coproporphyrinogen III by the enzyme uroporphyrinogen III decarboxylase
5. Four molecules of porphobilinogen (PBG) are combined to form a linear tetrapyrrole molecule
6. The first step in heme synthesis takes place in the cytosol
7. Formation of delta-aminolevulinic acid (ALA) is catalyzed by the enzyme ALA synthase
8. Coproporphyrinogen III is transported to the mitochondria, where it is converted to delta-aminolevulinic acid (ALA)
9. The synthesis of heme starts in the mitochondria with the formation of delta-aminolevulinic acid (ALA) from glycine and succinyl CoA
10. The final reaction involves the incorporation of ferrous iron ($Fe^{2+}$) into protoporphyrin in a reaction catalyzed by ALA dehydratase

201. Regarding the following image it can be stated:




1. The reaction is catalyzed by the enzyme delta-aminolevulinate (ALA) synthase
2. The process shown in the image is involved in heme degradation
3. Compound X is acetyl-CoA
4. Compound Y is glycine
5. X is also used in the synthesis of creatine
6. Compound Y is named alanine
7. Compound Z is named arginine
8. Compound Z is named delta-aminolevulinic acid (ALA)
9. Compound X is named succinyl-CoA
10. The process shown in the image is first reaction in heme synthesis

202. In the following image:




1. Compound X is succinyl-CoA
2. Compound X is protoporphyrin IX
3. Enzyme Z catalyzes the condensation of succinyl-CoA with glycine
4. Compound Y is glycine
5. Compound Y is serine
6. Enzyme Z is delta-aminolevulinate (ALA) synthase
7. Delta-aminolevulinic acid (ALA) is obtained by removing the CoA-SH with energy consumption in a reversible reaction
8. This is the rate-limiting step in heme synthesis
9. The process shown in the image is the last reaction in heme synthesis
10. Enzyme Z catalyzes the condensation of succinyl-CoA with alanine

203. Regarding porphyria, it can be stated:

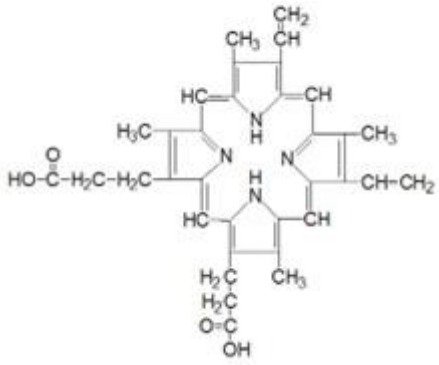
1. Porphyria is caused by a deficiency of vitamin B12
2. Porphyria can be acquired in case of lead (Pb) intoxication, because lead inhibits two enzymes of the heme synthesis pathway
3. Porphyria is a group of rare genetic disorders that affect the production of heme
4. Porphyria is caused by a deficiency of folic acid

5. Porphyria is caused by a deficiency of the enzymes involved in heme degradation
6. Porphyria is caused exclusively by a decreased activity of heme synthase (ferrochelatase)
7. The symptoms of porphyria are caused by excessive accumulation of the intermediates before the deficient enzyme in the pathway
8. Porphyria is caused by enzyme deficiencies in the pathway of heme synthesis
9. In porphyria, the cells do not convert delta-aminolevulinate to heme in a normal manner
10. Porphyria is a common disorder

204. Which of the following statements about the regulation of heme synthesis are correct?

1. The enzyme ferrochelatase (heme synthase), which catalyzes the final step in heme synthesis, is inhibited by heavy metals (e.g., Pb)
2. High levels of iron can lead to a decrease in heme synthesis
3. High levels of heme have an inhibitory effect on delta-ALA synthase
4. Heme synthesis is activated by the end product, heme
5. The key regulatory enzyme of heme biosynthesis in the liver is delta-ALA synthase
6. The rate-limiting step in heme synthesis is the formation of delta-aminolevulinic acid (ALA) from succinyl-CoA and glycine
7. The availability of substrates for heme synthesis cannot affect the rate of heme synthesis
8. The enzyme ferrochelatase, which catalyzes the final step in heme synthesis is regulated by covalent regulation
9. The decreased availability of pyridoxal phosphate may affect heme synthesis
10. Heme is a positive modulator for delta-ALA synthase

205. Regarding the compound presented in the image it can be stated:



1. Deficiencies in ferrochelatase (heme synthase) activity can lead to a type of porphyria, which is characterized by an accumulation of protoporphyrin IX
2. In some types of porphyria, there can be an accumulation of the compound
3. It is named protoporphyrin IX
4. It is a molecule composed of a porphyrin ring and an iron atom
5. It is an important intermediate in the biosynthesis of heme
6. It is composed of four pyrrole rings that are connected by methenyl (methine) bridges
7. It is heme
8. It is an intermediate in the cholesterol synthesis
9. It is the product of the reaction catalyzed by delta-aminolevulinate (ALA) synthase
10. Is the final product of heme degradation

206. Which of the following statements about the heme degradation are correct?

1. Biliverdin reductase reduces biliverdin using NADH
2. Biliverdin is reduced to bilirubin by the enzyme biliverdin reductase
3. Heme oxygenase cleaves the heme molecule to produce bilirubin, iron, and carbon monoxide
4. After the first step of heme degradation, the products will be: biliverdin, free iron and carbon monoxide

5. Bilirubin is transported to the liver, where it is conjugated with glucuronic acid
6. Bilirubin is converted into biliverdin by the enzyme biliverdin reductase
7. Conjugated bilirubin (bilirubin glucuronide) is nonpolar
8. Defects in glucuronyl transferase can lead to a buildup of bilirubin in the body, causing jaundice
9. The first step of heme degradation is the cleavage of the heme molecule by the heme oxygenase
10. The first step of heme degradation is catalyzed by the enzyme biliverdin reductase

207. About the bilirubin, we can state:

1. Conjugated bilirubin is water-soluble and can be eliminated from the body
2. Ring cleavage by heme oxygenase system produces biliverdin
3. Bilirubin is a waste product of heme catabolism
4. Biliverdin reductase uses pyridoxal-5'-phosphate (PLP) as a cofactor
5. Jaundice may be caused by a deficiency of heme oxygenase
6. Biliverdin is transported to the liver, where it is conjugated with glucuronic acid
7. Conjugated bilirubin is not water-soluble
8. There are two forms of bilirubin: unconjugated (also called indirect) bilirubin and conjugated (also called direct) bilirubin
9. Unconjugated bilirubin is transported in plasma by transferrin
10. Unconjugated bilirubin is not water-soluble and is transported in the blood bound to albumin

208. Regarding the heme degradation it can be stated:

1. Elevated levels of bilirubin in the blood can lead to a condition called jaundice
2. Bilirubin is a yellow pigment that is produced during the breakdown of heme

3. Urobilinogen is produced in the liver
4. Heme oxygenase requires biotin as a cofactor
5. In newborns, high levels of bilirubin can lead to a condition called neonatal jaundice
6. Unconjugated bilirubin is not soluble in water and cannot be eliminated from the body
7. Biliverdin reductase reduces biliverdin using molecular oxygen (O2)
8. In the intestine, UDP-glucuronyl transferase forms conjugated bilirubin
9. Bilirubin is transported to the liver, where it is conjugated with glucuronic acid by the enzyme UDP-glucuronyl-transferase
10. Stercobilin is excreted in urine in significant amounts

209. Regarding the bilirubin it can be stated:

1. Conjugated bilirubin is an insoluble (nonpolar) form of bilirubin that has been modified in the liver by the addition of glucuronic acid
2. In hepatocytes, UDP-glucuronyl transferase adds two glucuronic acid molecules to bilirubin to produce bilirubin diglucuronide derivative (conjugated bilirubin)
3. Heme oxygenase is an enzyme that catalyzes the breakdown of heme into biliverdin, iron, and carbon monoxide
4. In the intestine, bilirubin is converted into urobilinogen
5. Bilirubin is essential for oxygen transport
6. Biliverdin is the precursor of bilirubin
7. Bilirubin is a bile acid
8. Heme oxygenase is an enzyme that catalyzes the breakdown of heme into bilirubin, iron, and carbon monoxide
9. Urobilinogen can be oxidized to form stercobilin
10. Reduction of biliverdin is a rate limiting step for the hepatic bilirubin metabolism

210. Regarding the jaundice it can be stated:

1. Hemolytic jaundice is characterized by an increase in conjugated bilirubin
2. Pre-hepatic jaundice is caused by excessive breakdown of red blood cells, leading to an increase in unconjugated bilirubin in the blood
3. Jaundice is characterized by yellowing of the skin and eyes
4. Jaundice associated with hepatitis may result from an inability to convert urobilinogen to stercobilin
5. Neonatal jaundice results from accelerated hemolysis and/or immature hepatic system
6. Unconjugated hyperbilirubinemia is caused by obstruction of the bile ducts, which prevents bilirubin from being excreted into the small intestine
7. Jaundice associated with hepatitis may result from an inability to form glucuronyl conjugates of bilirubin
8. Crigler-Najjar syndrome is caused by mutations in the gene encoding biliverdin reductase
9. Obstruction of the bile ducts can lead to jaundice, as bilirubin cannot be properly excreted from the body
10. Gilbert syndrome is caused by a genetic deficiency of delta-aminolevulinate (ALA) synthase

211. About the jaundice, we can state:

1. Hemolytic jaundice is characterized by an increase in unconjugated bilirubin and an increase in urobilinogen in the urine
2. Jaundice is a condition characterized by yellowing of the skin
3. Neonatal jaundice is treated by phenobarbital and phototherapy
4. Jaundice associated with hepatitis may result from an inability to convert bilirubin to urobilinogen
5. Obstruction of the bile ducts lead to an increase in unconjugated bilirubin
6. Jaundice with increased conjugated bilirubin is caused by obstruction of the bile ducts
7. The pale color of the feces in obstructive jaundice is caused by the absence of bilirubin

8. In case of hemolytic jaundice, conjugated bilirubin is eliminated in the urine, which is hyperchromic
9. Hepatic (hepatocellular) jaundice is caused by liver damage or disease
10. Jaundice with increased conjugated bilirubin is caused by excessive breakdown of red blood cells

212. Regarding the heme degradation, the following statement(s) is/are correct:

1. Unconjugated bilirubin is secreted into the bile
2. In the liver, bilirubin is conjugated with glucuronic acid by the enzyme UDP-glucuronyl-transferase to form water-soluble bilirubin glucuronide, which can be excreted
3. Dysfunction in any of the enzymes or processes involved in bilirubin metabolism can lead to various types of jaundice
4. In the intestine, bilirubin is converted by bacterial enzymes to urobilinogen, which can be further metabolized to form stercobilin, the pigment responsible for the brown color of feces
5. Heme degradation involves the breakdown of the heme molecule into biliverdin, carbon monoxide (CO), and iron
6. Gilbert syndrome is a genetic condition characterized by a deficiency in albumin
7. Defects in heme synthesis can lead to hyperbilirubinemia
8. Individuals with Gilbert syndrome have elevated levels of conjugated bilirubin in their blood
9. Biliverdin is rapidly converted to bilirubin by the enzyme biliverdin reductase, and bilirubin is then transported to the liver bound to albumin
10. Biliverdin is conjugated with glucuronic acid to form bilirubin diglucuronide

213. Which of the following is a nucleotide?

1. GDP
2. Thymidine

3. Guanine
4. Thymidine triphosphate
5. Cytosine monophosphate
6. Adenosine monophosphate
7. Guanosine diphosphate
8. Uridine
9. UMP
10. Adenine

214. Choose the correct statements about the de novo synthesis of purine nucleotides:

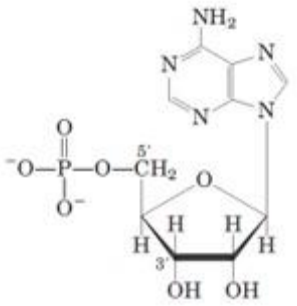
1. It is located in the cytosol
2. It starts with ribose-5-phosphate
3. It is located in the mitochondria
4. The pathway produces adenine and guanine
5. The pathway produces directly nucleotides (not free bases)
6. The pathway produces CMP and UMP
7. The pathway produces adenosine and guanosine
8. It is energy-consuming
9. It starts with ribose
10. It produces ribonucleotides, from which deoxyribonucleotides can be obtained

215. What can be said about the de novo synthesis of purine nucleotides?

1. PRPP (phospho-ribosyl-pyrophosphate) is an intermediate of the pathway
2. It first synthesizes IMP (the nucleotide containing hypoxanthine)
3. The conversion of IMP to AMP uses GTP as an energy source
4. It uses asparagine, glutamate and glycine
5. It produces adenine and guanine, which then react with ribose-5-phosphate to generate AMP and GMP
6. It uses aspartate, glutamine and glycine
7. It is inhibited by high amounts of AMP and GMP

8. It first produces nucleosides, which are then phosphorylated to generate nucleotides
9. It starts with deoxyribose-5-phosphate
10. It is inhibited by CTP and UTP

216. What can be said about the compound presented in the image?



1. It is adenine-monophosphate
2. Its synthesis from IMP (by the de novo pathway) uses GTP as an energy donor
3. Two C atoms of the purine ring are provided by a derivative of tetrahydrofolate
4. It is AMP
5. It is found in DNA structure
6. It is cyclic AMP
7. It may be obtained by the reaction between adenine and PRPP (phospho-ribosyl-pyrophosphate)
8. The nitrogenous base contained in this compound is degraded to urea
9. It may be converted to ATP
10. It is adenosine

217. What can be said about ribonucleotide reductase?

1. It converts GTP to GDP
2. It provides the building blocks for DNA synthesis
3. It uses FADH2 as a hydrogen donor

4. It uses a protein cofactor called thioredoxin
5. It uses nucleoside diphosphates as substrates
6. It converts ribonucleotides to deoxyribonucleotides
7. It converts ADP to deoxy-ADP
8. It is involved in the degradation of ribonucleotides
9. It provides the building blocks for RNA synthesis
10. It converts ribonucleotides to ribonucleosides

218. What can be said about PRPP (phospho-ribosyl-pyrophosphate)?

1. It is used in the salvage pathways of purine bases
2. It is a product of the pentose-phosphate pathway
3. It can be used to convert adenine to AMP
4. It is an intermediate in the pathway of purine nucleotide degradation
5. It is used for the synthesis of pyrimidine nucleotides
6. The pathway of pyrimidine nucleotide synthesis begins with PRPP
7. It is an intermediate in the de novo pathway of purine nucleotide synthesis
8. It is formed by the reaction between ribose-5-phosphate and ATP
9. It is synthesized by the transfer of a pyrophosphate group from GTP to ribose-5-phosphate
10. It is an allosteric inhibitor of the key enzyme in the pathway of purine synthesis

219. Select the correct answer(s) about the process of purine degradation:

1. Guanine from GMP is converted to xanthine
2. Hypoxanthine derives from guanine oxidation
3. The final product of this process is xanthine
4. Uric acid derives from xanthine under the action of xanthine oxidase
5. Xanthine oxidase converts hypoxanthine to xanthine
6. It involves the cleavage of the purine ring
7. Xanthine oxidase is inhibited by allopurinol

8. Xanthine oxidase, the last enzyme in the pathway, is activated by a hypoxanthine analog
9. Uric acid, the final product of this process, can be reutilized in the salvage pathway for the synthesis of purine nucleotides
10. Adenine from AMP is converted to hypoxanthine

220. What can be said about the pathway of pyrimidine synthesis?

1. It first produces carbamoyl phosphate, using NH3 as a nitrogen donor
2. It starts with ribose-5-phosphate, on which the pyrimidine ring is built
3. It is feedback inhibited by the final products (UTP) and activated by PRPP
4. The first reaction is catalyzed by carbamoyl-synthetase II, which uses glutamine as a substrate that donates the -NH2 from the amide group
5. PRPP (phospho-ribosyl-pyrophosphate) participates in the process
6. The first intermediate is carbamoyl-phosphate, which is produced by the same carbamoyl-phosphate synthetase that also acts in the urea cycle
7. It starts with the synthesis of the pyrimidine ring, followed by the addition of ribose-5-P
8. A derivative of tetrahydrofolate donates 1 C atom of the pyrimidine ring
9. It produces AMP and GMP
10. The atoms of the pyrimidine ring are provided by glutamine, aspartate and CO2

221. Select the correct statements about TMP synthesis:

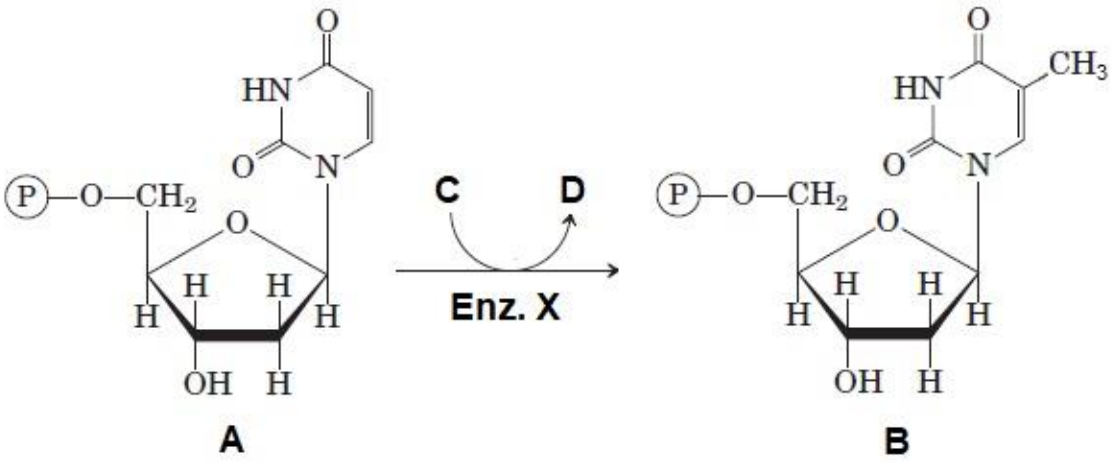
1. It is catalyzed by thymidylate synthase
2. The donor of the methyl group for TMP synthesis is N5-methyl tetrahydrofolate

3. Dihydrofolate is the second product of the reaction, and is reduced to FH4 by dihydrofolate reductase
4. TMP is used in DNA synthesis
5. It is synthesized from dUMP
6. It is feedback inhibited by excess TMP
7. dUDP is converted to TDP, which is then dephosphorylated to TMP
8. TMP is used in RNA synthesis
9. TMP is a purine nucleotide
10. The donor of the methyl group is N5,N10-methylene-tetrahydrofolate

222. What can be said about the salvage pathways of nitrogenous bases?

1. They are much more rapid and economic than the de novo pathways for nucleotide synthesis
2. Guanine is converted to GMP by GMP synthase
3. The general reaction is “Base + PRPP → Nucleotide + PPi”
4. Comprise the reaction “Base + ribose-5-phosphate → Nucleotide”
5. They convert the free nitrogenous bases to the corresponding nucleotides
6. They consist of a sequence of two reactions
7. They are catalyzed by phosphoribosyl transferases
8. They convert nitrogenous bases to nucleosides, which are then phosphorylated to nucleotides
9. APRT (adenine phosphoribosyl transferase) converts adenine to AMP
10. Through such a pathway, guanine is converted to GTP

223. What can be said about the reaction presented in the image?




1. C is N5-methyl tetrahydrofolate
2. X is dUMP methylase
3. B is dTMP
4. D is tetrahydrofolate
5. B is found in RNA structure
6. C is N5,N10-methylene-tetrahydrofolate
7. D is dihydrofolate
8. A is dUMP
9. A is a ribonucleotide
10. X is thymidylate synthase

224. Functions of nucleotides in biochemistry are:

1. NAD+ (nicotinamide adenine dinucleotide) and FAD (flavin adenine dinucleotide), serve as cofactors for enzymes
2. They are important components of coenzymes such as Coenzyme A
3. cGMP (cyclic guanosine monophosphate) acts as second messenger
4. All nucleotides have the same sugar component
5. Nucleotides cannot function as signaling molecules
6. All nucleotides serve exclusively as building blocks for DNA and RNA

7. cAMP (cyclic adenosine monophosphate) acts as second messenger
8. Nucleotides are not involved in energy storage or transfer in cells
9. Nucleotides are only found in DNA and not in RNA
10. Building blocks of DNA and RNA

225. Which of the following are required for the synthesis of purine nucleotides?

1. Glutamate
2. Glutamine
3. Aspartate
4. Oxygen
5. Glycine
6. Carbamoyl phosphate synthetase II
7. Biotin
8. Acyl-CoA
9. Carbon dioxide ($CO_2$)
10. Tetrahydrofolate ($FH_4$)

226. In the de novo synthesis of purine nucleotides:

1. The first step is the formation of phosphoribosyl pyrophosphate (PRPP) from ribose 5-phosphate
2. The formation of phosphoribosyl pyrophosphate (PRPP) from ribose 5-phosphate, requires the enzyme PRPP synthetase and ATP as a substrate
3. Hypoxanthine is linked to ribose-1-phosphate
4. The second step is the formation of 5-phosphoribosylamine (PRA) from PRPP
5. IMP can be further converted to other purine nucleotides, such as AMP and GMP
6. The purine ring is assembled first, and then linked to ribose phosphate
7. ATP is necessary for the conversion of IMP to AMP
8. AMP is converted to GMP

9. The synthesis of IMP requires the cofactor tetrahydrofolate (THF or FH4)
10. The purine ring is synthesized first followed by the attachment of ribose-5-P

227. Regarding phosphoribosyl pyrophosphate (PRPP), we can state the following:

1. PRPP participates in both the de novo synthesis and the salvage pathway of purine nucleotide synthesis
2. PRPP is needed in the mitochondria for the synthesis of citrulline
3. Purine nucleotides are activators for the enzyme PRPP synthetase
4. PRPP synthesis is catalyzed by the enzyme xanthine oxidase
5. PRPP is synthesized from uric acid
6. PRPP levels are regulated by the enzyme PRPP synthetase, which is subject to feedback inhibition by purine nucleotides
7. PRPP is a precursor for the synthesis of both purine and pyrimidine nucleotides
8. PRPP is involved in the urea synthesis
9. PRPP is synthesized from ribose-5-phosphate
10. In the purine nucleotide biosynthesis pathway, PRPP is the precursor for the formation of inosine monophosphate (IMP)

228. Regarding the uric acid we can state:

1. A deficiency of xanthine oxidase leads to the deposition of uric acid crystals in the joints
2. High levels of uric acid in the blood can lead to a condition called hyperuricemia
3. Uric acid is partly degraded in humans to allantoin
4. The drug allopurinol (a purine analog) inhibits the enzyme xanthine oxidase
5. The reaction that generates the end product uric acid is catalyzed by xanthine oxidase
6. Uric acid formation contributes to the energy production of the cell

7. The reaction that generates the uric acid is catalyzed by phosphoribosyl-pyrophosphate (PRPP) synthetase
8. An increased synthesis or decreased excretion of uric acid can lead to gout
9. It is formed by the degradation of adenine and guanine
10. Intermediate of uric acid formation is orotic acid

229. The precursors in the biosynthesis of pyrimidines are:

1. Aspartate
2. Glutamate
3. Methotrexate
4. Amino-isobutyrate
5. Alanine
6. Carbamoyl phosphate
7. Glutamine
8. Uric acid
9. Carbon dioxide (CO2)
10. Phosphoribosyl-pyrophosphate (PRPP)

230. Regarding the biosynthesis of pyrimidine nucleotides, we can state:

1. Carbamoyl phosphate synthetase II (CPS II) is also involved in urea cycle
2. Pyrimidine biosynthesis occurs both through the de novo pathway and the salvage pathway
3. The second step involves the condensation of carbamoyl phosphate and aspartate to form dihydroorotate
4. Orotic acid is converted to AMP
5. Biosynthesis of pyrimidine nucleotides takes place in mitochondria
6. The first step in the de novo synthesis of pyrimidines is the formation of IMP
7. Carbamoyl phosphate synthetase II (CPS II) is activated by UTP
8. In the de novo pathway, pyrimidine rings are synthesized from simple precursors such as glutamine, aspartate
9. The first step is the formation of carbamoyl phosphate

10. Carbamoyl phosphate synthetase II (CPS II) catalyzes the first step in biosynthesis of pyrimidines

231. Regarding the biosynthesis of pyrimidine nucleotides, we can state:

1. The rate-limiting step of pyrimidine biosynthesis is the formation of carbamoyl phosphate from ammonia and bicarbonate
2. The end product of the pathway is uric acid
3. The first enzyme in the pathway is aspartate transcarbamoylase
4. The pyrimidine ring is synthesized first, in the form of orotic acid
5. The key enzyme in the pathway is carbamoyl phosphate synthetase II (CPS II)
6. Inhibitors of ATP synthesis are used in cancer therapy
7. The first nucleotide in the biosynthesis of pyrimidine is OMP
8. OMP is decarboxylated to form uridine 5'-monophosphate (UMP)
9. TMP exists only as ribonucleotide
10. UMP is the precursor of CTP and dTMP

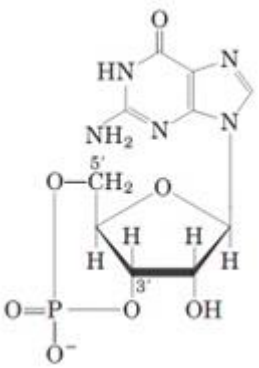
232. Regarding UMP (uridine monophosphate), we can state:

1. UMP contains a pyrimidine base
2. UMP is formed by decarboxylation of orotidine monophosphate (OMP)
3. UMP is precursor for the synthesis of ATP
4. UMP is precursor for the synthesis of UTP
5. UMP is released by the hydrolysis of RNA
6. UMP contains a purine base
7. UMP reacts with methylene-dihydrofolate to generate dTMP
8. UMP contains an N-glycosidic linkage
9. UMP is not involved in the biosynthesis of pyrimidines
10. Its precursor is dUMP

233. Regarding the metabolism of purine and pyrimidine nucleotides, we can state:

1. Adenine phosphoribosyl-transferase (APRT) is responsible for the reutilization of adenine
2. The formation of PRPP from adenosine diphosphate (ADP) and ribose is catalyzed by ribonucleotide reductase
3. Imbalances in nucleotide metabolism can lead to a variety of diseases, including gout and Lesch-Nyhan syndrome
4. The degradation of purine nucleotides produces uric acid
5. The degradation of pyrimidine nucleotides results in the formation of uric acid
6. The conversion of ADP to dADP is catalyzed by ribonucleotide reductase
7. In the degradation pathway, purine nucleotides are first phosphorylated to their respective nucleosides
8. The conversion of xanthine to uric acid is catalyzed by carbamoyl phosphate synthetase II (CPS II)
9. ATP is necessary for the conversion of IMP to AMP
10. The biosynthesis of pyrimidines and purines demands a high energy input

234. What can be stated about the compound presented in the image, involved in hormone signaling?




1. It is adenosine monophosphate
2. It activates a tyrosine-protein kinase called protein kinase A
3. It may be hydrolyzed to 3’-AMP
4. It is formed from ATP in a reaction catalyzed by adenylate cyclase

5. Its role is to activate protein kinase A
6. It is formed from ATP by the action of phosphodiesterase
7. It is a second messenger
8. It is cyclic AMP
9. It is involved in the signaling pathways of many water-soluble hormones
10. It is involved in the signaling pathways of lipid-soluble hormones

235. The following hormones/enzymes lead to the formation of second messengers:

1. Cortisol
2. Phosphodiesterase
3. Many water-soluble hormones
4. Phospholipase C
5. Thyroid hormones
6. Adenylate cyclase
7. Lipid-soluble hormones
8. Protein kinase A
9. Glucagon
10. Adrenaline (epinephrine)

236. Which of the following is/are involved in the signaling pathway triggered by the binding of adrenaline (epinephrine) to the beta-adrenergic receptor?

1. Protein kinase A
2. cAMP-dependent protein kinase
3. Adenylate cyclase
4. Gs protein
5. Gq protein
6. Phospholipase C
7. Inositol triphosphate
8. Cyclic AMP
9. Diacylglycerol
10. Calcium ions

237. Which of the following is/are involved in the signaling pathway triggered by the binding of adrenaline (epinephrine) to the alpha1-adrenergic receptor?

    1. Gs protein
    2. Inositol triphosphate
    3. Calcium ions
    4. Adenylate cyclase
    5. Gq protein
    6. cAMP-dependent protein kinase
    7. Cyclic AMP
    8. Phospholipase C
    9. Protein kinase A
    10. Diacylglycerol

238. Which of the following statements about the signaling pathways of water-soluble hormones is/are correct?

    1. cAMP is a second messenger
    2. Adenylate cyclase directly activates protein kinase A
    3. Protein kinase A phosphorylates proteins at serine/threonine residues
    4. Adenylate cyclase converts AMP to cAMP
    5. Gs protein activates phospholipase C
    6. Protein kinase A leads to the formation of cAMP
    7. Phospholipase C leads to the formation of inositol triphosphate (IP3) and diacylglycerol (DAG)
    8. Gs protein activates adenylate cyclase
    9. Diacylglycerol (DAG) activates protein kinase A
    10. Adenylate cyclase converts ATP into cAMP

239. Choose the correct statement(s) about adenylate cyclase (AC) and phospholipase C (PLC):

    1. Activation of AC is triggered by the binding of certain hormones to membrane receptors, followed by the activation of the Gs protein

2. The products of PLC are diacylglycerol (DAG) and inositol triphosphate (IP3)
3. The substrate of AC is ATP
4. AC is part of the signaling pathway that is triggered by many lipid-soluble hormones
5. PLC is activated by the Gq protein
6. AC is a serine/threonine protein kinase
7. The product of AC is cAMP
8. PLC is a second messenger involved in hormone signaling pathways
9. AC is directly activated by certain membrane receptors that have bound specific hormones
10. PLC converts CTP to cyclic CMP

240. What can be said about the G proteins (involved in hormone signaling pathways)?

1. In the active form, they have GTP attached to the alpha subunit
2. In the active form, they have GDP attached to the alpha subunit
3. They are located in the plasma membrane of cells
4. Gs protein is activated by adenylate cyclase
5. Gs protein activates adenylate cyclase and Gq protein activates phospholipase C
6. They are enzymes that form second messengers
7. They are activated by membrane receptors that have bound their specific hormones
8. They are monomeric proteins
9. Gs protein leads to an increase in the concentration of inositol triphosphate inside cells
10. After being activated, they activate specific enzymes that lead to the formation of second messengers

241. Select the correct statement(s) about the mechanism of action of lipid-soluble hormones:

1. It does not involve second messengers

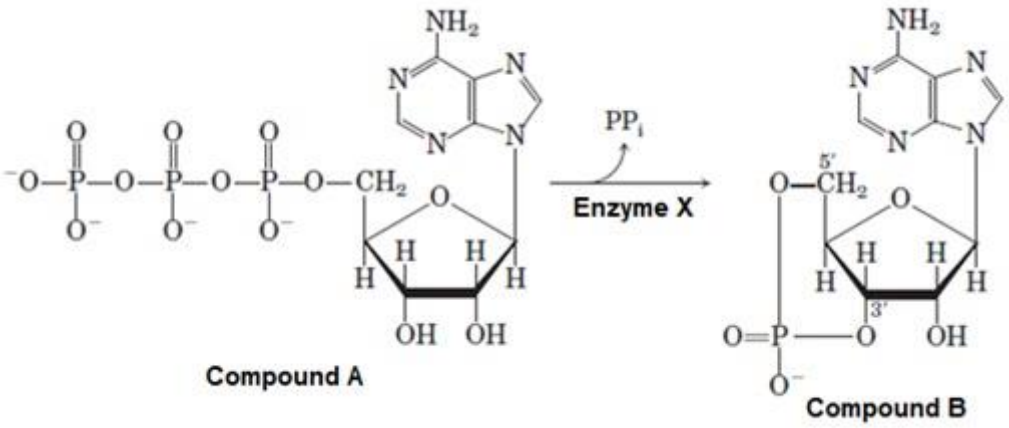
2. The hormone-receptor complex binds to specific regulatory sequences in DNA
3. After binding the lipid-soluble hormones, the receptors phosphorylate specific proteins on tyrosine residues
4. It involves the participation of adenylate cyclase and protein kinase A
5. The ultimate effect of lipid-soluble hormones is a change in the amount of specific proteins/enzymes inside the target cells
6. The receptors of lipid-soluble hormones are located in the cytosol or in the nucleus
7. The receptors of lipid-soluble hormones are located in the plasma membrane of target cells
8. The hormone-receptor complex activates or inhibits the transcription of specific genes
9. It involves the utilization of second messengers
10. The hormone-receptor complex binds to specific sequences in mRNA and change the rate of translation

242. Choose the correct answer(s) regarding water-soluble hormones (WH) and lipid-soluble hormones (LH):

1. The ultimate effect of WH is usually a change in the activity of enzymes by phosphorylation, while that of LH is a change in the amount of specific proteins
2. LH-receptor complex binds to hormone response elements (HRE) located in the membrane of endoplasmic reticulum
3. LH always lead to an increase in the transcription rates of specific genes
4. WH have their receptors in the plasma membrane, while LH have their receptors in the cytosol or nucleus
5. WH use second messengers, while LH do not
6. Both WH and LH have their receptors in the plasma membrane
7. Both WH and LH utilize second messengers to exert their effects
8. The LH-receptor complex binds to regulatory sequences in DNA called hormone-response elements (HRE)
9. LH exert their effects by changing the expression of specific genes

10. LH bind to receptors located in the mitochondria

243. What can be said about the reaction presented in the image?




1. Enzyme X is protein kinase A
2. B is 5’-AMP
3. Enzyme X is activated by several hormones by means of Gq protein
4. It is part of a hormone signaling pathway and enzyme X is activated by the Gs protein
5. Compound B activates protein kinase A
6. It is part of a signaling pathway that is used by several lipid-soluble hormones
7. B is a second messenger that activates protein phosphatase-1
8. B is cyclic AMP
9. A is ATP
10. X is adenylate cyclase

244. Which of the following statements about the hormones are correct?

1. Hormones have the same effect on all cells in the body
2. Hormones can be classified into several categories based on their chemical structure, including steroid hormones, peptide hormones
3. They are biochemical messengers that have important roles in regulating metabolism and other processes in the body

4. Peptide hormones are made up of chains of amino acids and include hormones such as insulin and glucagon
5. Hormones are only released in response to stress
6. Hormones act on target cells by means of specific receptors
7. Hormones only affect target cells that are in close proximity to the site of hormone production
8. Steroid hormones are derived from cholesterol and include hormones such as testosterone, estrogen, and cortisol
9. All hormones are proteins
10. All hormones are transported in the blood by specific proteins

245. Regarding the hormones it can be stated:

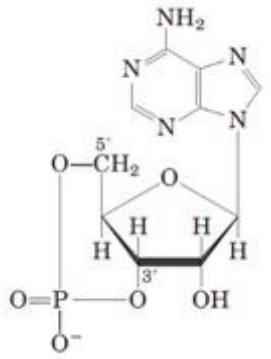
1. A given hormone may act upon more than one target tissue
2. Water-soluble hormones include peptide hormones and hormones derived from amino acids
3. Thyroid hormones are water soluble hormones
4. A given hormone receptor is able to bind several hormones having different structures
5. Endocrine hormones act on the same cells that produce them
6. Peptide and protein hormones bind to receptors on the cell surface, triggering second messenger systems that activate various signaling pathways within the cell
7. Endocrine hormones are produced by specialized cells in endocrine glands and are released into the bloodstream, which carries them to their target cells
8. Steroid hormones pass through the cell membrane and bind to intracellular receptors, leading to changes in gene expression
9. Epinephrine (adrenaline) circulates in blood bound to albumin
10. Testosterone and estrogen are derived from amino acids

246. Regarding the hormones it can be stated:

1. Insulin is derived from the amino acid tyrosine
2. Steroid hormones act via intracellular receptors
3. Eicosanoids are a group of peptide hormones

4. Peptide hormones act via cell surface receptors
5. Epinephrine (adrenaline) and glucagon are steroid hormones
6. Peptide hormones can easily cross cell membranes to interact with intracellular receptors
7. Steroid hormones are derived from cholesterol and are lipid-soluble
8. Aldosterone and cortisol are water-soluble hormones
9. Peptide hormones are made up of chains of amino acids and are water-soluble
10. Steroid hormones are derived from cholesterol and include sex hormones such as testosterone, estrogen, and progesterone

247. Regarding the following compound, it can be stated:




1. It is synthesized by the enzyme adenylate cyclase
2. It is GMP structure
3. It is guanine phosphate
4. It is 3',5'-cyclic guanosine monophosphate
5. When the cellular level of cAMP increases, protein kinase A is activated
6. It is formed from ATP by the enzyme adenylate cyclase
7. It is cAMP structure
8. It represents adenine phosphate
9. It is AMP
10. It is a second messenger

248. Which of the following statements about hormones are correct?

1. Water soluble hormones act through membrane receptors
2. Water soluble hormones are hormones that can diffuse through the lipid bilayer of the cell membrane and bind to intracellular receptors inside the cell
3. Lipid soluble hormones travel in the bloodstream bound to transport proteins
4. Water soluble hormones bind to their specific intracellular receptors, forming a hormone-receptor complex
5. Water soluble hormones include glucagon and epinephrine (adrenaline)
6. Examples of water soluble hormones include steroid hormones
7. Lipid soluble hormones are hormones that are soluble in the blood and other extracellular fluids
8. Water soluble hormones travel freely in the bloodstream
9. Lipid soluble hormones diffuse across cell membranes
10. Lipid soluble hormones are hormones that bind to membrane-bound receptors on target cells

249. Regarding the plasma membrane receptors, it can be stated:

1. All plasma membrane receptors are G protein-coupled receptors
2. A given receptor specifically binds a given hormone
3. They most commonly bind steroid hormones
4. The receptor kinase phosphorylates an amino acid residue on the receptor (auto phosphorylation) or an associated protein
5. They bind the specific hormones with high affinity
6. They directly activate enzymes that generate second messengers, such as adenylate cyclase and phospholipase C
7. Triiodotyronine acts by an auto-phosphorylation process of the tyrosine residues from the intracellular part of the receptor
8. All plasma membrane receptors lead to the activation of adenylate cyclase

9. Receptor tyrosine kinases have an extracellular domain that binds to ligands and an intracellular domain that possesses tyrosine kinase activity
10. Heptahelical receptors (which contain 7-membrane spanning -helices) are themost common type of plasma membrane receptor

250. Regarding the G proteins we can state:

1. G proteins are heterotrimeric, composed of three subunits: alpha, beta, and gamma
2. The alpha subunit of G proteins contains a site for binding guanine-nucleotides (GDP/GTP)
3. The alpha subunit can then interact with and regulate the activity of effector proteins, such as adenylate cyclase or phospholipase C
4. Are a family of proteins located on the inner face of the plasma membrane
5. G proteins can directly activate enzyme activity without the involvement of a second messenger
6. G proteins are only activated by hormone binding to intracellular receptors
7. G proteins only activate adenylate cyclase
8. G proteins are composed of three identical subunits
9. The activity of the alpha subunit is terminated by the intrinsic GTPase activity of the alpha subunit, which hydrolyzes GTP to GDP
10. G proteins are always in an active state

251. Which of the following statements about the cAMP are correct?

1. cAMP acts as a transcription factor in the nucleus
2. Adenosine-5’-triphosphate (ATP) is substrate for cAMP synthesis, by the adenylate cyclase
3. cAMP is a type of hormone
4. cAMP is degraded by adenylate cyclase
5. cAMP binds directly to DNA to activate gene expression
6. cAMP can be degraded by the enzyme phosphodiesterase

7. cAMP activates protein kinase A by binding to its regulatory subunits
8. cAMP is the intracellular messenger for glucagon
9. cAMP is a lipid molecule
10. Phosphodiesterase hydrolyzes cAMP to its inactive form, AMP

252. Which of the following intermediates and enzymes is/are found in the pathway of epinephrine (adrenaline) synthesis from tyrosine?

1. A methyl-transferase
2. Phenylalanine
3. Dopaquinone
4. Monoamine oxidase (MAO)
5. Dopamine
6. Phenylalanine hydroxylase
7. DOPA
8. Metanephrine
9. Norepinephrine
10. DOPA decarboxylase

253. Select the correct statement(s) about epinephrine (adrenaline):

1. It is synthesized from tryptophan
2. Its action through beta-adrenergic receptors involves cAMP as a second messenger
3. Its synthesis pathway involves dopamine as an intermediate
4. It binds to adrenergic receptors on target cells, which are of four types: α1, α2, β1, β2
5. It is synthesized from tyrosine
6. It may be converted to norepinephrine
7. Its synthesis pathway involves the action of monoamine oxidase (MAO)
8. It acts upon target cells through G-protein coupled receptors
9. It is synthesized in the adrenal cortex glands
10. Its action through beta-adrenergic receptors involves DAG and IP3 as second messengers

254. Which of the following is/are involved in the synthesis of epinephrine (adrenaline)?

1. Pyridoxal phosphate (active vitamin B6)
2. Tyrosine hydroxylase
3. Tetrahydrobiopterin (BH4)
4. Vitamin C
5. Vitamin B12
6. NADPH
7. Epinephrine synthase
8. Tetrahydrofolate (FH4)
9. Tryptophan hydroxylase
10. SAM (S-adenosyl-methionine)

255. What can be said about epinephrine (adrenaline)?

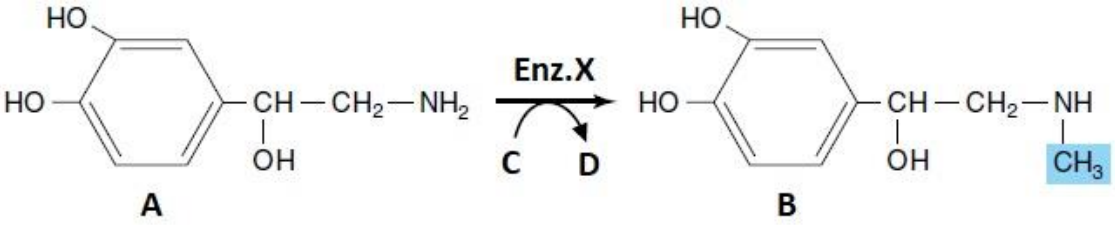
1. It is a hypoglycemiant hormone
2. It regulates glycogen metabolism both in the liver and muscle
3. It acts upon target tissues by using intracellular receptors
4. It regulates glycogen metabolism in the liver, but not in the muscle
5. Its effects upon glycogen metabolism in the liver are opposed to those of glucagon
6. Its immediate precursor is norepinephrine
7. It is produced in the adrenal medulla glands
8. It is involved in the stress response of the organism
9. It is a peptide hormone
10. It can be synthesized starting from phenylalanine or tyrosine

256. Epinephrine (adrenaline) has the following effect(s):

1. Stimulation of gluconeogenesis
2. Activation of lipolysis in the adipose tissue
3. Adaptation to stress situations
4. Inhibition of glucose synthesis from amino acids
5. Inhibition of glycogenolysis
6. Increase of glycemia

7. Stimulation of protein degradation in the muscle
8. Decrease of glycemia
9. Inhibition of triglyceride degradation in the adipose tissue
10. Stimulation of glycogen degradation

257. What can be stated about the reaction presented in the image?




1. C is SAM (S-adenosyl-methionine)
2. It takes place in the adrenal medulla
3. Enzyme X is a methyl transferase
4. B is epinephrine (adrenaline)
5. It is part of the cortisol synthesis pathway
6. It takes place in the adrenal cortex glands
7. A is norepinephrine (noradrenaline)
8. C is N5-CH3 FH4 (N5-methyl-tetrahydrofolate)
9. A is thyroxine
10. B is norepinephrine (noradrenaline)

258. What can be said about epinephrine (adrenaline):

1. Its synthesis pathway involves DOPA as an intermediate
2. Is synthesized from the amino acid tyrosine
3. Its synthesis pathway involves tryptamine as an intermediate
4. It acts by binding to adrenergic receptors that lead to the stimulation of adenylate cyclase or phospholipase C
5. It stimulates glycogen synthesis
6. It has a stimulatory effect on lipolysis in the adipose tissue
7. It stimulates protein degradation to provide metabolic fuel for the stress response

8. Is synthesized from the amino acid tryptophan
9. It exerts its effects by binding to intracellular receptors that lead to an increase in cAMP levels
10. It stimulates glycogen degradation in the muscle and liver

259. Select the correct answers about glucagon:

1. It stimulates glycogen degradation in the muscle
2. It stimulates glycogen degradation in the liver
3. It is synthesized by the alpha cells of the pancreas
4. It modifies the activity of enzymes by promoting their dephosphorylated forms (in the context of covalent regulation)
5. It is synthesized by the beta cells of the pancreas
6. It is a peptide hormone
7. It inhibits gluconeogenesis
8. It stimulates lipolysis in the adipose tissue
9. It is secreted in response to hypoglycemia
10. Its secretion is stimulated by hyperglycemia

260. What can be said about glucagon?

1. It is secreted after carbohydrate meals
2. It is a water-soluble hormone
3. It uses cAMP as a second messenger to exert its effects
4. It acts by nuclear receptors
5. It is synthesized from the amino acid glutamate
6. It stimulates glycogenolysis both in the liver and muscle
7. It changes the activity of enzymes by promoting their phosphorylated forms (in the context of covalent regulation)
8. It is a hyperglycemiant hormone
9. Its metabolic effects are opposite to those of epinephrine (adrenaline)
10. It is secreted during fasting periods

261. What can be said about insulin?

1. It is produced in the beta cells of the pancreas
2. It acts upon target cells by binding to receptors that have protein-phosphatase activity
3. It stimulates triglyceride degradation in the adipose tissue
4. It is secreted in response to hyperglycemia
5. Its metabolic effects are similar to those of glucagon because these two hormones are both produced by the pancreas
6. It is secreted in response to hypoglycemia
7. It stimulates glycogenolysis
8. It inhibits lipolysis in the adipose tissue
9. It stimulates glycogen synthesis
10. It results from the cleavage of proinsulin to insulin and C-peptide

262. Choose the correct statements regarding insulin:

1. It stimulates triglyceride breakdown in the adipose tissue
2. It acts through membrane receptors that have tyrosine kinase activity
3. It stimulates gluconeogenesis
4. It stimulates glucose uptake in the muscle and adipose cells
5. It is composed of a single polypeptide chain
6. It is a peptide hormone composed of two chains, A and B, connected by disulfide bridges
7. It acts by binding to intracellular receptors
8. It regulates the activity of several enzymes by stimulating protein phosphatase-1
9. It is secreted during fasting
10. It inhibits glycogen breakdown

263. The following can be stated about insulin:

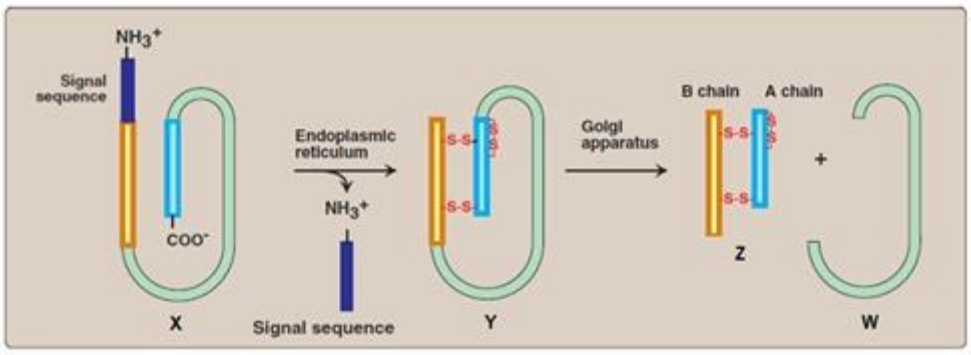
1. It acts by binding to tetrameric membrane receptors
2. It inhibits gluconeogenesis
3. It is initially synthesized as a larger precursor called pre-pro-insulin
4. It stimulates glucose synthesis from amino acids

5. It has metabolic effects that are opposed to those of glucagon
6. It inhibits triglyceride synthesis in the adipose tissue
7. It is secreted in response to hypoglycemia
8. Many of its metabolic effects are exerted by promoting the phosphorylated forms of enzymes (in the context of covalent regulation)
9. It stimulates glycolysis
10. Its effects on glycogen metabolism are similar to those that are exerted through beta-adrenergic receptors

264. Select the correct statement(s) about the mechanism of insulin action:

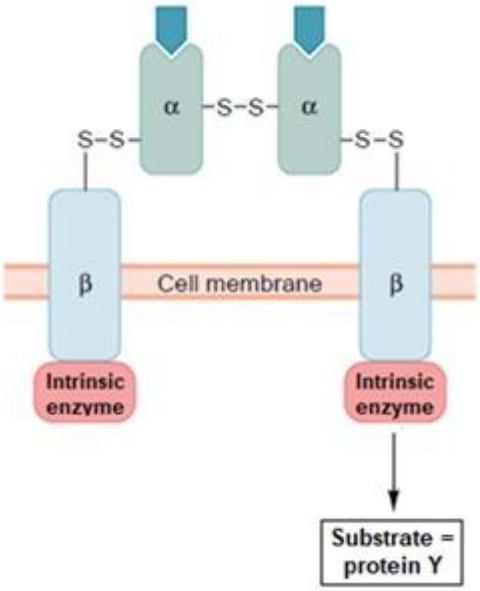
1. The insulin receptor is coupled to the Gs protein
2. The insulin receptor phosphorylates intracellular proteins called IRS (insulin receptor substrates)
3. The insulin receptor is a monomeric membrane protein
4. The insulin signaling pathway is similar to that of other water-soluble hormones, like glucagon and adrenaline
5. Insulin leads to the stimulation of protein kinase A
6. The insulin receptor has intrinsic tyrosine kinase activity
7. The insulin receptor is composed of four subunits: two alpha and two beta
8. Insulin uses cAMP as a second messenger to exert its effects
9. The insulin signaling pathway leads to the stimulation of protein phosphatase-1
10. Insulin regulates the activity of enzymes by promoting their dephosphorylated forms

265. The image below represents the process by which a hormone (Z) is synthesized. What can be stated about this process?




1. X is preproinsulin
2. It takes place in the alpha cells of the pancreas
3. Z is a lipid-soluble hormone
4. The process takes place in the beta cells of the pancreas
5. Y is proinsulin
6. The A and B chains must be dissociated for the hormone to become active
7. Hormone Z is released into the blood during fasting periods
8. W is C-peptide
9. Z is insulin
10. It represents glucagon synthesis

266. The image below represents the specific receptor of a hormone. Select the correct statement(s):




1. The disulfide bridges must be broken for the receptor to be able to bind the hormone
2. The signaling pathway that is initiated by this receptor leads to the stimulation of protein phosphatase-1
3. The intrinsic enzyme is protein phosphatase
4. The ligand for this receptor is a lipid-soluble hormone
5. The intrinsic enzyme contained in the beta subunits is tyrosine kinase
6. Protein Y is called IRS (insulin receptor substrate)
7. Protein Y is protein kinase A
8. The alpha subunits bind insulin
9. It is the insulin receptor
10. It is the beta-adrenergic receptor

267. Regarding insulin, it can be stated:

1. Insulin promotes the uptake and storage of glucose, amino acids, and fatty acids in target tissues
2. Insulin is produced by the adrenal gland
3. Insulin binds to insulin receptors on the surface of target cells, leading to a cascade of intracellular signaling events

4. Insulin decreases blood glucose levels by stimulating gluconeogenesis in the liver
5. Insulin is a steroid hormone
6. Insulin secretion is regulated by blood glucose levels
7. Insulin has no effect on lipid metabolism
8. High glucose levels stimulate insulin release and low glucose levels inhibit it
9. Insulin is a hormone that plays a critical role in regulating glucose homeostasis
10. Insulin is secreted by the alpha cells of the pancreas

268. Which of the following statements about insulin are correct?

1. Insulin resistance is a condition in which the body produces too much insulin
2. Insulin stimulates lipolysis in adipose tissue
3. It transfers the signal into the cell through a receptor that has tyrosine kinase activity
4. Insulin is a lipid-soluble hormone
5. It is initially synthesized as a single-chain pre-pro-insulin
6. Glucose is the key regulator of insulin secretion by the pancreatic beta cell
7. Cleavage of an internal fragment from proinsulin generates the C peptide
8. Insulin is synthesized and secreted by the adrenal gland
9. Insulin stimulates glycogenolysis
10. Proinsulin is a polypeptide containing the A and B fragments connected by the C-peptide

269. Which of the following statements about the insulin secretion are correct?

1. Insulin secretion increases after carbohydrate meals
2. Insulin is a lipid-soluble hormone
3. Elevated plasma arginine stimulates insulin secretion
4. Insulin binds to a receptor with tyrosine phosphatase activity

5. Insulin secretion is increased by gastro-intestinal hormones, e.g., GLP-1
6. Insulin secretion increases during fasting periods
7. Insulin primarily acts to increase blood glucose levels
8. Insulin secretion is stimulated by certain amino acids
9. Insulin secretion is stimulated by high levels of glucagon
10. Increased plasma glucose levels stimulate insulin secretion

270. Regarding the function of insulin, we can state:

1. In the adipose tissue insulin stimulates lipogenesis
2. The place of insulin production is pancreas, inside the α-cells
3. It stimulates the breakdown of fatty acids
4. Insulin is produced using insulinase
5. In the liver insulin stimulates glycogenogenesis
6. In the muscle insulin stimulates the uptake of amino acids and their incorporation into proteins
7. In the liver insulin stimulates glycolysis
8. In the liver insulin inhibits gluconeogenesis
9. Insulin inhibits glycogen synthesis in the liver
10. It stimulates lipolysis

271. About insulin, we can state:

1. It is the primary hormone responsible for increasing blood glucose levels
2. Insulin secretion is stimulated by low blood glucose levels
3. Insulin acts on the liver to stimulate the breakdown of glycogen into glucose
4. Stimulates glycogen synthesis
5. Insulin molecule results from the cleavage of proinsulin
6. Inhibits lipolysis
7. It is a hormone produced by the alpha cells of the pancreas
8. Its secretion is stimulated by hypoglycemia
9. Has a special membrane receptor with 4 subunits: 2 alpha and 2 beta

10. The first form in which it is synthesized is called preproinsulin

272. The correct statements about the insulin are:

1. Proinsulin is the precursor molecule of insulin
2. It is produced by the alpha cells of the pancreas
3. For signaling, insulin uses a pathway that contains protein kinases
4. It increases the number of GLUT4 transporters for glucose transport into the cells
5. Insulin acts by means of receptors coupled with G-protein
6. It inactivates hormone-sensitive lipase
7. Insulin stimulates the breakdown of glycogen in the liver and muscle
8. It increases lipolysis in adipose tissue
9. Insulin has no effect on protein synthesis in the body
10. It activates HMG-CoA reductase

273. The correct statements about the insulin receptor are:

1. Insulin receptor mediates the action of insulin
2. Insulin receptor is a G protein-coupled receptor
3. Insulin receptor is located on the surface of mitochondria
4. Insulin receptor is a transmembrane receptor composed of four beta subunits
5. Insulin receptor is located in the plasma membrane of cells
6. Insulin receptor mediates the action of glucagon
7. Insulin receptor is a transmembrane receptor composed of two alpha and two beta subunits
8. Insulin stimulates the translocation of GLUT4 glucose transporters from the cytosol to the plasma membrane
9. Insulin receptor is a tyrosine kinase receptor
10. By autophosphorylation the insulin receptor is inactivated and becomes insulin resistant

274. Which statements about the insulin receptor are NOT true?

1. The insulin receptor has intrinsic tyrosine phosphatase activity
2. Binding of insulin to the alpha subunits induces the activation of tyrosine kinase function in the beta subunits
3. The insulin receptor is a tyrosine kinase receptor
4. The insulin receptor is essential for glucose homeostasis
5. The insulin receptor is a G protein-coupled receptor
6. The insulin receptor is expressed in many tissues, including liver, muscle, and adipose tissue
7. The insulin receptor is not essential for glucose homeostasis
8. The insulin receptor is activated by binding to glucagon
9. The insulin receptor has intrinsic tyrosine kinase activity
10. The insulin receptor is expressed only in adipose tissue

275. Regarding the metabolic effects of insulin on lipid metabolism, we can state:

1. Promotes lipid breakdown
2. Increases triglycerides degradation
3. Insulin inhibits hormone-sensitive lipase
4. Increases triglycerides synthesis
5. Decreases triglycerides degradation
6. Decreases triglycerides synthesis
7. Insulin increases the transport and metabolism of glucose into adipocytes
8. Decreases fatty acid synthesis
9. Insulin activates hormone-sensitive lipase
10. Stimulates fatty acid synthesis (liver) by activating acetyl-CoA carboxylase

276. Glucagon is a pancreatic hormone that has several biochemical effects on the body, including:

1. Stimulation of glycogenolysis
2. Inhibition of lipolysis
3. Increase of glycemia
4. Stimulation of lipolysis in the adipose tissue

5. Inhibition of glycogenolysis
6. Inhibition of gluconeogenesis
7. Inhibition of glycogenesis
8. Stimulation of glycogenesis
9. Stimulation of glycolysis
10. Stimulation of gluconeogenesis

277. The pancreatic hormone glucagon has the following biochemical effects:

1. Glucagon is one of the hyperglycemic hormones
2. Glucagon is a hormone produced by the beta cells of the pancreas
3. Glucagon promotes the synthesis of fatty acids and triglycerides in adipose tissue
4. Glucagon inhibits the breakdown of glycogen into glucose in the liver
5. Glucagon suppresses glycolysis, the breakdown of glucose into pyruvate, in the liver
6. Promotes the breakdown of stored triglycerides
7. Glucagon binds to specific receptors on liver cells and activates a signaling pathway that leads to the breakdown of glycogen
8. Glucagon stimulates the storage of glucose as glycogen in the liver
9. Glucagon stimulates the production of glucose from non-carbohydrate precursors
10. Glucagon promotes the uptake of glucose by muscle cells

278. What can be stated about glucagon?

1. Glucagon is released in response to high blood glucose levels
2. Glucagon promotes lipogenesis in adipose tissue
3. In the liver, glucagon decreases hepatic glucose production
4. Glucagon promotes glycolysis in the liver
5. In the liver, glucagon increases hepatic glucose production
6. Glucagon stimulates glycogenesis

7. It stimulates the degradation of glucose deposits in the liver
8. It is composed of amino acids
9. It acts by increasing cAMP concentration in the target cells
10. It acts by means of a membrane receptor coupled with the Gs protein

279. Epinephrine, also known as adrenaline, is a hormone produced by the adrenal glands. It has several effects on various systems of the body, including:

1. Stimulation of glycogen degradation in the muscle
2. Inhibition of glycogen degradation in the muscle
3. Adaptation to stress situations
4. Epinephrine stimulates the breakdown of glycogen to glucose
5. Inhibition of lipolysis in the adipose tissue
6. Promotes the breakdown of fat (lipolysis) in adipose tissue
7. Decrease of glycemia
8. It stimulates hepatic glycogen synthesis
9. Epinephrine decreases heart rate
10. Increase of glycemia

280. Correct statements regarding catecholamine degradation are:

1. Catecholamine degradation only occurs in the kidneys
2. Catecholamine degradation is not an important regulatory mechanism for maintaining physiological balance
3. The final product of epinephrine degradation (VMA) is eliminated in the urine
4. The final product of adrenaline degradation is vanillyl-mandelic acid (VMA)
5. Monoamine oxidase (MAO) is an enzyme that catalyzes the oxidative deamination of monoamines, including the catecholamines
6. Catechol-O-methyl transferase (COMT) is involved in the degradation of adrenaline (epinephrine)

7. Adrenaline and noradrenaline are eliminated in the urine without prior metabolization
8. Adrenaline is converted to noradrenaline and the latter is subjected to the action of monoamine oxidase (MAO) and catechol-O-methyl transferase (COMT)
9. Catecholamine degradation occurs only in the liver
10. Catecholamines are a class of neurotransmitters and hormones that include dopamine, norepinephrine (noradrenaline), and epinephrine (adrenaline)

281. Correct statements regarding catecholamines are:

1. Catecholamines are synthesized from the amino acid tyrosine
2. The rate-limiting step in epinephrine biosynthesis is catalyzed by tyrosine hydroxylase
3. Catecholamines act on cholinergic receptors
4. Catecholamines bind to membrane receptors that have tyrosine kinase activity
5. Adrenaline acts by means of heptahelical receptors coupled with various types of G-proteins
6. Catecholamines bind to adrenergic receptors on target cells
7. Catecholamines biosynthesis is dependent on cytochrome P450
8. Catecholamines only bind to steroid receptors
9. The final product of catecholamine degradation is vanillyl mandelic acid
10. Can easily pass through the phospholipid plasma membrane of target cells

282. What can be said about the lipid-soluble hormones?

1. Steroid hormones (e.g., cortisol) are produced from cholesterol
2. The adrenal medulla produces lipid-soluble hormones
3. They bind to receptors located in the plasma membrane of target cells
4. They act upon target cells by using intracellular receptors
5. They directly influence the process of mRNA translation

6. They circulate in plasma bound to proteins
7. They act at the level of transcription of specific genes
8. Thyroid hormones derive from tyrosine residues in thyroglobulin
9. Adrenaline (epinephrine) is a lipid-soluble hormone
10. Their role is to allosterically activate specific enzymes

283. Which of the following hormones bind to intracellular receptors:

1. Androgens and estrogens
2. Adrenaline (epinephrine)
3. Insulin
4. Aldosterone
5. Water-soluble hormones
6. Norepinephrine (noradrenaline)
7. Glucagon
8. Cortisol
9. Thyroxine
10. Triiodothyronine (T4)

284. What can be said about steroid hormones?

1. They derive from cholesterol
2. The adrenal cortex produces cortisol and aldosterone
3. The regulatory step in their synthesis is the first reaction that converts cholesterol to pregnenolone
4. They derive from fatty acids
5. They circulate in plasma bound to transport proteins
6. They exert their effects on target cells by binding to intracellular receptors
7. They are water-soluble hormones
8. They are taken up inside cells by receptor-mediated endocytosis
9. They directly bind to regulatory sequences in DNA to influence gene transcription
10. They act upon target cells by means of second messengers (e.g., cAMP)

285. Choose the correct statement(s) about cortisol:

1. It acts by using intracellular receptors
2. Its synthesis is inhibited by excess ACTH
3. Its synthesis is stimulated by ACTH from the anterior pituitary
4. It is produced by the adrenal medulla
5. High levels of plasma cortisol inhibit its own synthesis by a negative feedback mechanism
6. It is produced in the adrenal cortex
7. Cortisol stimulates in target cells the formation of cAMP from ATP
8. It stimulates gluconeogenesis
9. It acts by means of membrane receptors
10. It circulates mostly in a free form in plasma

286. What can be said about thyroid hormones?

1. They are lipid-soluble hormones
2. One step in their synthesis is the binding of iodine to tyrosine residues in thyroglobulin
3. They are triiodothyronine (T3) and thyroxine (T4)
4. They decrease the rate of basal metabolism
5. They contain iodine
6. They act by means of intracellular receptors
7. They are monoiodotyrosine (MIT) and diiodotyrosine (DIT)
8. They are peptide hormones
9. They derive from cholesterol
10. They contain iodine bound to phenylalanine residues in thyroglobulin

287. Choose the correct answer(s) about the hormones T3 and T4:

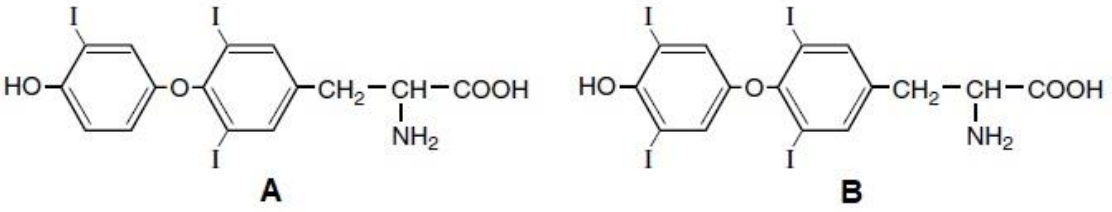
1. They influence the transcription of specific genes
2. They circulate mostly in a free form in plasma because they are water-soluble
3. They are composed of a tyrosine molecule that contain 3 or 4 iodine atoms

4. Their synthesis is inhibited by excess TSH
5. Their circulation in blood is ensured by transporter proteins
6. They are produced in the parathyroids
7. Although derived from an amino acid, they are lipid-soluble hormones
8. They are essential during the development of the organism up to maturity
9. The synthesis of T3 and T4 is stimulated by TSH from the anterior pituitary
10. They are involved in the covalent regulation of enzyme activity

288. Which of the following is/are common features of cortisol and thyroxine (T4)?

1. They exert allosteric effects on specific enzymes
2. They derive from cholesterol
3. They are peptide hormones
4. They circulate mostly in free forms in plasma
5. They directly bind to HRE (hormone response elements) in DNA to regulate gene transcription
6. They lead to changes in the transcription of specific genes in target cells
7. Their synthesis and secretion is stimulated by hormones of the anterior pituitary
8. They have metabolic effects
9. To exert their effects, they must bind to intracellular receptors
10. They are lipid-soluble hormones

289. What can be stated about the compounds presented in the image?



1. A may be converted to B by the adding of an extra iodine atom
2. Their deficiency during childhood does not have any pathologic effect
3. They are produced by the thyroid gland
4. They are hydrofilic hormones that act via plasma membrane receptors
5. They involve the iodination of tyrosyl residues belonging to a protein called thyroglobulin
6. Their synthesis is stimulated by TSH
7. B is named thyroxine
8. They decrease the basal metabolic rate
9. They are hormones produced by the adrenal medulla
10. A is named triiodothyronine (T3)

290. Choose the correct statement(s) about the hormones of the adrenal glands:

1. Cortisol is a mineralocorticoid hormone
2. Steroid hormones are produced by the adrenal medulla
3. Cortisol is a steroid hormone that stimulates gluconeogenesis
4. The synthesis of hormones of the adrenal medulla is stimulated by hormones of the anterior pituitary
5. The adrenal cortex produces hormones derived from cholesterol
6. The hormones of the adrenal cortex act via intracellular receptors
7. The adrenal medulla produces epinephrine (adrenaline)
8. Cortisol and aldosterone exert their effects by regulating the transcription of specific genes
9. Cortisol is a hypoglycemiant hormone
10. Aldosterone and cortisol are produced in the same region of the adrenal cortex

291. Select the correct statement(s) about the lipid-soluble hormones:

1. Cortisol and thyroxine are lipid-soluble hormones
2. Thyroid hormones are synthesized from free tyrosine molecules

3. Their receptors are located in the cytosol or in the nucleus of target cells
4. After binding to specific receptors, the hormone-receptor complex binds to regulatory DNA sequences called HRE (hormone response elements)
5. They act by means of receptors having tyrosine kinase activity
6. They act by changing the conformation of specific proteins in target cells
7. All of them are synthesized from cholesterol
8. They do not have metabolic effects
9. They may derive from cholesterol or from tyrosine residues of a specific glycoprotein
10. They regulate the transcription rate of specific genes

292. The following can be said about glucocorticoid hormones:

1. They derive from cholesterol
2. Cortisol stimulates the secretion of adrenocorticotropin (ACTH)
3. Cortisol has a hypoglycemic effect
4. Cortisol is a glucocorticoid
5. Cortisol inhibits gluconeogenesis
6. Cortisol is a glucocorticoid synthesized in the zona fasciculata of the adrenal cortex
7. Pregnenolone is an intermediate involved in the synthesis of cortisol
8. Cortisol stimulates aldosterone secretion
9. They are synthesized in the adrenal cortex
10. Cortisol has a pro-inflammatory action by activating phospholipase A2

293. The following can be said about thyroid hormones:

1. T4 - is a thyroid hormone also called thyroxine
2. T3 is activated by removing iodine (deiodination)
3. Thyroid hormones stimulate calcium fixation in bones

4. T3 and T4 hormones are released into the blood following the hydrolysis of iodinated thyroglobulin
5. In the synthesis of thyroid hormones, iodine is used for the iodination of phenylalanine residues
6. They are synthesized by the thyroid gland
7. Thyroid hormones have intracellular receptors
8. Thyroid hormones can contain both Iodine and Chlorine ions in their structure
9. Thyroid hormones act on the cyclic AMP pathway
10. T3 - is a thyroid hormone also called triiodothyronine

294. The following can be said about thyroid hormones:

1. Thyroglobulin is a protein rich in phenylalanine residues
2. Thyroid hormones are eliminated exclusively through the intestinal tract
3. Following the attachment of iodine to thyroid hormones, they become water-soluble and can be eliminated through the kidneys
4. Thyroglobulin is a protein rich in tyrosine residues
5. The secretion of T3 and T4 is controlled by a negative feed-back mechanism, by the hypothalamic-pituitary axis
6. The MIT compound is formed by coupling Methionine and Isoleucine with Tyrosine
7. Iodine is used in the iodination of tyrosine residues in thyroglobulin
8. Thyroid hormones circulate bonded to a specific protein (TBG) and to albumin
9. Thyroid hormones are also called parathormones because they come from the parathyroid gland
10. TSH accelerates the synthesis of thyroid hormones T3 and T4 in the thyroid

295. Which statements about the thyroid hormones are true?

1. Thyroid hormones are water-soluble hormones because they derive from tyrosine

2. In the synthesis of thyroid hormones, iodine is used for the iodination of tyrosine residues
3. Thyroid hormones are lipid-soluble hormones
4. The coupling of two DIT (diiodotyrosine) residues generates T4
5. T3 is formed by the reaction between monoiodotyrosine (MIT) and diiodotyrosine (DIT)
6. The decrease in thyroid hormones in the blood will cause the blood concentration of TSH to decrease
7. Thyroid hormones are synthesized by the parathyroid gland
8. Thyroid hormones can circulate coupled with a specific protein (thyroxine binding globulin - TBG)
9. In the synthesis of thyroid hormones, iodine atoms are used in the iodination of tyramine residues
10. By replacing the Iodine ion with the Chlorine ion, the thyroid hormones are inactivated

296. Which statements about the iodine are true?

1. Iodine deficiency can lead to low levels of thyroid hormones in the body
2. In the thyroid hormones T3 and T4, iodine is found covalently bound to phenylalanine
3. Iodine is not important for human health
4. Iodine deficiency can have serious effects on brain development and cognitive function
5. Iodine is a key component in the synthesis of thyroid hormone
6. The thyroid gland is the primary site of iodine uptake and utilization in the body
7. Iodine is a metal
8. Iodine deficiency is the most common cause of hypothyroidism
9. For the synthesis of thyroid hormones, iodine binds to free tyrosine molecules
10. Iodine deficiency can lead to an increase in the basal metabolic rate

297. Which of the following are steroid hormones?

1. Cortisol
2. Epinephrine (adrenaline)
3. Norepinephrine (noradrenaline)
4. Glucagon
5. Progesterone
6. Testosterone
7. Aldosterone
8. Estradiol
9. Triiodothyronine (T3)
10. Insulin

298. Which of the following are nonsteroid hormones?

1. Epinephrine (adrenaline)
2. Cortisol
3. Testosterone
4. Glucagon
5. Thyroxine (T4)
6. Aldosterone
7. Norepinephrine (noradrenaline)
8. Estrogen
9. Progesterone
10. Triiodothyronine (T3)

299. Thyroid hormone synthesis and secretion involve several steps, including:

1. Capture of plasma iodine ions
2. Endocytosis of colloid thyroglobulin in cells
3. Thyroglobulin is a small protein containing a low number of tyrosine residues
4. The release of thyroid-stimulating hormone (TSH) is inhibited by low levels of thyroid hormones in the blood
5. Triiodothyronine (T3) is synthesized by combining two diiodotyrosine (DIT) molecules
6. Thyroid hormone synthesis occurs in the parathyroid gland

7. Oxidation of iodine and iodination of tyrosyl residues from thyroglobulin
8. Iodide uptake into the thyroid follicular cells occurs passively
9. Release of T3 and T4 and their secretion into the blood
10. Coupling of iodinated tyrosine residues and formation of T3 and T4 as components of thyroglobulin

300. Which of the following hormones contain peptide bonds?

1. Triiodothyronine (T3)
2. ACTH
3. Glucagon
4. Estrogen
5. Insulin
6. Testosterone
7. Thyroxine (T4)
8. Parathormone (PTH)
9. Cortisol
10. TSH