Biochemistry Collection

Question Bank with Answers & Explanations

Generated on April 18, 2026

38 Topics • 973 Questions

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Viewer Guide

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DIRECTORY EXPLORER

Click a folder below to open it.

» **01_Introduction**

» **02_Carbohydrate Chemistry**

» **03_Lipid Chemistry**

» **04_Amino Acids and Proteins Chemistry**

» **05_Enzymes**

» **06_Free Radicals_ Antioxidants _ Trace Elements**

» **07_ Genetics**

» **08_Lipid Metabolism**

» **09_Amino Acids Metabolism**

» **10_Heme Metabolism**

» **11_Vitamins**

» **12_Heme Synthesis**

» **13_Amino Acid and Protein Metabolism**

» **14_Clinical Biochemistry**

» **15_Biochemistry**

» **16_Concepts**

» **17_Miscellaneous**

» 01_Introduction

« Back to Parent Directory

Path: 01_Introduction

	Questions	*Answers*
• 001 Previous Year Questions		

» 02_Carbohydrate Chemistry

« Back to Parent Directory

Path: 02_Carbohydrate Chemistry

• 002 Carbohydrates	***Questions***	***Answers***
• 003 Fructose And Galactose Metabolism Hmp Pathway	***Questions***	***Answers***
• 004 Fates Of Pyruvate Pdh Complex Electron Transport Chain	***Questions***	***Answers***
• 005 Glut Transporters And Glycolysis	***Questions***	***Answers***
• 006 Glycogen Metabolism And Glycogen Storage Disorders	***Questions***	***Answers***
• 007 Citric Acid Cycle And Gluconeogenesis	***Questions***	***Answers***
• 008 Previous Year Questions	***Questions***	***Answers***

» 03_Lipid Chemistry

« Back to Parent Directory

Path: 03_Lipid Chemistry

• 009 Lipid Chemistry	***Questions***	***Answers***
• 010 Fatty Acid Synthesis And Oxidation - I	***Questions***	***Answers***
• 011 Cholesterol Metabolism Lipoproteins - I	***Questions***	***Answers***
• 012 Previous Year Questions	***Questions***	***Answers***

» 04_Amino Acids and Proteins Chemistry

« Back to Parent Directory

Path: 04_Amino Acids and Proteins Chemistry

• 013 Protein Chemistry Hemoglobin Myoglobin Epp Chromatography	***Questions***	***Answers***
• 014 Amino Acid Catabolism Urea Cycle And Hyperammonemia	***Questions***	***Answers***
• 015 Amino Acid Metabolic Disorder - I	***Questions***	***Answers***
• 016 Glycine And One Carbon Pool	***Questions***	***Answers***
• 017 Previous Year Questions	***Questions***	***Answers***

» 05_Enzymes

« Back to Parent Directory

Path: 05_Enzymes

• 018 Enzymes	***Questions***	***Answers***
• 019 Previous Year Questions	***Questions***	***Answers***

» 06_Free Radicals_ Antioxidants _ Trace Elements

« Back to Parent Directory

Path: 06_Free Radicals_ Antioxidants _ Trace Elements

	Questions	*Answers*
• 020 Previous Year Questions		

» 07_ Genetics

« Back to Parent Directory

Path: 07_ Genetics

• 021 Dna And Chromosome Biochemistry Structure Replication And Repair	***Questions***	***Answers***
• 022 Rna Transcription Genetic Code Mutation Translation Inhibitors	***Questions***	***Answers***
• 023 Pcr	***Questions***	***Answers***
• 024 Advanced Molecular Techniques Recombinant Dna Technology Microarray Crispr Lac Operon	***Questions***	***Answers***
• 025 Previous Year Questions	***Questions***	***Answers***

» 08_Lipid Metabolism

« Back to Parent Directory

Path: 08_Lipid Metabolism

	Questions	*Answers*
• 026 Previous Year Questions		

» 09_Amino Acids Metabolism

« Back to Parent Directory

Path: 09_Amino Acids Metabolism

	Questions	*Answers*
• 027 Previous Year Questions		

» 10_Heme Metabolism

« Back to Parent Directory

Path: 10_Heme Metabolism

	Questions	*Answers*
• 028 Previous Year Questions		

» 11_Vitamins

« Back to Parent Directory

Path: 11_Vitamins

• 029 Fat-Soluble Vitamins - I	***Questions***	***Answers***
• 030 Water-Soluble Vitamins - I	***Questions***	***Answers***
• 031 Previous Year Questions	***Questions***	***Answers***

» 12_Heme Synthesis

« Back to Parent Directory

Path: 12_Heme Synthesis

• 032 Heme Synthesis And Porphyrias	***Questions***	***Answers***
• 033 Previous Year Questions	***Questions***	***Answers***

» 13_Amino Acid and Protein Metabolism

« Back to Parent Directory

Path: 13_Amino Acid and Protein Metabolism

	Questions	*Answers*
• 034 Previous Year Questions		

» 14_Clinical Biochemistry

« Back to Parent Directory

Path: 14_Clinical Biochemistry

• 035 Previous Year Questions	***Questions***	***Answers***

» 15_Biochemistry

« Back to Parent Directory

Path: 15_Biochemistry

	Questions	*Answers*
• 036 Previous Year Questions		

» 16_Concepts

« Back to Parent Directory

Path: 16_Concepts

	Questions	***Answers***
• 037 Previous Year Questions		

» 17_Miscellaneous

« Back to Parent Directory

Path: 17_Miscellaneous

	Questions	*Answers*
• 038 Previous Year Questions		

Part A — All Questions

• [01_Introduction] 001 Previous Year Questions

« Back to Folder Directory

[01_Introduction] 001 Previous Year Questions — Quick Jump

Q1 (p. 21) *Q2 (p. 21)* *Q3 (p. 22)* *Q4 (p. 22)* *Q5 (p. 22)* *Q6 (p. 23)*
Q7 (p. 23) *Q8 (p. 24)* *Q9 (p. 24)* *Q10 (p. 25)* *Q11 (p. 25)* *Q12 (p. 26)*
Q13 (p. 26) *Q14 (p. 26)*

Q1: In which organelle is DNA present? (Q820383)

A. Mitochondria

B. Lysosome

C. Endoplasmic reticulum

D. Cytoplasm

See Answer in Part B → Q1

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Q2: Zellweger syndrome results from abnormality in formation of: (Q855055)

A. Peroxisomes

B. Lysosomes

C. Mitochondria

D. Nucleus

See Answer in Part B → Q2

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Q3: Refsum disease is caused due to absence of which of the following enzymes? (Q855061)

A. Absence of alpha oxidation in peroxisomes.

B. Absence of beta oxidation in peroxisomes.

C. Absence of alpha oxidation in lysosomes.

D. Absence of beta oxidation in lysosomes.

See Answer in Part B → Q3

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Q4: What is the cause of elevated H+ ions in the intermembrane space of mitochondria? (Q855538)

A. Decreased oxidative phosphorylation

B. Increased ATP production

C. Reduced chemiosmosis

D. Water retention in inner mitochondrial membrane

See Answer in Part B → Q4

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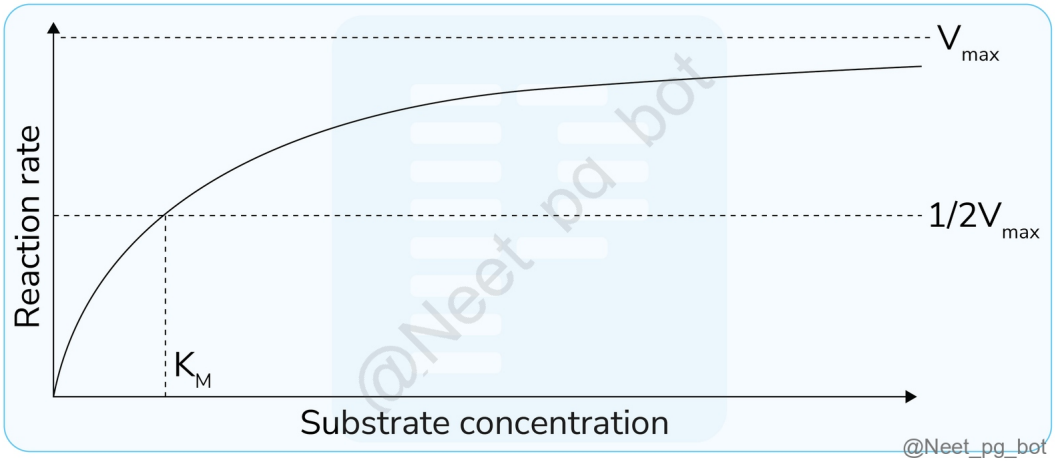
Q5: The enzyme kinetics depicted in the graph is suggestive of (Q855904)

A. Allosteric activation

B. Cooperative binding

C. Michaelis Menten kinetics

D. Sigmoid curve



See Answer in Part B → Q5

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Q6: Which of the following enzymes has the highest catalytic efficiency? (Q856482)

A. Km of an enzyme = 10 micromole and Kcat = 20 per sec

B. Km of an enzyme = 2000 nanomole and Kcat = 50 per sec

C. Km of an enzyme = 2 micromole and Kcat = 200 per sec

D. Km of an enzyme = 4 micromole and Kcat = 200 per sec

See Answer in Part B → Q6

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Q7: Which mechanism is responsible for the inhibition of the acetylcholinesterase enzyme by organophosphorus compounds? (Q856597)

A. Non-competitive inhibition

B. Reversible competitive inhibition

C. Irreversibleinhibition

D. Suicidal inhibition

See Answer in Part B → Q7

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Q8: A TPN bag consists of 100g glucose, 30g amino acids, and 40g lipids. What is the amount of calories delivered? (Q856864)

A. 840 KCal

B. 880 KCal

C. 640 KCal

D. 680 KCal

See Answer in Part B → Q8

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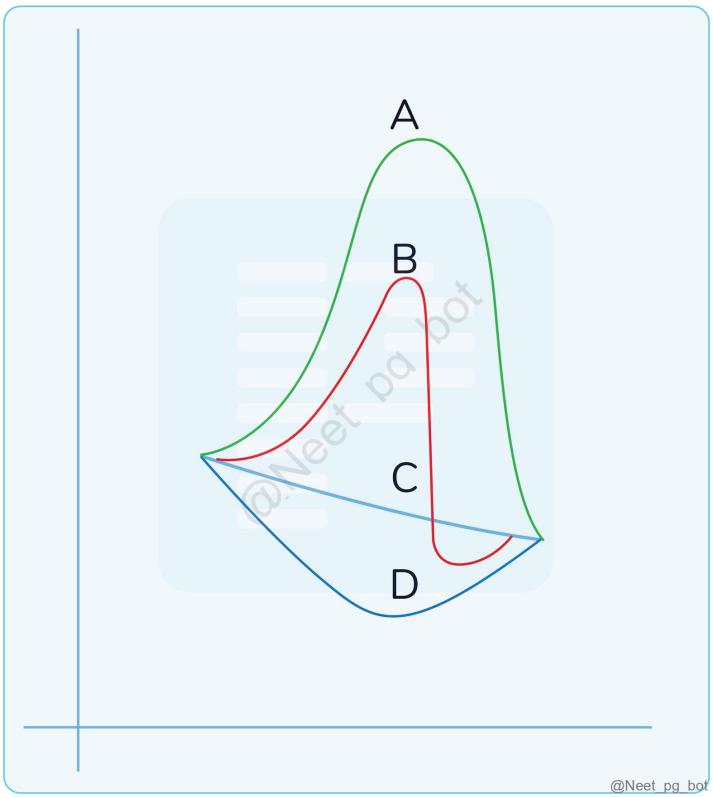
Q9: The Gibbs free energy curve of an enzyme-catalysed reaction is given by: (Q856930)

A. Curve B

B. Curve A

C. Curve D

D. Curve C



See Answer in Part B → Q9

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Q10: The active site of an enzyme is most complementary to? (Q857356)

A. Allosteric activator

B. Substrate

C. Product

D. Transition state

See Answer in Part B → Q10

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Q11: The subcellular organelle which is associated with the killing of viruses and bacteria is? (Q857485)

A. Peroxisome

B. Mitochondria

C. Lysosome

D. Golgi apparatus

See Answer in Part B → Q11

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Q12: In which organelle is DNA present? (Q857869)

A. Mitochondria

B. Lysosome

C. Endoplasmic reticulum

D. Cytoplasm

See Answer in Part B → Q12

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Q13: What is the process of converting unconjugated substances into conjugated forms called? (Q860059)

A. Glycation

B. Glucuronidation

C. Glycosylation

D. Oxidation

See Answer in Part B → Q13

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Q14: Which substance is involved in the conjugation process in the liver? (Q860320)

A. Hyaluronic Acid

B. Glucuronic Acid

C. Gluconic Acid

D. Glycolic Acid

See Answer in Part B → Q14

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• [02_Carbohydrate Chemistry] 002 Carbohydrates

« Back to Folder Directory

[02_Carbohydrate Chemistry] 002 Carbohydrates — Quick Jump

Q1 (p. 28)	*Q2 (p. 28)*	*Q3 (p. 29)*	*Q4 (p. 29)*	*Q5 (p. 29)*	*Q6 (p. 30)*
Q7 (p. 30)	*Q8 (p. 31)*	*Q9 (p. 32)*	*Q10 (p. 33)*	*Q11 (p. 33)*	*Q12 (p. 34)*
Q13 (p. 34)	*Q14 (p. 35)*	*Q15 (p. 35)*	*Q16 (p. 36)*	*Q17 (p. 36)*	*Q18 (p. 36)*
Q19 (p. 37)	*Q20 (p. 38)*	*Q21 (p. 38)*	*Q22 (p. 38)*	*Q23 (p. 39)*	*Q24 (p. 39)*

Q1: Which of the following statements is correct? (Q844975)

A. Glycoproteins contain long mucopolysaccharide chains

B. Proteoglycans have long mucopolysaccharide chains and high carbohydrate content.

C. Glycoproteins have long mucopolysaccharide chains and higher carbohydrate content

D. Proteoglycans contain short oligosaccharide chains and lower carbohydrate content

See Answer in Part B → Q1

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Q2: A 15-year-old girl presents with fatigue and excessive thirst. Urine tests like Benedict's, Barfoed's, and Glucose Oxidase Peroxidase tests are positive. What is the most likely diagnosis? (Q844986)

A. Diabetes Mellitus

B. Essential Fructosuria

C. Lactose due to pregnancy and lactation

D. Classical Galactosemia

See Answer in Part B → Q2

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Q3: A laboratory technician performs an Osazone test on a patient's urine sample and observes fluffy, hedgehog-shaped crystals. Which sugar is most likely present in the urine? (Q844985)

A. Glucose

B. Lactose

C. Maltose

D. Sucrose

See Answer in Part B → Q3

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Q4: What is the reagent used in Barfoed's test to detect monosaccharides? (Q844984)

A. Sodium carbonate, copper sulfate, and sodium citrate

B. Cupric acetate in acetic acid solution

C. Nitric acid

D. Phenolphthalein blue

See Answer in Part B → Q4

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Q5: A new mother comes to the pediatric OPD with her infant as she notices dark staining in diapers after the baby urinates. What is the possible enzyme defect here? (Q844983)

A. Fumarylacetoacetase hydrolase

B. Homogentisic acid oxidase

C. Cystathionine β-synthase

D. Phenylalanine hydroxylase

See Answer in Part B → Q5

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Q6: A urine sample of a diabetes patient was tested to see the presence of reducing sugars. Initial color of solution is blue. What would be the color of the solution after the test? (Q844982)

A. Blue

B. Orange

C. Colourless

D. Black

See Answer in Part B → Q6

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Q7: A 55-year-old woman presents with polydipsia, polyuria, and weight loss. Diabetes mellitus is suspected, and her urine is tested for reducing sugars Image A: Benedict's Test Results Image B: Fehling's Test Results Based on the images provided, which of the following statements is correct? (Q844981)

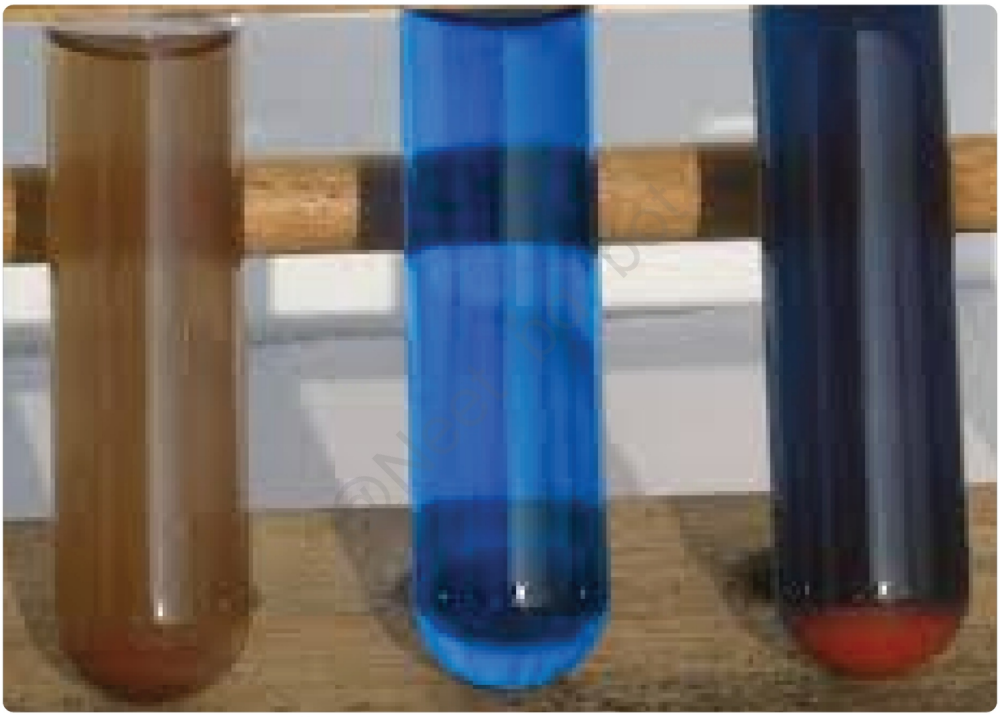
A. Both tests indicate absence of reducing sugars

B. Both tests indicate presence of reducing sugars

C. Benedict's test indicates presence, while Fehling's test indicates absence of reducing sugars.

D. Benedict's test indicates absence, while Fehling's test indicates presence of reducing sugars.





See Answer in Part B → Q7

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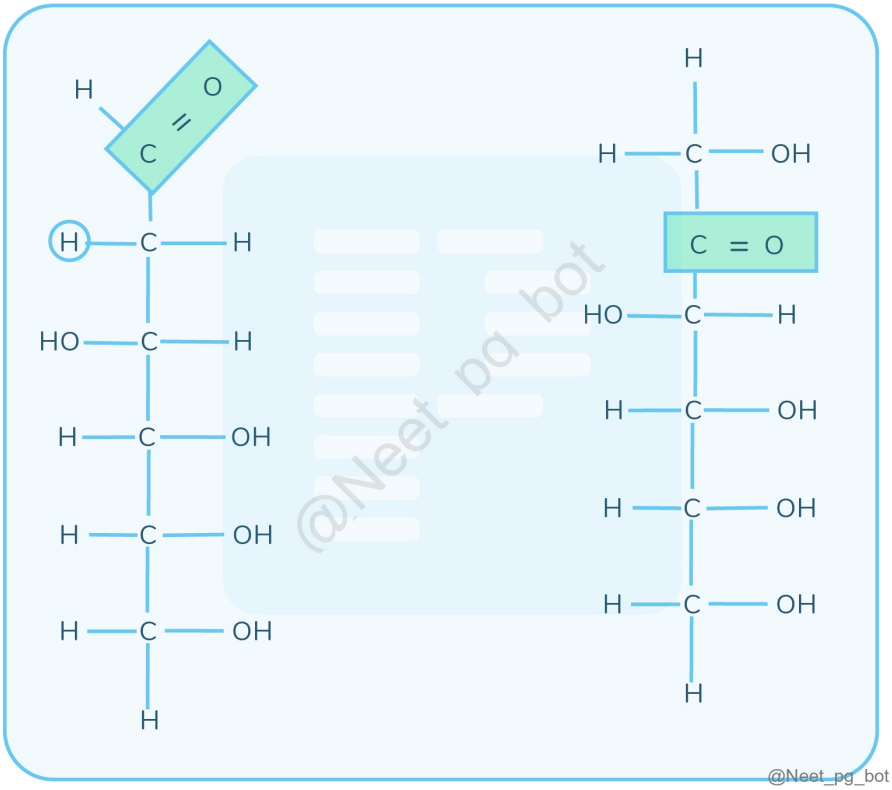
Q8: Which type of isomerism is exhibited by these two compounds? (Q844980)

A. Functional Isomerism

B. Epimerism

C. Enantiomerism

D. Anomerism



See Answer in Part B → Q8

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Q9: Xylose and arabinose are examples of? (Q844979)

A. Optical isomerism

B. Diastereoisomerism

C. Enantiomerism

D. Epimerism

See Answer in Part B → Q9

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Q10: What type of isomerism occurs when two molecules have the same molecular and structural formula but differ in the three-dimensional arrangement of their groups? (Q844978)

A. Optical Isomerism

B. Functional Isomerism

C. Stereoisomerism

D. All of the above

See Answer in Part B → Q10

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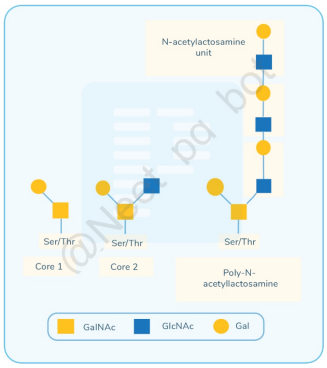
Q11: Identify the type of linkage in the given Structure. (Q844977)

A. O-linked glycoproteins

B. N- N-linked glycoproteins

C. GPI anchored Glycoproteins

D. None of the above



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See Answer in Part B → Q11

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Q12: A 45-year-old with fatigue, abdominal pain, and morning dark urine shows hemoglobinuria. Which test confirms Paroxysmal Nocturnal Hemoglobinuria (PNH)? (Q844976)

A. Bone Marrow Biopsy

B. Flow Cytometry

C. Serum Ferritin Test

D. Haemoglobin Electrophoresis

See Answer in Part B → Q12

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Q13: Which class of carbohydrates cannot be further hydrolyzed into simpler sugar units? (Q844962)

A. Monosaccharides

B. Disaccharides

C. Oligosaccharides

D. Polysaccharides

See Answer in Part B → Q13

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Q14: Which of the following is a distinguishing feature between Hunter syndrome and Hurler syndrome? (Q844974)

A. Hunter is associated with corneal clouding; Hurler is not.

B. Hurler is due to a deficiency in iduronate-2-sulfatase; Hunter is due to a deficiency in alpha-L-iduronidase.

C. Both result in the accumulation of heparan and dermatan sulfate.

D. Hunter is X-linked ; Hurler is autosomal recessive.

See Answer in Part B → Q14

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Q15: A 4-year-old child shows developmental delay, behavioral problems, and progressive cognitive decline. Examination reveals coarse facial features and hepatosplenomegaly, but normal ocular findings. Which type of mucopolysaccharidosis is most likely? (Q844973)

A. MPS I (Hurler syndrome)

B. MPS IX (Natowicz syndrome)

C. MPS III (Sanfilippo syndrome)

D. MPS IV (Morquio syndrome)

See Answer in Part B → Q15

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Q16: A patient with joint pain and restricted movement is found to have reduced levels of a key mucopolysaccharide in their synovial fluid responsible for lubrication. Which glycosaminoglycan is most likely deficient? (Q844972)

A. Heparin

B. Chondroitin Sulfate

C. Dermatan Sulfate

D. Hyaluronic Acid

See Answer in Part B → Q16

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Q17: A patient presents with hyperextensible skin and fragile blood vessels due to a rare genetic disorder affecting connective tissue. Which glycosaminoglycan (mucopolysaccharide) found in the skin and vascular tissue is most likely implicated in this condition?? (Q844971)

A. Hyaluronic Acid

B. Heparin

C. Chondroitin Sulfate

D. Dermatan Sulfate

See Answer in Part B → Q17

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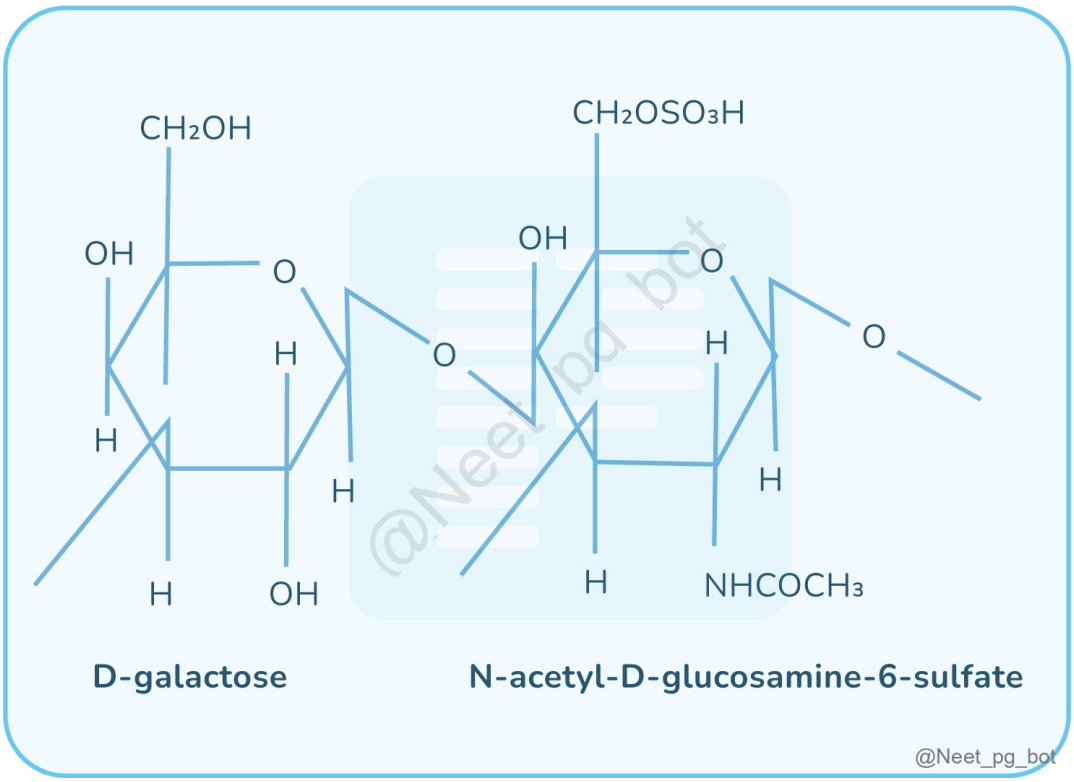
Q18: Analyse the structure of the mucopolysaccharide shown below: Which mucopolysaccharide is represented in this image? (Q844970)

A. Heparan sulfate

B. Keratan sulfate

C. Chondroitin sulfate

D. Dermatan sulfate



See Answer in Part B → Q18

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Q19: Mucopolysaccharides are heteropolysaccharides containing uronic acid and amino sugars. Which among the following mucopolysaccharides contradicts the above statement? (Q844969)

A. Keratan sulfate

B. Chondroitin sulfate

C. Dermatan sulfate

D. Heparin

See Answer in Part B → Q19

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Q20: Which of the following is accurate about dietary fibres? (Q844968)

A. Insoluble fibre is fermented in the large intestine

B. Dietary fibre provides a significant source of energy

C. Soluble fibre helps lower LDL cholesterol

D. Soluble fibre promotes bowel movements

See Answer in Part B → Q20

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Q21: Which of the following pairs correctly distinguishes between homoglycans and heteroglycans? (Q844967)

A. Homoglycans - repeated units of the same monosaccharide; Heteroglycans - two or more different types of monosaccharide units.

B. Homoglycans - used for structural purposes; Heteroglycans - used forenergy storage.

C. Homoglycans- typically soluble in water; Heteroglycans - insoluble in water

D. Homoglycans - only in plant cells; Heteroglycans - only in animal cells.

See Answer in Part B → Q21

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Q22: A patient comes with complaints of abdominal pain, bloating, flatulence and frothy stools. It is noticed that these symptoms arise only when he consumes milk or other dairy products. Which of the following is the IOC in this situation to diagnose

him? (Q844966)

A. Lactose tolerance test

B. Hydrogen breath test

C. Stool osmolarity test

D. Serum calcium test

See Answer in Part B → Q22

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Q23: Which of the following is non-reducing sugar? (Q844964)

A. Maltose

B. Glucose

C. Sucrose

D. Fructose

See Answer in Part B → Q23

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Q24: Which monosaccharides combine to form the disaccharide lactose? (Q844963)

A. Galactose and Glucose

B. Glucose and Fructose

C. Galactose and Sucrose

D. Glucose and Glucose

See Answer in Part B → Q24

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• [02_Carbohydrate Chemistry] 003 Fructose And Galactose Metabolism Hmp Pathway

« Back to Folder Directory

[02_Carbohydrate Chemistry] 003 Fructose And Galactose Metabolism Hmp Pathway — Quick Jump

Q1 (p. 40)	*Q2 (p. 40)*	*Q3 (p. 40)*	*Q4 (p. 41)*	*Q5 (p. 41)*	*Q6 (p. 42)*
Q7 (p. 42)	*Q8 (p. 43)*	*Q9 (p. 43)*	*Q10 (p. 44)*	*Q11 (p. 44)*	*Q12 (p. 44)*
Q13 (p. 45)	*Q14 (p. 45)*	*Q15 (p. 45)*	*Q16 (p. 46)*	*Q17 (p. 46)*	

Q1: How many ATPs are produced from the HMP shunt pathway? (Q875991)

A. Zero

B. One

C. Two

D. Four

See Answer in Part B → Q1

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Q2: Which of the following enzymes is not involved in the reversible phase of the pentose phosphate pathway? (Q845033)

A. Ribulose-5-phosphate epimerase

B. Transketolase

C. 6-phosphogluconate dehydrogenase

D. Transaldolase

See Answer in Part B → Q2

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Q3: Which of the following statements about the pentose phosphate (HMP) pathway in skeletal muscle is most accurate? (Q891569)

A. It is completely absent because muscle lacks glucose-6-phosphate dehydrogenase

B. It is highly active because muscle requires large amounts of NADPH for lipogenesis

C. It is present but has low activity because muscle has low demand for NADPH

D. It functions only through the non-oxidative phase and cannot generate NADPH

See Answer in Part B → Q3

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Q4: The HMP shunt pathway occurs in which cellular compartment? (Q891568)

A. Mitochondria

B. Nucleus

C. Cytosol

D. Endoplasmic Reticulum

See Answer in Part B → Q4

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Q5: Which phase of the hexose monophosphate (HMP) shunt pathway is reversible? (Q891567)

A. Oxidative phase

B. Non-oxidative phase

C. Both phases

D. Neither phase

See Answer in Part B → Q5

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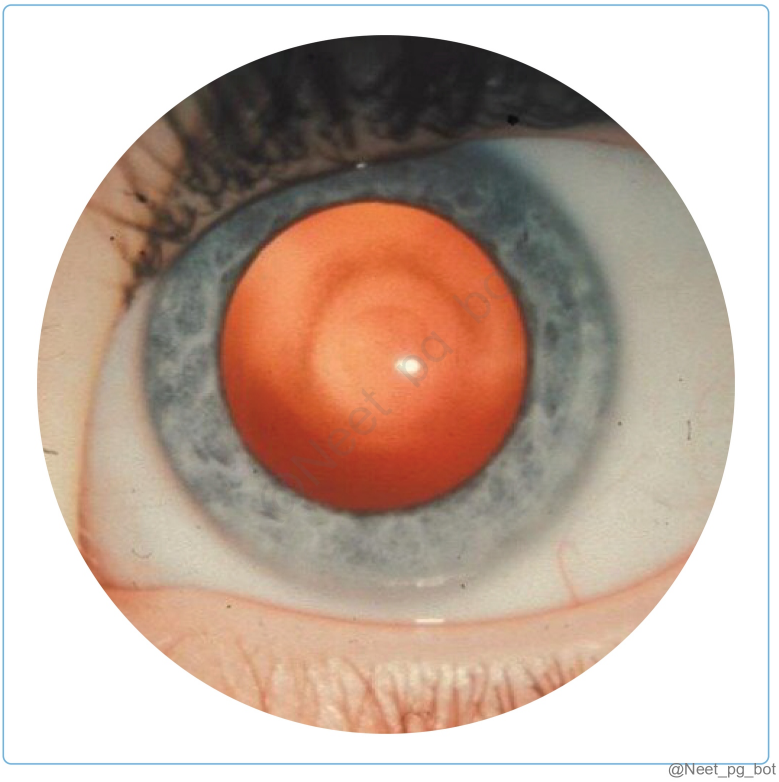
Q6: A newborn presents with bilateral lens opacities showing an “oil droplet” appearance on retroillumination. Which metabolic disorder is classically associated with this finding? (Q845037)

A. Phenylketonuria

B. Galactosemia

C. Maple Syrup Urine Disease

D. Homocystinuria



See Answer in Part B → Q6

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Q7: A diabetic patient develops snowflake cataracts. Sorbitol accumulation in the lens fibers is responsible. Deficiency or low activity of which enzyme in the lens leads to this complication? (Q845036)

A. Aldose Reductase

B. Glucose-6-Phosphate Dehydrogenase

C. Sorbitol Dehydrogenase

D. Hexokinase

See Answer in Part B → Q7

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Q8: What is the role of inosine monophosphate in stored blood? (Q845035)

A. It increases plasma Na and K levels.

B. It stimulates 2,3-DPG synthesis.

C. It prevents lactic acid buildup.

D. It stabilizes hemoglobin concentration.

See Answer in Part B → Q8

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Q9: A 30-year-old woman with G6PD deficiency presents with fatigue, jaundice, dark urine, and hemolytic anemia after eating fava beans. How does G6PD deficiency lead to these symptoms? (Q845034)

A. Reduced NADPH impairs glutathione regeneration, causing hemolytic anemia.

B. Increased ribulose-5-phosphate overloads nucleotides, leading to renal failure.

C. Impaired glucose-6-phosphate conversion disrupts glycolysis, causing hypoglycemia.

D. Decreased ATP production affects muscle function, causing myopathy.

See Answer in Part B → Q9

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Q10: Which is the rate-limiting enzyme in galactose metabolism? (Q845025)

A. Galactokinase

B. Galactose-1-phosphate uridyltransferase

C. UDP-hexose-4-epimerase

D. Aldolase Reductase

See Answer in Part B → Q10

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Q11: All the following enzymes catalyze oxidative decarboxylation reactions except? (Q845032)

A. α-Ketoglutarate dehydrogenase

B. Malate dehydrogenase

C. 6-Phosphogluconate dehydrogenase

D. Isocitrate dehydrogenase

See Answer in Part B → Q11

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Q12: Which statement is true regarding NADH and NADPH? (Q845031)

A. Both are produced in the Pentose Phosphate Pathway.

B. NADH is used in lipid biosynthesis, while NADPH is not.

C. NADPH is used for anabolic reactions, while NADH is primarily for catabolic reactions.

D. Both serve same function in the cell.

See Answer in Part B → Q12

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Q13: Which sequence correctly represents the steps of the oxidative phase of the pentose phosphate pathway (PPP)? (Q845030)

A. 6-Phosphogluconolactone → Glucose-6-phosphate → 6-Phosphogluconate → Ribulose-5-phosphate

B. Glucose-6-phosphate → 6-Phosphogluconate → 6-Phosphogluconolactone → Ribulose-5-phosphate

C. Glucose-6-phosphate → 6-Phosphogluconolactone → 6-Phosphogluconate → Ribulose-5-phosphate

D. 6-Phosphogluconate → 6-Phosphogluconolactone → Glucose-6-phosphate → Ribulose-5-phosphate

See Answer in Part B → Q13

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Q14: A 45-year-old man with hemolytic anemia shows RBCs susceptible to oxidative damage. What is the main function of the pentose phosphate pathway in RBCs? (Q845029)

A. To produce ATP for energy metabolism

B. To provide NADPH for fatty acid and cholesterol synthesis

C. To provide NADPH for maintaining reduced glutathione levels

D. To synthesize hemoglobin

See Answer in Part B → Q14

@Neet_pg_bot

Q15: Which of the following statements is true regarding Hereditary Fructose Intolerance (HFI) and Classical Galactosemia? (Q845028)

A. Both present with hyperglycemia.

B. Both require lifelong avoidance of lactose.

C. Both present with hypophosphatemia.

D. Both have symptoms that appear at birth.

See Answer in Part B → Q15

@Neet_pg_bot

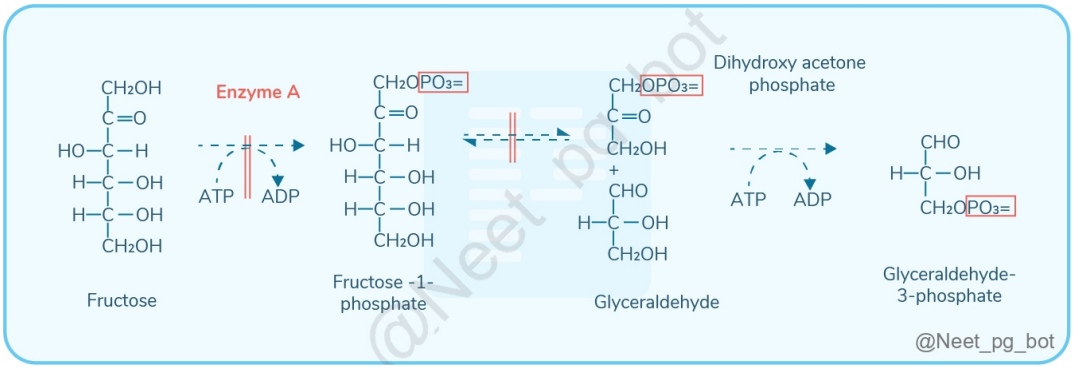
Q16: A child is diagnosed with essential fructosuria during a routine health check. Name the enzyme A responsible for this condition. (Q845027)

A. Aldolase B

B. Hexokinase

C. Fructokinase

D. Glucokinase



See Answer in Part B → Q16

@Neet_pg_bot

Q17: An infant is diagnosed with classic galactosemia. Which enzyme deficiency is responsible for this condition? (Q845026)

A. Galactokinase

B. Galactose-1-phosphate uridyltransferase

C. UDP-galactose 4-epimerase

D. Aldose reductase

See Answer in Part B → Q17

@Neet_pg_bot

• [02_Carbohydrate Chemistry] 004 Fates Of Pyruvate Pdh Complex Electron Transport Chain

« Back to Folder Directory

[02_Carbohydrate Chemistry] 004 Fates Of Pyruvate Pdh Complex Electron Transport Chain — Quick Jump

Q1 (p. 48)	*Q2 (p. 48)*	*Q3 (p. 49)*	*Q4 (p. 49)*	*Q5 (p. 49)*	*Q6 (p. 50)*
Q7 (p. 50)	*Q8 (p. 50)*	*Q9 (p. 51)*	*Q10 (p. 51)*	*Q11 (p. 52)*	*Q12 (p. 52)*
Q13 (p. 52)	*Q14 (p. 53)*	*Q15 (p. 53)*	*Q16 (p. 54)*	*Q17 (p. 54)*	*Q18 (p. 54)*
Q19 (p. 55)	*Q20 (p. 55)*				

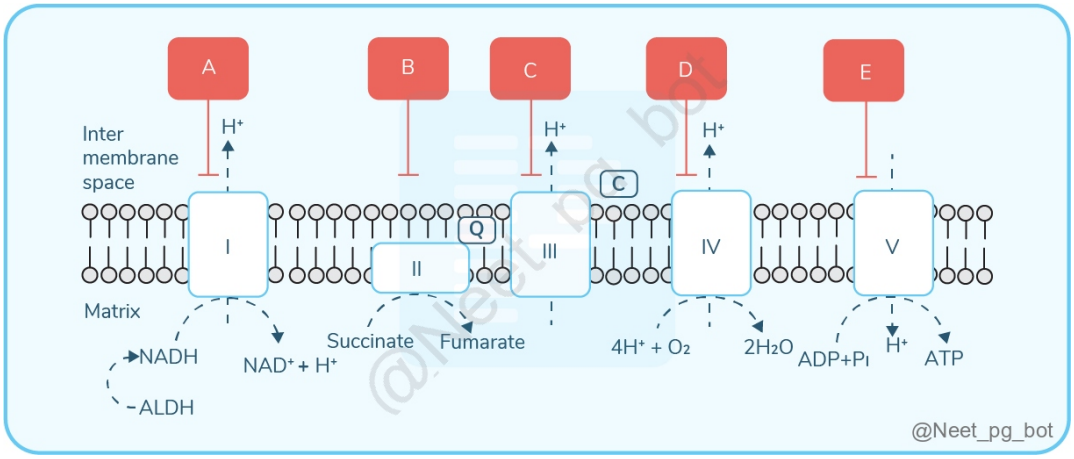
Q1: The image below shows the sites of action for different ETC inhibitors. Based on the image, which of the following is the site of action of Cyanide? (Q845065)

A. A

B. B

C. C

D. D



See Answer in Part B → Q1

@Neet_pg_bot

Q2: Oligomycin inhibits oxidative phosphorylation by: (Q891582)

A. Blocking electron transport at Complex I

B. Binding to ATP synthase (F■ domain), blocking proton flow

C. Uncoupling proton gradient

D. Inhibiting ADP-ATP translocase

See Answer in Part B → Q2

@Neet_pg_bot

Q3: According to the chemiosmotic theory, ATP synthesis is driven by: (Q891580)

A. Substrate-level phosphorylation

B. Direct transfer of phosphate from NADH

C. Proton gradient across inner mitochondrial membrane

D. Electron transfer without gradient

See Answer in Part B → Q3

@Neet_pg_bot

Q4: Which metal ion is a component of cytochrome oxidase (Complex IV)? (Q891577)

A. Iron only

B. Magnesium

C. Zinc

D. Copper

See Answer in Part B → Q4

@Neet_pg_bot

Q5: Succinate-Q reductase belongs to which ETC complex? (Q891575)

A. Complex I

B. Complex II

C. Complex III

D. Complex IV

See Answer in Part B → Q5

@Neet_pg_bot

Q6: Complex I of the electron transport chain is also known as: (Q891574)

A. Succinate-Q reductase

B. Cytochrome c oxidase

C. NADH-Q oxidoreductase

D. Q-cytochrome c oxidoreductase

See Answer in Part B → Q6

@Neet_pg_bot

Q7: Where is ATP synthase (Complex V) is located? (Q891572)

A. Mitochondrial matrix

B. Outer mitochondrial membrane

C. Inner mitochondrial membrane

D. Intermembrane space

See Answer in Part B → Q7

@Neet_pg_bot

Q8: The electron transport chain (ETC) carriers are located on which structure? (Q891571)

A. Inner mitochondrial membrane

B. Outer mitochondrial membrane

C. Mitochondrial matrix

D. Cytosol

See Answer in Part B → Q8

@Neet_pg_bot

Q9: A 28-year-old woman presents with recurrent episodes of neurologic disturbances and lactic acidosis, particularly after carbohydrate-rich meals. Genetic testing reveals a mutation affecting one of the components of the pyruvate dehydrogenase complex. Which of the following is most likely to be directly affected by this mutation? (Q845067)

A. Flavin adenine dinucleotide (FAD)

B. Dihydrolipoyl transacetylase

C. Thiamine diphosphate

D. Coenzyme A

See Answer in Part B → Q9

@Neet_pg_bot

Q10: Which of the following is a physiological uncoupler? (Q845066)

A. 2,4-dinitrophenol

B. Uncoupling protein 1 [UCP1]

C. Valinomycin

D. Nigericin

See Answer in Part B → Q10

@Neet_pg_bot

Q11: In red blood cells under low energy states, which of the following best describes the fate of pyruvate? (Q845038)

A. Converted to acetyl-CoA to enter the citric acid cycle.

B. Converted to oxaloacetate to replenish the citric acid cycle.

C. Converted to lactate by lactate dehydrogenase to regenerate NAD + .

D. Converted to alanine via transamination.

See Answer in Part B → Q11

@Neet_pg_bot

Q12: In the electron transport chain, how many ATP molecules are produced per NADH and FADH 2 , respectively? How many protons (H+) are translocated across the inner mitochondrial membrane for each? (Q845047)

A. 2.5 ATP per NADH and 1.5 ATP per FADH 2 ; with 10 H + and 6 H + translocated, respectively.

B. 3 ATP per NADH and 2 ATP per FADH 2 ; with 12 H + and 8 H + translocated, respectively.

C. 2 ATP per NADH and 1 ATP per FADH 2 ; with 8 H + and 4 H + translocated, respectively.

D. 1 ATP per NADH and 0.5 ATP per FADH 2 ; with 6 H + and 3 H + translocated, respectively.

See Answer in Part B → Q12

@Neet_pg_bot

Q13: A patient, with a history of attempting weight loss, presents with fatigue, muscle weakness, and vision problems. Genetic testing confirms MELAS (mitochondrial encephalopathy, lactic acidosis, and stroke). Which electron transport chain complex is most likely affected in this patient (Q845046)

A. Complex I

B. Complex II

C. Complex III

D. Complex V

See Answer in Part B → Q13

@Neet_pg_bot

Q14: Which of the following statements regarding the electron transport chain (ETC) is incorrect? (Q845045)

A. Electrons from NADH enter the ETC through Complex I

B. Complex II does not participate in electron transfer

C. Coenzyme Q transfers electrons between Complex I/II and III

D. Cytochrome c transfers electrons between Complex III and IV

See Answer in Part B → Q14

@Neet_pg_bot

Q15: A 25-year-old athlete experiences exercise intolerance and muscle fatigue during high-intensity activity. Genetic testing shows a deficiency in the enzyme that transfers electrons from cytosolic NADH to mitochondrial FADH■ in skeletal muscle.Which mitochondrial shuttle system is most likely impaired? (Q845044)

A. Malate shuttle

B. Glycerol phosphate shuttle

C. Citrate shuttle

D. Aspartate shuttle

See Answer in Part B → Q15

@Neet_pg_bot

Q16: Which of the following regulatory effects best explains the anti-neoplastic effect of the ketogenic diet? (Q845043)

A. Acetyl CoA stimulates pyruvate carboxylase.

B. Malonyl CoA inhibits carnitine acyltransferase-1.

C. Citrate inhibits PFK1.

D. Acyl CoA stimulates carnitine acyltransferase-1.

See Answer in Part B → Q16

@Neet_pg_bot

Q17: A 5-year-old boy with lethargy, vomiting, and ataxia shows lactic acidosis and elevated pyruvate levels. Genetic testing confirms pyruvate dehydrogenase complex deficiency. What is the underlying mechanism for his metabolic derangements? (Q845042)

A. Impaired conversion of pyruvate to acetyl-CoA.

B. Increased conversion of pyruvate to ribose-5-phosphate.

C. Reduced activity of the electron transport chain.

D. Inhibition of gluconeogenesis in the liver.

See Answer in Part B → Q17

@Neet_pg_bot

Q18: Which of the following is the primary site for peripheral lipolysis? (Q845041)

A. Liver

B. Skeletal muscle

C. Adipose tissue

D. Pancreas

See Answer in Part B → Q18

@Neet_pg_bot

Q19: During fasting, after how many hours does gluconeogenesis become the primary source of blood glucose? (Q845040)

A. 8 to 12 hours

B. 12 to 24 hours

C. 16 to 48 hours

D. 48 to 72 hours

See Answer in Part B → Q19

@Neet_pg_bot

Q20: Which of the following metabolic disturbances is most likely to be seen in a patient with pyruvate dehydrogenase deficiency? (Q845039)

A. Hypoglycemia due to impaired gluconeogenesis

B. Hyperglycemia due to increased gluconeogenesis

C. Ketoacidosis due to excessive fatty acid oxidation

D. Lactic acidosis due to impaired conversion of pyruvate to acetyl-CoA

See Answer in Part B → Q20

@Neet_pg_bot

• [02_Carbohydrate Chemistry] 005 Glut Transporters And Glycolysis

« Back to Folder Directory

[02_Carbohydrate Chemistry] 005 Glut Transporters And Glycolysis — Quick Jump

Q1 (p. 56) *Q2 (p. 56)* *Q3 (p. 57)* *Q4 (p. 57)* *Q5 (p. 57)* *Q6 (p. 58)*
Q7 (p. 58) *Q8 (p. 58)* *Q9 (p. 59)* *Q10 (p. 59)* *Q11 (p. 59)* *Q12 (p. 60)*

Q1: Match the following GLUT transporters with their primary locations: 1. GLUT1 a) Liver 2. GLUT2 b) Neurons 3. GLUT3 c) Erythrocytes 4.GLUT4 d) Skeletal Muscle (Q844950)

A. 1-c, 2-a, 3-b, 4-d

B. 1-d, 2-b, 3-c, 4-a

C. 1-b, 2-d, 3-a, 4-c

D. 1-a, 2-c, 3-d, 4-b

See Answer in Part B → Q1

@Neet_pg_bot

Q2: Which of the following mechanisms is primarily responsible for glucose uptake into enterocytes? (Q844951)

A. Facilitated diffusion via GLUT5 transporter

B. Secondary active transport via SGLT1 transporter

C. Primary active transport via Na+/K+ ATPase

D. Simple diffusion through the lipid bilayer

See Answer in Part B → Q2

@Neet_pg_bot

Q3: Which of the following tissues is unable to transport glucose independently of insulin? (Q844952)

A. Hepatocytes

B. Cardiac muscle

C. RBC

D. Neurons

See Answer in Part B → Q3

@Neet_pg_bot

Q4: All of the following are true about glycolysis except: (Q844953)

A. Aerobic glycolysis produces 2 pyruvates and 7 ATPs while anaerobic glycolysis produces 2 lactates and 2 ATPs

B. Retinal and corneal cells can produce energy using glycolysis in hypoxic conditions

C. Tumor cells rely mainly on fatty acid oxidation

D. Fast twitch muscle fibers primarily rely on anaerobic glycolysis for ATP sources

See Answer in Part B → Q4

@Neet_pg_bot

Q5: Which of the following statements is false? (Q844954)

A. RBCs solely depend on glycolysis for ATP

B. White muscle fibers depend on anaerobic glycolysis during muscular contraction

C. Myoglobin is the storage form of oxygen in white muscle fibers

D. Slow-twitch muscle fibers contain high amounts of myoglobin

See Answer in Part B → Q5

@Neet_pg_bot

Q6: Which of the following statements regarding aerobic glycolysis is true? (Q844955)

A. Each acetyl CoA molecule yields 5 ATPs by converting to pyruvate

B. Each molecule of NADPH yields 2.5 ATP

C. Aerobic glycolysis of one molecule of glucose yields a net total of 7 ATP molecules.

D. Each turn of the citric acid cycle produces 20 ATP molecules.

See Answer in Part B → Q6

@Neet_pg_bot

Q7: Which of the following enzymes catalyzes the conversion of glucose to glucose-6-phosphate in the glycolysis pathway? (Q844956)

A. Hexokinase

B. Phosphofructokinase

C. Aldolase

D. Enolase

See Answer in Part B → Q7

@Neet_pg_bot

Q8: Which of the following steps in glycolysis is irreversible? (Q844957)

A. Conversion of glucose-6-phosphate to fructose-6-phosphate

B. Conversion of 3-phosphoglycerate to 2-phosphoglycerate

C. Conversion of phosphoenolpyruvate to pyruvate

D. Conversion of fructose-1,6-bisphosphate to glyceraldehyde-3-phosphate and dihydroxyacetone phosphate

See Answer in Part B → Q8

@Neet_pg_bot

Q9: Which of the following statements accurately describes the difference between hexokinase and glucokinase? (Q844958)

A. Hexokinase has a higher Km and Vmax compared to glucokinase.

B. Glucokinase is inhibited by glucose-6-phosphate, whereas hexokinase is not.

C. Hexokinase is primarily found in the liver and pancreatic β-cells, while glucokinase is found in most tissues.

D. Glucokinase has a higher Km and Vmax compared to hexokinase.

See Answer in Part B → Q9

@Neet_pg_bot

Q10: Which of the following chemical compounds inhibits glycolysis by inhibiting the enzyme enolase? (Q844959)

A. Iodoacetate

B. Fluoride

C. Arsenate

D. 2-Deoxyglucose

See Answer in Part B → Q10

@Neet_pg_bot

Q11: What is the primary function of the Rapoport-Luebering Shunt in red blood cells? (Q844960)

A. To generate ATP from 2,3-bisphosphoglycerate

B. To produce 2,3-bisphosphoglycerate, which regulates oxygen binding to hemoglobin

C. To convert pyruvate to lactate

D. To facilitate the conversion of glucose to glucose-6-phosphate

See Answer in Part B → Q11

@Neet_pg_bot

Q12: Which of the following is a key positive regulator of the glycolytic enzyme phosphofructokinase-1 (PFK-1)? (Q844961)

A. ATP

B. Citrate

C. Fructose-2,6-bisphosphate

D. Acetyl-CoA

See Answer in Part B → Q12

@Neet_pg_bot

• [02_Carbohydrate Chemistry] 006 Glycogen Metabolism And Glycogen Storage Disorders

« Back to Folder Directory

[02_Carbohydrate Chemistry] 006 Glycogen Metabolism And Glycogen Storage Disorders — Quick Jump

Q1 (p. 61)	*Q2 (p. 61)*	*Q3 (p. 62)*	*Q4 (p. 62)*	*Q5 (p. 62)*	*Q6 (p. 63)*
Q7 (p. 63)	*Q8 (p. 63)*	*Q9 (p. 64)*	*Q10 (p. 64)*	*Q11 (p. 64)*	*Q12 (p. 65)*
Q13 (p. 65)	*Q14 (p. 66)*	*Q15 (p. 66)*	*Q16 (p. 67)*	*Q17 (p. 67)*	*Q18 (p. 67)*
Q19 (p. 68)	*Q20 (p. 68)*	*Q21 (p. 68)*	*Q22 (p. 69)*	*Q23 (p. 69)*	*Q24 (p. 69)*

Q1: A 5-year-old child presents with muscle weakness, mild hepatomegaly, and cardiomegaly. Labs show mild hypoglycemia and elevated creatine kinase. Which enzyme deficiency is most likely responsible? (Q844869)

A. Glycogen synthase

B. Glucose-6-phosphatase

C. Debranching enzyme

D. Muscle phosphorylase

See Answer in Part B → Q1

@Neet_pg_bot

Q2: Glycogen phosphorylase requires which vitamin-derived coenzyme? (Q891558)

A. Thiamine pyrophosphate

B. Pyridoxal phosphate (Vitamin B6)

C. Biotin

D. Coenzyme A

See Answer in Part B → Q2

@Neet_pg_bot

Q3: Muscle cannot release glucose into the bloodstream because it lacks which enzyme? (Q891554)

A. Glycogen phosphorylase

B. Hexokinase

C. Glucose-6-phosphatase

D. Phosphoglucomutase

See Answer in Part B → Q3

@Neet_pg_bot

Q4: The correct sequence of enzymes involved in glycogenolysis (glycogen breakdown) is: (Q891549)

A. Phosphorylase → Glucose-6-phosphatase → Phosphoglucomutase → Glucan transferase

B. Glucan transferase → Phosphorylase → Amylo-1,6-glucosidase → Phosphoglucomutase

C. Phosphorylase → Phosphoglucomutase → Glucan transferase → Glucose-6-phosphatase

D. Phosphorylase → Debranching enzyme → Phosphoglucomutase → Glucose-6-phosphatase

See Answer in Part B → Q4

@Neet_pg_bot

Q5: The activated form of glucose used for addition to the growing glycogen chain is: (Q891547)

A. Glucose-1-phosphate

B. Glucose-6-phosphate

C. UDP-glucose

D. ADP-glucose

See Answer in Part B → Q5

@Neet_pg_bot

Q6: Which protein serves as the primer for glycogen synthesis, accepting the first glucose residues? (Q891544)

A. Glycogen synthase

B. Glycogenin

C. Branching enzyme

D. UDP-glucose pyrophosphorylase

See Answer in Part B → Q6

@Neet_pg_bot

Q7: Glycogen synthesis occurs in which cellular compartment? (Q891541)

A. Mitochondria

B. Nucleus

C. Cytosol

D. Endoplasmic Reticulum

See Answer in Part B → Q7

@Neet_pg_bot

Q8: Glycogenesis (glycogen synthesis) occurs predominantly in which two tissues? (Q891539)

A. Liver and kidney

B. Skeletal muscle and liver

C. Brain and adipose tissue

D. Heart and pancreas

See Answer in Part B → Q8

@Neet_pg_bot

Q9: Which glycogen storage disease does not involve hypoglycemia or exercise intolerance? (Q844873)

A. Von Gierke Disease

B. McArdle Disease

C. Andersen's Disease

D. Tarui Disease

See Answer in Part B → Q9

@Neet_pg_bot

Q10: A 6-month-old infant presents with recurrent episodes of hypoglycemia and elevated ketone levels. Genetic testing was done and diagnosed as Type 0 Glycogen storage disorder. Which of the following statements about Glycogen Storage Disease Type 0 is true? (Q844872)

A. The enzyme deficiency is glycogen synthase.

B. The affected genes are GYS2 and GYS1.

C. It is an autosomal recessive disorder

D. All of the above

See Answer in Part B → Q10

@Neet_pg_bot

Q11: A 1 year old child presents with failure to thrive, weak bones, and renal dysfunction. The physician suspected glycogen storage disorder and further identified a mutation in the SLC2A2 gene while genetic testing. Which among the following defects is associated with it? (Q844871)

A. GLUT 2

B. Liver phosphorylase

C. Muscle and erythrocyte phosphofructokinase 1

D. Muscle Phosphorylase

See Answer in Part B → Q11

@Neet_pg_bot

Q12: A 6-month-old infant presents with muscle hypotonia and heart failure. Laboratory tests reveal an accumulation of glycogen in lysosomes. Which of the following is the most likely diagnosis? (Q844870)

A. McArdle Disease

B. Pompe Disease

C. Von Gierke Disease

D. Cori Disease

See Answer in Part B → Q12

@Neet_pg_bot

Q13: Which of the following statements is true on the structure of glycogen? Arranged in 12 concentric layers Glucose residues are connected by ■-1,4 linkage Branching points formed by α-1,6 linkage (Q844857)

A. 1,2

B. 2,3

C. 1,2,3

D. 1,3

See Answer in Part B → Q13

@Neet_pg_bot

Q14: A 6-month-old infant presents with severe hypoglycemia, hepatomegaly, growth retardation, irritability, and lethargy between feedings. Labs show elevated lactate, triglycerides, and uric acid, and the child requires frequent feedings to prevent hypoglycemia. Which condition is most likely? (Q844868)

A. Von Gierke's disease

B. McArdle's disease

C. Tarui's Disease

D. Hers' Disease

See Answer in Part B → Q14

@Neet_pg_bot

Q15: Match the glycogen storage disorders with their respective enzyme defects. Glycogen Storage Disorders: Enzyme Defects: 1. Pompe Disease a. Debranching enzyme 2. McArdle Disease b. Acid maltase 3. Von Gierke Disease c. Glucose-6-phosphatase 4. Cori Disease d. Myophosphorylase (Q844867)

A. 1-b, 2-d, 3-c, 4-a

B. 1-a, 2-c, 3-b, 4-d

C. 1-d, 2-b, 3-a, 4-c

D. 1-c, 2-a, 3-d, 4-b

See Answer in Part B → Q15

@Neet_pg_bot

Q16: A 32-year-old woman consumes a high-carbohydrate diet. You are asked to explain the impact of her dietary habits on glycogen metabolism. Which of the following statements best describes glycogen metabolism? (Q844866)

A. Increased Gluconeogenesis

B. Reduced peripheral lipolysis

C. Low rate of glycogen synthesis

D. High rate of glycogenolysis

See Answer in Part B → Q16

@Neet_pg_bot

Q17: Which of the following enzymes is responsible for converting the glycogen molecule from a branched structure to a linear structure during glycogenolysis? (Q844865)

A. Glycogen Phosphorylase

B. Branching Enzyme

C. Debranching Enzyme

D. Glycogen Synthase

See Answer in Part B → Q17

@Neet_pg_bot

Q18: A 25-year-old male athlete develops fatigue and muscle cramps after intense exercise. Blood tests show elevated lactic acid. What is the net ATP yield from one molecule of muscle glycogen converted to lactic acid during strenuous exercise? (Q844864)

A. 1 ATP

B. 2 ATP

C. 3 ATP

D. 4 ATP

See Answer in Part B → Q18

@Neet_pg_bot

Q19: Which of the following statements correctly differentiates liver glycogen phosphorylase (LGP) and muscle glycogen phosphorylase (MGP)? (Q844863)

A. LGP is activated by glucagon, while MGP is activated by epinephrine.

B. LGP releases glucose-6-phosphate into the bloodstream, while MGP releases free glucose into the muscle.

C. LGP activity is regulated by AMP, while MGP is regulated by glucagon and epinephrine.

D. MGP is highly active during fasting compared to LGP.

See Answer in Part B → Q19

@Neet_pg_bot

Q20: Which of the following enzymes catalyzes the rate-limiting step of glycogenolysis (Q844862)

A. Phosphoglucomutase

B. Glucose-6-phosphatase

C. Glucan transferase

D. Glycogen phosphorylase

See Answer in Part B → Q20

@Neet_pg_bot

Q21: Which of the following statements is false regarding glycogenolysis? (Q844861)

A. Glucose-6-phosphatase acts in liver

B. Glycogen phosphorylase cleaves ■-1,4 linkages

C. Phosphoglucomutase converts glucose-1-phosphate into glucose-6-phosphate

D. Glycogenolysis is the reverse process of glycogenesis.

See Answer in Part B → Q21

@Neet_pg_bot

Q22: Which of the following enzymes is activated by phosphorylation? (Q844860)

A. Acetyl-CoA carboxylase

B. Glycogen synthase

C. Pyruvate dehydrogenase

D. Glycogen phosphorylase

See Answer in Part B → Q22

@Neet_pg_bot

Q23: What is the correct order of enzymes that act in glycogenesis? (Q844859)

A. Hexokinase→ Phosphoglucomutase → UDP-glucose pyrophosphorylase

B. Phosphoglucomutase → UDP-glucose pyrophosphorylase → Hexokinase

C. Hexokinase→ UDP-glucose pyrophosphorylase → Phosphoglucomutase

D. UDP-glucose pyrophosphorylase → Phosphoglucomutase → Hexokinase

See Answer in Part B → Q23

@Neet_pg_bot

Q24: Assertion (A): Glycogen and amylopectin are both branched polysaccharides composed of glucose units. Reason (R): Both contain α-1,4 glycosidic linkages in the linear chains and α-1,6 glycosidic linkages at the branch points. (Q844858)

A. Both the assertion and the reason are correct, and the reason is the correct explanation for the assertion.

B. Both the assertion and the reason are correct, but the reason is not the correct explanation for the assertion.

C. The assertion is correct, but the reason is incorrect.

D. The assertion is incorrect, but the reason is correct.

See Answer in Part B → Q24

@Neet_pg_bot

• [02_Carbohydrate Chemistry] 007 Citric Acid Cycle And Gluconeogenesis

« Back to Folder Directory

[02_Carbohydrate Chemistry] 007 Citric Acid Cycle And Gluconeogenesis — Quick Jump

Q1 (p. 71)	*Q2 (p. 71)*	*Q3 (p. 71)*	*Q4 (p. 72)*	*Q5 (p. 72)*	*Q6 (p. 73)*
Q7 (p. 73)	*Q8 (p. 73)*	*Q9 (p. 74)*	*Q10 (p. 74)*	*Q11 (p. 74)*	*Q12 (p. 75)*
Q13 (p. 75)	*Q14 (p. 76)*	*Q15 (p. 76)*	*Q16 (p. 76)*	*Q17 (p. 77)*	*Q18 (p. 77)*

Q1: Which enzyme is the rate-limiting enzyme of gluconeogenesis? (Q845015)

A. PEP Carboxykinase

B. Pyruvate Carboxylase

C. Glucose-6-phosphatase

D. Fructose 1,6-bisphosphatase

See Answer in Part B → Q1

@Neet_pg_bot

Q2: In patients with uncontrolled diabetes, which of the following intermediates is depleted, suppressing the TCA cycle? (Q845024)

A. Acetyl CoA

B. Oxaloacetate

C. Succinyl-CoA

D. Malate

See Answer in Part B → Q2

@Neet_pg_bot

Q3: Which of the following factors regulates the expression of PEPCK in liver cells? (Q845023)

A. Glucagon

B. Insulin

C. Citrate

D. Acetyl-CoA

See Answer in Part B → Q3

@Neet_pg_bot

Q4: A 45-year-old woman presents with fatigue and muscle weakness. Laboratory tests reveal hypoglycemia and elevated lactate levels. The physician suspects a deficiency in a coenzyme essential for gluconeogenesis. Which coenzyme is most likely deficient in this patient? (Q845022)

A. Magnesium

B. Thiamine Pyrophosphate

C. Biotin

D. Fluoride

See Answer in Part B → Q4

@Neet_pg_bot

Q5: Which of the following enzymes in gluconeogenesis is considered a "tandem enzyme" with fructose-2,6-bisphosphatase, functioning in the opposite direction? (Q845021)

A. Pyruvate carboxylase

B. Phosphoenolpyruvate carboxykinase

C. Phosphofructokinase I

D. Phosphofructokinase-2

See Answer in Part B → Q5

@Neet_pg_bot

Q6: A patient presents with symptoms of hypoglycemia shortly after consuming a high-carbohydrate meal. Laboratory tests reveal elevated insulin levels. Which of the following enzymes in the gluconeogenesis pathway is most likely to be strongly inhibited in this scenario? (Q845020)

A. Pyruvate carboxylase

B. Phosphoenolpyruvate carboxykinase (PEPCK)

C. Fructose-1,6-bisphosphatase

D. Glucose-6-phosphatase

See Answer in Part B → Q6

@Neet_pg_bot

Q7: Which of the following statements about the malate shuttle in gluconeogenesis is not true? (Q845018)

A. Helps transfer reducing equivalents from the cytoplasm to mitochondria

B. Involves the interconversion of oxaloacetate and malate

C. Requires the enzyme malate dehydrogenase in both mitochondria and cytosol

D. Directly transports NADH across the mitochondrial membrane

See Answer in Part B → Q7

@Neet_pg_bot

Q8: Which of the following enzymes involved in gluconeogenesis is located in the endoplasmic reticulum? (Q845017)

A. Pyruvate carboxylase

B. Phosphoenolpyruvate carboxykinase (PEPCK)

C. Fructose-1,6-bisphosphatase

D. Glucose-6-phosphatase

See Answer in Part B → Q8

@Neet_pg_bot

Q9: Which of the following is NOT a substrate for gluconeogenesis? (Q845016)

A. Lactate

B. Glycerol

C. Alanine

D. Acetyl-CoA

See Answer in Part B → Q9

@Neet_pg_bot

Q10: All of the following metabolic pathways occur in the cytoplasm, except? (Q844990)

A. Glycolysis

B. Pentose Phosphate Pathway

C. Fatty Acid Synthesis

D. Citric Acid Cycle

See Answer in Part B → Q10

@Neet_pg_bot

Q11: Which of the following best describes the amphibolic nature of the citric acid cycle? (Q845014)

A. It generates ATP and produces citrate for fatty acid synthesis.

B. It exclusively produces NADH for oxidative phosphorylation.

C. It breaks down fatty acids and synthesises only cholesterol.

D. It provides intermediates solely for glucose synthesis.

See Answer in Part B → Q11

@Neet_pg_bot

Q12: Which of the following is correct regarding anaplerotic reactions? (Q845013)

A. Produce ATP

B. Replenish TCA intermediates

C. Reduction of NAD+ to NADH

D. Breakdown of fatty acids

See Answer in Part B → Q12

@Neet_pg_bot

Q13: ATP inhibits all of the following enzymes in the citric acid cycle except: (Q845012)

A. Citrate synthase

B. Isocitrate dehydrogenase

C. α-Ketoglutarate dehydrogenase

D. Succinate dehydrogenase

See Answer in Part B → Q13

@Neet_pg_bot

Q14: Which of the following toxins directly inhibits the activity of the enzyme aconitase in the Citric Acid Cycle? (Q845011)

A. Cyanide

B. Fluoroacetate

C. Rotenone

D. Antimycin A

See Answer in Part B → Q14

@Neet_pg_bot

Q15: Calculate the total amount of ATP produced from the Citric Acid Cycle alone for one glucose molecule. (Q845008)

A. 30 ATP

B. 9 ATP

C. 18 ATP

D. 20 ATP

See Answer in Part B → Q15

@Neet_pg_bot

Q16: Which of the following by-products from the Krebs Cycle is involved in heme synthesis? (Q845007)

A. Citrate

B. α-Ketoglutarate

C. Succinyl-CoA

D. Malate

See Answer in Part B → Q16

@Neet_pg_bot

Q17: Which of the following steps in the Citric acid cycle involves oxidative phosphorylation and decarboxylation? Isocitrate → α-Ketoglutarate Succinate → Fumarate Malate → Oxaloacetate α-Ketoglutarate → Succinyl-CoA (Q845006)

A. 1 and 4 are correct

B. 2 and 3 are correct

C. 1, 3, and 4 are correct

D. All are correct

See Answer in Part B → Q17

@Neet_pg_bot

Q18: Which of the following is not a cofactor for the enzyme catalysing α-Ketoglutarate → Succinyl-CoA? (Q844991)

A. Fe2+

B. Thiamine

C. Lipoic Acid

D. Pantothenic acid

See Answer in Part B → Q18

@Neet_pg_bot

• [02_Carbohydrate Chemistry] 008 Previous Year Questions

« Back to Folder Directory

[02_Carbohydrate Chemistry] 008 Previous Year Questions — Quick Jump

Q1 (p. 78)	*Q2 (p. 79)*	*Q3 (p. 79)*	*Q4 (p. 80)*	*Q5 (p. 80)*	*Q6 (p. 80)*
Q7 (p. 81)	*Q8 (p. 82)*	*Q9 (p. 82)*	*Q10 (p. 83)*	*Q11 (p. 83)*	*Q12 (p. 83)*
Q13 (p. 84)	*Q14 (p. 84)*	*Q15 (p. 84)*	*Q16 (p. 85)*	*Q17 (p. 85)*	*Q18 (p. 86)*
Q19 (p. 86)	*Q20 (p. 87)*	*Q21 (p. 87)*	*Q22 (p. 88)*	*Q23 (p. 88)*	*Q24 (p. 88)*
Q25 (p. 89)	*Q26 (p. 89)*	*Q27 (p. 89)*	*Q28 (p. 90)*	*Q29 (p. 91)*	*Q30 (p. 91)*
Q31 (p. 91)	*Q32 (p. 92)*	*Q33 (p. 92)*	*Q34 (p. 92)*	*Q35 (p. 93)*	*Q36 (p. 93)*
Q37 (p. 94)	*Q38 (p. 94)*	*Q39 (p. 94)*	*Q40 (p. 95)*	*Q41 (p. 95)*	*Q42 (p. 96)*
Q43 (p. 96)	*Q44 (p. 96)*	*Q45 (p. 97)*	*Q46 (p. 97)*	*Q47 (p. 97)*	*Q48 (p. 98)*
Q49 (p. 98)	*Q50 (p. 99)*	*Q51 (p. 99)*	*Q52 (p. 99)*	*Q53 (p. 100)*	*Q54 (p. 100)*
Q55 (p. 100)	*Q56 (p. 101)*	*Q57 (p. 101)*	*Q58 (p. 101)*	*Q59 (p. 102)*	*Q60 (p. 102)*
Q61 (p. 103)	*Q62 (p. 103)*	*Q63 (p. 103)*	*Q64 (p. 104)*	*Q65 (p. 104)*	*Q66 (p. 105)*
Q67 (p. 105)	*Q68 (p. 105)*				

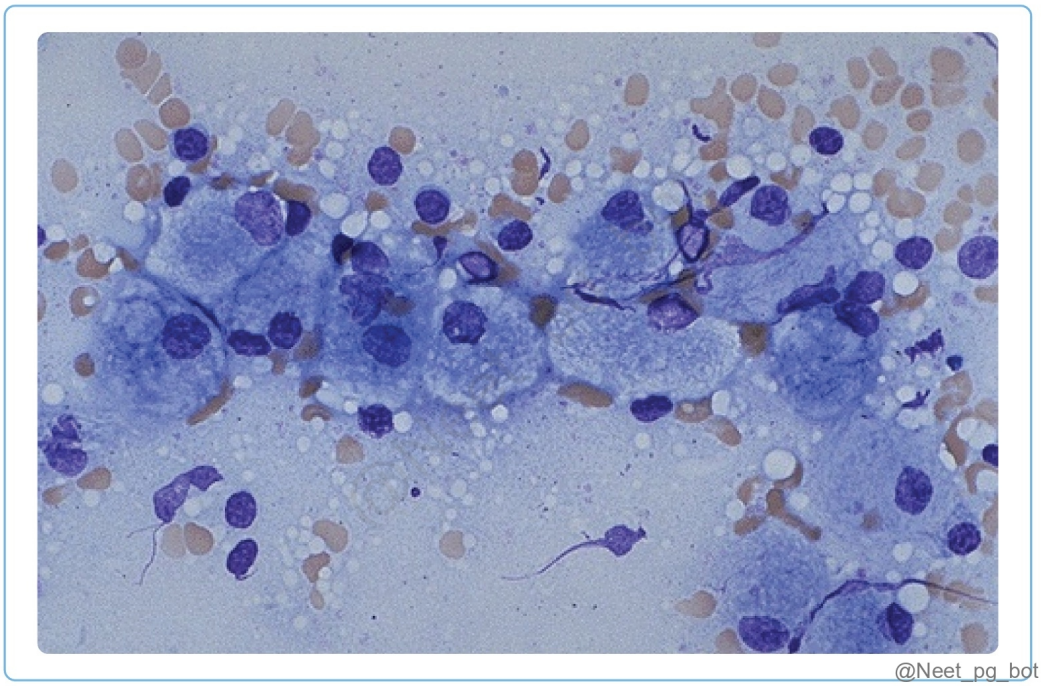
Q1: A child presents with bone pain and hepatosplenomegaly. A trephine biopsy and aspirate shows the following finding. Which of the following is the most likely enzyme deficient in this condition? (Q855278)

A. Hexosaminidase

B. Glucocerebrosidase

C. Sphingomyelinase

D. Alpha 1,4-glucosidase



See Answer in Part B → Q1

@Neet_pg_bot

Q2: Which enzyme deficiency causesVon Gierke's disease? (Q857492)

A. Liver phosphorylase

B. Lysosomal glucosidase

C. Hepatic glycogen synthase

D. Glucose 6-phosphatase

See Answer in Part B → Q2

@Neet_pg_bot

Q3: Which enzyme is responsible for the deficiency causing classical galactosemia? (Q856998)

A. Aldose reductase

B. Galactokinase

C. Galactose-1-phosphate uridyl transferase

D. UDP galactose-4-epimerase

See Answer in Part B → Q3

@Neet_pg_bot

Q4: Which one of the following choices does not represent a function of glucagon? (Q856992)

A. Glycolysis

B. Gluconeogenesis

C. Glycogenolysis

D. Ketogenesis

See Answer in Part B → Q4

@Neet_pg_bot

Q5: Glycogen phosphorylase is activated by: (Q855065)

A. Calcium

B. Glucose-6-phosphate

C. Insulin

D. Protein phosphatase

See Answer in Part B → Q5

@Neet_pg_bot

Q6: A baby is being evaluated for delayed developmental milestones. On examination, there is hepatosplenomegaly. Microscopic image of the bone marrow evaluation

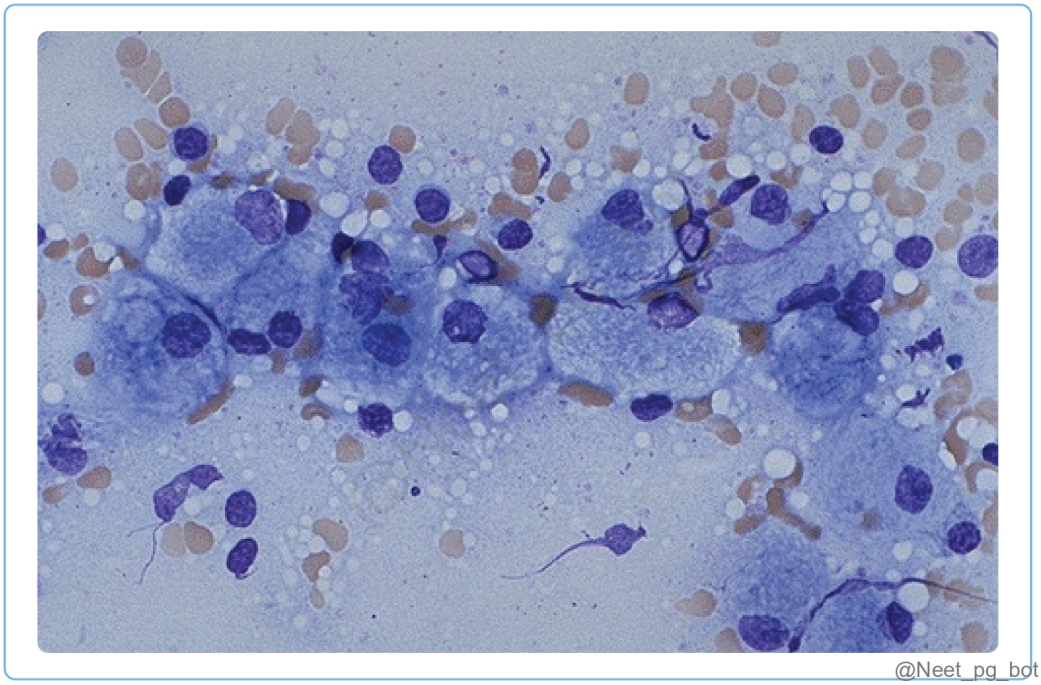
shown below. What is the diagnosis? (Q855544)

A. Gaucher disease

B. Niemann-pick disease

C. Hunter disease

D. Hurler disease



See Answer in Part B → Q6

@Neet_pg_bot

Q7: A newborn infant is brought to the clinic with jaundice and a white, punctate, and scattered opacity in the central lens. The condition is consistent with Oil drop cataract. Which of the following metabolic disorders is most likely responsible for this presentation? (Q857861)

A. Phenylketonuria (PKU)

B. Maple syrup urine disease

C. Galactosemia

D. Wilson's disease

See Answer in Part B → Q7

@Neet_pg_bot

Q8: A 2-month-old infant is brought to the pediatrician due to feeding difficulties, vomiting, and failure to thrive. The child's history reveals that the infant was born full-term and had a normal birth weight. On physical examination, the infant appears jaundiced, and the liver is palpable below the costal margin. Oil drop Cataracts are also noted on eye examination. Laboratory tests show elevated levels of galactose in the blood and urine. Based on these clinical findings, which enzyme deficiency is most likely responsible for this presentation? (Q857857)

A. G6PD (Glucose-6-phosphate dehydrogenase)

B. GALT (Galactose-1-phosphate uridylyltransferase)

C. ALDH (Aldehyde dehydrogenase)

D. PKU (Phenylalanine hydroxylase)

See Answer in Part B → Q8

@Neet_pg_bot

Q9: A child presents with developmental delay, bone pain, and inability to walk. On fundoscopy, a cherry-red spot is observed. There is no organomegaly. What is the most likely diagnosis? (Q855287)

A. Tay-Sachs disease

B. Niemann-Pick disease

C. Hurler syndrome

D. Gaucher disease

See Answer in Part B → Q9

@Neet_pg_bot

Q10: What are the main substrate and metabolic fuel used by RBCs during fasting? (Q855902)

A. Ketone bodies

B. Glucose

C. Free fatty acids

D. Amino acids

See Answer in Part B → Q10

@Neet_pg_bot

Q11: Which of the following is seen in low insulin:glucagon ratio? (Q855240)

A. Activation of lipoprotein lipase

B. Activation of glycogen synthase

C. Activation of phosphofructokinase

D. Activation of hormone-sensitive lipase

See Answer in Part B → Q11

@Neet_pg_bot

Q12: A 4-year-old child presents with easy fatiguability. The mother also complains that the child has increased hunger between meals, which is relieved after food. Liver examination revealed no glycogen. The enzyme most likely deficient is (Q855224)

A. Glycogen phosphorylase

B. Glycogen synthase

C. Debranching enzyme

D. Glucose 6 phosphatase

See Answer in Part B → Q12

@Neet_pg_bot

Q13: A young boy presents with muscle cramps and dark-colored urine after strenuous exercise. He reports that his symptoms improve after brief rest. Laboratory tests show normal blood glucose levels, no rise in serum lactate after exercise, and elevated creatine kinase. Which of the following is the most likely diagnosis? (Q855193)

A. McArdle disease

B. Hers disease

C. Cori's disease

D. Andersen disease

See Answer in Part B → Q13

@Neet_pg_bot

Q14: Glycogen phosphorylase is activated by? (Q854622)

A. Glucose 6 phosphatase

B. ATP

C. Calcium

D. Glucose

See Answer in Part B → Q14

@Neet_pg_bot

Q15: Which of the following organs or tissues demonstrate insulin-dependent glucose uptake ? (Q854604)

A. RBC

B. Pancreas

C. Adipose tissue

D. Hepatocytes

See Answer in Part B → Q15

@Neet_pg_bot

Q16: A newborn infant is brought to the clinic with jaundice and a white, punctate, and scattered opacity in the central lens. The condition is consistent with Oil drop cataract. Which of the following metabolic disorders is most likely responsible for this presentation? (Q820353)

A. Phenylketonuria (PKU)

B. Maple syrup urine disease

C. Galactosemia

D. Wilson's disease

See Answer in Part B → Q16

@Neet_pg_bot

Q17: A patient with a history of strenuous exercise forgot to eat a meal and later consumed alcohol at a party. Which of the following metabolic processes is inhibited by alcohol consumption? (Q860060)

A. Gluconeogenesis

B. Glycogenolysis

C. HMP shunt

D. Ketone body formation

See Answer in Part B → Q17

@Neet_pg_bot

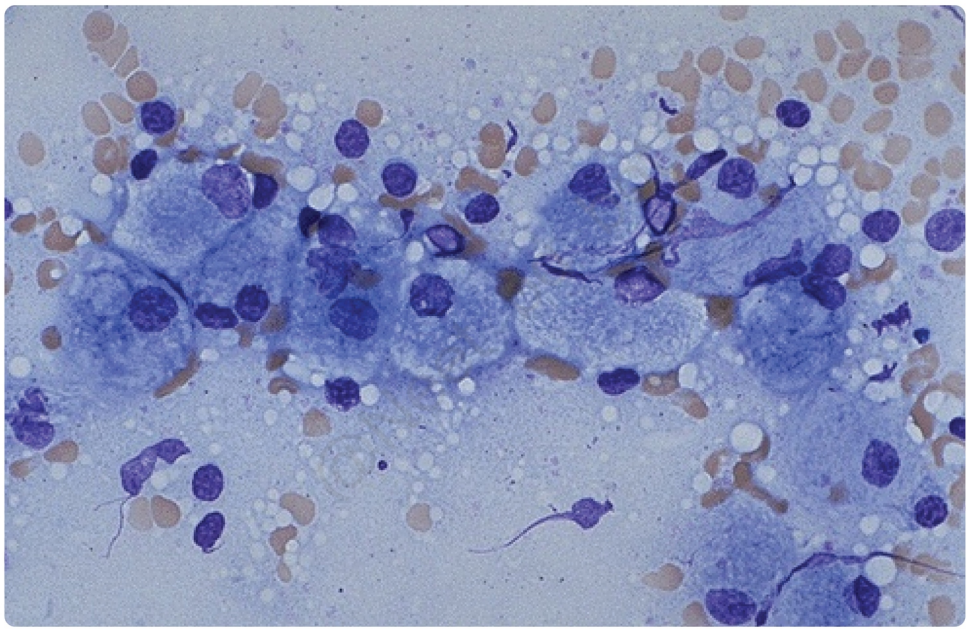
Q18: A 5 year old boy presented with easy fatigability, irritability and inability to concentrate. Labs revealed the following findings a and b (light and oil immersion respectively) on the bone marrow aspiration. Which of the following is the most likely enzyme deficient in this condition? (Q856005)

A. Hexosaminidase

B. Glucocerebrosidase

C. Sphingomyelinase

D. N-acetyglucosaminidase



See Answer in Part B → Q18

@Neet_pg_bot

Q19: A A 6-year-old child presents with recurrent abdominal pain. Examination reveals hepatomegaly, short stature and a doll-like facies. The laboratory studies showed anemia, acidosis and a deficiency of glucose-6-phosphatase. Which of the following features is most likely to be seen in this patient? (Q879291)

A. Fasting hypoglycemia

B. Fasting hypoketosis

C. Decreased hepatocellular cytoplasmic accumulation of glycogen

D. Muscle cramps

See Answer in Part B → Q19

@Neet_pg_bot

Q20: A 5-year-old girl was washing her doll with shampoo containing rotenone. Her mother noticed her in unconscious state. Which is inhibited by the above chemical? (Q844251)

A. NADH dehydrogenase

B. Succinate dehydrogenase

C. Cytochrome C

D. Cytochrome oxidase

See Answer in Part B → Q20

@Neet_pg_bot

Q21: Which of the following doesn't inhibit Cytochrome? (Q853002)

A. Nitric oxide

B. CO

C. H2S

D. Cyanide

See Answer in Part B → Q21

@Neet_pg_bot

Q22: Which of the following substances inhibits cytochrome complex IV, a component of the electron transport chain responsible for ATP synthesis? (Q855551)

A. Cyanide

B. Carbon dioxide

C. Oligomycin

D. Ouabain

See Answer in Part B → Q22

@Neet_pg_bot

Q23: Which enzyme deficiency could potentially be the cause of exercise intolerance and cramps in adolescent male patient? (Q855537)

A. Myophosphorylase

B. Hexokinase

C. Glucose-6-phosphatase

D. Hepatic glycogen phosphorylase

See Answer in Part B → Q23

@Neet_pg_bot

Q24: Ketosis is common in type 1 DM due to: - Decreased beta-oxidation of fatty acids Increased mobilization of fat from the adipose cell Decreased acetyl CoA in TCA cycle Increase beta-hydroxy butyryl synthase (Q856515)

A. 2,3 and 4

B. 1,2,3 and 4

C. 4 only

D. 1 and 2 only

See Answer in Part B → Q24

@Neet_pg_bot

Q25: Which enzyme is shared by both glycolysis and gluconeogenesis? (Q856507)

A. Pyruvate kinase

B. Phosphofructokinase

C. Phosphoenolpyruvate carboxy kinase

D. Phosphoglycerate kinase

See Answer in Part B → Q25

@Neet_pg_bot

Q26: Glucose is stored in the form of glycogen because (Q856876)

A. It is compact

B. It has multiple reducing ends

C. It can be stored at multiple organs

D. It can provide glucose for about 1 week when needed

See Answer in Part B → Q26

@Neet_pg_bot

Q27: Which of the following deficiency is found in congenital lactic acidosis? (Q839919)

A. Transketolase

B. Branched chain ketoacid dehydrogenase

C. Isocitrate dehydrogenase

D. Pyruvate dehydrogenase

See Answer in Part B → Q27

@Neet_pg_bot

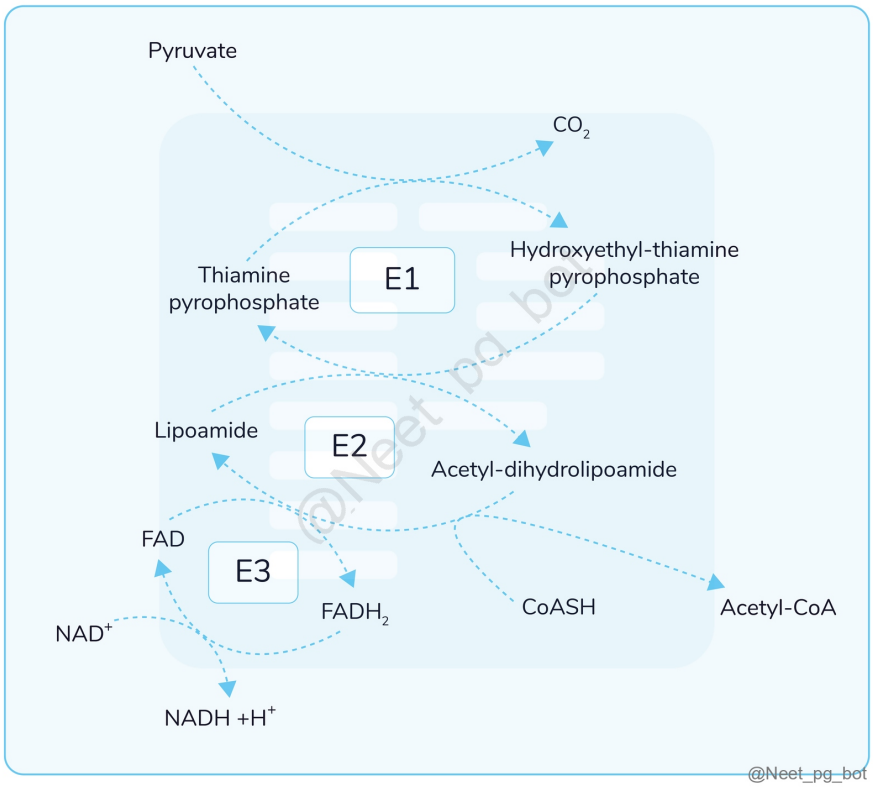
Q28: In the diagram provided, which enzymes are identified as E1 and E2, responsible for the conversion of pyruvate to acetyl CoA? (Q856000)

A. Pyruvate dehydrogenase and dihydrolipoyl transacetylase

B. Pyruvate carboxylase and dihydrolipoyl transacetylase

C. Pyruvate dehydrogenase and dihydrolipoyl dehydrogenase

D. Pyruvate carboxylase and dihydrolipoyl dehydrogenase



See Answer in Part B → Q28

@Neet_pg_bot

Q29: In this scenario, what is the origin of the blood glucose measured at 7 am the following morning for an individual who had dinner at 8 pm the previous night? (Q855985)

A. Hepatic glycogenolysis

B. Hepatic gluconeogenesis

C. Muscle glycogenolysis

D. Dietary glucose

See Answer in Part B → Q29

@Neet_pg_bot

Q30: You are conducting an experiment on mitochondrial respiration. You add malate/pyruvate and respiration is normal. You add succinate and respiration is normal. When you add another substance in the presence of pyruvate/succinate and malate, respiration is blocked. Which of the following substances is most likely added? (Q855978)

A. Rotenone

B. Antimycin A

C. Oligomycin

D. 2,4-dinitrophenol

See Answer in Part B → Q30

@Neet_pg_bot

Q31: Which one of the options provided does not serve as a substrate for the synthesis of glucose? (Q856613)

A. Acetyl coenzyme A

B. Glycerol

C. Alanine

D. Lactate

See Answer in Part B → Q31

@Neet_pg_bot

Q32: Which of the following poisoning agent does not inhibit cytochrome oxidase? (Q856558)

A. Hydrogen sulfide

B. Carbon monoxide

C. Methane

D. Cyanide

See Answer in Part B → Q32

@Neet_pg_bot

Q33: A patient with thiamine deficiency develops lactic acidosis. Which of the following enzymes is affected? (Q855957)

A. Pyruvate carboxylase

B. Pyruvate dehydrogenase

C. Phosphoenolpyruvate carboxykinase

D. Transketolase

See Answer in Part B → Q33

@Neet_pg_bot

Q34: Which of the following diagnostic markers is /are incorrectly paired? 1. Beta glucocerebrosidase - Von Gierke disease 2. Lipase - Acute pancreatitis 3. Ceruloplasmin - Hepatolenticular degeneration 4. High LDH1: LDH2 ratio - Myocardial infarction (Q855916)

A. 1 only

B. 1 and 2

C. 1, 2 and 4

D. 2, 3 and 4

See Answer in Part B → Q34

@Neet_pg_bot

Q35: Which among the following absorbs the least water? (Q858469)

A. Mucilage

B. Cellulose

C. Gums

D. Pectin

See Answer in Part B → Q35

@Neet_pg_bot

Q36: Which of the following is deficient in the mucopolysaccharidosis associated with Natowicz disease? (Q852553)

A. Dermatan sulfate

B. Heparan sulfate

C. Keratan sulfate

D. Hyaluronic acid

See Answer in Part B → Q36

@Neet_pg_bot

Q37: A 13-year-old boy who accompanied his father to a fish farming region in his hometown accidentally consumes a white odourless chemical. He was under some medication and post-meal he developed toxicity of the drug. Which of the following toxin is most likely to cause this condition? (Q857895)

A. Alkyl guanides

B. BAL

C. Antimycin A

D. Strychnine

See Answer in Part B → Q37

@Neet_pg_bot

Q38: In which of the following locations can cardiolipin be found? (Q830710)

A. Outer mitochondrial membrane

B. Inner mitochondrial membrane

C. Cytosol

D. Mitochondrial matrix

See Answer in Part B → Q38

@Neet_pg_bot

Q39: A 13-year-old boy who accompanied his father to a fish farming region in his hometown accidentally consumes a white odourless chemical. He was under some medication and post-meal he developed toxicity of the drug. Which of the the following toxin is most likely to cause this condition? (Q820415)

A. Alkyl guanides

B. BAL

C. Antimycin A

D. Strychnine

See Answer in Part B → Q39

@Neet_pg_bot

Q40: Which of the following minerals is best known for insulin secretion stabilisation? (Q820373)

A. Cadmium

B. Copper

C. Iron

D. Zinc

See Answer in Part B → Q40

@Neet_pg_bot

Q41: Cholera is known to cause diarrhea through its interaction with specific receptors in the human body. Which of the following is known to make it easier for cholera toxin to enter the body through the intestinal mucosa? (Q820337)

A. GLUT 1

B. GM2 ganglioside

C. Cephalin

D. GM1 ganglioside

See Answer in Part B → Q41

@Neet_pg_bot

Q42: What is the primary mechanism behind tissue damage in long-standing diabetes? (Q852558)

A. Insulin resistance

B. Sorbitol accumulation

C. Hyperglycemia

D. Decreased insulin secretion

See Answer in Part B → Q42

@Neet_pg_bot

Q43: A patient who hails from Mediterranean country and travels to Africa, they contact malaria. After receiving treatment with primaquine, the patient subsequently develops haemolytic anaemia. Which pathway deficiency in the enzyme could potentially be responsible for this condition? (Q855524)

A. Glycolysis

B. Gluconeogenesis

C. Hexose monophosphate (HMP) pathway

D. Luebering-Rapoport pathway

See Answer in Part B → Q43

@Neet_pg_bot

Q44: Complex IV is inhibited by --? (Q859423)

A. Rotenone

B. Cyanide

C. Carboxin

D. Antimycin A

See Answer in Part B → Q44

@Neet_pg_bot

Q45: Which is the enzyme activated by a decrease in insulin: glucagon ratio? (Q854882)

A. Glucokinase

B. Hexokinase

C. Phosphoructokinase

D. Glucose 6-phosphatase

See Answer in Part B → Q45

@Neet_pg_bot

Q46: Which of the following enzymes are involved in gluconeogenesis? Pyruvate carboxylase Phosphoenol pyruvate kinase Hexokinase Pyruvate kinase Glucose 6 phosphatase (Q856927)

A. 1, 2, and 5 only

B. 2 and 3 only

C. 1 and 5 only

D. 1, 2, and 3 only

See Answer in Part B → Q46

@Neet_pg_bot

Q47: Which of the following glycosaminoglycans (GAGs) are responsible for corneal transparency? (Q855981)

A. Keratan sulfate

B. Chondroitin sulfate

C. Heparin sulfate

D. Hyaluronic acid

See Answer in Part B → Q47

@Neet_pg_bot

Q48: Which of the following options is correct with respect to a fed state? 1. Insulin secretion is more 2. Glucagon secretion is less 3. Insulin secretions is less 4. Glucagon secretion is more (Q857486)

A. Only 1

B. Only 3

C. Option 1 and 2 are correct

D. Option 3 and 4 are correct

See Answer in Part B → Q48

@Neet_pg_bot

Q49: A 10-year-old boy rapidly develops hypoglycemia after moderate physical activity. Laboratory evaluation shows elevated ketone bodies, lactic acid, and triglycerides. On examination, hepatomegaly and renomegaly are noted. Which of the following enzyme deficiencies is most likely present in the liver of this child? (Q856947)

A. Glucose-6-phosphatase

B. Phosphoglucomutase

C. Phosphofructokinase

D. Defect in glycogen branching enzyme

See Answer in Part B → Q49

@Neet_pg_bot

Q50: Complex IV is inhibited by? (Q793381)

A. Rotenone

B. Cyanide

C. Carboxin

D. Antimycin A

See Answer in Part B → Q50

@Neet_pg_bot

Q51: What is the composition of lactulose? (Q860047)

A. Glucose + Galactose

B. Glucose + Fructose

C. Galactose + Fructose

D. Fructose + Ribulose

See Answer in Part B → Q51

@Neet_pg_bot

Q52: A patient’s X-ray shows cardiomegaly, and they present with a "floppy baby" syndrome, hypotonia, macroglossia, and hepatomegaly. What is the most likely diagnosis? (Q844238)

A. Pompe disease

B. Ebstein anomaly

C. Down syndrome

D. Cori's Disease

See Answer in Part B → Q52

@Neet_pg_bot

Q53: Match the following with the most appropriate feature. 1. Von Gierke's a. Branched structure in liver biopsy 2. Pompe's b. Glycogen deposition in lysosomes 3. McArdle's c. Hypoglycemia with lactic acidosis 4. Cori's d. Exercise intolerance (Q839906)

A. 1c, 2a,3d,4b

B. 1c, 2b,3d,4a

C. 1a, 2c,3d,4b

D. 1d, 2c,3b,4d

See Answer in Part B → Q53

@Neet_pg_bot

Q54: Which receptor plays a crucial role in glucose uptake by skeletal muscle? (Q860412)

A. GLUT 1

B. GLUT 2

C. GLUT 3

D. GLUT 4

See Answer in Part B → Q54

@Neet_pg_bot

Q55: What is the rate-limiting enzyme of cholesterol synthesis? (Q860403)

A. HMG-CoA reductase

B. 7-alpha hydroxylase

C. Carnitine acyltransferase 1

D. Carnitine acyltransferase 2

See Answer in Part B → Q55

@Neet_pg_bot

Q56: Which of the following is the rate-limiting enzyme of the hexose monophosphate (HMP) pathway? (Q860387)

A. Glucose-6-phosphatase

B. Glucose-6-phosphate dehydrogenase

C. Phosphofructokinase-1

D. Glycogen phosphorylase

See Answer in Part B → Q56

@Neet_pg_bot

Q57: A diabetic patient is advised to exercise. Which of the following glucose transporters is insulin-sensitive? (Q844249)

A. GLUT4

B. GLUT1

C. GLUT3

D. GLUT2

See Answer in Part B → Q57

@Neet_pg_bot

Q58: A neonate presents with poor feeding and vomiting, cataract, and acidosis with a positive Benedict's test on urine and negative urinary glucose. Which enzyme is likely defective? (Q844276)

A. Galactose-1-phosphate uridyl transferase

B. Aldolase B

C. Fructokinase

D. Glucose-6-phosphatase

See Answer in Part B → Q58

@Neet_pg_bot

Q59: A patient with high triglycerides esterified with long-chain fatty acids presents with fatigue, and muscle biopsy shows fat vacuoles. What is the most likely diagnosis? (Q844255)

A. Carnitine deficiency

B. Fatty Acid Synthase defect

C. Lipoprotein Lipase (LPL) deficiency

D. Medium-chain acyl-CoA dehydrogenase (MCAD) deficiency

See Answer in Part B → Q59

@Neet_pg_bot

Q60: A breastfed infant presents with lethargy, hepatomegaly, and cataracts. Which of the following enzyme deficiencies is most likely responsible for this presentation? (Q866651)

A. Galactokinase

B. Galactose-1-phosphate uridyltransferase (GALPUT)

C. Aldolase B

D. Fructokinase

See Answer in Part B → Q60

@Neet_pg_bot

Q61: A patient with a history of strenuous exercise skipped a meal and later consumed alcohol at a party. Alcohol inhibits which of the following biochemical processes, potentially leading to hypoglycemia? (Q852549)

A. Gluconeogenesis

B. Glycogenolysis

C. Hexose Monophosphate (HMP) Shunt

D. Ketone Body Formation

See Answer in Part B → Q61

@Neet_pg_bot

Q62: Which of the following is excreted in the urine of patients with pentosuria? (Q852461)

A. L-Xylulose

B. D-Ribose

C. D-Ribulose

D. D-Lactose

See Answer in Part B → Q62

@Neet_pg_bot

Q63: During starvation, which of the following hormones is primarily produced to maintain blood glucose levels? (Q852554)

A. Glucagon

B. Insulin

C. Amylase

D. Somatostatin

See Answer in Part B → Q63

@Neet_pg_bot

Q64: When blood glucose concentration is suddenly increased to 2–3 times the normal level and maintained at that level, what will be the typical pattern of blood glucose change observed over time? (Q866337)

A. Rapid rise followed by sustained high glucose levels

B. Continuous increase in insulin secretion without plateau

C. Rise in glucose followed by a fall below baseline

D. Initial rise and then stabilization slightly above baseline

See Answer in Part B → Q64

@Neet_pg_bot

Q65: After a high-carbohydrate meal, weight gain is explained by all except? (Q865420)

A. Malonyl CoA

B. Tricarboxylate transporter

C. Acetyl-CoA carboxylase

D. Mitochondrial carnitine

See Answer in Part B → Q65

@Neet_pg_bot

Q66: Cancer cells take up excess glucose because? (Q865417)

A. Lactate is produced even in the presence of Oxygen

B. High NADH/NAD ratio

C. High GLUT2

D. Absence of Oxygen

See Answer in Part B → Q66

@Neet_pg_bot

Q67: A child presents with fatigue and hepatomegaly. Liver enzymes (ALT, AST) are elevated. Ketosis was significant. Liver biopsy shows excess glycogen accumulation. After feeding, blood glucose levels rise, but there is no rise in glucose after overnight fasting. Which of the following enzyme deficiencies is most likely responsible for this presentation? (Q865410)

A. Glucose-6-phosphatase

B. Liver phosphorylase

C. Muscle phosphorylase

D. Phosphofructokinase

See Answer in Part B → Q67

@Neet_pg_bot

Q68: In a fasting state, glucagon promotes the production of glucose by which of the following pathways? (Q793392)

A. Glycolysis and gluconeogenesis

B. Gluconeogenesis and glycogenolysis

C. Glycogenesis and gluconeogenesis

D. Only gluconeogenesis

See Answer in Part B → Q68

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• [03_Lipid Chemistry] 009 Lipid Chemistry

« Back to Folder Directory

[03_Lipid Chemistry] 009 Lipid Chemistry — Quick Jump

Q1 (p. 107)	*Q2 (p. 107)*	*Q3 (p. 108)*	*Q4 (p. 108)*	*Q5 (p. 109)*	*Q6 (p. 109)*
Q7 (p. 110)	*Q8 (p. 110)*	*Q9 (p. 111)*	*Q10 (p. 112)*	*Q11 (p. 112)*	*Q12 (p. 112)*
Q13 (p. 113)	*Q14 (p. 114)*	*Q15 (p. 114)*	*Q16 (p. 114)*	*Q17 (p. 115)*	*Q18 (p. 115)*
Q19 (p. 116)					

Q1: In a patient with a history of recurring attacks of pancreatitis, eruptive xanthomas, increased plasma triglyceride levels (2,000 mg/dL), and milky plasma. Which of the following is the most likely deficiency? (Q882115)

A. Lipoprotein Lipase

B. LDL receptors

C. HMG-CoA reductase

D. ABCA1 receptor

See Answer in Part B → Q1

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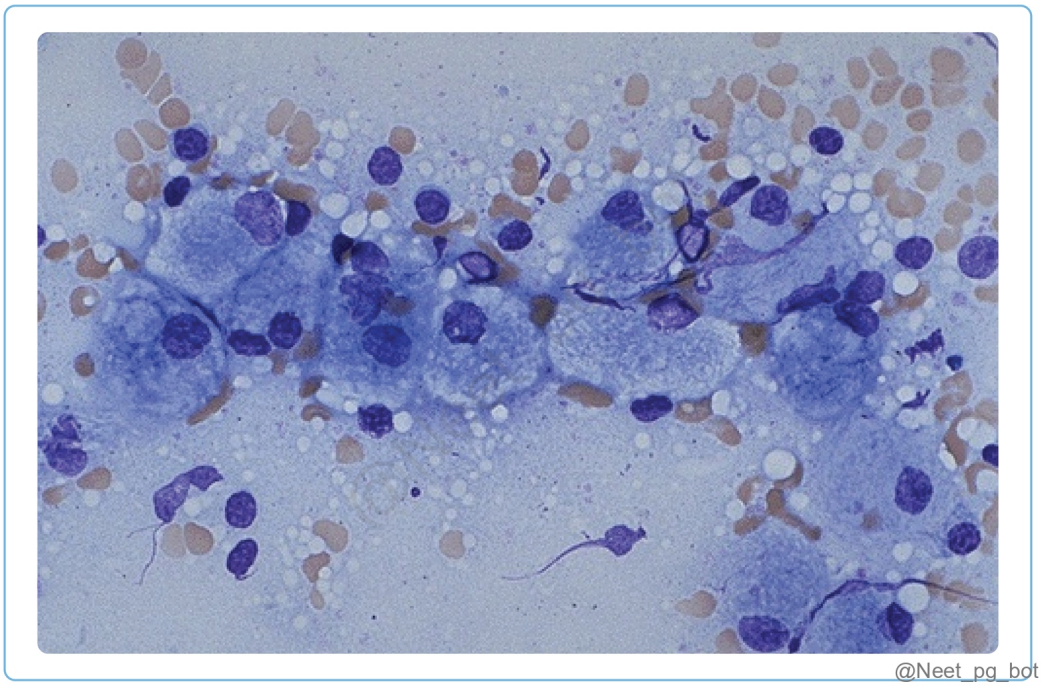
Q2: A patient presents with bone pain, fatigue and hepatosplenomegaly. Blood tests reveal anemia and thrombocytopenia. Bone marrow biopsy shows macrophages with a characteristic "wrinkled paper" appearance. Which substrate accumulates in Gaucher's disease due to the deficient enzyme activity? (Q843064)

A. Sphingomyelin

B. Glucosylceramide

C. Ganglioside

D. Ceramide



See Answer in Part B → Q2

@Neet_pg_bot

Q3: In a patient with a history of recurring attacks of pancreatitis, eruptive xanthomas, increased plasma triglyceride levels (2,000 mg/dL), and milky plasma. Which of the following is the most likely deficiency? (Q882162)

A. Lipoprotein Lipase

B. LDL receptors

C. HMG-CoA reductase

D. ABCA1 receptor

See Answer in Part B → Q3

@Neet_pg_bot

Q4: Which enzyme in snake venom hydrolyzes only sn-2 acyl bond of phospholipids? (Q843438)

A. Phospholipase A2

B. Phospholipase B

C. Phospholipase C

D. Phospholipase D

See Answer in Part B → Q4

@Neet_pg_bot

Q5: Which of the following fatty acids is needed for humans from diet as humans cannot create double bonds beyond the 9th carbon from the carboxyl end of a fatty acid chain? (Q843434)

A. Alpha-linolenic acid

B. Palmitic acid

C. Oleic acid

D. Stearic acid

See Answer in Part B → Q5

@Neet_pg_bot

Q6: Which of the following lipids is characterized by its amphipathic nature, allowing it to form micelles in aqueous solutions? (Q843070)

A. Triacylglycerols

B. Cholesteryl esters

C. Phospholipids

D. Nonpolar lipids

See Answer in Part B → Q6

@Neet_pg_bot

Q7: Which of the following pairs correctly associates a disease with its corresponding enzyme deficiency? (Q843068)

A. Tay-Sachs disease - α-Galactosidase

B. Gaucher disease - β-Glucosidase

C. Farber disease - Sphingomyelinase

D. Fabry disease - Ceramidase

See Answer in Part B → Q7

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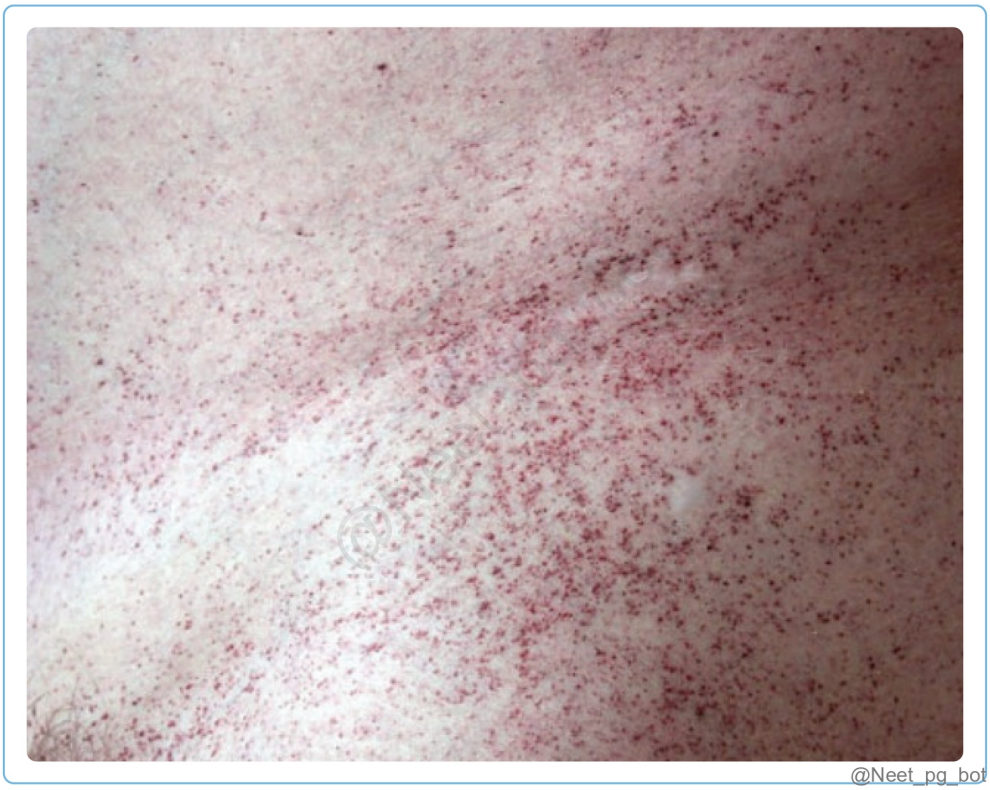
Q8: A 9-year-old boy presents with episodic pain in his extremities, skin finding as shown in the image, and progressive renal dysfunction. A biopsy of the skin reveals the accumulation of ceramide trihexoside. An α-galactosidase deficiency is suspected. What is the diagnosis? (Q843067)

A. Tay-Sachs disease

B. Fabry disease

C. Krabbe disease

D. Gaucher disease



See Answer in Part B → Q8

@Neet_pg_bot

Q9: A 6-month-old infant presents with a hoarse cry, painful and swollen joints, and skin nodules. The child also shows signs of developmental delay and mental retardation. Histological analysis reveals the accumulation of a certain substance in the tissues, which confirms the diagnosis of Farber disease. Which enzyme deficiency is responsible for this condition? (Q843066)

A. Hexosaminidase A

B. Arylsulfatase A

C. Ceramidase

D. Sphingomyelinase

See Answer in Part B → Q9

@Neet_pg_bot

Q10: A 6-month-old infant presents with hepatosplenomegaly, progressive neurodegeneration, and a history of frequent respiratory infections. A biopsy shows lipid-laden macrophages and a genetic test confirms the diagnosis. Which of the following enzyme deficiencies is most likely responsible for this condition? (Q843065)

A. Hexosaminidase A

B. Arylsulfatase A

C. Sphingomyelinase

D. β-Glucosidase

See Answer in Part B → Q10

@Neet_pg_bot

Q11: Which of the following correctly matches the type of lipid with its description? (Q843051)

A. Fats - Liquid at room temperature, often containing polyunsaturated fatty acids.

B. Phospholipids - Contain glycerol and are the main lipid constituents of cell membranes.

C. Glycolipids - Esters of fatty acids that never contain sphingosine.

D. Sphingophospholipids - Cerebrosides is a subtype

See Answer in Part B → Q11

@Neet_pg_bot

Q12: A 4-month-old infant presents developmental delay. MRI shows demyelination in the central nervous system. Which of the following is the most likely diagnosis, and what enzyme deficiency causes this condition? (Q843063)

A. Niemann-Pick disease, Sphingomyelinase

B. Krabbe disease, β-Galactosidase

C. Farber disease, Ceramidase

D. Gaucher disease, β-Glucosidase

See Answer in Part B → Q12

@Neet_pg_bot

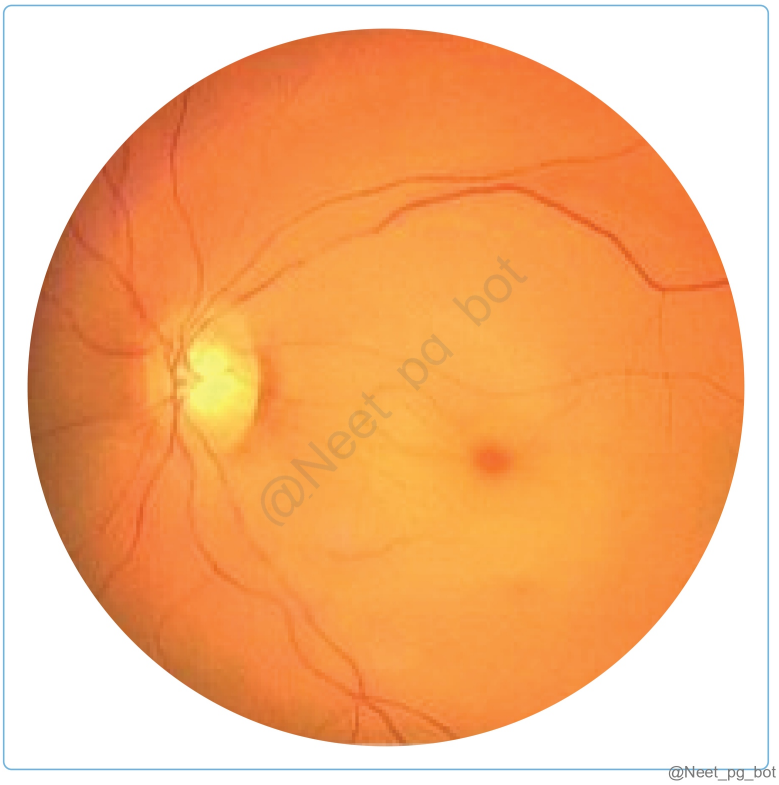
Q13: A 6-month-old infant has developmental delay, muscle weakness, and on eye examination, a cherry-red spot on the macula is noted. What is the most likely diagnosis, and which enzyme deficiency is responsible? (Q843062)

A. Niemann-Pick disease, Sphingomyelinase

B. Gaucher disease, Glucocerebrosidase

C. Tay-Sachs disease, Hexosaminidase A

D. Sandhoff disease, Beta-galactosidase



See Answer in Part B → Q13

@Neet_pg_bot

Q14: Which enzyme deficiency is responsible for GM1 gangliosidosis? (Q843060)

A. Beta-glucosidase

B. Beta-galactosidase

C. Alpha-galactosidase

D. Hexosaminidase

See Answer in Part B → Q14

@Neet_pg_bot

Q15: Which component is present in gangliosides but not in cerebrosides or globosides? (Q843059)

A. Monosaccharides

B. Ceramide

C. Fatty acid

D. N-acetyl Neuraminic Acid

See Answer in Part B → Q15

@Neet_pg_bot

Q16: Which of the following are the correct components of sphingomyelin? (Q843057)

A. Sphingosine + Fatty acid + Choline

B. Ceramide + Phosphate + Choline

C. Sphingosine + Phosphate + Choline

D. Ceramide + Phosphate + Fatty acids

See Answer in Part B → Q16

@Neet_pg_bot

Q17: A 30-year-old woman presents with a history of four consecutive miscarriages over the past three years. After a thorough evaluation, a diagnosis is made . Which of the following phospholipids is specifically antigenic in this disease? (Q843056)

A. Phosphatidylserine

B. Phosphatidylinositol

C. Cardiolipin

D. Phosphatidylethanolamine

See Answer in Part B → Q17

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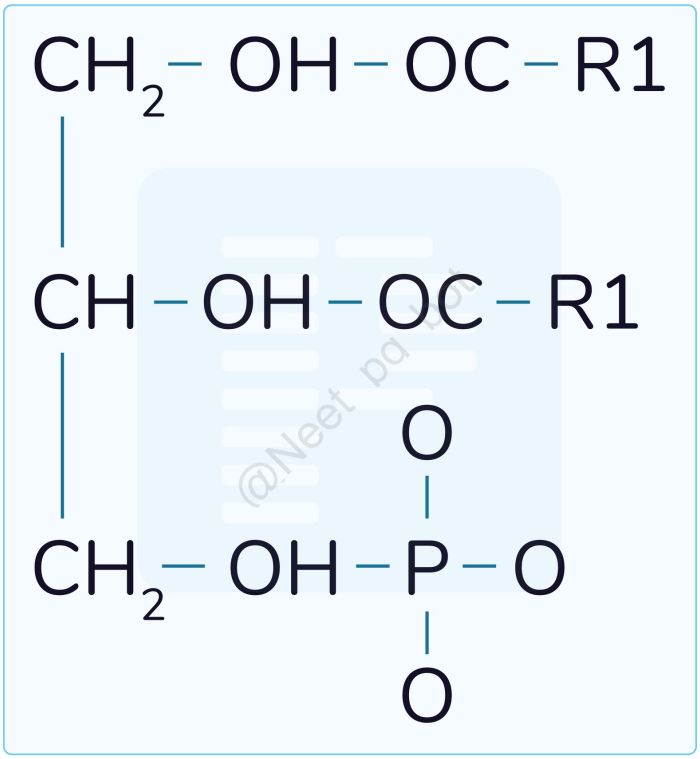
Q18: What compound is formed when choline is added to the following structure? (Q843054)

A. Lecithin

B. Cephalin

C. Cardiolipin

D. Inositol



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See Answer in Part B → Q18

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Q19: A premature baby presents with infant respiratory distress syndrome. The neonatologist says the neonate is yet to produce a type of phospholipid adequately. All of the following belong to the same class except? (Q843052)

A. Cephalin

B. Cardiolipin

C. Lecithin

D. Cerebrosides

See Answer in Part B → Q19

@Neet_pg_bot

• [03_Lipid Chemistry] 010 Fatty Acid Synthesis And Oxidation - I

« Back to Folder Directory

[03_Lipid Chemistry] 010 Fatty Acid Synthesis And Oxidation - I — Quick Jump

Q1 (p. 117)	*Q2 (p. 117)*	*Q3 (p. 118)*	*Q4 (p. 118)*	*Q5 (p. 118)*	*Q6 (p. 119)*
Q7 (p. 119)	*Q8 (p. 119)*	*Q9 (p. 120)*	*Q10 (p. 120)*	*Q11 (p. 121)*	*Q12 (p. 121)*
Q13 (p. 121)	*Q14 (p. 122)*	*Q15 (p. 122)*	*Q16 (p. 122)*	*Q17 (p. 123)*	*Q18 (p. 123)*
Q19 (p. 123)	*Q20 (p. 124)*	*Q21 (p. 124)*	*Q22 (p. 125)*	*Q23 (p. 125)*	*Q24 (p. 125)*

Q1: Which of the following is an end product of beta-oxidation of odd-chain fatty acids? (Q845005)

A. Propionyl-CoA

B. Malonyl CoA

C. Succinyl CoA

D. Pyruvate

See Answer in Part B → Q1

@Neet_pg_bot

Q2: Which of the following organs can utilize ketone bodies for energy, and which enzyme is involved in their metabolism? (Q845004)

A. Liver- Thiophorase

B. Heart muscle- Acetyl-CoA carboxylase

C. Brain- Thiophorase

D. Liver- Carboxylase

See Answer in Part B → Q2

@Neet_pg_bot

Q3: Which of the following statements accurately describes the role of glucagon in ketogenesis during starvation? (Q845003)

A. Glucagon decreases lipolysis and reduces ketogenesis.

B. Glucagon inhibits gluconeogenesis and promotes ketogenesis.

C. Glucagon increases lipolysis and stimulates ketogenesis.

D. Glucagon decreases fatty acid entry into mitochondria and inhibits ketogenesis.

See Answer in Part B → Q3

@Neet_pg_bot

Q4: Which one of the following is the major regulator of carnitine acyltransferase I (CAT-I) activity? (Q844996)

A. Insulin

B. Glucagon

C. Malonyl CoA

D. ATP

See Answer in Part B → Q4

@Neet_pg_bot

Q5: Which one of the following does not occur during β-oxidation of fatty acids? (Q844998)

A. Dehydrogenation of fatty acyl-CoA to trans-enoyl-CoA

B. Formation of acetyl-CoA from β-keto fatty acyl-CoA

C. Conversion of trans-enoyl-CoA to saturated fatty acyl-CoA

D. Hydration of trans-enoyl-CoA to β-hydroxy fatty acyl-CoA

See Answer in Part B → Q5

@Neet_pg_bot

Q6: What is the Total and Net ATP used in the complete oxidation of Arachidonic acid? (Q844999)

A. 136, 134

B. 134, 136

C. 120, 122

D. 120, 118

See Answer in Part B → Q6

@Neet_pg_bot

Q7: Which of the following is NOT an inhibitor of Carnitine Acyl Transferase-1 (CAT-1)? (Q845000)

A. Sulfonylureas

B. Malonyl-CoA

C. AMPK

D. Insulin

See Answer in Part B → Q7

@Neet_pg_bot

Q8: A 3-y/o child is brought to the emergency department with symptoms of severe fatigue, vomiting, and episodes of hypoglycemia. Blood tests reveal low glucose levels without elevated ketone bodies, as indicated by a negative Rothera and Benedict tests. Further examination indicates a fatty liver. The child's mother gives h/o acute fatty liver during her pregnancy with the child. Which of the following deficiencies is most

likely responsible for these symptoms? (Q845001)

A. CPT-I Deficiency

B. Long-Chain 3-Hydroxyacyl-CoA Dehydrogenase Deficiency

C. Jamaican Vomiting Sickness

D. Carnitine Deficiency

See Answer in Part B → Q8

@Neet_pg_bot

Q9: Which enzyme requires vitamin B12 for the isomerization of L-methylmalonyl-CoA to succinyl-CoA during odd-chain fatty acid oxidation? (Q845002)

A. Propionyl-CoA carboxylase

B. Methyl malonyl-CoA mutase

C. Methyl malonyl-CoA racemase

D. Biotin

See Answer in Part B → Q9

@Neet_pg_bot

Q10: During activation of long-chain fatty acids, ATP is hydrolyzed directly to AMP and pyrophosphate (PPi) by which enzyme? (Q885194)

A. Acyl-CoA dehydrogenase

B. Acyl-CoA synthetase (thiokinase)

C. Carnitine palmitoyltransferase I

D. Inorganic pyrophosphatase

See Answer in Part B → Q10

@Neet_pg_bot

Q11: A newborn presents with characteristic facial features (mongoloid facies, hypertelorism), hypotonia, seizures, and hepatomegaly. Accumulation of very-long-chain fatty acids (VLCFA) is found. The most likely diagnosis is: (Q885207)

A. Refsum disease

B. MCAD deficiency

C. Zellweger Syndrome

D. Adrenoleukodystrophy

See Answer in Part B → Q11

@Neet_pg_bot

Q12: During fatty acid oxidation, acyl-carnitine is formed as an intermediate in which process? (Q885195)

A. Activation of fatty acids

B. Transport across the inner mitochondrial membrane

C. Beta-oxidation spiral

D. Ketone body synthesis

See Answer in Part B → Q12

@Neet_pg_bot

Q13: Which enzyme catalyses the initial energy-requiring "activation" step necessary for fatty acid oxidation to occur? (Q885201)

A. Acyl-CoA dehydrogenase

B. Enoyl-CoA hydratase

C. Thiolase

D. Fatty Acyl-CoA synthetase (Thiokinase)

See Answer in Part B → Q13

@Neet_pg_bot

Q14: In each cycle of beta-oxidation, which of the following is not produced? (Q885202)

A. 1 FADH■

B. 1 NADH

C. 1 Acetyl-CoA

D. 1 GTP

See Answer in Part B → Q14

@Neet_pg_bot

Q15: A straight-chain fatty acid with 2N carbon atoms undergoes beta-oxidation. The net ATP yield can be calculated by which formula? (Q885204)

A. 10N - 2

B. 12N - 4

C. 14N - 6

D. 17N - 8

See Answer in Part B → Q15

@Neet_pg_bot

Q16: A child presents with hypoketotic hypoglycemia and dicarboxylic aciduria (C6-C10 dicarboxylic acids in urine). The most likely diagnosis is: (Q885205)

A. Refsum disease

B. Zellweger syndrome

C. Medium-chain acyl-CoA dehydrogenase (MCAD) deficiency

D. CPT-I deficiency

See Answer in Part B → Q16

@Neet_pg_bot

Q17: Which of the following enzymes is correctly paired with its function in metabolic processes? (Q844988)

A. Acetyl CoA Carboxylase (ACC) converts acetyl-CoA to citrate, initiating fatty acid synthesis.

B. Carnitine Palmitoyltransferase I (CPT-I) regulates entry of all fatty acids into mitochondria for beta-oxidation.

C. HMG-CoA Synthase catalyzes ketone body production from acetyl-CoA.

D. Acetyl CoA Carboxylase (ACC) converts acetyl-CoA to malonyl-CoA, the rate-limiting step in fatty acid Oxidation

See Answer in Part B → Q17

@Neet_pg_bot

Q18: Which of the following statements about alpha-oxidation is true? (Q885186)

A. It is a major energy-yielding process

B. It occurs primarily in mitochondria

C. It is not an energy-yielding process

D. It produces FADH■ and NADH

See Answer in Part B → Q18

@Neet_pg_bot

Q19: Which of the following enzymes is the most important target for the regulation of fatty acid synthesis by citrate? (Q875985)

A. Hydratase

B. Acetyl-CoA carboxylase

C. Enoyl reductase

D. Acetyl transacetylase

See Answer in Part B → Q19

@Neet_pg_bot

Q20: Which of the following fatty acid metabolism primarily takes place in the peroxisome? (Q844995)

A. Very long chain FA

B. Medium Chain FA

C. Short chain FA

D. Long chain FA

See Answer in Part B → Q20

@Neet_pg_bot

Q21: Which of the following inhibits Acetyl CoA Carboxylase? (Q844994)

A. Glucagon

B. NADH

C. ATP

D. Insulin

See Answer in Part B → Q21

@Neet_pg_bot

Q22: Which of the following correctly describes the products and by-products of the palmitic acid synthesis reaction? (Q844993)

A. 1 Palmitate + 7 CO■ + 6 H■O + 8 CoA–SH + 14 NADPH

B. 1 Palmitate + 7 CO■ + 6 H■O + 8 CoA–SH + 14 NADP+

C. 1 Palmitate + 7 CO■ + 8 H■O + 8 CoA–SH + 14 NADP■

D. 1 Palmitate + 7 CO■ + 6 H■O + 8 CoA–SH + 14 NADP■ + 14 H■

See Answer in Part B → Q22

@Neet_pg_bot

Q23: Which of the following statements about the fatty acid synthesis mechanism is NOT TRUE? (Q844992)

A. Acetyl-CoA is converted to malonyl-CoA by acetyl-CoA carboxylase.

B. The FAS complex is a homodimer with two identical subunits and seven enzymes

C. NADPH is primarily generated by the citric acid cycle.

D. Palmitate is the main product, which can be converted into shorter fatty acids.

See Answer in Part B → Q23

@Neet_pg_bot

Q24: Which of the following statements about the Fatty acid synthase (FAS) complex is true? (Q844989)

A. The FAS complex synthesizes fatty acids exclusively in the mitochondria.

B. The primary product of the FAS complex is palmitic acid, a 16-carbon saturated fatty acid.

C. The FAS complex utilizes the same enzymes and intermediates as those found in β-oxidation.

D. Each subunit of the FAS complex consists of a single enzyme responsible for fatty acid synthesis.

See Answer in Part B → Q24

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• [03_Lipid Chemistry] 011 Cholesterol Metabolism Lipoproteins - I

« Back to Folder Directory

[03_Lipid Chemistry] 011 Cholesterol Metabolism Lipoproteins - I — Quick Jump

Q1 (p. 127)	*Q2 (p. 127)*	*Q3 (p. 128)*	*Q4 (p. 128)*	*Q5 (p. 129)*	*Q6 (p. 129)*
Q7 (p. 129)	*Q8 (p. 130)*	*Q9 (p. 130)*	*Q10 (p. 131)*	*Q11 (p. 131)*	*Q12 (p. 131)*
Q13 (p. 132)	*Q14 (p. 132)*	*Q15 (p. 133)*	*Q16 (p. 133)*	*Q17 (p. 133)*	*Q18 (p. 134)*
Q19 (p. 134)	*Q20 (p. 135)*				

Q1: Which of the following best describes Tangier disease? (Q845019)

A. Low VLDL levels

B. Low HDL levels

C. Low LDL levels

D. Low LDL receptor activity

See Answer in Part B → Q1

@Neet_pg_bot

Q2: A 12-year-old boy presents with developmental delays, visual impairment, and recurrent episodes of ataxia. Laboratory tests reveal low serum levels of triglycerides, cholesterol, and fat-soluble vitamins. A blood smear shows the presence of acanthocytes. Which of the following findings is most likely present in this patient? (Q844854)

A. Elevated levels of chylomicrons in the bloodstream.

B. Absence of apolipoprotein B-containing lipoproteins.

C. Normal absorption of dietary fats.

D. High serum levels of low-density lipoprotein (LDL).

See Answer in Part B → Q2

@Neet_pg_bot

Q3: A researcher is studying the mechanisms of lipoprotein lipase (LPL) activity in post-heparinized blood samples. They conduct a mixing study to differentiate between a deficiency of apolipoprotein C-II (Apo C-II) and LPL deficiency. Which of the following outcomes from the mixing study would most accurately indicate an Apo C-II deficiency? (Q843472)

A. The addition of pooled normal plasma results in no change in LPL activity.

B. LPL activity normalizes after mixing with normal plasma.

C. A significant increase in triglyceride levels is observed after mixing.

D. LPL activity decreases following the addition of normal plasma.

See Answer in Part B → Q3

@Neet_pg_bot

Q4: Match the following contributing factors with their corresponding effects on fatty liver: A) Choline Deficiency 1) Increases NADH, hindering fatty acid oxidation and promoting triacylglycerol accumulation. B) Orotic Acid Interference 2) Impairs VLDL secretion, resulting in triacylglycerol accumulation and a fatty liver. C) Vitamin E and Selenium 3) Involved in pyrimidine synthesis. Disrupts VLDL glycosylation, hindering release. D) Ethanol Consumption 4) Protect against liver damage from lipid peroxidation (Q843471)

A. A-2, B-3, C-4, D-1

B. A-3, B-4, C-2, D-1

C. A-4, B-1, C-3, D-2

D. A-1, B-2, C-4, D-3

See Answer in Part B → Q4

@Neet_pg_bot

Q5: Which among the following is a true statement on electrophoretic mobility of lipoproteins seen during serum electrophoresis? (Q843470)

A. HDL shows the least mobility due to high lipid content.

B. Lipoproteins with higher protein content move slower towards the anode.

C. Lipoproteins with higher protein content migrate faster towards the anode

D. Lipoproteins identified by dye color after staining with lipid dyes, not electrophoretic mobility.

See Answer in Part B → Q5

@Neet_pg_bot

Q6: A 45-year-old male presents with symptoms of peripheral neuropathy, enlarged tonsils, and orange-colored tonsils. His lipid profile shows very low levels of high-density lipoprotein (HDL) cholesterol. Further investigation reveals that the patient has a rare genetic condition leading to severe HDL deficiency. Given these findings, which of the following gene mutations is most likely responsible for this condition? (Q843026)

A. ABCA1 gene mutation

B. LDLR gene mutation

C. APOE gene mutation

D. PCSK9 gene mutation

See Answer in Part B → Q6

@Neet_pg_bot

Q7: A 45-year-old woman presents with a history of palmar xanthoma. She reports a family history of similar symptoms. Laboratory tests reveal elevated cholesterol and triglyceride levels, with a cloudy appearance of her plasma.What is the most likely diagnosis for this patient? (Q843191)

A. Type I (Familial chylomicronemia syndrome)

B. Type IIa (Familial hypercholesterolemia)

C. Type III (Familial dysbetalipoproteinemia)

D. Type IV (Familial hypertriglyceridemia)

See Answer in Part B → Q7

@Neet_pg_bot

Q8: Which of the following statements accurately distinguishes Type IIa from Type IIb Hyperlipoproteinemias? (Q843190)

A. Plasma after 24 hrs- Type IIa: clear; Type IIb: slightly cloudy.

B. Clinical Features- Type IIa: eruptive xanthoma ; Type IIb: corneal arcus.

C. Type IIa: VLDL overproduction; Type IIb: LDL and VLDL overproduction.

D. Management - Type IIa: restrict fat, supplement MCT; Type IIb: weight reduction, drug therapy.

See Answer in Part B → Q8

@Neet_pg_bot

Q9: A 5-year-old boy presents with recurrent episodes of severe abdominal pain and hepatomegaly. Physical examination reveals eruptive xanthomas on his trunk and limbs. Blood tests show a creamy layer over clear plasma after overnight refrigeration. What metabolic defect is most likely present in this patient? (Q843189)

A. LDL receptor defect

B. Apo B-100 mutation

C. Lipoprotein lipase deficiency

D. Apo E abnormality

See Answer in Part B → Q9

@Neet_pg_bot

Q10: Match the apolipoproteins with their respective functions in lipid metabolism: Apolipoprotein Function 1) Apolipoprotein A-I a. Enhances lipoprotein lipase activity, facilitating triglyceride hydrolysis. 2) Apolipoprotein B-100 b. Involved in the transport of dietary lipids from the intestine to other tissues. 3) Apolipoprotein C-II c. Helps in reverse cholesterol transport, removing excess cholesterol from tissue back to liver. 4) Apolipoprotein E d. Essential for binding to LDL receptors on various tissues. (Q843188)

A. 1-c, 2-d, 3-a, 4-b

B. 1-b, 2-c, 3-d, 4-a

C. 1-c, 2-d, 3-d, 4-a

D. 1-d, 2-a, 3-c, 4-b

See Answer in Part B → Q10

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Q11: Following are the substrates of cholesterol synthesis. Arrange them in order of appearance in the cholesterol synthesis pathway. Mevalonate Squalene Lanosterol Zymosterol Desmosterol (Q843016)

A. 1,2,3,4,5

B. 5,4,3,2,1

C. 4,3,1,2,5

D. 1,3,5,2,4

See Answer in Part B → Q11

@Neet_pg_bot

Q12: A 45-year-old male presents with a history of progressive kidney dysfunction and recurrent episodes of hemolytic anemia. On examination, the patient is found to

have corneal opacification on slit-lamp examination. His lipid profile shows high levels of cholesterol and lipoprotein X. What is the most likely diagnosis? (Q843025)

A. Tangier disease

B. Familial hypercholesterolemia

C. LCAT deficiency

D. Niemann-Pick disease

See Answer in Part B → Q12

@Neet_pg_bot

Q13: Which of the following best describes the primary function of Very Low-Density Lipoprotein (VLDL) in the body? (Q843024)

A. Transport of dietary cholesterol to the liver

B. Transport of triglycerides from the liver to peripheral tissues

C. Removal of cholesterol from peripheral tissues to the liver

D. Storage of excess cholesterol in adipose tissue

See Answer in Part B → Q13

@Neet_pg_bot

Q14: The lipid profile of 55 Y/O female is given below: Triglycerides 150 mg/dL Total cholesterol 350 mg/dL HDL 90 mg/dL Calculate LDL level of this patient. (Q843023)

A. 250 mg/dL

B. 230 mg/dL

C. 200 mg/dL

D. 270 mg/dL

See Answer in Part B → Q14

@Neet_pg_bot

Q15: Which enzyme facilitates the conversion of discoidal HDL into spheroidal HDL in HDL metabolism? (Q843022)

A. Lipoprotein lipase (LPL)

B. Lecithin-cholesterol acyltransferase (LCAT)

C. Hepatic lipase

D. Cholesteryl ester transfer protein (CETP)

See Answer in Part B → Q15

@Neet_pg_bot

Q16: What is Apo B48 primarily associated with? (Q843021)

A. High-density lipoproteins (HDL)

B. Chylomicrons

C. Very low-density lipoproteins (VLDL)

D. Low-density lipoproteins (LDL)

See Answer in Part B → Q16

@Neet_pg_bot

Q17: Match the lipoprotein with its function. Lipoprotein Function 1.Chylomicron a.Transports triglycerides from the liver to muscle and adipose tissue 2.Very low-density lipoprotein (VLDL) b.Transports cholesterol from peripheral tissue to the liver 3. Low-density lipoprotein (LDL) c.Transports cholesterol from the liver to peripheral tissues 4. High-density lipoprotein (HDL) d.Transports triglycerides from the gut to muscle and adipose tissue (Q843020)

A. 1-d, 2-a, 3-c, 4-b

B. 1-a, 2-b, 3-c, 4-d

C. 1-a, 2-b, 3-d, 4-c

D. 1-c, 2-d, 3-b, 4-a

See Answer in Part B → Q17

@Neet_pg_bot

Q18: Which of the following is true regarding bile acid synthesis regulation? 7α-hydroxylation is the step which is suppressed Cholic acid suppresses the synthesis Farnesoid X receptor is activated Transcription of the CYP7A1 gene is suppressed (Q843019)

A. 1,2,3

B. 1,3,4

C. 2,3

D. 2,3,4

See Answer in Part B → Q18

@Neet_pg_bot

Q19: Which of the following statements is true regarding the fate of cholesterol? Chenodeoxycholic acid and cholic acid are the primary bile acids Glycine-taurine conjugation happens in liver peroxisomes Intestinal bacteria produce secondary bile acids (Q843018)

A. 1, 2

B. 1, 2, 3

C. 2, 3

D. Only 1

See Answer in Part B → Q19

@Neet_pg_bot

Q20: Which of the following enzymes catalyzes the principal regulatory step of cholesterol synthesis? (Q843017)

A. HMG CoA synthase

B. HMG CoA reductase

C. Cytosolic thiolase

D. Squalene epoxidase

See Answer in Part B → Q20

@Neet_pg_bot

• [03_Lipid Chemistry] 012 Previous Year Questions

« Back to Folder Directory

[03_Lipid Chemistry] 012 Previous Year Questions — Quick Jump

Q1 (p. 136)	*Q2 (p. 136)*	*Q3 (p. 137)*	*Q4 (p. 137)*	*Q5 (p. 137)*	*Q6 (p. 138)*
Q7 (p. 138)	*Q8 (p. 139)*	*Q9 (p. 139)*	*Q10 (p. 139)*	*Q11 (p. 140)*	*Q12 (p. 140)*
Q13 (p. 141)	*Q14 (p. 142)*	*Q15 (p. 142)*	*Q16 (p. 143)*	*Q17 (p. 143)*	*Q18 (p. 144)*
Q19 (p. 144)	*Q20 (p. 144)*	*Q21 (p. 145)*	*Q22 (p. 145)*	*Q23 (p. 146)*	*Q24 (p. 146)*
Q25 (p. 146)	*Q26 (p. 147)*	*Q27 (p. 147)*	*Q28 (p. 147)*	*Q29 (p. 148)*	*Q30 (p. 148)*
Q31 (p. 148)	*Q32 (p. 149)*	*Q33 (p. 149)*	*Q34 (p. 150)*	*Q35 (p. 150)*	*Q36 (p. 150)*
Q37 (p. 151)	*Q38 (p. 151)*	*Q39 (p. 151)*	*Q40 (p. 152)*	*Q41 (p. 152)*	*Q42 (p. 152)*
Q43 (p. 153)	*Q44 (p. 153)*	*Q45 (p. 154)*	*Q46 (p. 154)*		

Q1: Which of the cycles listed below is absent in muscle tissue? (Q856932)

A. Glycogenolysis

B. Ketogenesis

C. Glycogenesis

D. Lipolysis

See Answer in Part B → Q1

@Neet_pg_bot

Q2: Which of the following enzymes has the highest catalytic efficiency? (Q856482)

A. Km of an enzyme = 10 micromole and Kcat = 20 per sec

B. Km of an enzyme = 2000 nanomole and Kcat = 50 per sec

C. Km of an enzyme = 2 micromole and Kcat = 200 per sec

D. Km of an enzyme = 4 micromole and Kcat = 200 per sec

See Answer in Part B → Q2

@Neet_pg_bot

Q3: A 6-month-old infant is brought to the emergency department with lethargy, vomiting, and poor feeding. The episode occurred after prolonged fasting. Laboratory results reveal hypoglycemia, low ketone levels (hypoketosis), and mild hepatomegaly. These findings suggest a defect in fat metabolism. Which of the following is the most likely underlying disorder? (Q866670)

A. Her's disease

B. Medium-chain acyl-CoA dehydrogenase (MCAD) deficiency

C. Hereditary fructose intolerance

D. Glycogen storage disease type I (Von Gierke disease)

See Answer in Part B → Q3

@Neet_pg_bot

Q4: A patient presents with orange-colored tonsils. Laboratory investigations reveal triglyceride level of 140 mg/dL and HDL cholesterol of 5 mg/dL. What is the most likely diagnosis? (Q866332)

A. Familial hypercholesterolemia

B. Tangier disease

C. Type I hyperlipoproteinemia

D. Abetalipoproteinemia

See Answer in Part B → Q4

@Neet_pg_bot

Q5: A patient presents with elevated total cholesterol, subcutaneous xanthomas, and a positive family history of similar findings. Triglyceride levels are normal (<140 mg/dL). What is the most likely type of familial dyslipidemia? (Q866333)

A. Type I

B. Type IIa

C. Type IIb

D. Type II

See Answer in Part B → Q5

@Neet_pg_bot

Q6: A patient presents with xanthomas on the Achilles tendon. Which of the following is the most likely diagnosis? (Q844257)

A. Familial hypercholesterolemia

B. Tangier disease

C. Familial hyperchylomicronemia

D. Type III hyperlipoproteinemia

See Answer in Part B → Q6

@Neet_pg_bot

Q7: A patient presents to the emergency room with severe abdominal pain, and serum triglyceride levels are 1500 mg/dL. What is the most likely underlying defect? (Q844261)

A. Apo B-48 defect

B. Apo B-100 defect

C. Apo C-II defect

D. LDL receptor defect

See Answer in Part B → Q7

@Neet_pg_bot

Q8: A patient with multiple tendon xanthomas is found to have a serum cholesterol level of 398 mg/dL and an LDL level of 220 mg/dL. What is the most likely defect? (Q844263)

A. Lipoprotein lipase deficiency

B. LDL receptor defect

C. Defective Apo B-100

D. PCSK9 gain-of-function mutation

See Answer in Part B → Q8

@Neet_pg_bot

Q9: Which gene defect causes familial hypercholesterolemia? (Q839947)

A. Apo E

B. Apo C II

C. LDL receptor

D. Apo B48

See Answer in Part B → Q9

@Neet_pg_bot

Q10: Hormone-sensitive lipase is activated by all of the following EXCEPT? (Q857872)

A. Glucagon

B. Epinephrine

C. ACTH

D. Insulin

See Answer in Part B → Q10

@Neet_pg_bot

Q11: A patient presented with reduced levels of high-density lipoprotein and an ABCA1 mutation. On examination, tonsils appeared as shown in the image. What is the diagnosis? (Q855549)

A. Tangier's disease

B. Abetalipoproteinemia

C. Hyperlipoproteinemia

D. Wolman's disease



See Answer in Part B → Q11

@Neet_pg_bot

Q12: What is the primary source of reducing equivalents for liver fatty acid synthesis? (Q856924)

A. Hexose monophosphate shunt pathway

B. Glycolysis

C. Tricarboxylic acid cycle

D. Uronic acid pathway

See Answer in Part B → Q12

@Neet_pg_bot

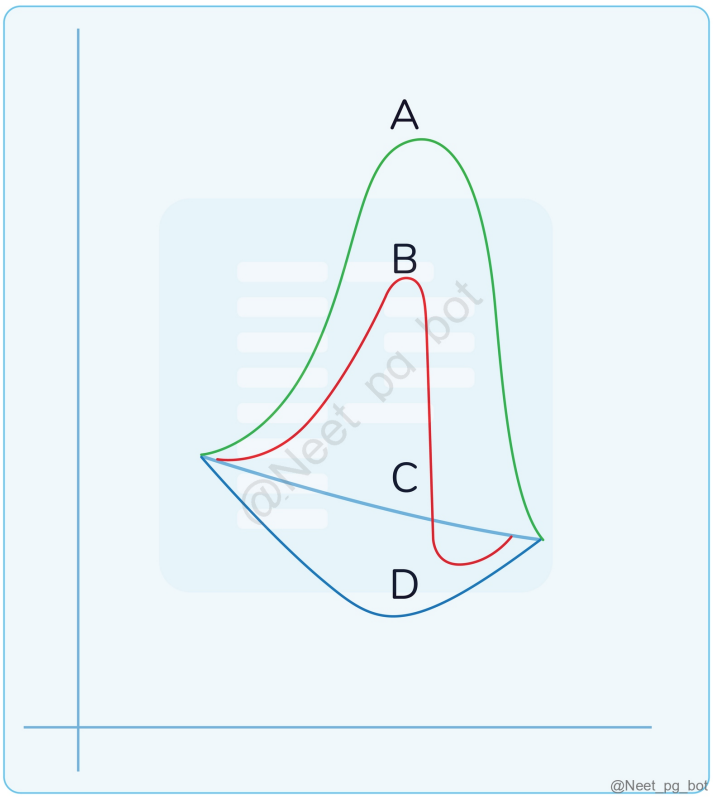
Q13: The Gibbs free energy curve of an enzyme-catalysed reaction is given by: (Q856930)

A. Curve B

B. Curve A

C. Curve D

D. Curve C



See Answer in Part B → Q13

@Neet_pg_bot

Q14: What is the primary source of cholesterol synthesis? (Q856980)

A. Malonyl-CoA

B. Pyruvate

C. Oxaloacetate

D. Acetyl-CoA

See Answer in Part B → Q14

@Neet_pg_bot

Q15: Enzyme deficient in Gaucher disease : (Q857482)

A. Betagalacto cerebrosidase

B. Glucose-6-phosphate

C. Betaglucocerebrosidase

D. Alpha glucocerebrosidase

See Answer in Part B → Q15

@Neet_pg_bot

Q16: A 5-year-old girl is brought to the clinic by her mother due to the abnormalities in the genital area. On examination, there is the presence of pubic and axillary hair, indicating early androgen effects. Which of the following enzyme deficiencies is the least likely cause of her symptoms? (Q857557)

A. 17α-hydroxylase deficiency

B. 21-hydroxylase deficiency

C. 11β-hydroxylase deficiency

D. 3β-hydroxy steroid dehydrogenase deficiency

See Answer in Part B → Q16

@Neet_pg_bot

Q17: Insulin inhibits which of the following lipase enzymes? (Q855896)

A. Lipoprotein lipase

B. Pancreatic lipase

C. Gastric lipase

D. Hormone sensitive lipase

See Answer in Part B → Q17

@Neet_pg_bot

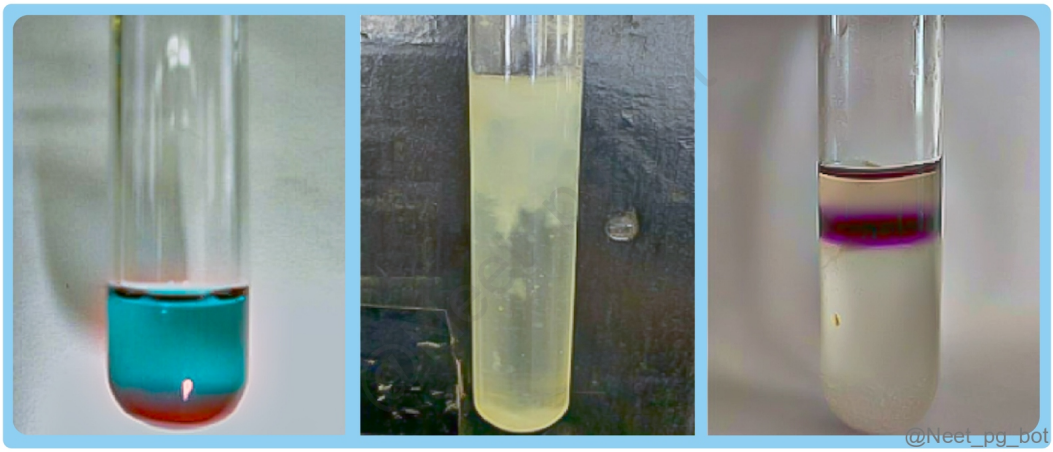
Q18: Which of the following tests will be positive in the urine of a patient who has undergone prolonged starvation? (Q855964)

A. 1 and 3

B. Only 3

C. 1, 2 and 3

D. 1 and 2



See Answer in Part B → Q18

@Neet_pg_bot

Q19: The enzyme that is not used in cholesterol synthesis is: (Q856879)

A. Thiolase

B. HMG CoA reductase

C. HMG CoA synthase

D. HMG CoA lyase

See Answer in Part B → Q19

@Neet_pg_bot

Q20: The process of apolipoprotein B48 synthesis involves which of the following? (Q854877)

A. RNA alternate splicing

B. RNA editing

C. DNA editing

D. RNA interference

See Answer in Part B → Q20

@Neet_pg_bot

Q21: Which of the options aids in the transportation of fatty acids through the inner mitochondria membrane? (Q855530)

A. Acyl carrier protein

B. Carnitine

C. Lecithin-Cholesterol acyltransferase

D. Albumin

See Answer in Part B → Q21

@Neet_pg_bot

Q22: What is the enzyme defect if a child with anemia, thrombocytopenia, bony pain and hepatosplenomegaly shows a "crumpled tissue paper" appearance on light microscopy of bone marrow aspirate? (Q855548)

A. Beta- glucosidase

B. Sphingomyelinase

C. Hexosaminidase

D. Glucose6-phosphatase

See Answer in Part B → Q22

@Neet_pg_bot

Q23: Which of the following statements about β-oxidation of fatty acids is incorrect? (Q879278)

A. CPT-1 and CPT-2 are in the mitochondrial membranes

B. ATP is directly produced during β-oxidation

C. CoA is released in the cytosol during acyl-carnitine formation

D. Carnitine is released into the matrix

See Answer in Part B → Q23

@Neet_pg_bot

Q24: What is the process of converting unconjugated substances into conjugated forms called? (Q860059)

A. Glycation

B. Glucuronidation

C. Glycosylation

D. Oxidation

See Answer in Part B → Q24

@Neet_pg_bot

Q25: A man is trapped in a tunnel for 5 consecutive days without access to food but survives. What is the primary source of energy for his brain during this period? (Q866318)

A. Gluconeogenesis

B. Glycogenolysis

C. Ketosis

D. Lipolysis

See Answer in Part B → Q25

@Neet_pg_bot

Q26: A diabetic patient presents with high VLDL due to the combination of which of the following? (Q853004)

A. High HSL, low LPL

B. High LPL, low HSL

C. High HSL, High LPL

D. Low LPL, Low HSL

See Answer in Part B → Q26

@Neet_pg_bot

Q27: Cholera is known to cause diarrhea through its interaction with specific receptors in the human body. Which of the following is known to make it easier for cholera toxin to enter the body through the intestinal mucosa? (Q857854)

A. GLUT 1

B. GM2 ganglioside

C. Cephalin

D. GM1 ganglioside

See Answer in Part B → Q27

@Neet_pg_bot

Q28: In which of the following locations can cardiolipin be found? (Q857900)

A. Outer mitochondrial membrane

B. Inner mitochondrial membrane

C. Cytosol

D. Mitochondrial matrix

See Answer in Part B → Q28

@Neet_pg_bot

Q29: Fat is stored in which form in tissue? (Q856950)

A. Fatty acid

B. Cholesterol

C. TGs

D. Cholesterol esters

See Answer in Part B → Q29

@Neet_pg_bot

Q30: Cholesterol is synthesized from? (Q857480)

A. Acetyl coA

B. Phenylalanine

C. Malonyl coA

D. Oxaloacetate

See Answer in Part B → Q30

@Neet_pg_bot

Q31: Which of the following substances is not characterized by amphipathicity? (Q856600)

A. Sphingolipids

B. Phosphoglycerol

C. Triglycerides

D. Glycolipids

See Answer in Part B → Q31

@Neet_pg_bot

Q32: What could be the possible diagnosis for a patient who exhibited tendon xanthomas, with his lipid profile indicating serum cholesterol levels of 283 mg/dL and serum LDL levels of 202 mg/dL, and subsequently initiated on lovastatin treatment? (Q856010)

A. Familial hypercholesterolemia

B. Tangier's disease

C. Lipoprotein lipase deficiency

D. Familial hyperchylomicronemia

See Answer in Part B → Q32

@Neet_pg_bot

Q33: What diagnostic tool is used to determine the cause of chest pain at rest for 6 hours and an electrocardiogram indicating myocardial ischemia in a patient? (Q859574)

A. LDH 2

B. LDH 3

C. LDH 1

D. LDH 4

See Answer in Part B → Q33

@Neet_pg_bot

Q34: Which substance is involved in the conjugation process in the liver? (Q860320)

A. Hyaluronic Acid

B. Glucuronic Acid

C. Gluconic Acid

D. Glycolic Acid

See Answer in Part B → Q34

@Neet_pg_bot

Q35: A man presents with high VLDL and chylomicrons, with eruptive xanthomas. What is the most likely diagnosis? (Q866645)

A. Familial hyperlipidemia

B. Familial dysbetalipoproteinemia

C. Familial hypertriglyceridemia

D. Familial hypercholesterolemia

See Answer in Part B → Q35

@Neet_pg_bot

Q36: The active site of an enzyme is most complementary to? (Q857356)

A. Allosteric activator

B. Substrate

C. Product

D. Transition state

See Answer in Part B → Q36

@Neet_pg_bot

Q37: In which organelle is DNA present? (Q820383)

A. Mitochondria

B. Lysosome

C. Endoplasmic reticulum

D. Cytoplasm

See Answer in Part B → Q37

@Neet_pg_bot

Q38: In which organelle is DNA present? (Q857869)

A. Mitochondria

B. Lysosome

C. Endoplasmic reticulum

D. Cytoplasm

See Answer in Part B → Q38

@Neet_pg_bot

Q39: What is the cause of elevated H+ ions in the intermembrane space of mitochondria? (Q855538)

A. Decreased oxidative phosphorylation

B. Increased ATP production

C. Reduced chemiosmosis

D. Water retention in inner mitochondrial membrane

See Answer in Part B → Q39

@Neet_pg_bot

Q40: Zellweger syndrome results from abnormality in formation of: (Q855055)

A. Peroxisomes

B. Lysosomes

C. Mitochondria

D. Nucleus

See Answer in Part B → Q40

@Neet_pg_bot

Q41: Refsum disease is caused due to absence of which of the following enzymes? (Q855061)

A. Absence of alpha oxidation in peroxisomes.

B. Absence of beta oxidation in peroxisomes.

C. Absence of alpha oxidation in lysosomes.

D. Absence of beta oxidation in lysosomes.

See Answer in Part B → Q41

@Neet_pg_bot

Q42: The subcellular organelle which is associated with the killing of viruses and bacteria is? (Q857485)

A. Peroxisome

B. Mitochondria

C. Lysosome

D. Golgi apparatus

See Answer in Part B → Q42

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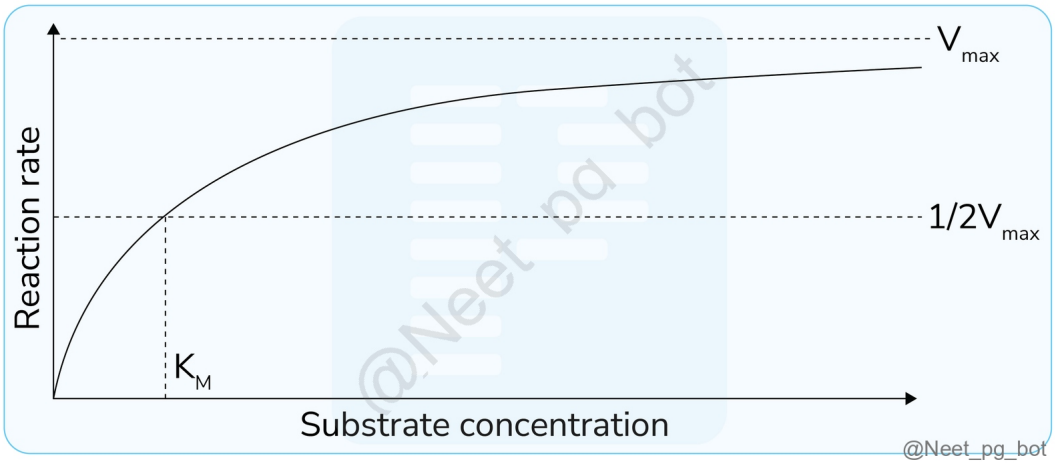
Q43: The enzyme kinetics depicted in the graph is suggestive of (Q855904)

A. Allosteric activation

B. Cooperative binding

C. Michaelis Menten kinetics

D. Sigmoid curve



See Answer in Part B → Q43

@Neet_pg_bot

Q44: Which mechanism is responsible for the inhibition of the acetylcholinesterase enzyme by organophosphorus compounds? (Q856597)

A. Non-competitive inhibition

B. Reversible competitive inhibition

C. Irreversibleinhibition

D. Suicidal inhibition

See Answer in Part B → Q44

@Neet_pg_bot

Q45: A TPN bag consists of 100g glucose, 30g amino acids, and 40g lipids. What is the amount of calories delivered? (Q856864)

A. 840 KCal

B. 880 KCal

C. 640 KCal

D. 680 KCal

See Answer in Part B → Q45

@Neet_pg_bot

Q46: In a patient who is tall, obese, known to have hypertension, and has recently been diagnosed with metabolic syndrome during a routine examination, which of the following findings are characteristic findings? (Q793475)

A. Increase in uric acid and insulin

B. Raised blood pressure and normal insulin

C. Increase in triglycerides and total cholesterol

D. Raised blood sugar and low insulin

See Answer in Part B → Q46

@Neet_pg_bot

• [04_Amino Acids and Proteins Chemistry] 013 Protein Chemistry Hemoglobin Myoglobin Epp Chromatography

« Back to Folder Directory

[04_Amino Acids and Proteins Chemistry] 013 Protein Chemistry Hemoglobin Myoglobin Epp Chromatography — Quick Jump

Q1 (p. 155)	*Q2 (p. 155)*	*Q3 (p. 156)*	*Q4 (p. 156)*	*Q5 (p. 156)*	*Q6 (p. 157)*
Q7 (p. 157)	*Q8 (p. 158)*	*Q9 (p. 158)*	*Q10 (p. 159)*	*Q11 (p. 159)*	*Q12 (p. 160)*
Q13 (p. 160)	*Q14 (p. 160)*	*Q15 (p. 161)*	*Q16 (p. 161)*	*Q17 (p. 161)*	*Q18 (p. 162)*
Q19 (p. 162)	*Q20 (p. 163)*	*Q21 (p. 163)*	*Q22 (p. 164)*	*Q23 (p. 164)*	*Q24 (p. 165)*
Q25 (p. 165)	*Q26 (p. 166)*				

Q1: Which of the following channels is primarily responsible for active magnesium reabsorption in the distal convoluted tubule? (Q876252)

A. TRPM6

B. ENaC

C. NKCC2

D. Aquaporin-2

See Answer in Part B → Q1

@Neet_pg_bot

Q2: Which of the following statements about selenocysteine and Pyrrolysine is correct? (Q844902)

A. Pyrrolysine is incorporated into proteins at UGA codons with the help of a specific tRNA.

B. The incorporation of selenocysteine requires a special mRNA structure known as the selenocysteine insertion sequence (SECIS).

C. Selenocysteine is synthesized from cysteine during protein translation.

D. Pyrrolysine is only found in eukaryotic organisms and not in prokaryotes.

See Answer in Part B → Q2

@Neet_pg_bot

Q3: Which of the following statements correctly describes the metabolic fate of specific amino acids? (Q844900)

A. All amino acids can be classified as either glucogenic or ketogenic without exceptions.

B. Aspartate is a purely ketogenic amino acid.

C. Phenylalanine can be converted to both glucose and ketone bodies.

D. Lysine is exclusively glucogenic.

See Answer in Part B → Q3

@Neet_pg_bot

Q4: A patient with anaemia has elevated levels of 2,3-Bisphosphoglycerate (2,3-BPG) in their red blood cells. How does this increased 2,3-BPG level affect the patient's haemoglobin in terms of oxygen delivery? (Q843515)

A. Forms salt bridges with positively charged residues forming a relaxed structure

B. Disrupts salt bridges formed in the taut structure, decreasing oxygen affinity

C. Forms salt bridges with positively charged residues stabilising taut structure

D. Binds to the heme directly, decreasing the oxygen affinity

See Answer in Part B → Q4

@Neet_pg_bot

Q5: Which of the following techniques is most widely used to estimate a patient's HbA1c level for monitoring long-term glucose control? (Q843526)

A. Cation exchange chromatography

B. Anion exchange chromatography

C. Gel filtration chromatography

D. Paper chromatography

See Answer in Part B → Q5

@Neet_pg_bot

Q6: Based on which characteristic the technique SDS-PAGE is used to separate proteins? (Q843525)

A. Charge

B. Molecular Weight

C. Size

D. Shape

See Answer in Part B → Q6

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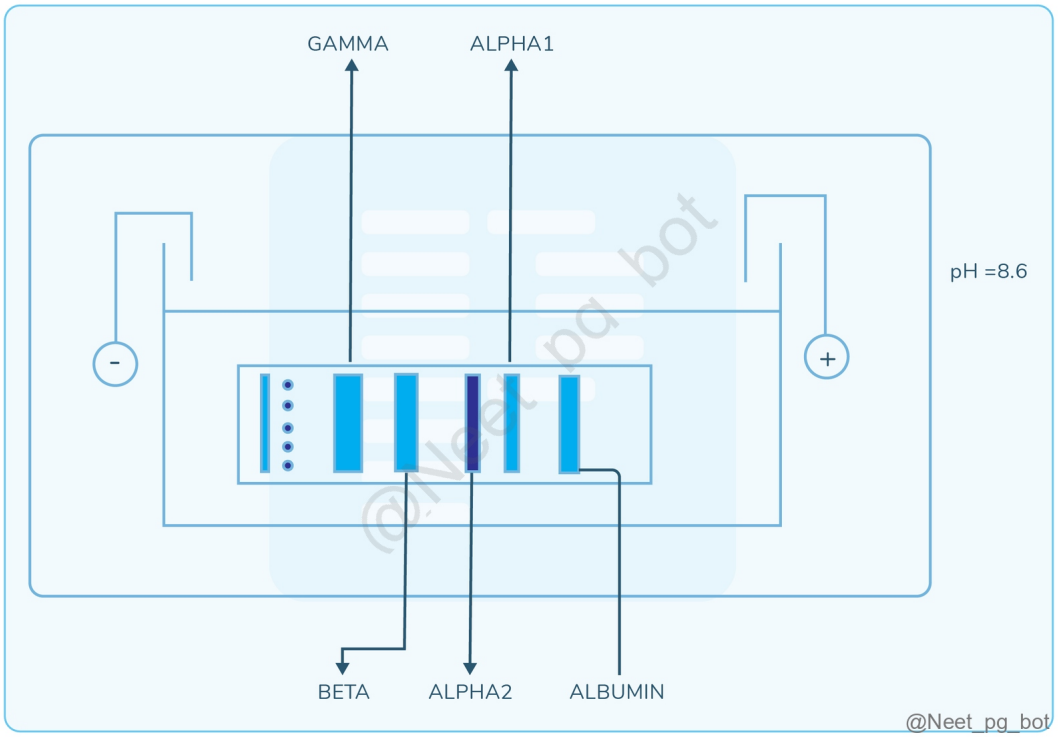
Q7: The electrophoresis pattern in the image shows a prominent alpha-2 band. Which disease is most likely associated with this finding? (Q843524)

A. Nephrotic syndrome

B. Multiple myeloma

C. Decompensated chronic liver disease

D. Nephritic syndrome



See Answer in Part B → Q7

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Q8: A laboratory technician prepares samples for electrophoresis as the person is suspected to auffer from multiple myeloma. Which sample type is preferred for this test to ensure accurate results? (Q843523)

A. Serum

B. Plasma

C. Whole blood

D. CSF

See Answer in Part B → Q8

@Neet_pg_bot

Q9: A 39-year-old male complains of unexplained fatigue and joint pain and visits his physician. The physician suspects chronic inflammatory disease and advises serum protein electrophoresis, which shows an increase in alpha 2 bands. Which of the following proteins shows an increase in alpha 2 bands? (Q843522)

A. Albumin

B. Macroglobulin

C. Gamma globulin

D. Transferrin

See Answer in Part B → Q9

@Neet_pg_bot

Q10: Which protein migrates the farthest from the application point towards the anode in serum protein electrophoresis? (Q843520)

A. Albumin

B. Gamma globulin

C. Alpha-1 globulin

D. Beta globulin

See Answer in Part B → Q10

@Neet_pg_bot

Q11: All the following factors determine the mobility of proteins in electrophoresis except? (Q843519)

A. Charge

B. Isoelectric pH

C. Molecular weight

D. Shape

See Answer in Part B → Q11

@Neet_pg_bot

Q12: What is the most likely cause of the rightward shift of the oxyhemoglobin dissociation curve? (Q843517)

A. Decreased 2,3 -BPG

B. Decreased CO2

C. Increased pH

D. Increased temperature

See Answer in Part B → Q12

@Neet_pg_bot

Q13: A newborn is diagnosed with a condition where their haemoglobin has an unusually high affinity for oxygen. What is the physiological basis for the increased oxygen affinity of fetal haemoglobin compared to adult haemoglobin? (Q843516)

A. Binds more 2,3-BPG, increasing oxygen affinity.

B. HbF contains serine that reduces the positive charge of the globin side chain.

C. Higher histidine content, reducing oxygen affinity.

D. Binds oxygen more loosely than adult Hb

See Answer in Part B → Q13

@Neet_pg_bot

Q14: Which of the following statements is TRUE regarding pKa and its implications in biochemistry? (Q843474)

A. The lower the pKa of the amino group, the weaker the acid.

B. pKa remains constant and does not change with the medium.

C. The net charge of a protein does not depend on the pKa value of the functional group.

D. When pH =pKa +/- 1, substances act as buffers.

See Answer in Part B → Q14

@Neet_pg_bot

Q15: An athlete undergoing high-altitude training adapts to decreased oxygen availability by altering haemoglobin to enhance oxygen delivery. Which structural form of haemoglobin is predominantly favoured to enhance oxygen release to tissues? (Q843493)

A. Taut structure

B. Relaxed structure

C. Globular structure

D. Tertiary structure

See Answer in Part B → Q15

@Neet_pg_bot

Q16: Which of the following correctly describes the role of histidine residues in myoglobin in relation to carbon monoxide (CO) binding? (Q843492)

A. Prevent CO binding by increasing the heme's affinity for oxygen.

B. Enhance CO binding, similar to oxygen.

C. Inhibit CO binding by sterically hindering the heme iron.

D. It does not influence the binding of CO to the heme.

See Answer in Part B → Q16

@Neet_pg_bot

Q17: Which cellular component is primarily responsible for degrading intracellular proteins tagged with ubiquitin? (Q843490)

A. Lysosomes

B. Proteasomes

C. Cathepsins

D. Endosomes

See Answer in Part B → Q17

@Neet_pg_bot

Q18: Which protein assists in proper folding by binding to hydrophobic regions of other proteins during the folding process? (Q843488)

A. Proteases

B. Chaperonins

C. Ubiquitin

D. Prions

See Answer in Part B → Q18

@Neet_pg_bot

Q19: Which of the following techniques provides the highest resolution for determining the primary structure of proteins? (Q843487)

A. Nuclear Magnetic Resonance (NMR) spectroscopy

B. X-ray crystallography

C. Sanger’s sequencing

D. Optical rotatory dispersion

See Answer in Part B → Q19

@Neet_pg_bot

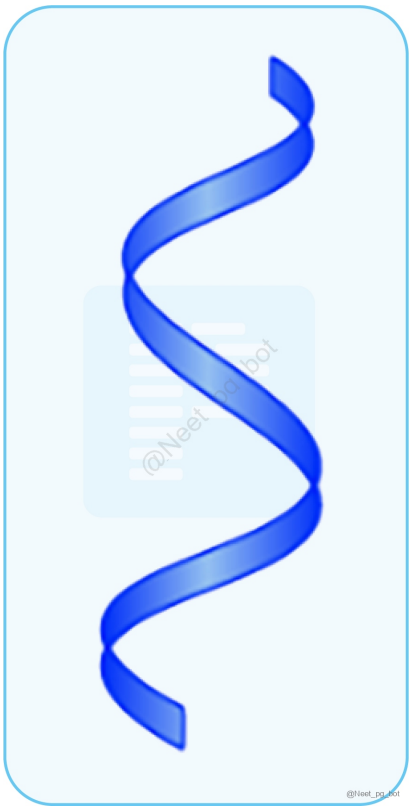
Q20: What type of bond is primarily responsible for stabilising the structure in proteins as shown in the image? (Q843486)

A. Covalent bonds between side chains

B. Ionic bonds between charged amino acids

C. Hydrogen bonds between backbone atoms

D. Disulfide bridges between cysteine residues



See Answer in Part B → Q20

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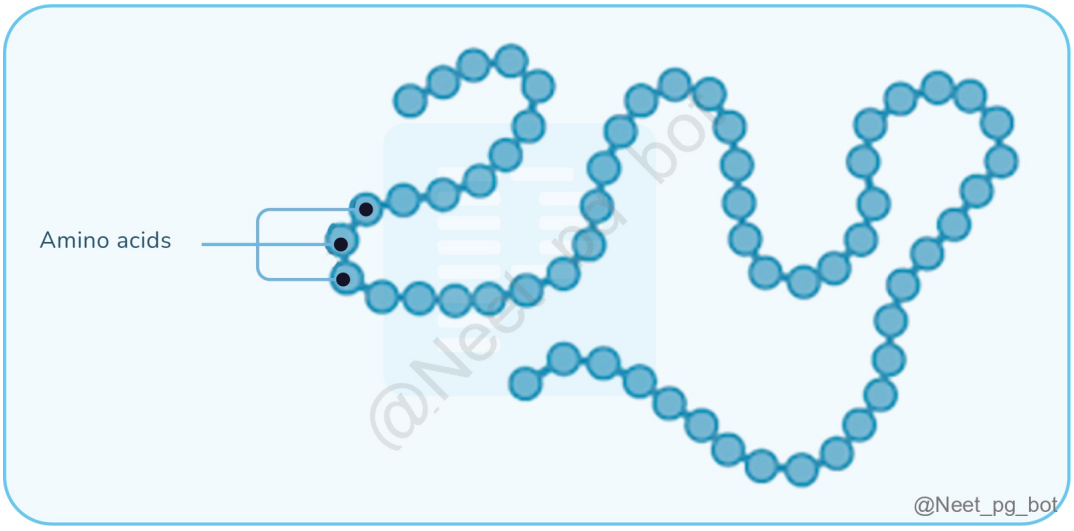
Q21: Which of the following interactions is primarily responsible for maintaining the structure of proteins, as shown in the image? (Q843485)

A. Peptide bonds

B. Hydrogen bonds

C. Disulfide bridges

D. Van der Waals interactions



See Answer in Part B → Q21

@Neet_pg_bot

Q22: Match the following protein tests with their primary purpose or detection target: 1.Ninhydrin test a. Detects compouds containing 2 or more peptide bonds 2.Xanthoproteic test b. Detects aromatic amino acids 3.Sakaguchi test c. Detects arginine 4.Biuret test d. Detects alpha-amino acids (Q843483)

A. 1-b, 2-c, 3-d, 4-a

B. 1-d, 2-b, 3-c, 4-a

C. 1-a, 2-d, 3-b, 4-c

D. 1-c, 2-a, 3-d, 4-b

See Answer in Part B → Q22

@Neet_pg_bot

Q23: Which of the following is true regarding the properties of glycine? (Q843482)

A. Has asymmetric carbon atoms

B. Doesn't exhibit stereoisomerism

C. Optically active

D. Complex amino acid

See Answer in Part B → Q23

@Neet_pg_bot

Q24: A 22-year-old woman presents with complaints of chronic fatigue and frequent infections. She follows a strict vegetarian diet and consumes less protein-rich food. Laboratory tests reveal a decreased level of a particular amino acid. Which of the following amino acids is likely deficient in this patient? (Q843477)

A. Threonine and Lysine

B. Tyrosine and Tryptophan

C. Alanine and Glycine

D. Cysteine and Methionine

See Answer in Part B → Q24

@Neet_pg_bot

Q25: Which of the following amino acids contains a hydroxyl group (OH) in its side chain? (Q843476)

A. Phenylalanine

B. Serine

C. Cysteine

D. Glutamine

See Answer in Part B → Q25

@Neet_pg_bot

Q26: Which of the following is not a characteristic of the isoelectric pH (pI) of a protein? (Q843475)

A. High solubility

B. Low buffering capacity

C. No net charge

D. Minimal mobility

See Answer in Part B → Q26

@Neet_pg_bot

• [04_Amino Acids and Proteins Chemistry] 014 Amino Acid Catabolism Urea Cycle And Hyperammonemia

« Back to Folder Directory

[04_Amino Acids and Proteins Chemistry] 014 Amino Acid Catabolism Urea Cycle And Hyperammonemia — Quick Jump

Q1 (p. 167)	*Q2 (p. 167)*	*Q3 (p. 168)*	*Q4 (p. 168)*	*Q5 (p. 168)*	*Q6 (p. 169)*
Q7 (p. 169)	*Q8 (p. 170)*	*Q9 (p. 170)*	*Q10 (p. 171)*	*Q11 (p. 172)*	*Q12 (p. 172)*
Q13 (p. 172)	*Q14 (p. 173)*	*Q15 (p. 173)*	*Q16 (p. 173)*	*Q17 (p. 174)*	*Q18 (p. 174)*
Q19 (p. 175)	*Q20 (p. 175)*	*Q21 (p. 176)*	*Q22 (p. 176)*	*Q23 (p. 176)*	*Q24 (p. 177)*
Q25 (p. 177)	*Q26 (p. 177)*				

Q1: A one-year-old boy presents with lethargy, poor muscle tone, and developmental delays. Blood tests reveal elevated levels of glycine. The physician suspects a defect in the glycine cleavage system. Which of the following metabolites, if found LOW, would support the diagnosis of a defective glycine cleavage system? (Q845049)

A. Serine

B. N5,N10 methylene tetrahydrofolate

C. Threonine

D. Succinyl CoA

See Answer in Part B → Q1

@Neet_pg_bot

Q2: A patient with a urea cycle disorder is being evaluated. Which urea cycle intermediate serves as the link between the urea cycle and the TCA cycle? (Q845061)

A. Argininosuccinate

B. Fumarate

C. Oxaloacetate

D. Succinate

See Answer in Part B → Q2

@Neet_pg_bot

Q3: A 3-week-old infant presents with poor feeding, lethargy, and vomiting. Lab results show elevated ammonia levels, increased glutamine and increased orotic acid in the blood, with no evidence of megaloblastic anaemia. Which enzyme deficiency is likely responsible for this condition? (Q845060)

A. Ornithine transcarbamylase (OTC)

B. Ornithine aminotransferase

C. Uridine monophosphate (UMP) synthase

D. Hypoxanthine guanine phosphoribosyltransferase (HGPRT)

See Answer in Part B → Q3

@Neet_pg_bot

Q4: Which enzyme in the histidine catabolism pathway facilitates incorporating the formimino group into the one-carbon pool, forming N5-formimino tetrahydrofolate? (Q845059)

A. Histidase

B. Urocanase

C. Glutamate formimino transferase

D. Glutamate dehydrogenase

See Answer in Part B → Q4

@Neet_pg_bot

Q5: In the kynurenine pathway of tryptophan catabolism, which of the following compounds directly contributes to the formation of N10-formyl tetrahydrofolate in the one-carbon pool? (Q845058)

A. Formyl kynurenine

B. Kynurenine

C. 3- Hydroxy kynurenine

D. THFA

See Answer in Part B → Q5

@Neet_pg_bot

Q6: A 62-year-old female patient presents with fatigue, pallor, and glossitis. Laboratory tests indicate macrocytic anaemia, and elevated homocysteine and methylmalonic acid levels. Given these findings, what is the most likely metabolic pathway disrupted by this patient's condition, leading to the observed anaemia? (Q845057)

A. Conversion of homocysteine to methionine

B. Reduction of folic acid to dihydrofolate

C. Deamination of histidine to formimino glutamate

D. Conversion of Phenylalanine to Tyrosine

See Answer in Part B → Q6

@Neet_pg_bot

Q7: Which of the following statements regarding one-carbon derivatives of tetrahydrofolic acid (THFA) and their associated enzymes and reactions is incorrect? (Q845056)

A. N5-formimino-THFA converts formiminoglutamate (FIGLU) to glutamate.

B. N5-methyl-THFA carries the methyl group for converting homocysteine to methionine.

C. N5N10-methylene-THFA is required to synthesise thymidylate (dTMP) from deoxyuridylate (dUMP).

D. N10-formyl-THFA transfers formyl groups in pyrimidine synthesis.

See Answer in Part B → Q7

@Neet_pg_bot

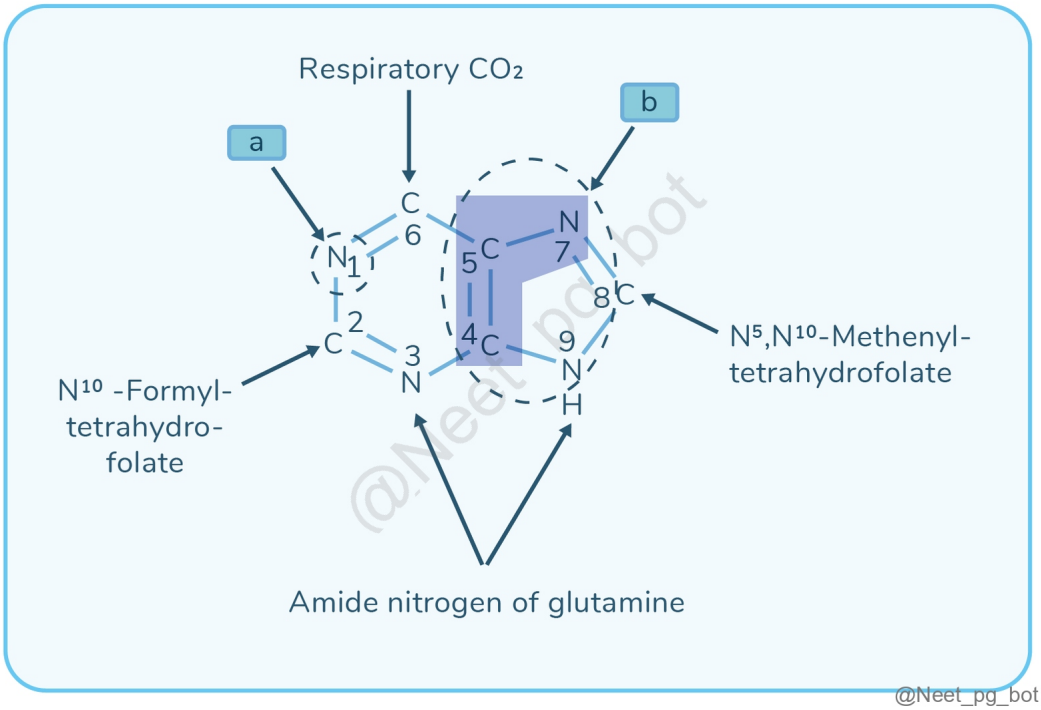
Q8: In the following figure, identify the sources of the marked components. (Q845055)

A. a- aspartate, b- glycine

B. a- glycine, b-aspartate

C. a- methionine, b-alanine

D. a- alanine, b- methionine



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See Answer in Part B → Q8

@Neet_pg_bot

Q9: Which of the following diseases is caused by the deficiency of the enzyme ■ ALA synthase? (Q845054)

A. Porphyria cutanea tarda

B. X linked sideroblastic anaemia

C. Acute intermittent porphyria

D. Congenital erythropoietic porphyria

See Answer in Part B → Q9

@Neet_pg_bot

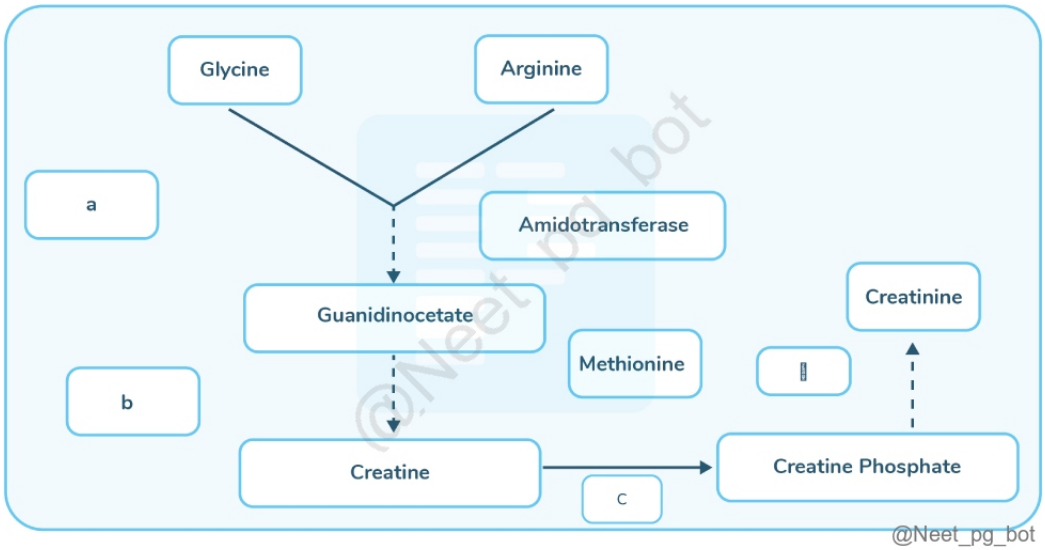
Q10: Based on the image given below, what are the sites at which the marked steps of creatinine synthesis occur? (Q845053)

A. a- brain, b- liver, c- kidney

B. a- kidney, b- liver, c- muscle

C. a- liver, b- kidney, c- muscle

D. a- muscle, b- liver, c-kidney



See Answer in Part B → Q10

@Neet_pg_bot

Q11: Which of the following statement is not a known use of glycine in humans? (Q845052)

A. Precursor for metabolites like creatine, glutathione, and heme.

B. Enhances the quality of sleep and supports neurological functions.

C. Inhibits synthesis of bile acid, reducing lipid absorption.

D. Provide structural integrity for collagen structure.

See Answer in Part B → Q11

@Neet_pg_bot

Q12: The glycine cleavage system in the body produces CO2, ammonia and a one-carbon molecule. Which of the following is a subunit in the system? (Q845051)

A. Tetrahydrofolate

B. Lipoamide

C. Tetrahydrofolate-dependent aminomethyltransferase (THFAAMT)

D. Pyridoxal phosphate

See Answer in Part B → Q12

@Neet_pg_bot

Q13: A 6-month-old infant presented with lethargy, vomiting, and irritability. Blood tests revealed elevated ammonia levels. The infant was diagnosed with a urea cycle disorder. Which of the following medications, acting as ammonia-scavenging agents, would be most effective in reducing the infant's blood ammonia levels? (Q845050)

A. Phenylacetate

B. N-acetyl glutamate

C. Lactulose

D. Neomycin

See Answer in Part B → Q13

@Neet_pg_bot

Q14: A 45-year-old man presents to the clinic with complaints of fatigue and abdominal discomfort. Blood tests reveal elevated levels of alanine aminotransferase (ALT) and aspartate aminotransferase (AST). The physician suspects liver disease. Which of the following statements best describes the role of transamination reactions in amino acid metabolism? (Q843343)

A. Transfer an amino group to one keto acid, producing new amino and keto acids.

B. Specific to lysine, threonine, and proline.

C. Elevated AST and ALT indicate myocardial infarction.

D. Occur exclusively in the liver mitochondria.

See Answer in Part B → Q14

@Neet_pg_bot

Q15: Which of the following is not a source of glycine? (Q845048)

A. Serine

B. Threonine

C. choline

D. Lysine

See Answer in Part B → Q15

@Neet_pg_bot

Q16: A 45-year-old male with microcytic anaemia is found to have a deficiency of a vitamin essential for the rate-limiting step of heme synthesis. Which vitamin is most likely deficient? (Q843442)

A. Vitamin B6

B. Vitamin B12

C. Vitamin C

D. Vitamin D

See Answer in Part B → Q16

@Neet_pg_bot

Q17: A 5-month-old boy presents with failure to thrive, developmental delay, and intermittent episodes of vomiting and lethargy. The episodes coincide with the introduction of solid foods. Blood tests reveal elevated ammonia levels and high levels of ornithine in blood. Urine analysis shows increased excretion of homocitrulline. What is the most likely diagnosis for this patient? (Q843441)

A. Citrullinemia

B. Argininosuccinate aciduria

C. HHH Syndrome

D. Argininemia

See Answer in Part B → Q17

@Neet_pg_bot

Q18: A 5-month-old male infant with a history of vomiting, irritability, and developmental delay. The onset of symptoms coincided with the introduction of formula feed. Physical examination revealed hepatomegaly and signs of encephalopathy. Blood tests revealed elevated ammonia levels and high glutamine, uracil, and orotic acid concentrations. Citrulline levels were normal. What is the most likely diagnosis? (Q843352)

A. Type 1 Hyperammonemia

B. Type 2 Hyperammonemia

C. Citrullinemia

D. Argininosuccinate aciduria

See Answer in Part B → Q18

@Neet_pg_bot

Q19: Which of the following statements about carbamoyl phosphate synthetases is correct? (Q843351)

A. CPS-I is activated by PRPP, and CPS-II is inhibited by UTP.

B. CPS-I is located in cytosol, and CPS-II in mitochondria.

C. CPS-I is involved in urea cycle, and CPS-II in pyrimidine synthesis.

D. Source of nitrogen for CPS-I is glutamine, and CPS-II is ammonia.

See Answer in Part B → Q19

@Neet_pg_bot

Q20: Which of the following is true about the rate-limiting step of the urea cycle? (Q843350)

A. It involves the formation of citrulline from ornithine.

B. It is catalysed by carbamoyl phosphate synthetase I (CPS I).

C. It is regulated by argininosuccinate synthetase.

D. It is controlled by the enzyme argininosuccinate lyase.

See Answer in Part B → Q20

@Neet_pg_bot

Q21: Which of the following statements about ammonia transport is correct? (Q843349)

A. Glutamine moves ammonia from liver to brain.

B. Alanine transports ammonia from liver to muscles.

C. Glutamine and alanine transport ammonia to the liver.

D. Alanine does not participate in ammonia transport.

See Answer in Part B → Q21

@Neet_pg_bot

Q22: A 45-year-old man with chronic liver disease is noted to have elevated blood ammonia on laboratory testing. Which of the following is least likely to result from ammonia neurotoxicity? (Q843348)

A. Slurred speech

B. Tremors

C. Muscle rigidity

D. Blurred vision

See Answer in Part B → Q22

@Neet_pg_bot

Q23: Which of the following pathways is primarily involved in the detoxification of ammonia in the brain? (Q843347)

A. Conversion of ammonia to urea in neurons

B. Direct excretion of ammonia through the blood-brain barrier

C. Conversion of ammonia to glutamine via glutamine synthetase in astrocytes

D. Storage of ammonia in neurons as glutamate

See Answer in Part B → Q23

@Neet_pg_bot

Q24: Which of the following reactions is not catalysed by glutamate dehydrogenase (GDH)? (Q843346)

A. Conversion of α-ketoglutarate and ammonia to glutamate

B. Oxidative deamination of glutamate

C. Formation of aspartate from oxaloacetate and ammonia

D. None of the above

See Answer in Part B → Q24

@Neet_pg_bot

Q25: Which of the following pairs of amino acids do not undergo transamination reactions? (Q843345)

A. Serine and threonine

B. Lysine and threonine

C. Proline and alanine

D. Hydroxyproline and asparagine

See Answer in Part B → Q25

@Neet_pg_bot

Q26: A 50-year-old woman presents with confusion and lethargy. Blood tests show elevated levels of ammonia, suggesting hyperammonemia. Further tests indicate liver dysfunction. Which of the following best describes the process of oxidative deamination in amino acid metabolism? (Q843344)

A. Removal of an amino group from an amino acid, resulting in the corresponding keto acid and ammonia.

B. Involves only transamination reactions in the cytoplasm.

C. Generates ammonia, which is directly excreted by the kidneys.

D. Predominantly occurs in the liver cytoplasm, producing glutamine.

See Answer in Part B → Q26

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• [04_Amino Acids and Proteins Chemistry] 015 Amino Acid Metabolic Disorder - I

« Back to Folder Directory

[04_Amino Acids and Proteins Chemistry] 015 Amino Acid Metabolic Disorder - I — Quick Jump

Q1 (p. 179)	*Q2 (p. 179)*	*Q3 (p. 180)*	*Q4 (p. 180)*	*Q5 (p. 180)*	*Q6 (p. 181)*
Q7 (p. 181)	*Q8 (p. 181)*	*Q9 (p. 182)*	*Q10 (p. 182)*	*Q11 (p. 183)*	*Q12 (p. 183)*
Q13 (p. 184)	*Q14 (p. 184)*	*Q15 (p. 184)*	*Q16 (p. 185)*	*Q17 (p. 185)*	*Q18 (p. 186)*
Q19 (p. 186)	*Q20 (p. 186)*	*Q21 (p. 187)*	*Q22 (p. 187)*	*Q23 (p. 188)*	*Q24 (p. 188)*
Q25 (p. 188)	*Q26 (p. 189)*	*Q27 (p. 189)*			

Q1: A 5-year-old boy is brought to recurrent kidney stones and abdominal pain. Urinalysis reveals hexagonal crystals, and a 24-hour urine test shows elevated levels of cystine. Which condition is the most likely diagnosis? (Q844923)

A. Cystinuria

B. Cystinosis

C. Hyperoxaluria

D. Nephrocalcinosis

See Answer in Part B → Q1

@Neet_pg_bot

Q2: A 10-year-old child presents with an acute stroke. He is tall and thin with long fingers. Ocular examination shows downward dislocation of the lens. The most likely diagnosis is: (Q885184)

A. Marfan syndrome

B. Homocystinuria

C. Ehlers-Danlos syndrome

D. Osteogenesis imperfecta

See Answer in Part B → Q2

@Neet_pg_bot

Q3: Deficiency of cystathionine β-synthase leads to the accumulation of which amino acid, increasing cardiovascular risk? (Q885183)

A. Methionine

B. Homocysteine

C. Cysteine

D. Taurine

See Answer in Part B → Q3

@Neet_pg_bot

Q4: Excessive urinary excretion of 5-HIAA (5-hydroxyindoleacetic acid) is characteristic of: (Q885182)

A. Pheochromocytoma

B. Carcinoid syndrome

C. Neuroblastoma

D. Mastocytosis

See Answer in Part B → Q4

@Neet_pg_bot

Q5: Xanthurenic aciduria after a tryptophan load test indicates deficiency of which vitamin? (Q885181)

A. Vitamin B■ (Thiamine)

B. Vitamin B■ (Riboflavin)

C. Vitamin B■ (Pyridoxine)

D. Niacin

See Answer in Part B → Q5

@Neet_pg_bot

Q6: A middle-aged patient presents with bluish-black discolouration of the ears and sclera, along with low back pain. X-ray shows calcification of intervertebral discs. Which enzyme is deficient? (Q885180)

A. Phenylalanine hydroxylase

B. Homogentisate oxidase

C. Tyrosinase

D. Cystathionine β-synthase

See Answer in Part B → Q6

@Neet_pg_bot

Q7: Neonatal tyrosinemia (transient) is due to a deficiency of which enzyme? (Q885179)

A. Tyrosine aminotransferase

B. Fumarylacetoacetate hydrolase

C. 4-Hydroxyphenylpyruvate dioxygenase

D. Phenylalanine hydroxylase

See Answer in Part B → Q7

@Neet_pg_bot

Q8: A child presents with hepatomegaly, jaundice, abdominal distension, and a "boiled cabbage" odour. Laboratory findings show elevated succinylacetone. The most likely diagnosis is: (Q885177)

A. Phenylketonuria

B. Alkaptonuria

C. Type I tyrosinemia

D. Homocystinuria

See Answer in Part B → Q8

@Neet_pg_bot

Q9: A child with developmental delay and seizures is found to have elevated serum phenylalanine. Which test detects the abnormal urinary metabolite in this condition? (Q885174)

A. Cyanide-nitroprusside test

B. Ferric chloride test

C. Obermeyer test

D. Nitrosonaphthol test

See Answer in Part B → Q9

@Neet_pg_bot

Q10: Match the following urinary odours with their corresponding diagnoses: Urinary Odour Diagnosis 1. Mousy odour a. DKA (Diabetic Ketoacidosis) 2. Fruity odour b. Hawkinsinuria 3. Cabbage odour c. Methionine malabsorption 4. Swimming pool odour d. PKU (Phenylketonuria) 5. Oast house odour e. Type 1 Tyrosinemia (Q844928)

A. 1-e, 2-a, 3-d, 4-b, 5-c

B. 1-d, 2-a, 3-e, 4-c, 5-b

C. 1-e, 2-a, 3-d, 4-c, 5-b

D. 1-d, 2-a, 3-e, 4-b, 5-c

See Answer in Part B → Q10

@Neet_pg_bot

Q11: A 45-year-old patient presents with episodes of palpitations, sweating, and severe headaches. These episodes occur intermittently and are associated with hypertension. Laboratory tests reveal elevated levels of plasma free metanephrines and urinary Vanillyl Mandelic Acid. Which of the following enzymes are directly involved in the synthesis of these metabolites? (Q844927)

A. Monoamine oxidase, Catechol-O-methyltransferase

B. Glutamate decarboxylase, Tyrosine hydroxylase

C. Tyrosine hydroxylase, Catechol-O-methyltransferase

D. Tyrosine hydroxylase, Monoamine oxidase

See Answer in Part B → Q11

@Neet_pg_bot

Q12: Which of the following statements about Segawa syndrome is incorrect? (Q844926)

A. Segawa syndrome is autosomal dominant.

B. Patients have a defect in the GTP cyclohydrolase enzyme.

C. CSF Biopterin levels are elevated.

D. Females are more commonly affected than males.

See Answer in Part B → Q12

@Neet_pg_bot

Q13: A newborn screening test reveals elevated phenylalanine levels, prompting further investigation. Genetic testing indicates a defect in dihydrobiopterin reductase. Based on this information, Which type of phenylketonuria does the patient most likely have? (Q844925)

A. Classic phenylketonuria

B. Non-classic phenylketonuria Type II

C. Non-classic phenylketonuria Type IV

D. Non-classic phenylketonuria Type V

See Answer in Part B → Q13

@Neet_pg_bot

Q14: A 12-year-old boy is brought to the ophthalmologist by his parents due to progressive vision loss, particularly at night. Fundoscopic examination reveals circular areas of chorioretinal atrophy and peripheral retinal degeneration. Laboratory tests indicate markedly elevated plasma ornithine levels. What is the primary biochemical defect in this patient's condition? (Q844924)

A. Deficiency of ornithine transcarbamylase

B. Deficiency of ornithine aminotransferase

C. Deficiency of ornithine decarboxylase

D. Deficiency of ornithine cyclodeaminase

See Answer in Part B → Q14

@Neet_pg_bot

Q15: Which of the following statements about the metabolism of phenylalanine is incorrect? (Q844910)

A. Phenylalanine is converted to tyrosine catalyzed by phenylalanine hydroxylase.

B. Phenylalanine on transamination gets converted to phenylpyruvate

C. Phenylpyruvate on oxidation yields phenyl lactate

D. Phenylpyruvate on oxidative decarboxylation forms phenylacetate.

See Answer in Part B → Q15

@Neet_pg_bot

Q16: A 10-year-old girl is evaluated for developmental delay. Physical examination reveals a tall, thin build, scoliosis, and ectopia lentis. Laboratory tests show elevated levels of homocysteine and methionine in the plasma. Treatment is initiated with vitamin B6 (pyridoxine) and folate. Homocysteine levels improve, but methionine levels remain elevated. Based on this scenario, what type of defect does the patient most likely have? (Q844922)

A. Type 1 Homocystinuria

B. Type 2 Homocystinuria

C. Type 3 Homocystinuria

D. Marfan Syndrome

See Answer in Part B → Q16

@Neet_pg_bot

Q17: A 7-year-old boy is brought with recurrent episodes of a red, scaly rash that worsens with sun exposure, as well as intermittent episodes of diarrhea and mood changes. A urine sample reveals an abnormal presence of neutral amino acids. The patient's diet is balanced, and there is no history of similar symptoms in the family. Laboratory tests indicate a deficiency in niacin despite adequate dietary intake. What is the underlying cause of the patient's condition? (Q844921)

A. Deficiency in tryptophan hydroxylase

B. Deficiency in the neutral amino acid transporter

C. Overactivity of serotonin decarboxylase

D. Excessive dietary leucine

See Answer in Part B → Q17

@Neet_pg_bot

Q18: A 45-year-old man presents with symptoms of diarrhea, dermatitis, and dementia. On further investigation, his diet is found to be predominantly corn-based, and he has a history of alcohol use disorder. Laboratory tests reveal low levels of tryptophan and niacin in his blood. Which enzyme deficiency is most likely contributing to the patient's condition? (Q844920)

A. Tryptophan hydroxylase

B. Kynureninase

C. Serotonin decarboxylase

D. Niacinamide adenine dinucleotide synthetase

See Answer in Part B → Q18

@Neet_pg_bot

Q19: Which of the following statements is true? (Q844919)

A. Tyrosine is the amino acid forming Serotonin

B. Niacin deficiency is seen in Carcinoid syndrome

C. Tryptophan hydroxylase is required for Niacin synthesis

D. Decarboxylation of 5 hydroxy tryptophan forms niacin.

See Answer in Part B → Q19

@Neet_pg_bot

Q20: Which of the following TCA intermediates is not formed by glucogenic amino acids? (Q844918)

A. Alpha Ketoglutarate

B. Isocitrate

C. Fumarate

D. Oxaloacetate

See Answer in Part B → Q20

@Neet_pg_bot

Q21: Which of the following amino acids are both glucogenic and ketogenic? (Q844917)

A. Leucine

B. Tryptophan

C. Lysine

D. Alanine

See Answer in Part B → Q21

@Neet_pg_bot

Q22: A 6-month-old infant has been having developmental delays. Further examination reveals a defect in the enzyme called phenylalanine hydroxylase. Which of the following urine odor is seen in this infant? (Q844916)

A. Fruity odour

B. Musty odour

C. Cabbage odour

D. Burnt sugar

See Answer in Part B → Q22

@Neet_pg_bot

Q23: A 6-month-old infant presents with a lack of pigmentation in their skin, hair, and eyes. The parents are concerned about the child's sensitivity to sunlight and the involuntary back-and-forth eye movements that the child exhibits. A significant decrease in melanin pigmentation is present. Which of the following enzymes is absent or deficient in this condition? (Q844915)

A. Alanine aminotransferase (ALT)

B. Tyrosinase

C. Phenylalanine hydroxylase

D. S-adenosylmethionine

See Answer in Part B → Q23

@Neet_pg_bot

Q24: Tyrosine is involved in the synthesis of multiple neurotransmitters. Match the following neurotransmitters with the corresponding enzymes involved in their synthesis? Neurotransmitter Enzyme 1. Dihydroxy phenylalanine (DOPA) a. Dopamine beta-hydroxylase 2. Dopamine b. N-methyl transferase 3. Norepinephrine c. Tyrosine hydroxylase 4. Epinephrine d. DOPA Decarboxylase (Q844914)

A. 1-d, 2-c, 3-b, 4-a

B. 1-c, 2-b, 3-a, 4-d

C. 1-d, 2-b, 3-c, 4-a

D. 1-c, 2-d, 3-a, 4-b

See Answer in Part B → Q24

@Neet_pg_bot

Q25: A 5-month-old infant is brought to the pediatrician due to poor weight gain, vomiting, and yellowing of the skin and eyes. Genetic testing confirms a deficiency in fumarylacetoacetate hydrolase. The physician recommends a low-protein diet and the drug Nitisinone. Which of the following is not an indication of Nitisinone? (Q844913)

A. Tyrosinemia type I

B. Alkaptonuria

C. Hawkinsinuria

D. Tyrosinemia type II

See Answer in Part B → Q25

@Neet_pg_bot

Q26: A 4-month-old infant presents with jaundice, hepatomegaly, and failure to thrive. The parents also report that the infant has episodes of vomiting and lethargy. Laboratory tests reveal elevated levels of tyrosine and succinylacetone in the blood and urine, along with signs of coagulopathy and renal tubular dysfunction. What is the most likely underlying metabolic disorder? (Q844912)

A. Phenylketonuria

B. Tyrosinemia type II

C. Tyrosinemia type I

D. Alkaptonuria

See Answer in Part B → Q26

@Neet_pg_bot

Q27: A 2-year-old child is brought to the clinic by his parents due to darkening of urine upon standing and brownish discolouration of the sclera and ear cartilage. The parents also report that their child has been experiencing joint pain. Upon further investigation, the physician suspects a tyrosine metabolic disorder and orders urine tests. Which of the following enzymes is most likely deficient in this patient?

(Q844911)

A. Parahydroxy phenylpyruvate dioxygenase

B. Tyrosine transaminase

C. Homogentisate oxidase

D. Fumarylacetoacetate hydrolase

See Answer in Part B → Q27

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• [04_Amino Acids and Proteins Chemistry] 016 Glycine And One Carbon Pool

« Back to Folder Directory

[04_Amino Acids and Proteins Chemistry] 016 Glycine And One Carbon Pool — Quick Jump

Q1 (p. 191) *Q2 (p. 191)* *Q3 (p. 192)* *Q4 (p. 192)* *Q5 (p. 192)* *Q6 (p. 193)*
Q7 (p. 193) *Q8 (p. 194)* *Q9 (p. 194)* *Q10 (p. 195)* *Q11 (p. 195)* *Q12 (p. 196)*
Q13 (p. 196) *Q14 (p. 197)* *Q15 (p. 197)*

Q1: Which of the following is not a source of glycine? (Q843353)

A. Serine

B. Threonine

C. Alanine

D. Lysine

See Answer in Part B → Q1

@Neet_pg_bot

Q2: A 1 year old boy presents with lethargy, poor muscle tone, and developmental delays. Blood tests reveal elevated levels of glycine. The physician suspects a defect in the glycine cleavage system. Which of the following metabolites if found LOW, would support the diagnosis of a defective glycine cleavage system? (Q843443)

A. Serine

B. N5,N10 methylene tetrahydrofolate

C. Threonine

D. Succinyl CoA

See Answer in Part B → Q2

@Neet_pg_bot

Q3: A 6-month-old infant presented with lethargy, vomiting, and irritability. Blood tests revealed elevated ammonia levels. The infant was diagnosed with a urea cycle disorder. Which of the following medications, acting as ammonia-scavenging agents, would be most effective in reducing the infant's blood ammonia levels? (Q843444)

A. Phenyl acetate

B. N-acetyl glutamate

C. Lactulose

D. Neomycin

See Answer in Part B → Q3

@Neet_pg_bot

Q4: The glycine cleavage system in the body produces CO2, ammonia and a one carbon molecule. Which of the following is a subunit in the system? (Q843446)

A. Tetrahydrofolate

B. Lipoamide

C. Tetrahydrofolate dependent aminomethyltransferase (THFAAMT)

D. Pyridoxal phosphate

See Answer in Part B → Q4

@Neet_pg_bot

Q5: Which of the following is not a known use of glycine in humans ? (Q843447)

A. Precursor for metabolites like creatine, glutathione, and heme.

B. Enhances the quality of sleep and supports neurological functions.

C. Inhibits synthesis of bile acid,reducing lipid absorption.

D. Provide structural integrity for collagen structure.

See Answer in Part B → Q5

@Neet_pg_bot

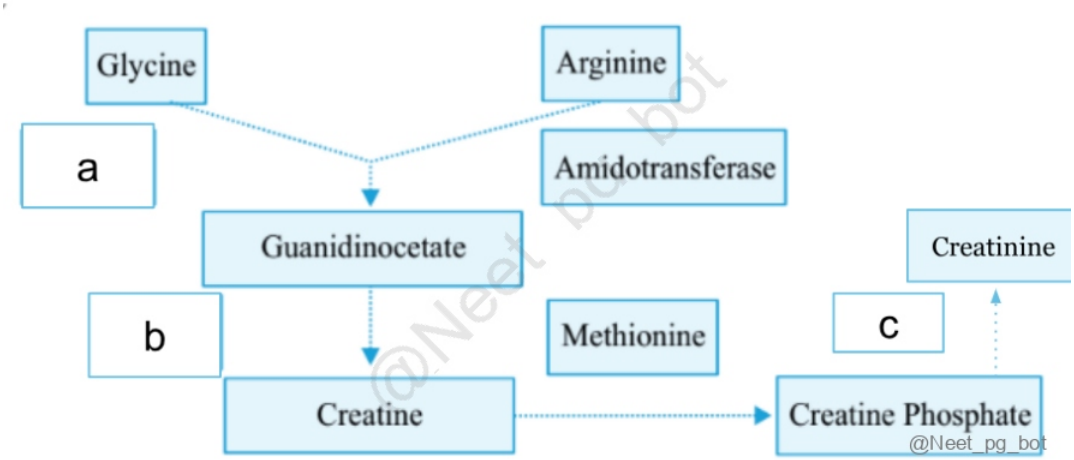
Q6: Based on the image given below, what are the sites at which the marked steps of creatinine synthesis occur? (Q843456)

A. a- brain, b- liver, c- kidney

B. a- kidney,b- liver, c- muscle

C. a- liver, b- kidney, c- muscle

D. a- muscle, b- liver, c-kidney



See Answer in Part B → Q6

@Neet_pg_bot

Q7: Which of the following diseases is caused by the deficiency of the enzyme ■ ALA synthase? (Q843457)

A. Porphyria cutanea tarda

B. X linked sideroblastic anemia

C. Acute intermittent porphyria

D. Congenital erythropoietic porphyria

See Answer in Part B → Q7

@Neet_pg_bot

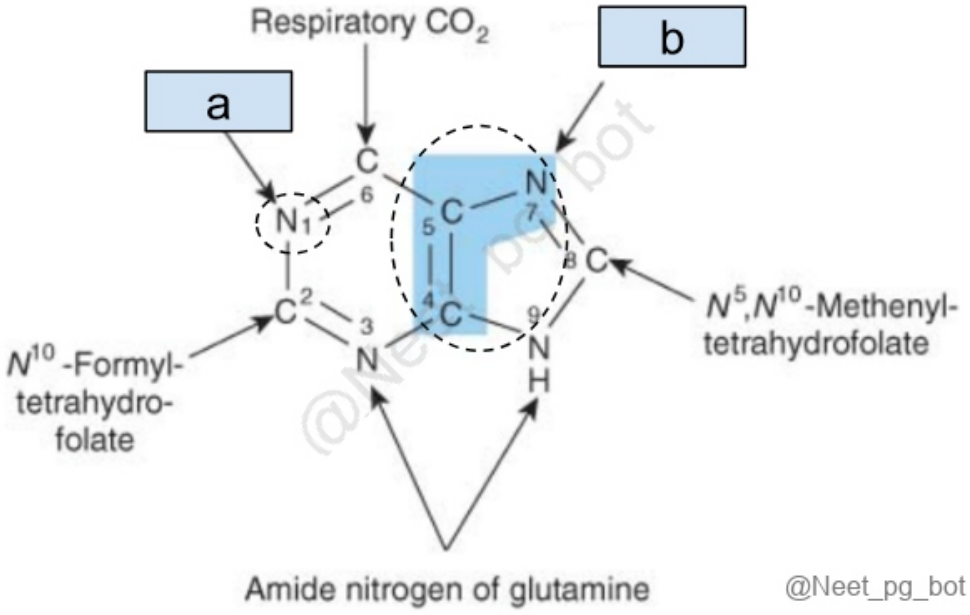
Q8: In the following figure, identify the sources of the marked components. (Q843458)

A. a- aspartate, b- glycine

B. a- glycine, b-aspartate

C. a- methionine, b-alanine

D. a- alanine, b- methionine



See Answer in Part B → Q8

@Neet_pg_bot

Q9: Which of the following statements is incorrect regarding one-carbon derivatives of tetrahydrofolic acid (THFA) and their associated enzymes and reactions? (Q843459)

A. N5-formimino-THFA converts formiminoglutamate (FIGLU) to glutamate.

B. N5-methyl-THFA carries the methyl group for converting homocysteine to methionine.

C. N5N10-methylene-THFA is required for the synthesis of thymidylate (dTMP) from deoxyuridylate (dUMP).

D. N10-formyl-THFA transfers formyl groups in pyrimidine synthesis.

See Answer in Part B → Q9

@Neet_pg_bot

Q10: A 62-year-old female patient presents with fatigue, pallor, and glossitis. Laboratory tests indicate macrocytic anemia, elevated homocysteine and methylmalonic acid levels. Given these findings, what is the most likely metabolic pathway disrupted by this patient's condition, leading to the observed anemia? (Q843462)

A. Conversion of homocysteine to methionine

B. Reduction of folic acid to dihydrofolate

C. Deamination of histidine to formimino glutamate

D. Conversion of Phenylalanine to Tyrosine

See Answer in Part B → Q10

@Neet_pg_bot

Q11: In the kynurenine pathway of tryptophan catabolism, which of the following compounds directly contributes to the formation of N10-formyl tetrahydrofolate in the one-carbon pool? (Q843463)

A. Formyl kynurenine

B. Kynurenine

C. 3- Hydroxy kynurenine

D. THFA

See Answer in Part B → Q11

@Neet_pg_bot

Q12: Which enzyme in the histidine catabolism pathway facilitates the incorporation of the formimino group into the one-carbon pool, forming N5-formimino tetrahydrofolate? (Q843464)

A. Histidase

B. Urocanase

C. Glutamate formimino transferase

D. Glutamate dehydrogenase

See Answer in Part B → Q12

@Neet_pg_bot

Q13: A 3-week-old infant presents with poor feeding, lethargy, and vomiting. Lab results show elevated ammonia levels, increased glutamine and increased orotic acid in the blood, with no evidence of megaloblastic anemia. Which enzyme deficiency is likely responsible for this condition? (Q845009)

A. Ornithine transcarbamylase (OTC)

B. Ornithine aminotransferase

C. Uridine monophosphate (UMP) synthase

D. Hypoxanthine guanine phosphoribosyltransferase (HGPRT)

See Answer in Part B → Q13

@Neet_pg_bot

Q14: A patient with a urea cycle disorder is being evaluated. Which urea cycle intermediate serves as the link between the urea cycle and the TCA cycle? (Q845010)

A. Argininosuccinate

B. Fumarate

C. Oxaloacetate

D. Succinate

See Answer in Part B → Q14

@Neet_pg_bot

Q15: Arrange the following steps of the synthesis of creatinine from glycine in the correct order: A. Transport of creatine from the liver to muscle via blood B. Formation of guanidino acetic acid from arginine and glycine C. Formation of creatine by methyl transferase D. Formation of creatine phosphate E. Conversion of creatine phosphate to creatinine (Q875957)

A. B→ C→A→ D → E

B. A→ D→C→ E → B

C. D→ A→E→ C → B

D. C→ D→B→ E → A

See Answer in Part B → Q15

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• [04_Amino Acids and Proteins Chemistry] 017 Previous Year Questions

« Back to Folder Directory

[04_Amino Acids and Proteins Chemistry] 017 Previous Year Questions — Quick Jump

Q1 (p. 198)	*Q2 (p. 199)*	*Q3 (p. 199)*	*Q4 (p. 200)*	*Q5 (p. 201)*	*Q6 (p. 201)*
Q7 (p. 202)	*Q8 (p. 202)*	*Q9 (p. 203)*	*Q10 (p. 203)*	*Q11 (p. 203)*	*Q12 (p. 204)*
Q13 (p. 204)	*Q14 (p. 204)*	*Q15 (p. 205)*	*Q16 (p. 205)*	*Q17 (p. 205)*	*Q18 (p. 206)*
Q19 (p. 206)	*Q20 (p. 207)*	*Q21 (p. 207)*	*Q22 (p. 207)*	*Q23 (p. 208)*	*Q24 (p. 208)*
Q25 (p. 208)	*Q26 (p. 209)*	*Q27 (p. 209)*	*Q28 (p. 209)*	*Q29 (p. 210)*	*Q30 (p. 211)*
Q31 (p. 211)	*Q32 (p. 211)*	*Q33 (p. 212)*	*Q34 (p. 212)*	*Q35 (p. 212)*	*Q36 (p. 213)*
Q37 (p. 213)	*Q38 (p. 214)*	*Q39 (p. 214)*	*Q40 (p. 214)*	*Q41 (p. 215)*	*Q42 (p. 215)*
Q43 (p. 215)	*Q44 (p. 216)*	*Q45 (p. 216)*	*Q46 (p. 217)*	*Q47 (p. 217)*	*Q48 (p. 217)*
Q49 (p. 218)	*Q50 (p. 218)*	*Q51 (p. 218)*	*Q52 (p. 219)*	*Q53 (p. 219)*	*Q54 (p. 219)*
Q55 (p. 220)	*Q56 (p. 220)*	*Q57 (p. 220)*	*Q58 (p. 221)*	*Q59 (p. 221)*	*Q60 (p. 222)*
Q61 (p. 222)	*Q62 (p. 222)*	*Q63 (p. 223)*	*Q64 (p. 223)*	*Q65 (p. 224)*	*Q66 (p. 224)*
Q67 (p. 224)	*Q68 (p. 225)*	*Q69 (p. 225)*	*Q70 (p. 226)*	*Q71 (p. 226)*	*Q72 (p. 226)*
Q73 (p. 227)	*Q74 (p. 227)*	*Q75 (p. 227)*	*Q76 (p. 228)*	*Q77 (p. 228)*	*Q78 (p. 229)*
Q79 (p. 229)	*Q80 (p. 229)*	*Q81 (p. 230)*			

Q1: Identify the test: (Q854626)

A. Rothera’s test

B. Benedict’s test

C. Fehling’s test

D. Barfoed’s test



See Answer in Part B → Q1

@Neet_pg_bot

Q2: A chronic alcoholic patient is diagnosed with gouty arthritis. What biochemical changes will be seen in this patient? (Q855230)

A. Decreased NADH/NAD+ ratio

B. Increased urea and urate levels

C. Increased urate and lactate levels

D. Increased level of alkaline phosphatase

See Answer in Part B → Q2

@Neet_pg_bot

Q3: Which of the following does not contribute to the repair of DNA double-stranded breaks? (Q857361)

A. ATP

B. RAD-51

C. ATM

D. Ku 70

See Answer in Part B → Q3

@Neet_pg_bot

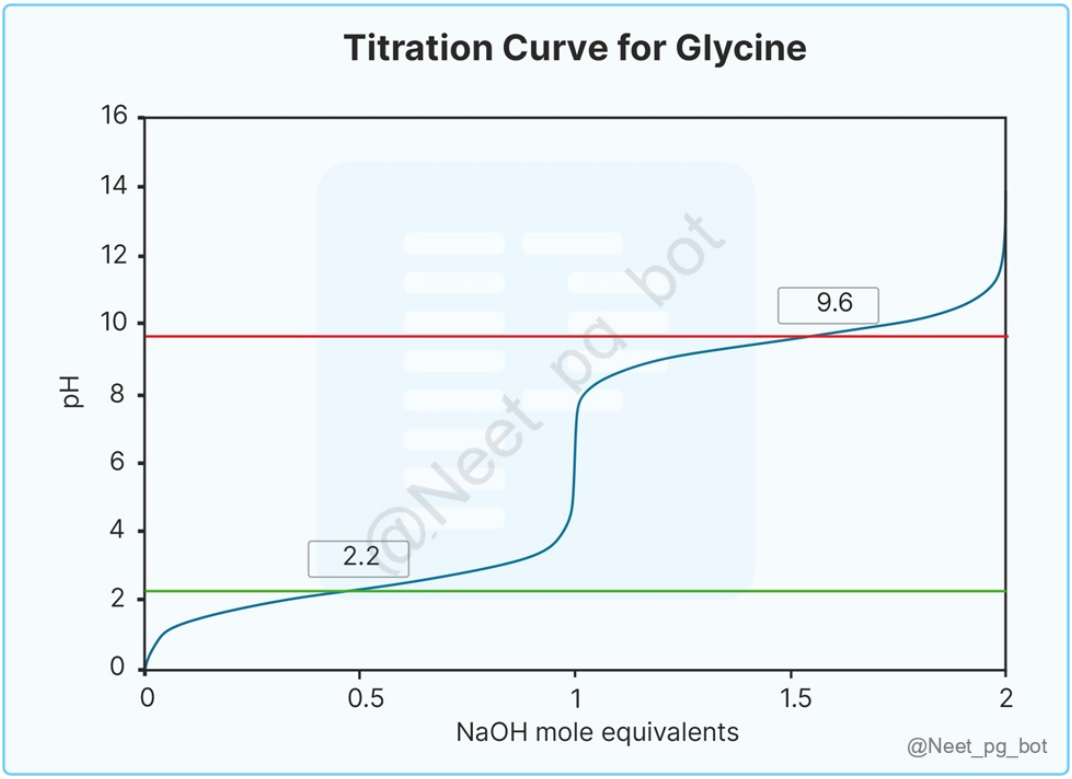
Q4: In the given glycine titration curve, calculate or identify the pH at which molecules exist in a zwitter ion form? (Q865428)

A. 4.4

B. 6

C. 9

D. 2.2



See Answer in Part B → Q4

@Neet_pg_bot

Q5: A female patient presented with pain and redness in the great toe. The serum uric acid level is 9.4mg/dl. The physician prescribed a drug for the treatment. Which of the following enzyme is likely to be inhibited by this drug? (Q859691)

A. Phosphoribosyl transferase

B. Thymidylate synthase

C. Xanthine oxidase

D. Dihydrofolate reductase

See Answer in Part B → Q5

@Neet_pg_bot

Q6: What is true about the Hayflick limit? (Q856475)

A. Human cells can divide a finite number of times only

B. Free radical oxidation level is unrestricted

C. Telomerase activity is unrestricted

D. RNA polymerase can function for a finite number of time only

See Answer in Part B → Q6

@Neet_pg_bot

Q7: Which of the following post-translational modifications is common to collagen, hemoglobin, and immunoglobulins? (Q860100)

A. Hydroxylation

B. Subunit aggregation

C. Poly A tail addition

D. DNA mismatch repair

See Answer in Part B → Q7

@Neet_pg_bot

Q8: A patient presents with dermatitis, dementia, and cognitive decline. Which micronutrient deficiency is most likely responsible? (Q844235)

A. Niacin

B. Thiamine

C. Tryptophan

D. Riboflavin

See Answer in Part B → Q8

@Neet_pg_bot

Q9: Which of the following is deficient in a patient with phenylketonuria with normal phenyl alanine hydroxylase levels? (Q844228)

A. Tyrosine transaminase

B. Dopa decarboxylase

C. Tyrosine hydroxylase

D. Dihydrobiopterine reductase

See Answer in Part B → Q9

@Neet_pg_bot

Q10: A 2-month-old infant presents with poor feeding, vomiting, and lethargy. Laboratory studies show hyperammonemia and elevated levels of orotic acid in the urine. Which of the following is the most likely enzyme deficiency? (Q866671)

A. Carbamoyl phosphate synthetase I deficiency

B. Ornithine transcarbamylase (OTC) deficiency

C. Orotidine 5'-phosphate decarboxylase deficiency

D. Argininosuccinate lyase deficiency

See Answer in Part B → Q10

@Neet_pg_bot

Q11: On centrifugation of a cell, LDH will get deposited in: (Q859706)

A. Mitochondria

B. Lysosome

C. Cytoplasm

D. Golgi complex

See Answer in Part B → Q11

@Neet_pg_bot

Q12: An infant is brought by his parents with complaints that his urine turns black on standing. Which of the following metabolic disorders is most likely? (Q855263)

A. Phenylketonuria

B. Alkaptonuria

C. Homocystinuria

D. Maple syrup urine disease

See Answer in Part B → Q12

@Neet_pg_bot

Q13: What happens to chloride levels in sweat and pancreatic secretions in cystic fibrosis? (Q854625)

A. Chloride is raised in sweat

B. Chloride is decreased in sweat

C. Chloride is decreased in pancreatic secretions

D. None of the above

See Answer in Part B → Q13

@Neet_pg_bot

Q14: A 45-year-old man presents with deep labored breathing and altered sensorium. His arterial blood gas shows a metabolic acidosis. The following serum electrolyte values are obtained: Na■: 145 mmol/L K■: 4 mmol/L Cl■: 90 mmol/L HCO■■: 15 mmol/L Calculate the anion gap (AG) and interpret the finding. (Q854606)

A. 16 mmol/L – Normal anion gap metabolic acidosis

B. 40mmol/L – High anion gap metabolic acidosis

C. 14 mmol/L – Normal anion gap metabolic acidosis

D. 32 mmol/L – Mixed metabolic acidosis

See Answer in Part B → Q14

@Neet_pg_bot

Q15: Which of the following enzyme defects leads to Pompe's disease? (Q857351)

A. Lysosomal alpha glucosidase

B. β-hexosaminidase -A

C. β-galactocerebrosidase

D. β-glucocerebrosidase

See Answer in Part B → Q15

@Neet_pg_bot

Q16: Xanthurenic aciduria may be treated with high doses of which vitamin? (Q857350)

A. Vitamin B6

B. Vitamin B12

C. Vitamin B5

D. Vitamin B7

See Answer in Part B → Q16

@Neet_pg_bot

Q17: Deficiency of which of the following causes Barth's syndrome? (Q857325)

A. Cardiolipin

B. Sphingomyelin

C. Glycolipids

D. Cerebroside

See Answer in Part B → Q17

@Neet_pg_bot

Q18: Liver produces ketones but cannot use it due to the deficiency of which of the following? (Q857321)

A. Thiophorase

B. Alkaline phosphatase

C. Alanine transaminase

D. Thoiolase

See Answer in Part B → Q18

@Neet_pg_bot

Q19: A 16-year-old patient presents with pellagra-like skin eruptions during the spring season for the past 5 years. Laboratory tests reveal normal levels of 5-Hydroxyindoleacetic Acid (HIAA), high aminoaciduria, and normal urinary indole levels. What is the most likely diagnosis? (Q860099)

A. Carcinoid tumor

B. Nutritional pellagra

C. Hartnup disorder

D. Essential fructosuria

See Answer in Part B → Q19

@Neet_pg_bot

Q20: Which of the following substances is used in a tube for collecting blood samples specifically for measuring alcohol concentration? (Q860092)

A. Heparin + EDTA

B. Sodium fluoride + Potassium dichromate

C. Heparin

D. Sodium citrate

See Answer in Part B → Q20

@Neet_pg_bot

Q21: Which of the following uses CRISPR-Cas9 technology? (Q860081)

A. DNA Editing

B. Post-transcriptional Modification

C. DNA Induction

D. DNA Editing Solution

See Answer in Part B → Q21

@Neet_pg_bot

Q22: A young girl presented to the OPD for an examination. She is tall and thin with slender fingers, and dislocated lenses. Workup revealed cystathione beta-synthase deficiency. Which amino acid supplement is required in this case? (Q855999)

A. Cysteine

B. Methionine

C. Serine

D. Tyrosine

See Answer in Part B → Q22

@Neet_pg_bot

Q23: Match the following amino acids with their derivatives: A. Tyrosine i. Melatonin B. Tryptophan ii. Taurine C. Glycine iii. Thyroid hormones D. Cysteine iv. Creatinine (Q879286)

A. A-i, B-ii, C-iii, D-iv

B. A-iii, B-i, C-iv, D-ii

C. A-iv, B-ii, C-i, D-iii

D. A-ii, B-i, C-iv, D-iii

See Answer in Part B → Q23

@Neet_pg_bot

Q24: All of the following are lipotropic factors except? (Q859698)

A. Choline

B. Lecithine

C. Arginine

D. Methionine

See Answer in Part B → Q24

@Neet_pg_bot

Q25: A patient came to the hospital with a complaint of abdominal pain. Routine investigations were unremarkable. On observation, the urine turned black on standing. Which of the following enzymes is likely to be defective in this patient? (Q858459)

A. Homogentisate oxidase

B. Xanthine oxidase

C. Phenylalanine hydroxylase

D. Dihydric orotate dehydrogenase

See Answer in Part B → Q25

@Neet_pg_bot

Q26: In a patient with obstructive jaundice, which of the following outcomes may be observed in Van der Bergh's reaction? (Q854880)

A. Direct positive

B. Indirect positive

C. Both positive

D. Coproporphyrin

See Answer in Part B → Q26

@Neet_pg_bot

Q27: Nitric oxide is synthesized from: (Q854869)

A. L-Arginine

B. L-Citrulline

C. Glycine

D. Lysine

See Answer in Part B → Q27

@Neet_pg_bot

Q28: The maximum thermic effect is seen with a meal that has (Q856500)

A. Protein

B. Fat

C. Carbohydrates

D. Mixed meal having all 3 in equal amount

See Answer in Part B → Q28

@Neet_pg_bot

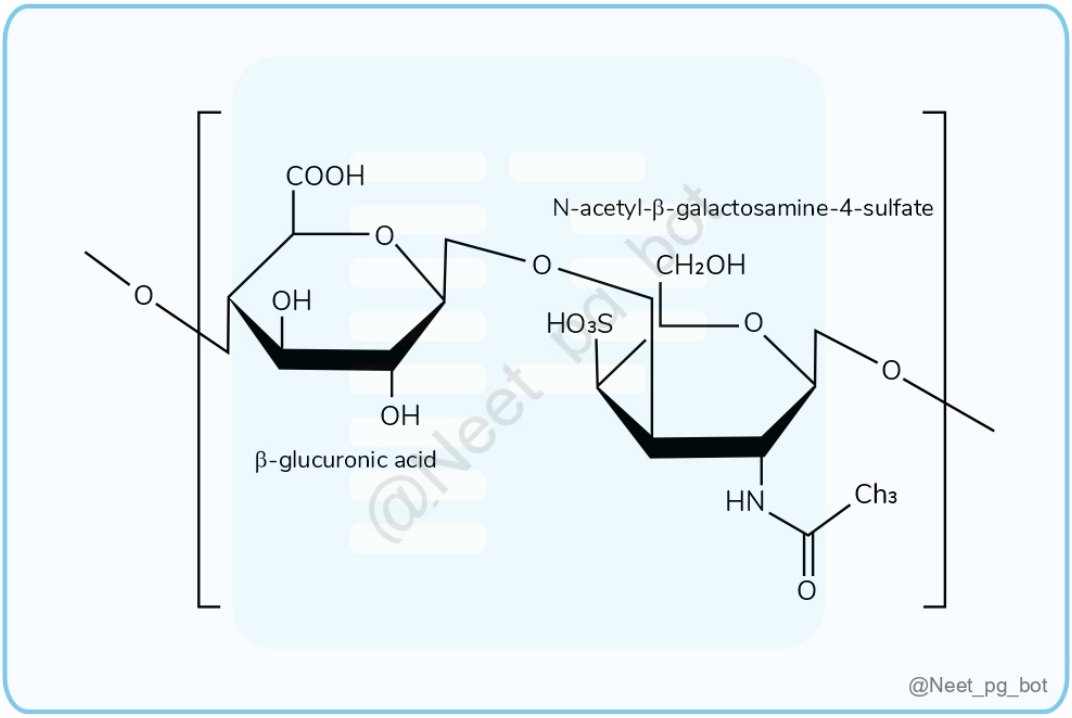
Q29: The given chemical structure is of: (Q856923)

A. Keratan sulfate

B. Chondroitin sulfate

C. Heparin

D. Dermatan sulfate



See Answer in Part B → Q29

@Neet_pg_bot

Q30: Which of the following is not a true statement regarding telomerase? (Q856880)

A. Has reverse transcriptase activity

B. Present only in Eukaryotes

C. Maintain the chromosome length

D. Involved in DNA repair

See Answer in Part B → Q30

@Neet_pg_bot

Q31: Ammonia in the brain is detoxified to form (Q856874)

A. Urea

B. Glutamine

C. Ammonium ions

D. Alanine

See Answer in Part B → Q31

@Neet_pg_bot

Q32: Which of the following disorders is characterized by the presence of a cherry red spot on the retina? (Q860068)

A. Tay-Sachs disease

B. Fabry's disease

C. Gaucher's disease

D. Von Gierke's disease

See Answer in Part B → Q32

@Neet_pg_bot

Q33: A patient presented with joint pain and on evaluation, urine turns black on standing. What is the likely enzyme deficiency? (Q855951)

A. Homogentisic acid 1,2-dioxygenase

B. Tyrosine transaminase

C. Fumarylacetoacetase hydrolase

D. 4-hydroxy-phenyl pyruvate dioxygenase

See Answer in Part B → Q33

@Neet_pg_bot

Q34: Which of the following options is false in a patient with advanced liver disease? (Q856619)

A. Hyperammonemia

B. Decreased ATP synthesis

C. Oxidative carboxylation of alpha-ketoglutarate

D. Increased synthesis of glutamine and glutamine from alpha-ketoglutarate

See Answer in Part B → Q34

@Neet_pg_bot

Q35: Which of the following consumes the highest energy during assimilation? (Q855919)

A. Proteins

B. Carbohydrates

C. Mono-unsaturated fatty acids

D. Polyunsaturated fatty acids

See Answer in Part B → Q35

@Neet_pg_bot

Q36: Which enzyme is likely to be deficient in a 4-year-old child who presents with a habit of biting their lips and fingers, and has a missing portion of fingers on their left hand? Further evaluation shows delayed developmental milestones. The parents report that two male cousins on the mother's side have similar symptoms and have been diagnosed with a genetic disorder. Laboratory reports indicate hyperuricemia. (Q857544)

A. Purine nucleoside phosphorylase

B. Hypoxanthine -guanine phosphoribosyl transferase.

C. Adenine phosphoribosyl transferase

D. Adenosine deaminase

See Answer in Part B → Q36

@Neet_pg_bot

Q37: Which of the following is considered a universal methyl group donor in various biochemical reactions? (Q856961)

A. Glucose-6-phosphate

B. Acetyl CoA

C. S-Adenosyl Methionine

D. S-Adenosyl Serine

See Answer in Part B → Q37

@Neet_pg_bot

Q38: What is the enzyme that is responsible for the defect causing Maple syrup urine disease? (Q856942)

A. Branched-chain a-ketoacid dehydrogenase

B. Isovaleryl CoA dehydrogenase

C. Pyruvate carboxylase

D. Acetyl CoA carboxylase

See Answer in Part B → Q38

@Neet_pg_bot

Q39: In a patient with Maple Syrup Urine Disease (MSUD), all of the following amino acids should be restricted in diet except? (Q855044)

A. Methionine

B. Valine

C. Leucine

D. Isoleucine

See Answer in Part B → Q39

@Neet_pg_bot

Q40: Which of the following options best describes the mutation seen in patients with cystic fibrosis? (Q855554)

A. Tryptophan at 708 position, chloride channel

B. Phenylalanine at 708 position, calcium channel

C. Phenylalanine at 508 position, chloride channel

D. Tryptophan at 508 position, calcium channel

See Answer in Part B → Q40

@Neet_pg_bot

Q41: A 62-year-old male with CAD comes to the physician for a follow-up examination. His blood pressure is 130/80 mm Hg, height of 182 cm, weight of 85 kg, and a BMI of 25 kg/m². Serum studies show a glucose concentration of 95 mg/dL and homocysteine concentration of 19.3 μmol/L (N=5–15). Serum lipid concentrations are within the normal range. Which of the following enzyme deficiencies is most likely to be found in this patient? (Q857892)

A. Homogentisate oxidase

B. Cystathionine beta synthase

C. Cystathionine gamma synthase

D. HGPRTase

See Answer in Part B → Q41

@Neet_pg_bot

Q42: Which of the following neurotransmitter is not derived from amino acid? (Q839939)

A. GABA

B. Acetylcholine

C. Dopamine

D. 5 hydroxytryptamine

See Answer in Part B → Q42

@Neet_pg_bot

Q43: Replacing alanine by which amino acid, will increase the UV absorbance of protein at 280nm wavelength? (Q854875)

A. Leucine

B. Proline

C. Arginine

D. Tryptophan

See Answer in Part B → Q43

@Neet_pg_bot

Q44: Tau protein inclusions are involved in: (Q866657)

A. Alzheimer’s disease

B. Huntington's disease

C. Amyotrophic lateral sclerosis

D. CNS lymphoma

See Answer in Part B → Q44

@Neet_pg_bot

Q45: Which of the following is the best method to detect HbA1c? (Q856564)

A. lon-exchange chromatography

B. Immunoassay

C. Electrophoresis

D. Affinity chromatography

See Answer in Part B → Q45

@Neet_pg_bot

Q46: Thermogenin protein is mainly found in: (Q857488)

A. Golgi complex

B. Mitochondria

C. Endoplasmic reticulum

D. Nucleus

See Answer in Part B → Q46

@Neet_pg_bot

Q47: Oxaloacetate is produced from which of the following amino acid? (Q857864)

A. Alanine

B. Lysine

C. Leucine

D. Aspartate

See Answer in Part B → Q47

@Neet_pg_bot

Q48: A farmer sprayed a chemical in the pool which causes inhibition of complex-III of the electron transport chain. Identify the inhibitor? (Q857851)

A. Rotenone

B. TTFA

C. Cyanide

D. Antimycin

See Answer in Part B → Q48

@Neet_pg_bot

Q49: A patient presents with recurrent episodes of severe body pain triggered by vigorous exercise. Laboratory findings reveal anemia and the presence of sickle-shaped red blood cells. He is diagnosed with sickle cell anemia. What amino acid substitution occurs in sickle cell anemia? (Q857841)

A. Substitution of valine by glutamic acid at the 6th position

B. Substitution of glutamic acid by valine at the 6th position

C. Substitution of valine by glutamic acid at the 5th position

D. Substitution of glutamic acid by valine at the 5th position

See Answer in Part B → Q49

@Neet_pg_bot

Q50: What do chaperones assist in? (Q855214)

A. Ubiquitinization of proteins

B. Protein modification

C. Protein folding

D. Protein cleavage

See Answer in Part B → Q50

@Neet_pg_bot

Q51: DNA sequence is determined by? (Q857323)

A. Flow cytometry

B. FISH

C. RFLP

D. PCR

See Answer in Part B → Q51

@Neet_pg_bot

Q52: Which of the following methods cannot be used to precipitate proteins? (Q856573)

A. Add alcohol and acetone

B. Using heavy metal ions

C. Moving pH away from isoelectric pH

D. Adding trichloroacetic acid

See Answer in Part B → Q52

@Neet_pg_bot

Q53: Aromatic ring is seen in which of the following amino acids? (Q860441)

A. Phenylalanine, valine, tryptophan

B. Phenylalanine, tryptophan, tyrosine

C. Tyrosine, cysteine, tryptophan

D. Lysine, methionine, valine

See Answer in Part B → Q53

@Neet_pg_bot

Q54: If the RNA sequence is CAAGGU, what is the sequence of the template strand of DNA? (Q860375)

A. CAAGGT

B. GTTCCA

C. ACCTTG

D. TGGAAC

See Answer in Part B → Q54

@Neet_pg_bot

Q55: Ammonia is transported from the brain to the liver in the form of which amino acid? (Q860348)

A. Glutamate

B. Alanine

C. Glycine

D. Glutamine

See Answer in Part B → Q55

@Neet_pg_bot

Q56: A patient presents with arthritis, and arthroscopy reveals blackening of the joints. Which enzyme deficiency is most likely responsible for this condition? (Q852546)

A. Homogentisate oxidase

B. Branched-chain alpha-ketoacid dehydrogenase

C. Tyrosinase

D. Fumarase

See Answer in Part B → Q56

@Neet_pg_bot

Q57: Which of the following amino acids primarily acts as a buffer in blood due to its ability to accept and donate protons at physiological pH? (Q852550)

A. Histidine

B. Arginine

C. Tryptophan

D. Tyrosine

See Answer in Part B → Q57

@Neet_pg_bot

Q58: Which amino acid donates nitrogen to sodium benzoate when given for urea cycle disorders? (Q853005)

A. Glutamine

B. Glutamate

C. Glycine

D. Arginine

See Answer in Part B → Q58

@Neet_pg_bot

Q59: Match the following amino acid with derivatives: 1. Histidine a.Catecholamines 2. Tyrosine b. Histamine 3. Tryptophan c. Glutathione 4. Cysteine d. Melatonin (Q852999)

A. 1-b, 2-a, 3-d, 4-c

B. 1-a, 2-c, 3-b, 4-d

C. 1-d, 2-c, 3-b, 4-a

D. 1-b, 2-c, 3-d, 4-a

See Answer in Part B → Q59

@Neet_pg_bot

Q60: A patient has elevated phenylalanine levels (40 mg/dL), but phenylalanine hydroxylase enzyme levels are normal. Which cofactor deficiency is most likely responsible for this condition? (Q866320)

A. Tetrahydrofolate

B. Tetrahydrobiopterin (BH4)

C. Thiamine

D. Pyridoxine

See Answer in Part B → Q60

@Neet_pg_bot

Q61: Which of the following is true about the structural organization of proteins? (Q858466)

A. Tertiary structure is three-dimensional

B. The secondary structure is stabilized by disulfide bonds.

C. Primary, secondary, and tertiary structures are destroyed by denaturation.

D. The secondary and tertiary structure depends upon amino acid sequence.

See Answer in Part B → Q61

@Neet_pg_bot

Q62: A 40-year-old female working as a senior executive leading a sedentary lifestyle presents to the OPD with palmoplantar pigmentation. X-ray spine shows calcification of intervertebral discs. On adding Benedict's reagent to the urine sample, it gives a greenish-brown precipitate and comes positive. What is the most likely enzyme

deficiency in this patient? (Q860064)

A. Phenylalanine hydroxylase

B. Tyrosine hydroxylase

C. Homogentisic acid oxidase

D. PHPP dioxygenase

See Answer in Part B → Q62

@Neet_pg_bot

Q63: A 3-month-old baby presents with severe hepatomegaly, cataracts in both eyes, lethargy, and hypotonia. Based on these symptoms, which enzyme deficiency is most likely involved? (Q860054)

A. Galactokinase

B. Hepatic Kinase

C. Hepatic Glucose-6-Phosphatase

D. Galactose-1-Phosphate Uridyl Transferase

See Answer in Part B → Q63

@Neet_pg_bot

Q64: A couple brings their 4-year-old child to the outpatient department (OPD) due to self-mutilating behavior and poor growth. Which of the following associated enzyme deficiencies could explain these symptoms? (Q860052)

A. HGPRT deficiency

B. Homogentisic Aciduria

C. Phenylalanine Hydroxylase deficiency

D. Adenosine deaminase deficiency

See Answer in Part B → Q64

@Neet_pg_bot

Q65: Zellweger syndrome is associated with which cellular organelle? (Q860043)

A. Peroxisomes

B. Nucleosomes

C. Lysosomes

D. Ribosomes

See Answer in Part B → Q65

@Neet_pg_bot

Q66: Which of the following is caused by the deficiency of the enzyme tyrosine transaminase? (Q860430)

A. Type I Tyrosinemia

B. Type II Tyrosinemia

C. Alkaptonuria

D. Type III Tyrosinemia

See Answer in Part B → Q66

@Neet_pg_bot

Q67: An infant presents to the Pediatrics OPD with a distended abdomen and jaundice. The mother reports that the baby has shown an increased appetite in recent months. During the clinical examination, the physician notes hepatomegaly. Which of the following enzyme deficiencies is most likely responsible for this condition? (Q860357)

A. Glycogen Synthase

B. Glycogen phosphorylase

C. Glucose-6-Phosphatase

D. Galactose-1-Phosphate Uridyl Transferase

See Answer in Part B → Q67

@Neet_pg_bot

Q68: A 1-month-old infant presents with recurrent vomiting, diarrhea, jaundice, and failure to thrive shortly after breastfeeding began. Examination shows hepatomegaly, and urine tests are positive for reducing substances but negative for glucose. What is the most likely diagnosis? (Q860334)

A. Lactose Intolerance

B. Duodenal Atresia

C. Galactosemia

D. Pyloric Stenosis

See Answer in Part B → Q68

@Neet_pg_bot

Q69: An infant presents with distended abdomen, jaundice, increased appetite, and hepatomegaly. Which enzyme deficiency is most likely responsible for this condition? (Q860329)

A. Glucose-6-phosphatase

B. Glycogen phosphorylase

C. Galactose-1-phosphate uridyl transferase

D. Glycogen synthase

See Answer in Part B → Q69

@Neet_pg_bot

Q70: A 7-year-old child of short stature presents with skin patches and learning difficulties. Diagnostic workup indicates a defect in DNA repair specifically related to double-strand breaks. What is the most likely diagnosis? (Q860325)

A. Bloom Syndrome

B. Xeroderma Pigmentosum

C. Cockayne Syndrome

D. Lynch Syndrome

See Answer in Part B → Q70

@Neet_pg_bot

Q71: A neonate presents with seizures and is found to have a cherry red spot on fundus examination. Enzyme assay reveals deficiency of hexosaminidase A. Which of the following substances is most likely to be accumulated in this patient? (Q866323)

A. GM1 ganglioside

B. GM2 ganglioside

C. Sphingomyelin

D. Glucocerebroside

See Answer in Part B → Q71

@Neet_pg_bot

Q72: In uncontrolled diabetes, in which of the following cells there is decreased glucose uptake due to insulin deficiency: (Q865446)

A. RBC

B. Skeletal muscle

C. Liver

D. Brain

See Answer in Part B → Q72

@Neet_pg_bot

Q73: For mycobacterium species differentiation, technique used is/are? Line probe assay MIRU (Mycobacterium Interspersed Rapatitive Units) Spoligotyping RFLP (Q865405)

A. 1, 2, 3

B. 2, 3, 4

C. 1, 2, 4

D. 1, 2, 3, 4

See Answer in Part B → Q73

@Neet_pg_bot

Q74: A child presents with abdominal distension and an enlarged liver by 8 cm below the costal margin. The liver is smooth on palpation. Which of the following is the most likely diagnosis? (Q866675)

A. Glycogen Storage Disease (GSD)

B. Lysosomal Storage Disease (LSD)

C. Hepatocellular Carcinoma (HCC)

D. Autoimmune Hepatitis

See Answer in Part B → Q74

@Neet_pg_bot

Q75: A patient presents with fatigue, anemia, and hepatomegaly. Hemoglobin electrophoresis reveals the presence of HbS. What will be the appropriate treatment? (Q866656)

A. Hydroxyurea

B. Blood transfusion

C. Iron supplementation

D. Vitamin B12 supplementation

See Answer in Part B → Q75

@Neet_pg_bot

Q76: The biochemical form of amyloid in hemodialysis-associated amyloidosis is: (Q859718)

A. AL

B. AA

C. Aß2m

D. ATTR

See Answer in Part B → Q76

@Neet_pg_bot

Q77: What do chaperones assist in? (Q859701)

A. Ubiquitinization of proteins

B. Protein modification

C. Protein folding

D. Protein cleavage

See Answer in Part B → Q77

@Neet_pg_bot

Q78: What is the function of proteasomes? (Q858454)

A. Protein folding

B. Post-translation modification

C. Protein degradation

D. Protein sorting

See Answer in Part B → Q78

@Neet_pg_bot

Q79: A farmer sprayed a chemical in the pool which causes inhibition of complex-III of the electron transport chain. Identify the inhibitor? (Q820323)

A. Rotenone

B. TTFA

C. Cyanide

D. Antimycin

See Answer in Part B → Q79

@Neet_pg_bot

Q80: Oxaloacetate is produced from which of the following amino acid? (Q820367)

A. Alanine

B. Lysine

C. Leucine

D. Aspartate

See Answer in Part B → Q80

@Neet_pg_bot

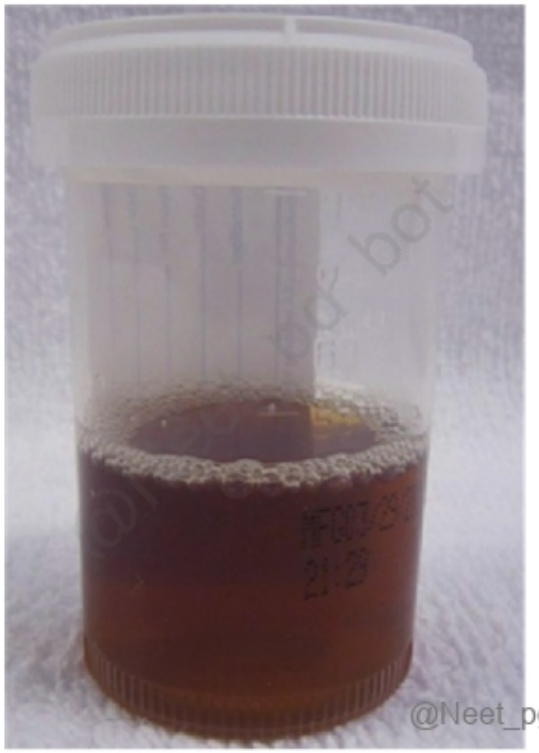
Q81: The lab technician observes that a urine sample obtained from a 4-year-old male appeared normal initially, but underwent a change in color as depicted in the image after a few hours. Subsequently, the color intensified over the following hours. Which deficiency can potentially lead to this condition? (Q793373)

A. Beta-carotene

B. Tyrosine aminotransferase

C. L-ascorbic acid

D. Homogentisic acid oxidase



See Answer in Part B → Q81

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• [05_Enzymes] 018 Enzymes

« Back to Folder Directory

[05_Enzymes] 018 Enzymes — Quick Jump

Q1 (p. 231)	*Q2 (p. 231)*	*Q3 (p. 232)*	*Q4 (p. 232)*	*Q5 (p. 232)*	*Q6 (p. 233)*
Q7 (p. 233)	*Q8 (p. 233)*	*Q9 (p. 234)*	*Q10 (p. 234)*	*Q11 (p. 235)*	*Q12 (p. 235)*
Q13 (p. 235)	*Q14 (p. 236)*	*Q15 (p. 236)*	*Q16 (p. 237)*	*Q17 (p. 237)*	*Q18 (p. 237)*
Q19 (p. 238)	*Q20 (p. 238)*	*Q21 (p. 238)*	*Q22 (p. 239)*	*Q23 (p. 239)*	*Q24 (p. 240)*
Q25 (p. 240)	*Q26 (p. 240)*				

Q1: Which of the following statements accurately describes the changes in Km (Michaelis constant) and Vmax (maximum reaction rate) in the presence of an uncompetitive inhibitor? (Q843311)

A. Km increases, Vmax increases

B. Km decreases, Vmax decreases

C. Km stays the same, Vmax decreases

D. Km increases, Vmax stays the same

See Answer in Part B → Q1

@Neet_pg_bot

Q2: A patient presents with hepatomegaly and joint pain. Investigations suggest abnormal intracellular accumulation of macromolecules such as lipids or glycoconjugates. Which cellular organelle is primarily responsible for this accumulation? (Q882197)

A. Golgi apparatu

B. Lysosome

C. Mitochondria

D. Peroxisome

See Answer in Part B → Q2

@Neet_pg_bot

Q3: Which of the following statements regarding creatine kinase (CK) is false? (Q843568)

A. Total CK is elevated in muscular dystrophies

B. CK levels start to rise within 3–6 hours after myocardial infarction

C. CK levels are increased in hemolysis and congestive heart failure

D. CK-MB is used as a marker for myocardial infarction

See Answer in Part B → Q3

@Neet_pg_bot

Q4: Which of the following is used to study the kinetics of oligomeric enzymes? (Q843567)

A. Lineweaver Burke equation

B. Michaelis Menten equation

C. Hill equation

D. Dixon equation

See Answer in Part B → Q4

@Neet_pg_bot

Q5: Which among the following statements regarding the induction and repression of enzymes is NOT true? (Q843566)

A. Repression acts at the gene level to decrease the amount of enzyme

B. Induction relieves repression on the operator site

C. Glucokinase is induced by insulin

D. ALA(Aminolevulinic acid) synthase is induced by heme

See Answer in Part B → Q5

@Neet_pg_bot

Q6: All of the following enzymes are proteins except? (Q843565)

A. Aminoacyl tRNA synthetase

B. Peptidyl transferase

C. RNA polymerase

D. Acetyl-CoA synthetase

See Answer in Part B → Q6

@Neet_pg_bot

Q7: In a Lineweaver-Burk plot for an enzyme experiencing non-competitive inhibition, which of the following statements is correct? (Q843312)

A. The slope of the Lineweaver-Burk plot increases, but the y-intercept remains the same.

B. The y-intercept increases while Km remains constant and the slope increases.

C. Both the slope and the y-intercept increase.

D. The slope of the Lineweaver-Burk plot decreases while the y-intercept increases.

See Answer in Part B → Q7

@Neet_pg_bot

Q8: Which of the following statements are true regarding enzymes? a) Enzymes are proteins that can accelerate reactions by factors of 10^6 to 10^9 or more. b) Enzymes act on a wide range of substrates. c) Enzymes stabilize the ground state. d) Enzymes can catalyze both forward and reverse reactions. their e) Enzymes are regulated by

pH, temperature, inhibitors, and activators. f) Enzyme are water insoluble (Q843236)

A. a, d, e, f ,c

B. a, b, d, e

C. a, d, e

D. a, e, f , c

See Answer in Part B → Q8

@Neet_pg_bot

Q9: Which of the following examples best illustrates competitive inhibition? (Q843310)

A. Methotrexate and Dihydrofolate Reductase

B. Cyanide and Cytochrome c Oxidase

C. Alanine and Pyruvate Kinase

D. Citrate and Phosphofructokinase-1(PFK-1)

See Answer in Part B → Q9

@Neet_pg_bot

Q10: Given the following enzyme kinetics parameters: Maximum velocity (Vmax): 100 µmol/min Michaelis constant (Km): 10 µM Initial velocity (Vo): 50 µmol/min What is the substrate concentration [S] of the given enzyme? (Q843309)

A. 5 µM

B. 10 µM

C. 20 µM

D. 25 µM

See Answer in Part B → Q10

@Neet_pg_bot

Q11: In enzyme kinetics, a double reciprocal plot (Lineweaver-Burk plot) is used to determine kinetic parameters. What do the x-intercept and y-intercept of a double reciprocal plot represent? (Q843308)

A. x-intercept: -1/Km; y-intercept: 1/Vmax

B. x-intercept: 1/Km; y-intercept: 1/Vmax

C. x-intercept:1/Vmax; y-intercept: -1/Km

D. x-intercept: -1/Km; y-intercept: -1/Vmax

See Answer in Part B → Q11

@Neet_pg_bot

Q12: Which of the following statements regarding Michaelis Menten equation is true? (Q843282)

A. Used to calculate the rate of reaction

B. Substrate concentration is required to calculate the rate using Michaelis Menten equation

C. Michaelis constant is the substrate concentration at which the velocity of the reaction is half the maximal velocity

D. All are true

See Answer in Part B → Q12

@Neet_pg_bot

Q13: What is the correct equation to calculate the catalytic efficiency of an enzyme? (Q843278)

A. Kcat/Km

B. Km/Kcat

C. Vmax/St

D. Vmax/Km

See Answer in Part B → Q13

@Neet_pg_bot

Q14: Which of the following statements is false Increase in enzyme concentration increases the rate of reaction Increase in substrate concentration increases the rate of reaction Increase in product concentration increases the rate of reaction (Q843277)

A. 1, 3

B. 3

C. 1, 2, 3

D. None

See Answer in Part B → Q14

@Neet_pg_bot

Q15: Which of the following statements is/are true regarding the mechanisms of action of enzymes? Enzymes that act by altering the strain of substrates catalyze lytic reactions Enzymes forming covalent bonds are seen commonly in group transfer reactions When the functional groups of aminoacyl side chains of enzymes act as an acid or base, the rate of reaction increases (Q843275)

A. 1, 2

B. 2, 3

C. 1, 3

D. 1, 2, 3

See Answer in Part B → Q15

@Neet_pg_bot

Q16: A 35-year-old female with chronic diarrhea, weight loss, and fatty stools complains that her symptoms worsen after a high-fat meal. Reduced activity of pancreatic lipase is suspected. Which of the following ions is most likely to be low in this patient? (Q843272)

A. Calcium

B. Copper

C. Iron

D. Zinc

See Answer in Part B → Q16

@Neet_pg_bot

Q17: A patient presents with symptoms of fatigue and muscle weakness. Laboratory tests indicate a deficiency in riboflavin. Which coenzyme's function is most likely affected? (Q843240)

A. NAD

B. FAD

C. Coenzyme A

D. Folic acid

See Answer in Part B → Q17

@Neet_pg_bot

Q18: Which of the following is not a function of prosthetic groups? (Q843239)

A. Substrate binding and orientation

B. Formation of covalent bonds

C. Electron transfer

D. Protein degradation

See Answer in Part B → Q18

@Neet_pg_bot

Q19: Match the enzyme class with its corresponding example: Enzyme Class Example A) Oxidoreductases 1) Hexokinase B) Transferases 2) Lactate dehydrogenase C) Hydrolases 3) DNA ligase D) Lyases 4) Trypsin E) Isomerases 5) Aldolase F) Ligases 6) Triosephosphate isomerase (Q843238)

A. A-2, B-1, C-4, D-5, E-6, F-3

B. A-1, B-2, C-4, D-5, E-3, F-6

C. A-2, B-4, C-5, D-1, E-6, F-3

D. A-3, B-2, C-4, D-5, E-6, F-1

See Answer in Part B → Q19

@Neet_pg_bot

Q20: What is the role of activation energy in a chemical reaction, and how do enzymes influence it? (Q843237)

A. Activation energy converts molecules from transition to ground state; enzymes increase it.

B. Activation energy converts molecules from ground state to transition state; enzymes lower it.

C. Activation energy is released during product formation; enzymes do not affect it.

D. Activation energy breaks bonds in products; enzymes increase it.

See Answer in Part B → Q20

@Neet_pg_bot

Q21: Which of the following statements about suicide inhibition is incorrect? (Q843320)

A. The inhibitor covalently binds to the enzyme after being converted to a reactive intermediate.

B. Suicide inhibition is a reversible form of enzyme inhibition.

C. Suicide inhibitors resemble the enzyme's natural substrate.

D. Allopurinol acts as a suicide inhibitor of xanthine oxidase to manage gout.

See Answer in Part B → Q21

@Neet_pg_bot

Q22: Which of the following biochemical reactions is not regulated by allosteric regulation? (Q843321)

A. Phosphofructokinase-1 in glycolysis

B. Aspartate transcarbamoylase in pyrimidine biosynthesis

C. Glycogen phosphorylase in glycogenolysis

D. Glucokinase in glycolysis

See Answer in Part B → Q22

@Neet_pg_bot

Q23: Which of the following shapes of the Michaelis-Menten plot is observed for allosteric regulation? (Q843322)

A. Hyperbolic curve

B. Sigmoidal curve

C. Linear increase

D. Parabolic curve

See Answer in Part B → Q23

@Neet_pg_bot

Q24: Match the following regulation mechanisms with their respective enzymatic reactions: Regulation mechanisms Enzymatic reactions 1. Compartmentation a. Phosphorylation of glycogen phosphorylase 2. Feedback inhibition b. Negative regulation of phosphofructokinase by ATP 3. Covalent modification c. Segregation of glycolytic enzymes in the cytosol 4. Proteolytic activation d. Activation of pepsinogen to pepsin (Q843323)

A. 1 - c, 2 - d, 3 - a, 4 - b

B. 1 - b, 2 - c, 3 - d, 4 - a

C. 1 - c, 2 - b, 3 - a, 4 - d

D. 1 - d, 2 - a, 3 - b, 4 - c

See Answer in Part B → Q24

@Neet_pg_bot

Q25: Which of the following statements about isoenzymes is incorrect? (Q843324)

A. Isoenzymes can be distinguished by their different amino acid sequences or structural properties

B. Allozymes are produced by different genes and can have distinct Km values or substrate affinities

C. Isoforms result from post-translational modifications, such as changes in sialic acid content in alkaline phosphatase (ALP)

D. Electrophoresis is a technique used to separate isoenzymes based on their mobility.

See Answer in Part B → Q25

@Neet_pg_bot

Q26: A 45-year-old male presents with severe pain and swelling in his right calf following an intense workout session two days ago. Laboratory tests, including measurements of lactate dehydrogenase (LDH) isoenzymes, are ordered. Which LDH isoenzyme is most indicative of the patient's condition? (Q843325)

A. LDH-1

B. LDH-2

C. LDH-3

D. LDH-5

See Answer in Part B → Q26

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• [05_Enzymes] 019 Previous Year Questions

« Back to Folder Directory

[05_Enzymes] 019 Previous Year Questions — Quick Jump

Q1 (p. 242)	*Q2 (p. 242)*	*Q3 (p. 242)*	*Q4 (p. 243)*	*Q5 (p. 243)*	*Q6 (p. 244)*
Q7 (p. 244)	*Q8 (p. 244)*	*Q9 (p. 245)*	*Q10 (p. 245)*	*Q11 (p. 246)*	*Q12 (p. 246)*
Q13 (p. 247)	*Q14 (p. 247)*	*Q15 (p. 248)*	*Q16 (p. 248)*	*Q17 (p. 248)*	*Q18 (p. 249)*
Q19 (p. 249)	*Q20 (p. 250)*	*Q21 (p. 250)*			

Q1: Deficiency of copper which is necessary for lysyl oxidase is seen in (Q853010)

A. Scurvy

B. Scoliosis

C. Ehler Danlos Syndrome

D. Menke's disease

See Answer in Part B → Q1

@Neet_pg_bot

Q2: Deficiency of which enzyme leads to increased 2,3-BPG levels and low glucose values in vitro? (Q879288)

A. Pyruvate kinase

B. Phosphoglycerate kinase

C. G6PD

D. Hexokinase

See Answer in Part B → Q2

@Neet_pg_bot

Q3: A 13-year-old presents with acute flaccid paralysis. Cerebrospinal fluid is collected, and an RT-PCR test is ordered to identify the causative virus. Which viral enzyme is responsible for replicating the genome of this RNA virus? (Q859716)

A. DNA-dependent RNA polymerase

B. RNA-dependent DNA polymerase

C. DNA-dependent DNA polymerase

D. RNA-dependent RNA polymerase

See Answer in Part B → Q3

@Neet_pg_bot

Q4: Which of the following is not a copper-containing enzyme? (Q859570)

A. Mitochondrial superoxide dismutase

B. Cytosolic superoxide dismutase

C. Tyrosinase

D. Lysyl oxidase

See Answer in Part B → Q4

@Neet_pg_bot

Q5: Which enzymes show deficiency in Von Gierke's disease? (Q859425)

A. Liver glycogen phosphorylase

B. Muscle glycogen phosphorylase

C. Glycogen debranching enzyme

D. Glucose-6-phosphatase

See Answer in Part B → Q5

@Neet_pg_bot

Q6: What is the likely diagnosis for a 36-year-old man who has been experiencing bluish-black discoloration of the sclera and pinna for the past 6 months, along with a history of black urine upon standing since childhood? (Q859421)

A. Phenylketonuria

B. Alkaptonuria

C. Tyrosinemia

D. Maple syrup urine disease

See Answer in Part B → Q6

@Neet_pg_bot

Q7: Which of the following is true regarding competitive inhibition? (Q854885)

A. The inhibitor binds to an allosteric site on the enzyme.

B. Competitive inhibition decreases the apparent Vmax (maximum reaction rate) of the enzyme.

C. Increasing substrate concentration can overcome competitive inhibition.

D. Competitive inhibition decreases the Km for the substrate

See Answer in Part B → Q7

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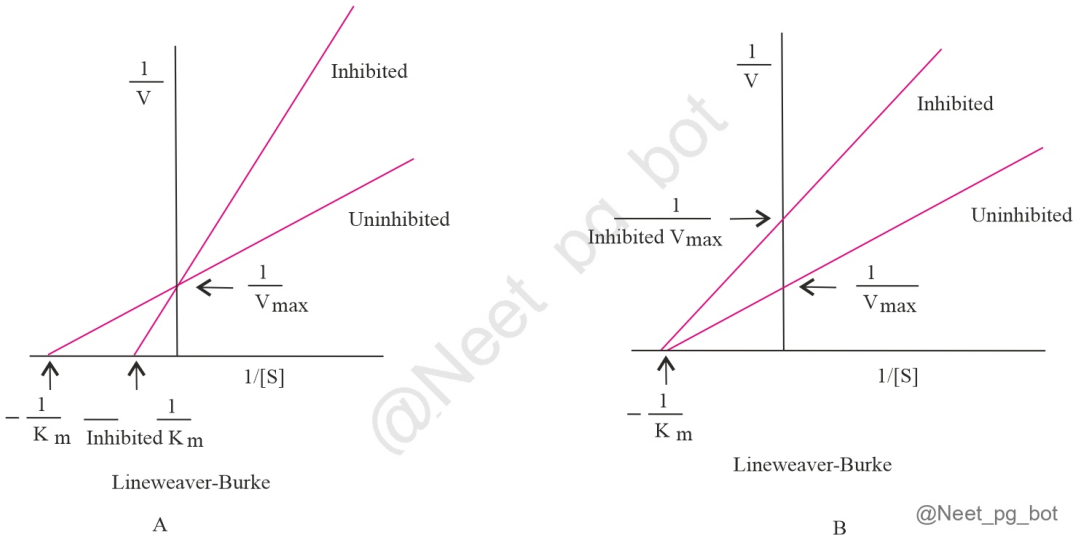
Q8: In the given enzyme kinetics graphs, identify the types of inhibitors labeled as A and B. (Q839952)

A. Competitive and Non-Competitive

B. Uncompetitive and Non-competitive

C. Non-Competitive and Uncompetitive

D. Non-competitive and competitive



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See Answer in Part B → Q8

@Neet_pg_bot

Q9: What is the most likely diagnosis of a hypertensive patient presenting with Tanner stage 4 development and hyperpigmentation? (Q839950)

A. 21 beta hydroxylase deficiency

B. 11 Beta hydroxylase deficiency

C. 17 beta hydroxylase deficiency

D. 17 alpha hydroxylase deficiency

See Answer in Part B → Q9

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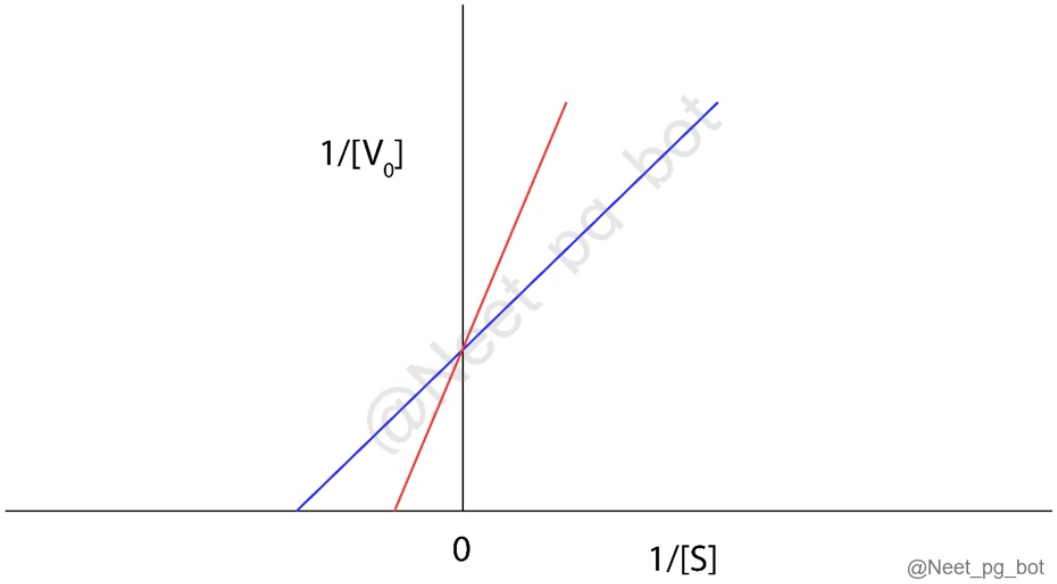
Q10: In the given graph, identify the type of inhibition: (Q844275)

A. Competitive

B. Non-competitive

C. Mixed

D. None of the above



See Answer in Part B → Q10

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Q11: A patient presents with severe abdominal pain and elevated lipase levels. Imaging reveals a stone in the common bile duct (CBD). Which enzyme is likely to be elevated in this condition? (Q844268)

A. Gamma-glutamyl transferase (GGT)

B. Lactate Dehydrogenase (LDH)

C. Alanine Aminotransferase (ALT)

D. Aspartate Aminotransferase (AST)

See Answer in Part B → Q11

@Neet_pg_bot

Q12: A 13-year-old boy presents with jaundice, fatigue, muscle stiffness, tremors, behavioral changes, hepatosplenomegaly, and Kayser-Fleischer rings. Which test is

definitive for the diagnosis? (Q844273)

A. Urinary copper

B. Serum ceruloplasmin

C. Hepatic parenchymal copper concentration

D. Genetic testing for ATP7B mutation

See Answer in Part B → Q12

@Neet_pg_bot

Q13: Which of the following enzymes is dependent on Vitamin C for its activity? (Q852557)

A. Lysyl dehydrogenase

B. Lysyl oxidase

C. Prolyl hydroxylase

D. Hydroxyprolyl kinase

See Answer in Part B → Q13

@Neet_pg_bot

Q14: Which of the following acts as a major intracellular antioxidant and helps in detoxifying reactive oxygen species? (Q866659)

A. Glutathione

B. Catalase

C. Superoxide dismutase

D. Peroxidase

See Answer in Part B → Q14

@Neet_pg_bot

Q15: A 2-month-old infant is brought to the pediatrician due to feeding difficulties, vomiting, and failure to thrive. The child's history reveals that the infant wasborn full-term and had a normal birth weight. On physical examination, the infant appears jaundiced, and the liver is palpable below the costal margin. Oil drop Cataracts are also noted on eye examination. Laboratory tests show elevated levels of galactose in the blood and urine. Based on these clinical findings, which enzyme deficiency is most likely responsible for this presentation? (Q820344)

A. G6PD (Glucose-6-phosphate dehydrogenase)

B. GALT (Galactose-1-phosphate uridylyltransferase)

C. ALDH (Aldehyde dehydrogenase)

D. PKU (Phenylalanine hydroxylase)

See Answer in Part B → Q15

@Neet_pg_bot

Q16: Hormone-sensitive lipase is activated by all of the following EXCEPT? (Q820391)

A. Glucagon

B. Epinephrine

C. ACTH

D. Insulin

See Answer in Part B → Q16

@Neet_pg_bot

Q17: A 62-year-old male with CAD comes to the physician for a follow-up examination. His blood pressure is 130/80 mm Hg, height of 182 cm, weight of 85 kg, and a BMI of 25 kg/m². Serum studies shows a glucose concentration of 95 mg/dL and

homocysteine concentration of 19.3 μmol/L (N=5–15). Serum lipid concentrations are within the normal range. which of the following enzyme deficiencies is most likely to be found in this patient? (Q820409)

A. Homogentisate oxidase

B. Cystathionine beta synthase

C. Cystathionine gamma synthase

D. HGPRTase

See Answer in Part B → Q17

@Neet_pg_bot

Q18: A 35-year-old battery factory worker presents with abdominal colic, a blue line on the gums, and anemia. Laboratory findings suggest lead poisoning. Which of the following enzymes is the most sensitive and primarily inhibited by lead in this condition? (Q830705)

A. ALA synthase

B. ALA dehydratase

C. Heme synthase

D. Coproporphyrinogen oxidase

See Answer in Part B → Q18

@Neet_pg_bot

Q19: Which of the statements below accurately describes the characteristics of the Michaelis-Menten graph? (Q793383)

A. Km is directly proportional to substrate concentration

B. Km will vary with the enzyme concentration

C. Km is indirectly proportional to substrate concentration

D. Km has no relation to substrate concentration

See Answer in Part B → Q19

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Q20: By the action of the enzyme aromatase, testosterone is transformed into which of the subsequent hormones? (Q793389)

A. 5-hydroxy testosterone

B. Estradiol

C. Cortisol

D. Cortisone

See Answer in Part B → Q20

@Neet_pg_bot

Q21: What is the substance that is most likely to be secreted by the hypothalamus? (Q793467)

A. Hexokinase I

B. Hexokinase II

C. Hexokinase III

D. Hexokinase IV

See Answer in Part B → Q21

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• [06_Free Radicals_ Antioxidants _ Trace Elements] 020 Previous Year Questions

« Back to Folder Directory

[06_Free Radicals_ Antioxidants _ Trace Elements] 020 Previous Year Questions — Quick Jump

Q1 (p. 251)	*Q2 (p. 251)*	*Q3 (p. 252)*	*Q4 (p. 252)*	*Q5 (p. 253)*	*Q6 (p. 254)*
Q7 (p. 254)	*Q8 (p. 255)*	*Q9 (p. 255)*	*Q10 (p. 255)*	*Q11 (p. 256)*	*Q12 (p. 256)*
Q13 (p. 257)	*Q14 (p. 257)*	*Q15 (p. 257)*	*Q16 (p. 258)*	*Q17 (p. 258)*	*Q18 (p. 259)*
Q19 (p. 259)	*Q20 (p. 259)*	*Q21 (p. 260)*	*Q22 (p. 260)*	*Q23 (p. 260)*	*Q24 (p. 261)*
Q25 (p. 261)	*Q26 (p. 261)*	*Q27 (p. 262)*	*Q28 (p. 262)*	*Q29 (p. 263)*	*Q30 (p. 263)*
Q31 (p. 263)	*Q32 (p. 264)*	*Q33 (p. 264)*	*Q34 (p. 265)*	*Q35 (p. 265)*	*Q36 (p. 265)*
Q37 (p. 266)	*Q38 (p. 266)*	*Q39 (p. 266)*	*Q40 (p. 267)*	*Q41 (p. 267)*	*Q42 (p. 268)*
Q43 (p. 268)	*Q44 (p. 269)*	*Q45 (p. 270)*	*Q46 (p. 270)*	*Q47 (p. 270)*	

Q1: Which enzyme deficiency causesVon Gierke's disease? (Q857492)

A. Liver phosphorylase

B. Lysosomal glucosidase

C. Hepatic glycogen synthase

D. Glucose 6-phosphatase

See Answer in Part B → Q1

@Neet_pg_bot

Q2: You are conducting an experiment on mitochondrial respiration. You add malate/pyruvate and respiration is normal. You add succinate and respiration is normal. When you add another substance in the presence of pyruvate/succinate and malate, respiration is blocked. Which of the following substances is most likely added? (Q855978)

A. Rotenone

B. Antimycin A

C. Oligomycin

D. 2,4-dinitrophenol

See Answer in Part B → Q2

@Neet_pg_bot

Q3: In this scenario, what is the origin of the blood glucose measured at 7 am the following morning for an individual who had dinner at 8 pm the previous night? (Q855985)

A. Hepatic glycogenolysis

B. Hepatic gluconeogenesis

C. Muscle glycogenolysis

D. Dietary glucose

See Answer in Part B → Q3

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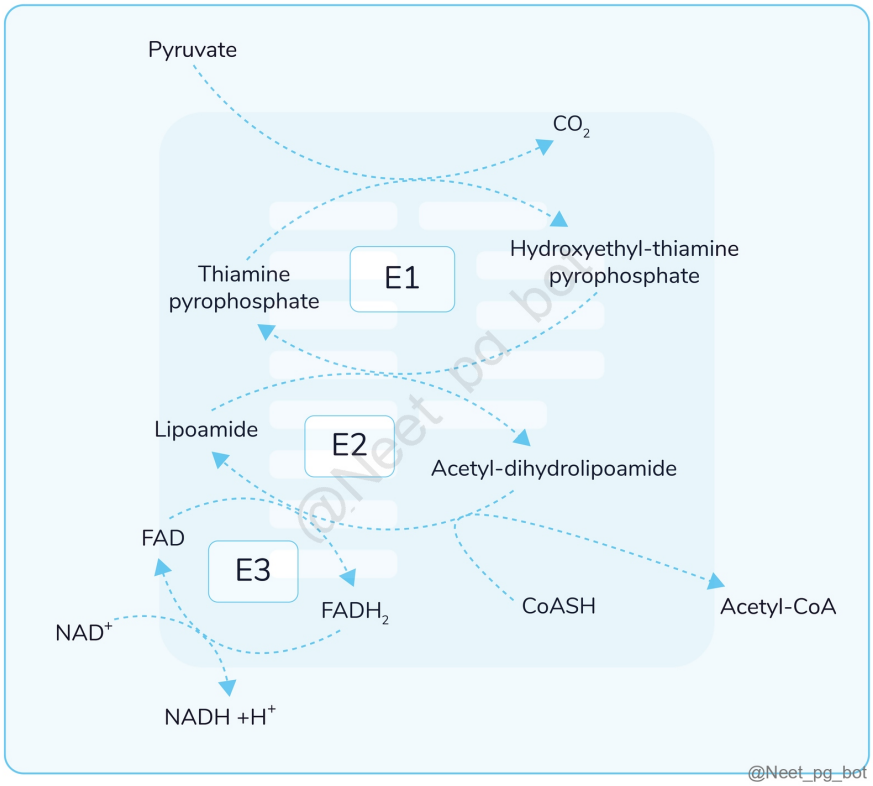
Q4: In the diagram provided, which enzymes are identified as E1 and E2, responsible for the conversion of pyruvate to acetyl CoA? (Q856000)

A. Pyruvate dehydrogenase and dihydrolipoyl transacetylase

B. Pyruvate carboxylase and dihydrolipoyl transacetylase

C. Pyruvate dehydrogenase and dihydrolipoyl dehydrogenase

D. Pyruvate carboxylase and dihydrolipoyl dehydrogenase



See Answer in Part B → Q4

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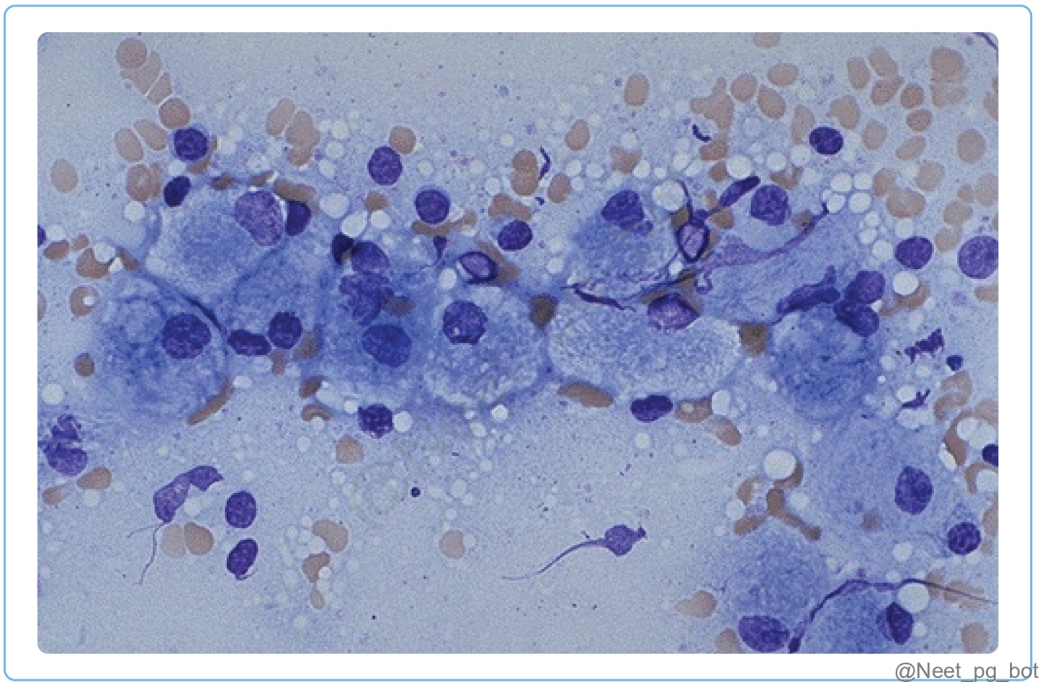
Q5: A 5 year old boy presented with easy fatigability, irritability and inability to concentrate. Labs revealed the following findings a and b (light and oil immersion respectively) on the bone marrow aspiration. Which of the following is the most likely enzyme deficient in this condition? (Q856005)

A. Hexosaminidase

B. Glucocerebrosidase

C. Sphingomyelinase

D. N-acetyglucosaminidase



See Answer in Part B → Q5

@Neet_pg_bot

Q6: Which enzyme is shared by both glycolysis and gluconeogenesis? (Q856507)

A. Pyruvate kinase

B. Phosphofructokinase

C. Phosphoenolpyruvate carboxy kinase

D. Phosphoglycerate kinase

See Answer in Part B → Q6

@Neet_pg_bot

Q7: Ketosis is common in type 1 DM due to: - Decreased beta-oxidation of fatty acids Increased mobilization of fat from the adipose cell Decreased acetyl CoA in TCA cycle Increase beta-hydroxy butyryl synthase (Q856515)

A. 2,3 and 4

B. 1,2,3 and 4

C. 4 only

D. 1 and 2 only

See Answer in Part B → Q7

@Neet_pg_bot

Q8: Which of the following poisoning agent does not inhibit cytochrome oxidase? (Q856558)

A. Hydrogen sulfide

B. Carbon monoxide

C. Methane

D. Cyanide

See Answer in Part B → Q8

@Neet_pg_bot

Q9: Which one of the options provided does not serve as a substrate for the synthesis of glucose? (Q856613)

A. Acetyl coenzyme A

B. Glycerol

C. Alanine

D. Lactate

See Answer in Part B → Q9

@Neet_pg_bot

Q10: Glucose is stored in the form of glycogen because (Q856876)

A. It is compact

B. It has multiple reducing ends

C. It can be stored at multiple organs

D. It can provide glucose for about 1 week when needed

See Answer in Part B → Q10

@Neet_pg_bot

Q11: Which one of the following choices does not represent a function of glucagon? (Q856992)

A. Glycolysis

B. Gluconeogenesis

C. Glycogenolysis

D. Ketogenesis

See Answer in Part B → Q11

@Neet_pg_bot

Q12: Which enzyme is responsible for the deficiency causing classical galactosemia? (Q856998)

A. Aldose reductase

B. Galactokinase

C. Galactose-1-phosphate uridyl transferase

D. UDP galactose-4-epimerase

See Answer in Part B → Q12

@Neet_pg_bot

Q13: A patient with thiamine deficiency develops lactic acidosis. Which of the following enzymes is affected? (Q855957)

A. Pyruvate carboxylase

B. Pyruvate dehydrogenase

C. Phosphoenolpyruvate carboxykinase

D. Transketolase

See Answer in Part B → Q13

@Neet_pg_bot

Q14: A 2-month-old infant is brought to the pediatrician due to feeding difficulties, vomiting, and failure to thrive. The child's history reveals that the infant was born full-term and had a normal birth weight. On physical examination, the infant appears jaundiced, and the liver is palpable below the costal margin. Oil drop Cataracts are also noted on eye examination. Laboratory tests show elevated levels of galactose in the blood and urine. Based on these clinical findings, which enzyme deficiency is most likely responsible for this presentation? (Q857857)

A. G6PD (Glucose-6-phosphate dehydrogenase)

B. GALT (Galactose-1-phosphate uridylyltransferase)

C. ALDH (Aldehyde dehydrogenase)

D. PKU (Phenylalanine hydroxylase)

See Answer in Part B → Q14

@Neet_pg_bot

Q15: A newborn infant is brought to the clinic with jaundice and a white, punctate, and scattered opacity in the central lens. The condition is consistent with Oil drop cataract. Which of the following metabolic disorders is most likely responsible for this presentation? (Q857861)

A. Phenylketonuria (PKU)

B. Maple syrup urine disease

C. Galactosemia

D. Wilson's disease

See Answer in Part B → Q15

@Neet_pg_bot

Q16: A patient with a history of strenuous exercise forgot to eat a meal and later consumed alcohol at a party. Which of the following metabolic processes is inhibited by alcohol consumption? (Q860060)

A. Gluconeogenesis

B. Glycogenolysis

C. HMP shunt

D. Ketone body formation

See Answer in Part B → Q16

@Neet_pg_bot

Q17: Which of the following is the rate-limiting enzyme of the hexose monophosphate (HMP) pathway? (Q860387)

A. Glucose-6-phosphatase

B. Glucose-6-phosphate dehydrogenase

C. Phosphofructokinase-1

D. Glycogen phosphorylase

See Answer in Part B → Q17

@Neet_pg_bot

Q18: What is the rate-limiting enzyme of cholesterol synthesis? (Q860403)

A. HMG-CoA reductase

B. 7-alpha hydroxylase

C. Carnitine acyltransferase 1

D. Carnitine acyltransferase 2

See Answer in Part B → Q18

@Neet_pg_bot

Q19: Which receptor plays a crucial role in glucose uptake by skeletal muscle? (Q860412)

A. GLUT 1

B. GLUT 2

C. GLUT 3

D. GLUT 4

See Answer in Part B → Q19

@Neet_pg_bot

Q20: A child presents with fatigue and hepatomegaly. Liver enzymes (ALT, AST) are elevated. Ketosis was significant. Liver biopsy shows excess glycogen accumulation. After feeding, blood glucose levels rise, but there is no rise in glucose after overnight fasting. Which of the following enzyme deficiencies is most likely responsible for this presentation? (Q865410)

A. Glucose-6-phosphatase

B. Liver phosphorylase

C. Muscle phosphorylase

D. Phosphofructokinase

See Answer in Part B → Q20

@Neet_pg_bot

Q21: Cancer cells take up excess glucose because? (Q865417)

A. Lactate is produced even in the presence of Oxygen

B. High NADH/NAD ratio

C. High GLUT2

D. Absence of Oxygen

See Answer in Part B → Q21

@Neet_pg_bot

Q22: After a high-carbohydrate meal, weight gain is explained by all except? (Q865420)

A. Malonyl CoA

B. Tricarboxylate transporter

C. Acetyl-CoA carboxylase

D. Mitochondrial carnitine

See Answer in Part B → Q22

@Neet_pg_bot

Q23: When blood glucose concentration is suddenly increased to 2–3 times the normal level and maintained at that level, what will be the typical pattern of blood glucose change observed over time? (Q866337)

A. Rapid rise followed by sustained high glucose levels

B. Continuous increase in insulin secretion without plateau

C. Rise in glucose followed by a fall below baseline

D. Initial rise and then stabilization slightly above baseline

See Answer in Part B → Q23

@Neet_pg_bot

Q24: A breastfed infant presents with lethargy, hepatomegaly, and cataracts. Which of the following enzyme deficiencies is most likely responsible for this presentation? (Q866651)

A. Galactokinase

B. Galactose-1-phosphate uridyltransferase (GALPUT)

C. Aldolase B

D. Fructokinase

See Answer in Part B → Q24

@Neet_pg_bot

Q25: Glycogen phosphorylase is activated by: (Q855065)

A. Calcium

B. Glucose-6-phosphate

C. Insulin

D. Protein phosphatase

See Answer in Part B → Q25

@Neet_pg_bot

Q26: Match the following with the most appropriate feature. 1. Von Gierke's a. Branched structure in liver biopsy 2. Pompe's b. Glycogen deposition in lysosomes 3. McArdle's c. Hypoglycemia with lactic acidosis 4. Cori's d. Exercise intolerance (Q839906)

A. 1c, 2a,3d,4b

B. 1c, 2b,3d,4a

C. 1a, 2c,3d,4b

D. 1d, 2c,3b,4d

See Answer in Part B → Q26

@Neet_pg_bot

Q27: Which of the following deficiency is found in congenital lactic acidosis? (Q839919)

A. Transketolase

B. Branched chain ketoacid dehydrogenase

C. Isocitrate dehydrogenase

D. Pyruvate dehydrogenase

See Answer in Part B → Q27

@Neet_pg_bot

Q28: A patient’s X-ray shows cardiomegaly, and they present with a "floppy baby" syndrome, hypotonia, macroglossia, and hepatomegaly. What is the most likely diagnosis? (Q844238)

A. Pompe disease

B. Ebstein anomaly

C. Down syndrome

D. Cori's Disease

See Answer in Part B → Q28

@Neet_pg_bot

Q29: A diabetic patient is advised to exercise. Which of the following glucose transporters is insulin-sensitive? (Q844249)

A. GLUT4

B. GLUT1

C. GLUT3

D. GLUT2

See Answer in Part B → Q29

@Neet_pg_bot

Q30: A patient with high triglycerides esterified with long-chain fatty acids presents with fatigue, and muscle biopsy shows fat vacuoles. What is the most likely diagnosis? (Q844255)

A. Carnitine deficiency

B. Fatty Acid Synthase defect

C. Lipoprotein Lipase (LPL) deficiency

D. Medium-chain acyl-CoA dehydrogenase (MCAD) deficiency

See Answer in Part B → Q30

@Neet_pg_bot

Q31: A neonate presents with poor feeding and vomiting, cataract, and acidosis with a positive Benedict's test on urine and negative urinary glucose. Which enzyme is likely defective? (Q844276)

A. Galactose-1-phosphate uridyl transferase

B. Aldolase B

C. Fructokinase

D. Glucose-6-phosphatase

See Answer in Part B → Q31

@Neet_pg_bot

Q32: Which of the following is excreted in the urine of patients with pentosuria? (Q852461)

A. L-Xylulose

B. D-Ribose

C. D-Ribulose

D. D-Lactose

See Answer in Part B → Q32

@Neet_pg_bot

Q33: A patient with a history of strenuous exercise skipped a meal and later consumed alcohol at a party. Alcohol inhibits which of the following biochemical processes, potentially leading to hypoglycemia? (Q852549)

A. Gluconeogenesis

B. Glycogenolysis

C. Hexose Monophosphate (HMP) Shunt

D. Ketone Body Formation

See Answer in Part B → Q33

@Neet_pg_bot

Q34: During starvation, which of the following hormones is primarily produced to maintain blood glucose levels? (Q852554)

A. Glucagon

B. Insulin

C. Amylase

D. Somatostatin

See Answer in Part B → Q34

@Neet_pg_bot

Q35: Which of the following organs or tissues demonstrate insulin-dependent glucose uptake ? (Q854604)

A. RBC

B. Pancreas

C. Adipose tissue

D. Hepatocytes

See Answer in Part B → Q35

@Neet_pg_bot

Q36: Glycogen phosphorylase is activated by? (Q854622)

A. Glucose 6 phosphatase

B. ATP

C. Calcium

D. Glucose

See Answer in Part B → Q36

@Neet_pg_bot

Q37: A newborn infant is brought to the clinic with jaundice and a white, punctate, and scattered opacity in the central lens. The condition is consistent with Oil drop cataract. Which of the following metabolic disorders is most likely responsible for this presentation? (Q820353)

A. Phenylketonuria (PKU)

B. Maple syrup urine disease

C. Galactosemia

D. Wilson's disease

See Answer in Part B → Q37

@Neet_pg_bot

Q38: A young boy presents with muscle cramps and dark-colored urine after strenuous exercise. He reports that his symptoms improve after brief rest. Laboratory tests show normal blood glucose levels, no rise in serum lactate after exercise, and elevated creatine kinase. Which of the following is the most likely diagnosis? (Q855193)

A. McArdle disease

B. Hers disease

C. Cori's disease

D. Andersen disease

See Answer in Part B → Q38

@Neet_pg_bot

Q39: A 4-year-old child presents with easy fatiguability. The mother also complains that the child has increased hunger between meals, which is relieved after food. Liver

examination revealed no glycogen. The enzyme most likely deficient is (Q855224)

A. Glycogen phosphorylase

B. Glycogen synthase

C. Debranching enzyme

D. Glucose 6 phosphatase

See Answer in Part B → Q39

@Neet_pg_bot

Q40: Which of the following is seen in low insulin:glucagon ratio? (Q855240)

A. Activation of lipoprotein lipase

B. Activation of glycogen synthase

C. Activation of phosphofructokinase

D. Activation of hormone-sensitive lipase

See Answer in Part B → Q40

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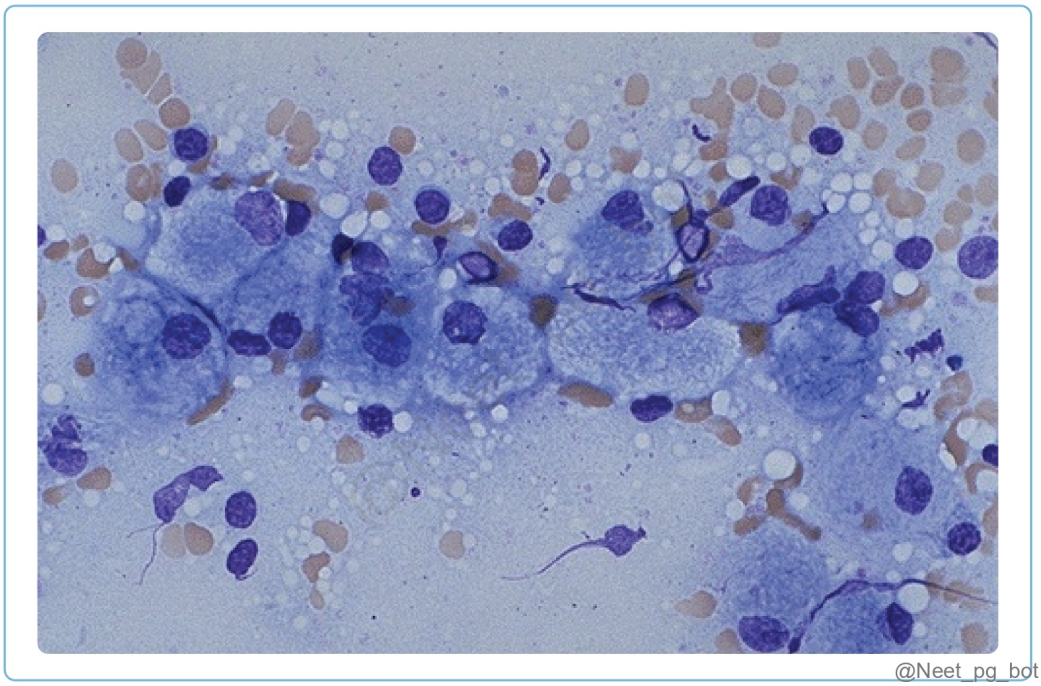
Q41: A child presents with bone pain and hepatosplenomegaly. A trephine biopsy and aspirate shows the following finding. Which of the following is the most likely enzyme deficient in this condition? (Q855278)

A. Hexosaminidase

B. Glucocerebrosidase

C. Sphingomyelinase

D. Alpha 1,4-glucosidase



See Answer in Part B → Q41

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Q42: A child presents with developmental delay, bone pain, and inability to walk. On fundoscopy, a cherry-red spot is observed. There is no organomegaly. What is the most likely diagnosis? (Q855287)

A. Tay-Sachs disease

B. Niemann-Pick disease

C. Hurler syndrome

D. Gaucher disease

See Answer in Part B → Q42

@Neet_pg_bot

Q43: Which enzyme deficiency could potentially be the cause of exercise intolerance and cramps in adolescent male patient? (Q855537)

A. Myophosphorylase

B. Hexokinase

C. Glucose-6-phosphatase

D. Hepatic glycogen phosphorylase

See Answer in Part B → Q43

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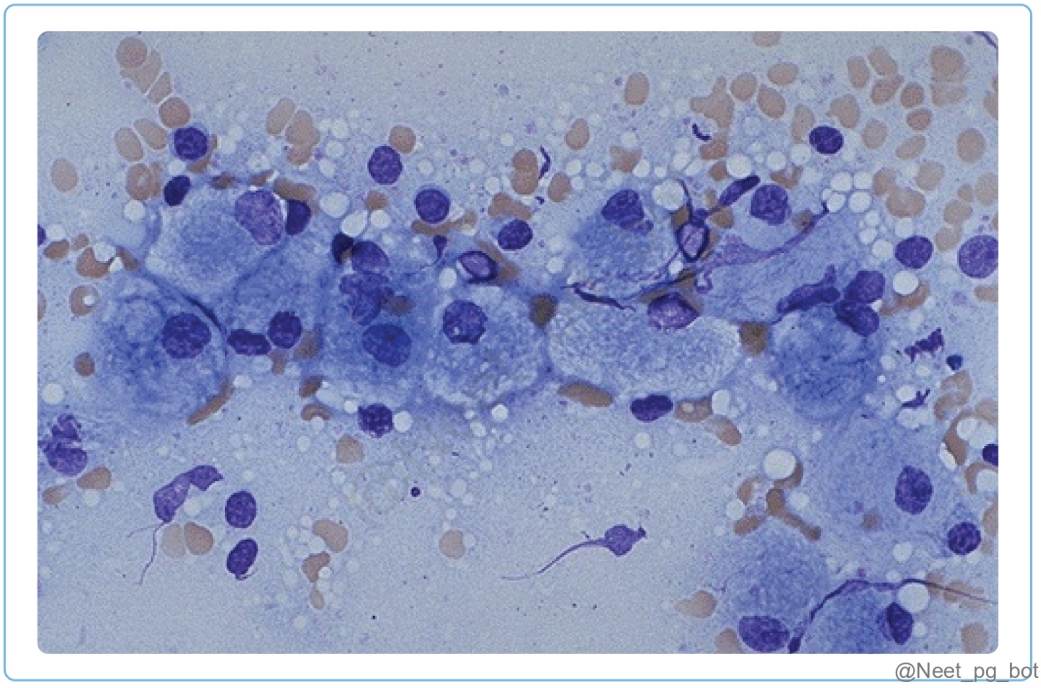
Q44: A baby is being evaluated for delayed developmental milestones. On examination, there is hepatosplenomegaly. Microscopic image of the bone marrow evaluation shown below. What is the diagnosis? (Q855544)

A. Gaucher disease

B. Niemann-pick disease

C. Hunter disease

D. Hurler disease



See Answer in Part B → Q44

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Q45: Which of the following substances inhibits cytochrome complex IV, a component of the electron transport chain responsible for ATP synthesis? (Q855551)

A. Cyanide

B. Carbon dioxide

C. Oligomycin

D. Ouabain

See Answer in Part B → Q45

@Neet_pg_bot

Q46: What are the main substrate and metabolic fuel used by RBCs during fasting? (Q855902)

A. Ketone bodies

B. Glucose

C. Free fatty acids

D. Amino acids

See Answer in Part B → Q46

@Neet_pg_bot

Q47: Which of the following diagnostic markers is /are incorrectly paired? 1. Beta glucocerebrosidase - Von Gierke disease 2. Lipase - Acute pancreatitis 3. Ceruloplasmin - Hepatolenticular degeneration 4. High LDH1: LDH2 ratio - Myocardial infarction (Q855916)

A. 1 only

B. 1 and 2

C. 1, 2 and 4

D. 2, 3 and 4

See Answer in Part B → Q47

@Neet_pg_bot

• [07_ Genetics] 021 Dna And Chromosome Biochemistry Structure Replication And Repair

« Back to Folder Directory

[07_ Genetics] 021 Dna And Chromosome Biochemistry Structure Replication And Repair — Quick Jump

Q1 (p. 272)	*Q2 (p. 272)*	*Q3 (p. 273)*	*Q4 (p. 273)*	*Q5 (p. 273)*	*Q6 (p. 274)*
Q7 (p. 274)	*Q8 (p. 274)*	*Q9 (p. 275)*	*Q10 (p. 275)*	*Q11 (p. 275)*	*Q12 (p. 276)*
Q13 (p. 276)	*Q14 (p. 277)*	*Q15 (p. 277)*	*Q16 (p. 277)*	*Q17 (p. 278)*	*Q18 (p. 278)*
Q19 (p. 279)	*Q20 (p. 279)*	*Q21 (p. 279)*	*Q22 (p. 280)*	*Q23 (p. 280)*	*Q24 (p. 280)*
Q25 (p. 281)	*Q26 (p. 281)*	*Q27 (p. 282)*	*Q28 (p. 282)*		

Q1: Steroid hormone receptors bind to specific sites on DNA by which of the following structures? (Q882301)

A. Zinc finger motif

B. RNA motif

C. Leucine zipper

D. Helix–turn–helix

See Answer in Part B → Q1

@Neet_pg_bot

Q2: Which of the following is not a requirement for DNA replication in eukaryotes? (Q843167)

A. Origin of replication (ori)

B. DNA helicase

C. RNA primer

D. RNA polymerase

See Answer in Part B → Q2

@Neet_pg_bot

Q3: A scientist observes the process of DNA replication in a human stem cell. He takes a sample of double-stranded DNA from the cell and studies it separately. He finds that the total amount of adenine in the sample is 24%. From the given information, what percentage of cytosine is present in the sample? (Q843584)

A. 52%

B. 24%

C. 30%

D. 26%

See Answer in Part B → Q3

@Neet_pg_bot

Q4: What is the wavelength at which nucleic acids show maximum absorbance of UV light? (Q843583)

A. 260 nm

B. 280 nm

C. 400 nm

D. 340 nm

See Answer in Part B → Q4

@Neet_pg_bot

Q5: What sequence is repeated in human telomeres? (Q843398)

A. 5'-TTAAGG-3'

B. 5'-GGTACC-3'

C. 5'-TTAGGG-3'

D. 5'-AGCTAG-3'

See Answer in Part B → Q5

@Neet_pg_bot

Q6: Which of the following is not caused by a defective NER mechanism? (Q843184)

A. Xeroderma pigmentosum (XP)

B. Cockayne syndrome (CS)

C. Trichothiodystrophy (TTD)

D. Werner syndrome (WS)

See Answer in Part B → Q6

@Neet_pg_bot

Q7: What is the role of UvrABC exinuclease in nucleotide excision repair (NER)? (Q843182)

A. It fills in the gap with new nucleotides

B. It recognizes and excises damaged DNA segments.

C. It seals the DNA strand after repair.

D. It unwinds the DNA double helix.

See Answer in Part B → Q7

@Neet_pg_bot

Q8: Mismatch repair defects are mostly associated with which of the following? (Q843180)

A. Hereditary Nonpolyposis Colon Cancer (HNPCC)

B. Familial Adenomatous Polyposis (FAP)

C. Li-Fraumeni syndrome

D. Retinoblastoma

See Answer in Part B → Q8

@Neet_pg_bot

Q9: Why are methylases important in the mismatch repair mechanism in prokaryotes like E. coli? (Q843178)

A. They add methyl groups to the newly synthesized DNA strand, marking it for repair.

B. They methylate the parental DNA strand, allowing the Mut proteins to distinguish it from the daughter strand.

C. They remove mismatched bases from the DNA strand.

D. They act as exonucleases during the repair process.

See Answer in Part B → Q9

@Neet_pg_bot

Q10: Which of the following enzymes helps remove the defective base during base excision repair (BER)? (Q843175)

A. DNA polymerase I

B. DNA ligase

C. DNA glycosylase

D. AP endonuclease

See Answer in Part B → Q10

@Neet_pg_bot

Q11: A 6-year-old child presents with recurrent respiratory infections and poor growth despite a normal diet. Genetic testing reveals Cystic fibrosis. What type of mutation causes cystic fibrosis? (Q843174)

A. Phenotypic heterogeneity

B. Pleiotropy

C. Allelic heterogeneity

D. Heteroplasmy

See Answer in Part B → Q11

@Neet_pg_bot

Q12: Which of the following statements accurately describes the role of DNA polymerase δ (Pol δ) in eukaryotic cells? (Q843172)

A. It synthesizes RNA primers necessary for DNA replication.

B. It is responsible for leading-strand DNA synthesis.

C. It requires a primer and provides proofreading during lagging-strand synthesis.

D. It directly repairs damaged DNA by filling in gaps.

See Answer in Part B → Q12

@Neet_pg_bot

Q13: Which of the following statements about telomeres and telomerase is correct? (Q843170)

A. Telomeres prevent chromosome ends from degrading but do not shorten during cell division.

B. Telomerase adds repetitive DNA sequences to the 5′ end of chromosomes

C. Telomeres are composed of repetitive sequences that help prevent chromosome fusion.

D. The shortening of telomeres occurs due to the continuous synthesis of the lagging strand.

See Answer in Part B → Q13

@Neet_pg_bot

Q14: Which of the following statements about DNA polymerases in E. coli is true? (Q843168)

A. DNA Polymerase I is the primary enzyme for DNA synthesis at the replication fork.

B. DNA Polymerase II is primarily responsible for gap filling following DNA replication

C. DNA Polymerase III has the highest rate of chain elongation and is involved in both leading and lagging strand synthesis.

D. All three DNA polymerases perform identical functions in DNA synthesis.

See Answer in Part B → Q14

@Neet_pg_bot

Q15: Which of the following disorders is primarily caused by a defect in the RecQ DNA helicase (BLM protein), leading to a profound deficiency in homologous recombination (HR) and characterized by increased sister chromatid exchanges? (Q842887)

A. Bloom syndrome

B. Ataxia telengectasia

C. LIG4 syndrome

D. Artemis-SCID

See Answer in Part B → Q15

@Neet_pg_bot

Q16: Which of the following is not a requirement for the activity of DNA polymerase during DNA replication? (Q843165)

A. A primer with a free 3′-OH group

B. A single-stranded DNA template

C. Free ribonucleoside triphosphates (rNTPs)

D. A divalent cation cofactor, typically Mg^{2}■

See Answer in Part B → Q16

@Neet_pg_bot

Q17: A 45 y/o woman presents with visual disturbances, including gradual vision loss and difficulty seeing in low light, along with muscle weakness and fatigue. She has a history of early-onset cardiac conduction issues and worsening symptoms over the years. Her family history shows similar symptoms in her mother. What genetic condition best explains her symptoms? (Q843164)

A. Leber’s Hereditary Optic Neuropathy (LHON)

B. Myoclonic Epilepsy with Ragged Red Fibers (MERRF)

C. Mitochondrial Encephalopathy, Lactic Acidosis, and Stroke-like Episodes (MELAS)

D. Leigh’s Syndrome.

See Answer in Part B → Q17

@Neet_pg_bot

Q18: Which of the following is a characteristic of mitochondrial DNA (mtDNA)? (Q843162)

A. It is linear and single-stranded

B. It has very few non-coding regions and a higher mutation rate compared to nuclear DNA.

C. It uses the standard genetic code, including AGA and AGG as codons for arginine (Arg).

D. It is composed of linear strands and encodes for all 67 subunits of the respiratory chain.

See Answer in Part B → Q18

@Neet_pg_bot

Q19: Which histone is least tightly bound to chromatin and can be easily removed with a salt solution? (Q843161)

A. H2A

B. H1

C. H3

D. H4

See Answer in Part B → Q19

@Neet_pg_bot

Q20: Which of the following factors can cause the denaturation of DNA? a) Increasing temperature b) Increasing salt concentration c) Decreasing temperature d) DNA with a high A-T base composition e) DNA with a high G-C base composition f) Adding Urea g) Adding Formamide h) Decreasing salt concentration (Q843160)

A. b, c, e, g, h

B. c, d, e, h

C. a, d, f, g, h

D. a, b, e, g

See Answer in Part B → Q20

@Neet_pg_bot

Q21: Which of the following statements about the different types of DNA is TRUE? (Q843159)

A. B-DNA has a left-handed helix and a full turn with 12 bases.

B. A-DNA forms during dehydration and has grooves of the same dimensions.

C. Z-DNA forms under normal physiological conditions and has a right-handed helix

D. A-DNA is the predominant form found in physiological conditions and has 11 bases per full turn.

See Answer in Part B → Q21

@Neet_pg_bot

Q22: In the analogy of the DNA double helix to a spiral staircase, what do the "handrails" and "steps" represent? (Q843157)

A. Handrails are nitrogenous bases; steps are phosphodiester bonds

B. Handrails are nitrogenous bases; steps are the sugar-phosphate backbone

C. Handrails are the sugar-phosphate backbone; steps are nitrogenous bases

D. Handrails are hydrogen bonds; steps are deoxyribose sugars

See Answer in Part B → Q22

@Neet_pg_bot

Q23: In the structure of nucleotides, where does the esterification of the phosphate group typically occur? (Q843154)

A. On the 1' carbon of the pentose sugar

B. On the 2' hydroxyl group of the pentose sugar

C. On the 5' or 3' hydroxyl group of the pentose sugar

D. On the N1 position of the nitrogenous base

See Answer in Part B → Q23

@Neet_pg_bot

Q24: What is the primary role of inosine monophosphate (IMP) in cellular metabolism? (Q843153)

A. Directly involved in energy transfer like ATP

B. Acts as a precursor for both adenosine monophosphate (AMP) and guanosine monophosphate (GMP)

C. Inhibits the de novo synthesis of purine nucleotides

D. Serves as an end product of the salvage pathway for purine nucleotides

See Answer in Part B → Q24

@Neet_pg_bot

Q25: What type of bond forms between a nitrogenous base and a pentose sugar in a nucleoside? (Q843151)

A. α-N-glycosidic bond

B. β-N-glycosidic bond

C. Phosphodiester bond

D. Hydrogen bond

See Answer in Part B → Q25

@Neet_pg_bot

Q26: Which of the following is a pyrimidine base found in nucleic acids? (Q843147)

A. Uracil

B. Hypoxanthine

C. Xanthine

D. Guanosine

See Answer in Part B → Q26

@Neet_pg_bot

Q27: Which of the following is not caused by a defect of ds DNA repair? (Q842892)

A. Cockayne syndrome (CS)

B. Fanconi’s anemia

C. Severe Combined Immunodeficiency (SCID)

D. Bloom's syndrome

See Answer in Part B → Q27

@Neet_pg_bot

Q28: Which of the following statements about Homologous DNA Repair (HR) is correct? (Q842890)

A. HR is active during G0/G1 phases of the cell cycle.

B. HR is an error-prone process compared to nonhomologous end joining (NHEJ).

C. HR uses a homologous DNA template for repair, making it highly accurate.

D. Defects in HR-related proteins decrease Ovarian and breast cancers

See Answer in Part B → Q28

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• [07_ Genetics] 022 Rna Transcription Genetic Code Mutation Translation Inhibitors

« Back to Folder Directory

[07_ Genetics] 022 Rna Transcription Genetic Code Mutation Translation Inhibitors — Quick Jump

Q1 (p. 283)	*Q2 (p. 283)*	*Q3 (p. 284)*	*Q4 (p. 284)*	*Q5 (p. 284)*	*Q6 (p. 285)*
Q7 (p. 285)	*Q8 (p. 285)*	*Q9 (p. 286)*	*Q10 (p. 286)*	*Q11 (p. 286)*	*Q12 (p. 287)*
Q13 (p. 287)	*Q14 (p. 288)*	*Q15 (p. 288)*	*Q16 (p. 288)*	*Q17 (p. 289)*	*Q18 (p. 289)*
Q19 (p. 289)	*Q20 (p. 290)*	*Q21 (p. 291)*	*Q22 (p. 291)*	*Q23 (p. 291)*	*Q24 (p. 292)*
Q25 (p. 292)	*Q26 (p. 293)*	*Q27 (p. 293)*	*Q28 (p. 293)*		

Q1: Which of the RNA Polymerase is responsible for synthesis of rRNA? (Q843574)

A. RNA polymerase I

B. RNA polymerase II

C. RNA polymerase III

D. RNA polymerase IV

See Answer in Part B → Q1

@Neet_pg_bot

Q2: Original DNA sequence: 5'-ATG GCA TCG GAA UAC-3'. Mutated sequence: 5'-ATG GCA TCG GAA UAA-3'. Identify the mutation. (Q843045)

A. Frameshift mutation

B. Nonsense mutation

C. Silent mutation

D. Missense mutation

See Answer in Part B → Q2

@Neet_pg_bot

Q3: Match the following phases of translation (Column A) with the events involved (Column B). Column A Column B 1. Elongation a. RF1 binds with RF3 and GTP 2. Initiation b. Binding of aminoacyl-tRNA to the ribosome's A site 3. Termination c. Methionine tRNA scans mRNA for AUG codon (Q843048)

A. 1-b, 2-c, 3-a

B. 1-c, 2-a, 3-b

C. 1-b, 2-a, 3-c

D. 1-a, 2-c, 3-b

See Answer in Part B → Q3

@Neet_pg_bot

Q4: Which of the following best describes the DNA segment from which the primary mRNA transcript is copied or transcribed? (Q876005)

A. Coding strand

B. Initiator methionine domain

C. Template strand

D. Translational Unit

See Answer in Part B → Q4

@Neet_pg_bot

Q5: Which of the following statements are false? (Q843580)

A. Ribosome is an example of ribozyme

B. Both ribozymes and protein enzymes exhibit specificity

C. Ribozymes exhibit greater structural complexity compared to protein enzymes

D. rRNA is directly involved in catalyzing the formation of peptide bonds

See Answer in Part B → Q5

@Neet_pg_bot

Q6: Which of the following belong to the group of Small non-coding RNAs? (Q843579)

A. mRNA

B. tRNA

C. rRNA

D. None of the above

See Answer in Part B → Q6

@Neet_pg_bot

Q7: Which of the following accurately describes the role of snRNAs in the spliceosome? (Q843578)

A. snRNAs bind to the poly-A tail of mRNA to stabilize the mature transcript.

B. snRNAs form ribonucleoprotein complexes with proteins to recognize and excise introns.

C. snRNAs bind to the 5' cap of mRNA to initiate translation.

D. snRNAs transport pre-mRNA from the nucleus to the cytoplasm for translation.

See Answer in Part B → Q7

@Neet_pg_bot

Q8: Which of the following statements accurately describes a difference between eukaryotic and prokaryotic transcription? (Q843577)

A. Eukaryotic transcription occurs in the cytoplasm.

B. Eukaryotic transcription uses multiple RNA polymerases.

C. Eukaryotic transcription lacks chromatin remodeling.

D. Eukaryotic transcription does not involve mRNA processing.

See Answer in Part B → Q8

@Neet_pg_bot

Q9: What is the primary role of the 7-methylguanosine cap added to the 5' end of eukaryotic mRNA? (Q843576)

A. To signal the termination of transcription

B. To aid in the transport of mRNA into the mitochondria

C. To protect the mRNA from exonucleases and facilitate ribosome recognition

D. To mark the mRNA for immediate degradation

See Answer in Part B → Q9

@Neet_pg_bot

Q10: Which of the following elements is typically found within the promoter region of a eukaryotic gene and is crucial for the initiation of transcription? (Q843575)

A. Pribnow box

B. TATA box

C. CAAT box

D. GC Box

See Answer in Part B → Q10

@Neet_pg_bot

Q11: Which technique is commonly used for DNA sequencing to identify mutations by determining the precise order of nucleotides in a DNA sample? (Q843150)

A. Fluorescence in situ hybridization (FISH)

B. Multiplex ligation-dependent probe amplification (MLPA)

C. Sanger technique

D. Single Strand Conformational Polymorphism (SSCP)

See Answer in Part B → Q11

@Neet_pg_bot

Q12: Which class of antibiotics inhibits bacterial protein synthesis by blocking the attachment of aminoacyl-tRNA to the ribosomal A site? (Q843149)

A. Tetracyclines

B. Chloramphenicol

C. Macrolides

D. Puromycin

See Answer in Part B → Q12

@Neet_pg_bot

Q13: What is the total number of ATPs required for activating amino acids by aminoacyl tRNA synthetase? (Q843148)

A. 3

B. 2

C. 4

D. 1

See Answer in Part B → Q13

@Neet_pg_bot

Q14: Which of the following statements is true about aminoacyl tRNA? (Q843146)

A. Attaches to the A site of 60s ribosomes

B. Needs 3 high-energy phosphates for attachment

C. It is guided by eIF2C

D. None of the above

See Answer in Part B → Q14

@Neet_pg_bot

Q15: Which of the following is NOT TRUE regarding the initiation of translation? (Q843145)

A. eIF3 and 1A stabilize 60s ribosomal subunit to prevent reassociation with 40s.

B. Methionine tRNA scans mRNA to identify the initiation AUG codon

C. ATP dependent helicase eIF4A/4B breaks hydrogen bonds to remove the hairpin loops

D. Association of mRNA with the 43S preinitiation complex forms the 48S initiation complex

See Answer in Part B → Q15

@Neet_pg_bot

Q16: Which of the following statements accurately reflects a difference between DNA and RNA, considering their roles in genetic information processing and stability? (Q843028)

A. RNA's 2'-hydroxyl group enhances its stability compared to DNA.

B. RNA polymerases need a primer; DNA polymerases do not.

C. RNA forms complex structures; DNA remains a double helix

D. DNA degrades more rapidly than RNA due to the presence of a 2'-hydroxyl group.

See Answer in Part B → Q16

@Neet_pg_bot

Q17: Sickle cell anaemia is suspected in a 25-year-old patient who presents with episodes of abdominal pain and fatigue. Genetic analysis reveals GAG in the 6th position is replaced with GTG. Which type of mutation here results in haemoglobin that is still functional but altered? (Q843047)

A. Unacceptable mutation

B. Partially acceptable mutation

C. Acceptable mutation

D. Nonsense mutation

See Answer in Part B → Q17

@Neet_pg_bot

Q18: A DNA sequence originally reads 5'-ATG GCA TCG TAC-3'. After a mutation, it reads 5'-ATG GCT CGT AC-3'. Identify the mutation that has occured? (Q843043)

A. Frameshift mutation

B. Point mutation

C. Transition mutation

D. Insertion mutation

See Answer in Part B → Q18

@Neet_pg_bot

Q19: Identify the type of mutation illustrated in the following DNA sequences? Starting sequence: Mutated sequence: (Q843039)

A. Point mutation

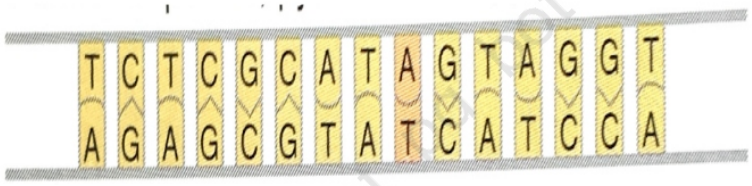
B. Frameshift mutation

C. Deletion mutation

D. Insertion mutation



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See Answer in Part B → Q19

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Q20: Which of the following statements are true regarding codons? A, G, T, C are RNA nucleotides A, G, T, C are DNA nucleotides A, G, U, C are RNA nucleotides A, G, U, C are DNA nucleotides (Q843037)

A. 1 and 3 are correct

B. 2 and 3 are correct

C. 1 and 4 are correct

D. 2 and 4 are correct

See Answer in Part B → Q20

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Q21: A 23-year-old medical student is studying molecular biology. She learns that a single tRNA molecule can recognize more than one codon for the same amino acid, because base-pairing at the third codon position is less stringent. Which of the following best explains this phenomenon? (Q843036)

A. Inosine forming non-standard base pairs at the third codon position.

B. The exact matching of all three nucleotides between codon and anticodon.

C. Single tRNA binds only to one specific codon.

D. The rigid Watson-Crick base pairing at the third position of the codon.

See Answer in Part B → Q21

@Neet_pg_bot

Q22: In the context of genetic code degeneracy, which of the following accurately describes its impact on protein synthesis? (Q843035)

A. Single codon specifies one amino acid.

B. Multiple codons can code for the same amino acid.

C. Flexibility occurs at the 2nd codon position.

D. Inosine in mRNA contributes to the flexibility in pairing.

See Answer in Part B → Q22

@Neet_pg_bot

Q23: Which of the following best describes the primary mechanisms and applications of RNA interference (RNAi)? (Q843034)

A. Degradation of mRNA; used for gene manipulation and therapy.

B. Enhancement of mRNA synthesis; used for genetic modifications.

C. Conversion of mRNA to DNA; used for protein structure studies.

D. Stabilisation of mRNA; used for treating infections.

See Answer in Part B → Q23

@Neet_pg_bot

Q24: A 25-year-old student is reviewing the roles of small RNAs in cellular processes. She learns about small nuclear RNAs (snRNAs) and small interfering RNAs (siRNAs). Which of the following statements about snRNAs and/or siRNAs is true? (Q843033)

A. Both snRNAs and siRNAs are involved in the degradation of mRNA.

B. snRNAs form complexes with the RNA-induced silencing complex (RISC).

C. snRNAs are primarily involved in mRNA splicing and are located in the nucleus.

D. snRNAs are typically found in the cytoplasm, while siRNAs are found in the nucleus.

See Answer in Part B → Q24

@Neet_pg_bot

Q25: Which of the following statements is true regarding the synthesis of mRNA? (Q843032)

A. Introns are the coding sequence in a mRNA

B. 7-methyl guanosine gets added to the 3’ end of mRNA

C. Process of splicing occurs in the cytoplasm

D. Heteronuclear RNA is not a functional mRNA

See Answer in Part B → Q25

@Neet_pg_bot

Q26: Which of the following steps in the process of tRNA synthesis occurs in the cytoplasm? (Q843031)

A. Rearrangement of glycosidic linkages giving rise to pseudouridine

B. Deamination creating inosine

C. Fourth post-transcriptional modification of random methylation

D. Aminoacyl-tRNA synthetases attach the amino acid

See Answer in Part B → Q26

@Neet_pg_bot

Q27: Which statement best describes the wobble hypothesis in relation to tRNA's function? (Q843030)

A. Allows multiple tRNAs to bind to a single codon.

B. Explains tRNA's ability to bind to any codon with perfect complementarity.

C. Single tRNA recognizes multiple codons due to flexible base pairing.

D. Accounts for varying tRNA lengths matching different mRNA sequences.

See Answer in Part B → Q27

@Neet_pg_bot

Q28: Which of the following types of rRNA is associated with the 40S ribosomal subunit in eukaryotic cells? (Q843029)

A. 5S rRNA

B. 18S rRNA

C. 28S rRNA

D. 16S rRNA

See Answer in Part B → Q28

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• [07_ Genetics] 023 Pcr

« Back to Folder Directory

[07_ Genetics] 023 Pcr — Quick Jump

Q1 (p. 295) *Q2 (p. 295)* *Q3 (p. 295)* *Q4 (p. 296)* *Q5 (p. 296)* *Q6 (p. 297)*
Q7 (p. 297) *Q8 (p. 297)* *Q9 (p. 298)* *Q10 (p. 298)* *Q11 (p. 298)*

Q1: In PCR cloning, the use of proofreading DNA polymerases is often preferred because: (Q843241)

A. They amplify DNA at a much faster rate than regular Taq polymerase

B. They have a lower error rate during DNA synthesis

C. They can amplify longer DNA fragments more efficiently

D. They are more tolerant to variations in PCR cycling conditions

See Answer in Part B → Q1

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Q2: Which of the following steps is NOT part of the standard PCR cloning process? (Q843242)

A. Denaturation

B. Annealing

C. Ligation

D. Elongation

See Answer in Part B → Q2

@Neet_pg_bot

Q3: Which of the following applications is a typical use of Polymerase Chain Reaction (PCR)? (Q843243)

A. Identifying specific genetic mutations

B. Determining protein structure

C. Measuring cell growth rates

D. Analyzing metabolite levels

See Answer in Part B → Q3

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Q4: What is the primary purpose of Reverse Transcription Polymerase Chain Reaction (RT-PCR) in molecular biology? (Q843244)

A. To amplify DNA sequences directly from the genome.

B. To convert RNA into complementary DNA (cDNA) and then amplify specific DNA sequences.

C. To sequence the entire genome of an organism.

D. To separate DNA fragments by size.

See Answer in Part B → Q4

@Neet_pg_bot

Q5: What is the primary advantage of using real-time PCR (qPCR) over traditional PCR techniques? (Q843245)

A. It allows for the detection of multiple targets in a single reaction.

B. It enables the quantification of DNA as the amplification progresses.

C. It eliminates the need for a thermal cycler.

D. It provides a higher specificity in amplifying DNA.

See Answer in Part B → Q5

@Neet_pg_bot

Q6: Which of the following best describes the main principle behind Sanger sequencing? (Q843597)

A. Amplification of DNA segments to facilitate sequencing.

B. Incorporation of dideoxynucleotides to terminate DNA synthesis at specific bases.

C. High-throughput sequencing of millions of DNA fragments simultaneously.

D. Insertion of a DNA fragment into a plasmid vector for replication and sequencing.

See Answer in Part B → Q6

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Q7: A 45-year-old patient diagnosed with COVID-19 has a real-time PCR test result showing a cycle threshold (Ct) value of 35. How should this Ct value be interpreted in the context of their infectivity and clinical management? (Q843598)

A. The patient has a high viral load and is highly infectious.

B. The patient has a low viral load and is less likely to be infectious.

C. The patient's Ct value indicates they should be immediately admitted to the ICU.

D. The Ct value alone is sufficient to determine the patient's disease severity and infectivity.

See Answer in Part B → Q7

@Neet_pg_bot

Q8: A forensic scientist is analyzing a DNA sample from a crime scene. The DNA is digested with restriction enzymes, separated by gel electrophoresis, and transferred to a nitrocellulose membrane. Labeled DNA probes are then used to detect specific fragments. Which technique is the scientist most likely using? (Q843599)

A. Restriction Fragment Length Polymorphism (RFLP)

B. Polymerase Chain Reaction (PCR)

C. Single-Strand Conformation Polymorphism (SSCP)

D. DNA Sequencing

See Answer in Part B → Q8

@Neet_pg_bot

Q9: Which of the following statements about Denaturing Gradient Gel Electrophoresis (DGGE) is false? (Q843600)

A. DGGE can separate DNA fragments based on their melting characteristics.

B. DGGE is effective for analyzing DNA segments of 200-300 base pairs.

C. DGGE requires the use of a denaturant gradient within a polyacrylamide gel.

D. DGGE is commonly used for detecting translocations.

See Answer in Part B → Q9

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Q10: Which of the following methods is specifically designed to detect differences in DNA sequences based on variations in the electrophoretic mobility of single-stranded DNA fragments? (Q843602)

A. RFLP (Restriction Fragment Length Polymorphism)

B. SSCP (Single-Strand Conformational Polymorphism)

C. Sequencing

D. All of the above methods are equally effective for detecting DNA sequence variations.

See Answer in Part B → Q10

@Neet_pg_bot

Q11: A researcher is experiencing issues with the accuracy and efficiency of DNA amplification during a PCR experiment. They suspect the problem might be related to magnesium ion concentration. What role does magnesium play in PCR, and how might its concentration affect the experiment? (Q843603)

A. Magnesium ions serve only to stabilize the DNA template and do not affect enzyme activity.

B. Excessive magnesium concentration enhances DNA polymerase activity, reducing the likelihood of errors.

C. Insufficient magnesium concentration can lead to decreased enzyme activity and lower DNA amplification efficiency.

D. Magnesium ions are not essential for the nucleophilic attack of the primer's 3' hydroxyl group during DNA synthesis.

See Answer in Part B → Q11

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• [07_ Genetics] 024 Advanced Molecular Techniques Recombinant Dna Technology Microarray Crispr Lac Operon

« Back to Folder Directory

[07_ Genetics] 024 Advanced Molecular Techniques Recombinant Dna Technology Microarray Crispr Lac Operon — Quick Jump

Q1 (p. 300)	*Q2 (p. 300)*	*Q3 (p. 301)*	*Q4 (p. 301)*	*Q5 (p. 301)*	*Q6 (p. 302)*
Q7 (p. 303)	*Q8 (p. 303)*	*Q9 (p. 303)*	*Q10 (p. 304)*	*Q11 (p. 304)*	*Q12 (p. 304)*
Q13 (p. 305)	*Q14 (p. 305)*	*Q15 (p. 306)*	*Q16 (p. 306)*	*Q17 (p. 307)*	*Q18 (p. 307)*

Q1: Which of the following best describes a mechanism involved in epigenetic changes that lead to heterochromatin formation? (Q843426)

A. Histone acetylation and DNA methylation of adenine residues

B. Histone deacetylation and methylation in CG islands

C. DNA replication

D. Histone phosphorylation

See Answer in Part B → Q1

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Q2: Which of the following statements about the lac operon is/are correct? Lac I is constitutively expressed. Lac Y is responsible for lactose transport into the cell. The structural genes are expressed when the CAP site is free. The ZYA genes are expressed when the operator site is free. (Q843437)

A. 1 and 2

B. 1, 2, and 3

C. 1, 2, and 4

D. 1, 2, 3, and 4

See Answer in Part B → Q2

@Neet_pg_bot

Q3: Which of the following statements about cloning vectors is correct? Plasmids can carry DNA inserts up to 10 kilobases in size. Phage vectors can carry DNA inserts up to approximately 50 kilobases. Cosmids are hybrid vectors that combine features of plasmids and phages. Artificial chromosomes are used to clone very large DNA fragments. (Q843435)

A. 1, 2, and 3

B. 1 and 4

C. All of the above

D. 1, 3, and 4

See Answer in Part B → Q3

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Q4: In their CRISPR-Cas9 experiment with a mammalian cell line, the research team observes the repair of DNA double-strand breaks. Which repair mechanisms are they most likely observing? Non-Homologous End Joining (NHEJ) Homologous End Joining (HEJ) Nucleotide Excision Repair (NER) Base Excision Repair (BER) (Q843432)

A. 1 and 2

B. 1, 2, 3

C. 1 and 4

D. All of the above

See Answer in Part B → Q4

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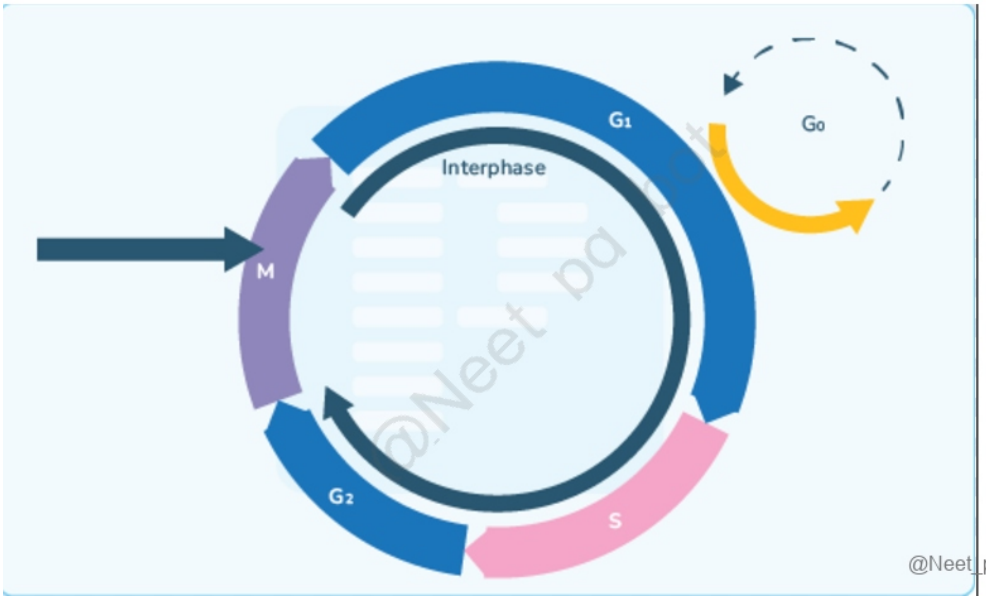
Q5: Which of the following is the feature of the ‘M’ phase in the image of the Cell cycle? (Q843431)

A. DNA replication occurs to duplicate the genome.

B. Chromosomes are highly condensed to facilitate segregation.

C. Cell growth and preparation for DNA synthesis take place.

D. Proofreading and repair of DNA replication errors are conducted.



See Answer in Part B → Q5

@Neet_pg_bot

Q6: A 32-year-old patient presents with a family history of progressive muscle weakness, cognitive decline, and involuntary movements. Genetic testing reveals a specific type of microsatellite instability disorder. Which of the following trinucleotide repeat sequences is most likely associated? (Q843430)

A. CAG

B. CTG

C. CGG

D. GAA

See Answer in Part B → Q6

@Neet_pg_bot

Q7: A 5-year-old boy presents with frequent falls, difficulty climbing stairs, and an unusual way of getting up from the floor. Genetic testing reveals a mutation in a gene associated with a severe muscle disorder. Which of the following statements about the involved gene is true? (Q843429)

A. Located on chromosome 7

B. Largest in the human genome.

C. Associated with a condition that primarily affects females

D. The Gene’s product is a component of the sarcomere.

See Answer in Part B → Q7

@Neet_pg_bot

Q8: Satellite DNA has various applications due to its unique repetitive sequences. Which of the following is not an application of satellite DNA? (Q843428)

A. Forensic identification and criminal investigations

B. Determining genetic susceptibility to cardiovascular diseases

C. Resolving paternal dispute cases

D. Studying evolutionary relationships and genetic diversity

See Answer in Part B → Q8

@Neet_pg_bot

Q9: Which of the following statements is not accurate regarding post-transcriptional modifications of eukaryotic RNA? (Q843427)

A. The addition of a poly-A tail at the 3′ end.

B. The 5′ capping involves a 5′ to 5′ triphosphate bridge.

C. Methylation of the N6 position of adenine.

D. Introns are removed, and exons are spliced within the cytoplasm.

See Answer in Part B → Q9

@Neet_pg_bot

Q10: A researcher aims to clone a large eukaryotic gene fragment (~600 kb) for use in functional genomics studies. Which of the following cloning vectors is the most appropriate for this purpose? (Q843416)

A. Plasmid

B. Bacteriophage λ

C. Yeast Artificial Chromosome (YAC)

D. Cosmid

See Answer in Part B → Q10

@Neet_pg_bot

Q11: In human somatic cells, which of the following statements accurately describes the chromatin and its characteristics? (Q843425)

A. Constitutive heterochromatin is always active

B. Facultative heterochromatin includes the Barr body

C. Euchromatin is found in centromeric regions

D. Heterochromatin is active and less stained

See Answer in Part B → Q11

@Neet_pg_bot

Q12: In a laboratory setting, E. coli bacteria are cultured in a medium where glucose and lactose is available. To increase the expression of the lac operon, which levels of lactose and cAMP would you expect to be present? (Q843424)

A. High lactose and low cAMP

B. Low lactose and high cAMP

C. High lactose and high cAMP

D. Low lactose and low cAMP

See Answer in Part B → Q12

@Neet_pg_bot

Q13: Which of the following techniques is specifically designed to introduce specific genetic modifications, such as gene knock-ins or knock-outs, into the genome of an organism? (Q843423)

A. FISH (Fluorescence In Situ Hybridization)

B. CRISPR-Cas

C. Recombinant DNA Technology

D. Comparative Genomic Hybridization

See Answer in Part B → Q13

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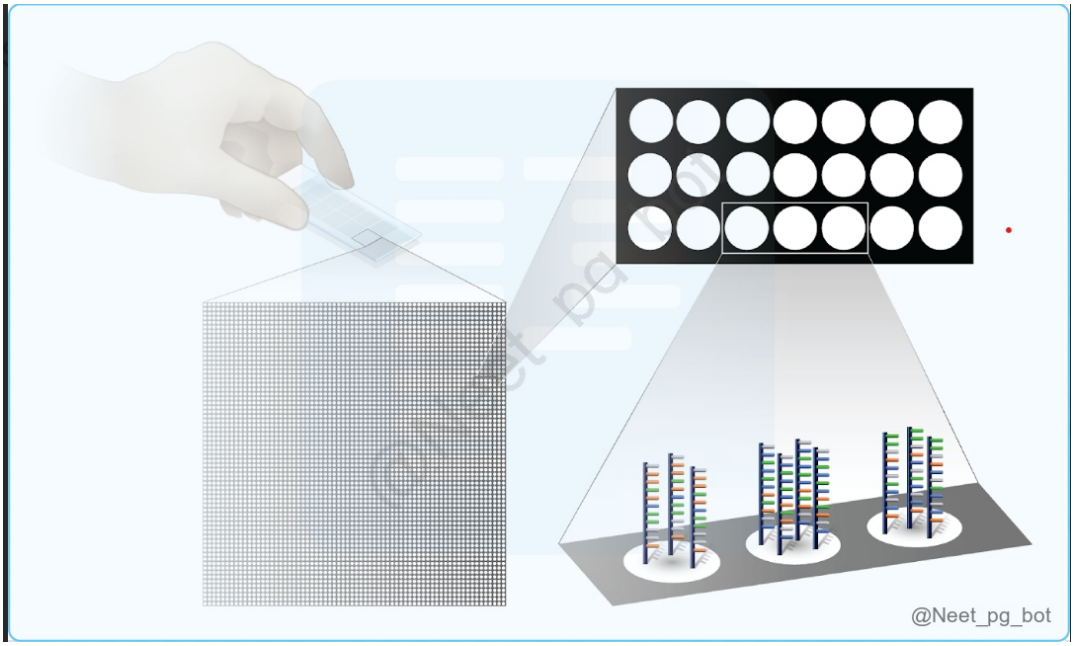
Q14: Which of the following is true about the significance of technology in the following image? {{caption_text}} (Q843422)

A. Used to study multiple gene expressions simultaneously

B. Not used to deciding the course of treatment

C. They have been completely replaced by next-generation sequencing.

D. Not used in proteomics and genotyping.



See Answer in Part B → Q14

@Neet_pg_bot

Q15: Which of the following vectors is most suitable for cloning large DNA fragments up to 300 kb in size? (Q843421)

A. Plasmids

B. Bacteriophages

C. Bacterial Artificial Chromosomes (BACs)

D. Yeast Artificial Chromosomes (YACs)

See Answer in Part B → Q15

@Neet_pg_bot

Q16: Which type of blotting technique is used to detect specific RNA sequences within a sample? (Q843420)

A. Southern Blotting

B. Northern Blotting

C. Western Blotting

D. Eastern blotting

See Answer in Part B → Q16

@Neet_pg_bot

Q17: Which type of restriction endonuclease creates staggered cuts in the DNA, producing "sticky ends"? (Q843418)

A. Type I restriction enzymes

B. Type II restriction enzymes

C. Type III restriction enzymes

D. Type IV restriction enzymes

See Answer in Part B → Q17

@Neet_pg_bot

Q18: Which of the following represents the correct sequence of steps in recombinant DNA technology? (Q843417)

A. Isolation of DNA → Insertion of recombinant DNA into host → Ligation → Cutting of DNA with restriction enzymes → Selection and screening.

B. Cutting of DNA with restriction enzymes → Ligation → Isolation of DNA → Insertion of recombinant DNA into host → Selection and screening.

C. Isolation of DNA → Cutting of DNA with restriction enzymes → Ligation → Insertion of recombinant DNA into host → Selection and screening.

D. Ligation → Isolation of DNA → Cutting of DNA with restriction enzymes → Insertion of recombinant DNA into host → Selection and screening.

See Answer in Part B → Q18

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• [07_ Genetics] 025 Previous Year Questions

« Back to Folder Directory

[07_ Genetics] 025 Previous Year Questions — Quick Jump

Q1 (p. 308)	*Q2 (p. 308)*	*Q3 (p. 309)*	*Q4 (p. 309)*	*Q5 (p. 309)*	*Q6 (p. 310)*
Q7 (p. 310)	*Q8 (p. 310)*	*Q9 (p. 311)*	*Q10 (p. 311)*	*Q11 (p. 311)*	*Q12 (p. 312)*
Q13 (p. 312)	*Q14 (p. 313)*	*Q15 (p. 314)*	*Q16 (p. 314)*	*Q17 (p. 315)*	*Q18 (p. 315)*
Q19 (p. 315)	*Q20 (p. 316)*	*Q21 (p. 316)*	*Q22 (p. 316)*	*Q23 (p. 317)*	*Q24 (p. 317)*
Q25 (p. 318)	*Q26 (p. 318)*	*Q27 (p. 318)*	*Q28 (p. 319)*	*Q29 (p. 319)*	*Q30 (p. 319)*
Q31 (p. 320)	*Q32 (p. 320)*	*Q33 (p. 321)*	*Q34 (p. 321)*	*Q35 (p. 321)*	*Q36 (p. 322)*
Q37 (p. 322)	*Q38 (p. 323)*	*Q39 (p. 323)*	*Q40 (p. 323)*	*Q41 (p. 324)*	*Q42 (p. 324)*
Q43 (p. 324)	*Q44 (p. 325)*	*Q45 (p. 325)*	*Q46 (p. 325)*	*Q47 (p. 326)*	*Q48 (p. 326)*
Q49 (p. 327)	*Q50 (p. 327)*	*Q51 (p. 327)*	*Q52 (p. 328)*	*Q53 (p. 328)*	*Q54 (p. 328)*
Q55 (p. 329)	*Q56 (p. 329)*	*Q57 (p. 329)*	*Q58 (p. 330)*	*Q59 (p. 330)*	*Q60 (p. 331)*
Q61 (p. 331)	*Q62 (p. 332)*	*Q63 (p. 332)*	*Q64 (p. 333)*		

Q1: Ascent to high altitude causes ? (Q865438)

A. Metabolic acidosis

B. Metabolic alkalosis

C. Respiratory acidosis

D. Respiratory alkalosis

See Answer in Part B → Q1

@Neet_pg_bot

Q2: A patient who has been diagnosed with multiple gastrointestinal polyps and carcinoma, with a positive family history/history of hereditary non-polyposis colon carcinoma has a defect in which of the following? (Q855998)

A. Mismatch repair

B. Nucleotide excision repair

C. Base excision repair

D. Point mutation

See Answer in Part B → Q2

@Neet_pg_bot

Q3: DNA methylation is not related to which of the following processes? (Q856919)

A. Mismatch repair

B. Chromatin remodeling

C. Gene silencing

D. DNA replication

See Answer in Part B → Q3

@Neet_pg_bot

Q4: Klenow fragment lacks the activity of? (Q857029)

A. 3’→5’ Exonuclease

B. 5’→3’ Exonuclease

C. 5’→3’ DNA polymerase

D. 3’→5’ DNA polymerase

See Answer in Part B → Q4

@Neet_pg_bot

Q5: DNA methylation is not related to which of the following processes? (Q856487)

A. Mismatch repair

B. Chromatin remodeling

C. DNA replication

D. Gene silencing

See Answer in Part B → Q5

@Neet_pg_bot

Q6: The correct order of steps in polymerase chain reaction (PCR) is:- (Q856494)

A. Hybridization, annealing, elongation, denaturation

B. Elongation, denaturation, Hybridization, annealing,

C. Denaturation, annealing, extension.

D. Annealing, extension, denaturation

See Answer in Part B → Q6

@Neet_pg_bot

Q7: Which type of DNA repair mechanism involves the removal and replacement of a damaged DNA strand with the help of an undamaged complementary strand as a template? (Q856514)

A. Base Excision Repair

B. Nucleotide Excision Repair

C. Mismatch Repair

D. Homologous Recombination

See Answer in Part B → Q7

@Neet_pg_bot

Q8: If the percentage of thymine residues in DNA is 28%. What is the % of cytosine? (Q858457)

A. 28%

B. 44%

C. 36%

D. 22%

See Answer in Part B → Q8

@Neet_pg_bot

Q9: DNA packing is done by which of the following? (Q855513)

A. Histone

B. Glycoprotein

C. Nucleus acid

D. Adenine

See Answer in Part B → Q9

@Neet_pg_bot

Q10: Which of the subsequent disorders, exhibit only autosomal recessive inheritance pattern? (Q855527)

A. Huntington’s disease

B. Treacher collins syndrome

C. Cystic fibrosis

D. Achondroplasia

See Answer in Part B → Q10

@Neet_pg_bot

Q11: What is the term for a single mutation in a nucleotide base pair that leads to formation of termination codon? (Q855533)

A. Missense mutation

B. Nonsense mutation

C. Stop mutation

D. Silent mutation

See Answer in Part B → Q11

@Neet_pg_bot

Q12: A 5-year-old child is brought to the clinic with abdominal pain, irritability, and developmental delay. On examination, the child has pallor and a bluish line on the gums. Laboratory tests reveal microcytic anemia, and a peripheral blood smear shows red blood cells with basophilic stippling. Which of the following is the most likely diagnosis? (Q866662)

A. Iron deficiency anemia

B. Sideroblastic anemia

C. Lead poisoning

D. Thalassemia minor

See Answer in Part B → Q12

@Neet_pg_bot

Q13: In a village of Assam, a man is observed walking with the help of a stick and has genu valgum. Many people in the same village present with similar symptoms. What is the most probable cause of this condition? (Q866664)

A. Calcium deficiency

B. Vitamin C deficiency

C. Skeletal fluorosis

D. Vitamin D deficiency (Osteomalacia)



See Answer in Part B → Q13

@Neet_pg_bot

Q14: Which of the following substances, at the given concentration, makes water unsuitable for human consumption according to safety standards? (Q866677)

A. Fluoride – 0.8 mg/L

B. Cadmium – 0.3 mg/L

C. Chloride - 200 mg/L

D. Calcium – 7 mg/L

See Answer in Part B → Q14

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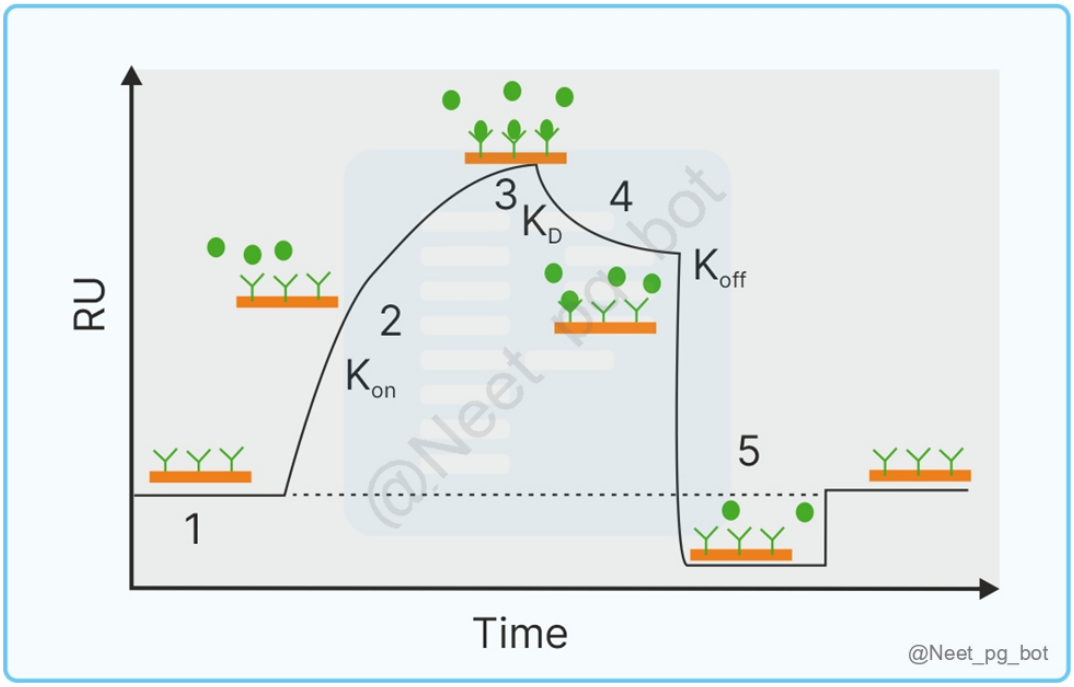
Q15: Name the graph and mention its application. (Q865396)

A. ELISA, Antibody concentration

B. ELISA, Antibody affinity

C. Plasma resonance, Ab concentration

D. Plasma resonance, Ab affinity



See Answer in Part B → Q15

@Neet_pg_bot

Q16: A child presents with hepatomegaly and a cherry-red spot on the macula. Neurological regression is also noted. These findings are suggestive of a lysosomal storage disorder. Which of the following enzyme deficiencies is most likely responsible? (Q865412)

A. Arylsulfatase A

B. Hexosaminidase A

C. Sphingomyelinase

D. Galactocerebrosidase

See Answer in Part B → Q16

@Neet_pg_bot

Q17: Wobble hypothesis explains which of the following concepts of the genetic code? (Q856607)

A. Universal

B. Unambiguous

C. Degeneracy

D. Non-punctuated

See Answer in Part B → Q17

@Neet_pg_bot

Q18: Cyanide and carbon monoxide act on which of the heme proteins: (Q865454)

A. Cytochrome c oxidase

B. Myoglobin

C. Catalase

D. NADH dehydrogenase

See Answer in Part B → Q18

@Neet_pg_bot

Q19: If you need to diagnose a syndromic illness such as meningitis, which of the following diagnostic techniques is most appropriate? (Q866328)

A. Multiplex PCR

B. Uniplex PCR

C. Attributed PCR

D. Nested PCR

See Answer in Part B → Q19

@Neet_pg_bot

Q20: A patient presents with a history of vomiting. Arterial blood gas analysis reveals the following: pH : 7.5 pCO■ : 48 mm Hg HCO■■ : 30 mm Hg What is the most likely acid-base abnormality? (Q852561)

A. Metabolic alkalosis

B. Metabolic acidosis

C. Respiratory acidosis

D. Respiratory alkalosis

See Answer in Part B → Q20

@Neet_pg_bot

Q21: The development of cataracts in patients with Diabetes Mellitus is primarily due to the accumulation of which of the following substances in the lens? (Q852565)

A. Sorbitol

B. Dulcitol

C. Mannitol

D. Galactitol

See Answer in Part B → Q21

@Neet_pg_bot

Q22: Which of the following enzymes is typically elevated in prostate cancer? (Q852562)

A. Acid phosphatase

B. Alkaline phosphatase

C. Lactate dehydrogenase

D. Alpha-fetoprotein

See Answer in Part B → Q22

@Neet_pg_bot

Q23: Which test should be done for detecting metabolite levels in inborn errors of metabolism in neonates? (Q839926)

A. Tandem mass spectroscopy

B. Complete blood count (CBC)

C. Urinalysis

D. Genetic testing

See Answer in Part B → Q23

@Neet_pg_bot

Q24: What is the order of the band in the sarcomere? (Q854616)

A. I-Z-M-A-H

B. Z-H-M-A-I

C. Z-I-A-H-M

D. Z-H-I-A-M

See Answer in Part B → Q24

@Neet_pg_bot

Q25: Nitric oxide is synthesized from (Q859428)

A. L-arginine

B. L-citrulline

C. Glycine

D. Lysine

See Answer in Part B → Q25

@Neet_pg_bot

Q26: A child with mental retardation, blindness, and muscle weakness was investigated and was found to be deficient for Hexosaminidase-A enzyme. She is most likely suffering from .. (Q859572)

A. Krabbe's disease

B. Fabry's disease

C. Tay-sachs disease

D. Gaucher's disease

See Answer in Part B → Q26

@Neet_pg_bot

Q27: In a 14-year-old male, a medical condition has been identified as a result of a defect in a gene responsible for DNA repair. The individual is experiencing increased pigmentation on the skin and is at a heightened risk of developing skin cancers, according to the physician. Which of the following mechanisms is most likely to be impaired in this particular condition? (Q859573)

A. Nonhomologous end joining repair defect

B. Nucleotide excision repair defect

C. DNA base excision repair defect

D. DNA mismatch repair defect

See Answer in Part B → Q27

@Neet_pg_bot

Q28: Nitrogenous waste is excreted from the body in the form of? (Q859710)

A. Urea

B. Nitric oxide

C. Ammonia

D. Glutamine

See Answer in Part B → Q28

@Neet_pg_bot

Q29: A scientist treats experimental cells with a histone acetylase inhibitor. Which of the following is the most immediate epigenetic change of this treatment? (Q879279)

A. Increased expression of H3K9me2/3

B. Increased acetylation of H3K9ac

C. Chromatin condensation

D. DNA methylation

See Answer in Part B → Q29

@Neet_pg_bot

Q30: In rheumatoid arthritis, synovial tissue from affected patients exhibits elevated expression of TNF-α and IL-6, which contribute to the inflammatory process. Which

epigenetic modification most likely accounts for this increased gene expression? (Q879280)

A. Activation of DNA methylation

B. Inhibition of DNA methylation

C. Activation of histone deacetylation

D. Inhibition of histone acetylation

See Answer in Part B → Q30

@Neet_pg_bot

Q31: Cancer cells exhibit an increased length of a particular RNA transcript or fragment compared to normal cells. The most likely explanation for this is: (Q879281)

A. Polyadenylation

B. Alternative RNA splicing

C. Gene amplification

D. Gene silencing

See Answer in Part B → Q31

@Neet_pg_bot

Q32: During B-cell development in the bone marrow, each B cell expresses only one immunoglobulin heavy chain allele to ensure that all antibodies produced by that cell possess a single antigen specificity. This process, known as allelic exclusion, is primarily mediated by: (Q879282)

A. V(D)J rearrangements

B. Alternate splicing of RNA transcripts

C. Alternate polyadenylation sites

D. Somatic hypermutation

See Answer in Part B → Q32

@Neet_pg_bot

Q33: Which type of mutation results in the reversal to the wild type of phenotype when the mutant gene is suppressed? (Q839921)

A. Frameshift of the mutatedgene

B. Addition of another normal gene

C. Deletion of mutanted gene

D. Mutation in tRNA

See Answer in Part B → Q33

@Neet_pg_bot

Q34: Which of the following enzymes is responsible for the immortality of cancer cells? (Q866654)

A. Telomerase

B. Topoisomerase

C. Helicase

D. RNA polymerase

See Answer in Part B → Q34

@Neet_pg_bot

Q35: Which of the following genetic mutations is most commonly associated with familial cases of Amyotrophic Lateral Sclerosis (ALS)? (Q866660)

A. CFTR gene mutation

B. SOD1 gene mutation

C. HTT gene mutation

D. FMR1 gene mutation

See Answer in Part B → Q35

@Neet_pg_bot

Q36: A woman with a strong family history of breast and ovarian cancer is being evaluated for BRCA1/BRCA2 mutations. Which of the following is the most appropriate test to detect multiple mutations at different codons in these genes? (Q865400)

A. qPCR - Measures BRCA1/2 expression levels

B. RT-PCR - Detects RNA-level mutations

C. Northern blot - Analyzes RNA expression

D. Next-generation sequencing (NGS)

See Answer in Part B → Q36

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Q37: Which of the following techniques are correctly matched with their uses: (Q865401)

A. Southern blot - RNA Identification, Northern blot - DNA Identification, Western blot - Protein Identification

B. Southern blot - DNA Identification, Northern blot - Protein Identification, Western blot - RNA Identification

C. Southern blot - Protein Identification, Northern blot - RNA Identification, Western blot - DNA Identification

D. Southern blot - DNA Identification, Northern blot - RNA Identification, Western blot - Protein Identification

See Answer in Part B → Q37

@Neet_pg_bot

Q38: After how many cycles of PCR, are we expected to have half the number of copies as found after 26 cycles? (Q865403)

A. 27

B. 13

C. 25

D. 35

See Answer in Part B → Q38

@Neet_pg_bot

Q39: Cancer cells consume more glucose by which of the following process: (Q865442)

A. Increased GLUT-2 transporters, oxidative phosphorylation

B. Increased conversion of pyruvate to Acetyl CoA

C. Increased lactate is produced even in the presence of oxygen

D. Increased fatty acid oxidation

See Answer in Part B → Q39

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Q40: Gene pool refers to all the alleles in a: (Q865448)

A. Family

B. Individual

C. Population

D. Tissue

See Answer in Part B → Q40

@Neet_pg_bot

Q41: A frameshift mutation occurs due to the insertion of a new nucleotide at the 4th position of an mRNA sequence with 900 nucleotides. What is the most likely outcome of this mutation? (Q866327)

A. No change in the final protein

B. Partial protein production

C. Complete change in protein production

D. No change due to RNA editing

See Answer in Part B → Q41

@Neet_pg_bot

Q42: A patient presents with skin cancer and hyperpigmentation that worsens with sunlight exposure. Which of the following DNA repair mechanisms is most likely defective in this condition? (Q866329)

A. Nucleotide excision repair

B. Base excision repair

C. Non-homologous end joining (NHEJ)

D. Mismatch repair

See Answer in Part B → Q42

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Q43: A 22-year-old tall male presents with long limbs, increased arm span, hypermobile joints, and a high-arched palate. On examination, he has lens subluxation and a diastolic murmur suggestive of aortic root dilation. Which of the following genes is most likely mutated in this condition? (Q866331)

A. Fibrillin-1 (FBN1)

B. COL4A5

C. COL1A1

D. Elastin

See Answer in Part B → Q43

@Neet_pg_bot

Q44: Which of the following is used in diagnosing aneuploidy? 1. FISH 2. Cytogenetics 3. Sangers 4. PCR (Q853000)

A. 1,2

B. 1,2,3

C. 2,3

D. 1,2,3,4

See Answer in Part B → Q44

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Q45: Enzymes used in recombinant DNA technology are: 1. Isomerase 2. Phosphatase 3. Terminal transferase. 4. CRISPR CASS (Q853001)

A. 1,2,3,4

B. 1,2,3

C. 2,3,4

D. 1,3,4

See Answer in Part B → Q45

@Neet_pg_bot

Q46: A child presents with micrognathia and low-set ears. These clinical features are commonly associated with which type of genetic abnormality? (Q852566)

A. Deletion

B. Translocation

C. Amplification

D. Inversion

See Answer in Part B → Q46

@Neet_pg_bot

Q47: A mother brings her 14-year-old mentally retarded son to the clinic, complaining of joint pain and an irresistible urge for the boy to bite his fingers and lips. Serum uric acid is elevated. What is the most likely diagnosis? (Q844280)

A. Lesch-Nyhan syndrome

B. Gout

C. Hyperuricemia

D. Juvenile arthritis

See Answer in Part B → Q47

@Neet_pg_bot

Q48: Which of the following is not associated with post-transcriptional modification? (Q839917)

A. Methylation

B. Endonuclease cleavage

C. 5' capping

D. Glycosylation

See Answer in Part B → Q48

@Neet_pg_bot

Q49: A patient presents with multiple colonic polyps and has been diagnosed with colorectal carcinoma. There is a strong family history of Hereditary Non-Polyposis Colorectal Cancer (HNPCC). Which DNA repair mechanism is most likely defective in this condition ? (Q866643)

A. Nucleotide excision repair

B. Mismatch repair

C. Base excision repair

D. Double-strand break repair

See Answer in Part B → Q49

@Neet_pg_bot

Q50: FISH technique helps to identify all except?? (Q839943)

A. Point mutation

B. Translocation

C. Deletion

D. Gene amplification

See Answer in Part B → Q50

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Q51: Which of the following is true about p43? (Q857848)

A. It increases telomere length in the liver

B. It synthesizes RNA

C. It stabilizes RNA

D. It retains the telomerase enzyme in the nucleus

See Answer in Part B → Q51

@Neet_pg_bot

Q52: Which is the correct sequence of steps in isolating desirable protein using recombinant DNA technology? 1. Expression of protein and lysis of the bacterial cell 2. Incorporation of genes into bacteria 3. SDS PAGE 4. Protein elution 5. Hybridization (Q855531)

A. 2, 1, 3, 5, 4

B. 1, 2, 4, 3, 5

C. 1, 5, 2, 4, 3

D. 2, 4, 5, 3, 1

See Answer in Part B → Q52

@Neet_pg_bot

Q53: Affinity maturation of antibodies is because of (Q855564)

A. Somatic hypermutation

B. Differential mRNA processing

C. CD40

D. Gene rearrangements

See Answer in Part B → Q53

@Neet_pg_bot

Q54: Which of the following techniques is used to detect DNA protein interaction? (Q855047)

A. DNA footprinting

B. DNA fingerprinting

C. ELISA

D. Northern Blotting

See Answer in Part B → Q54

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Q55: Misincorporation of uracil into DNA is seen in deficiency of (Q855066)

A. Vitamin B12

B. Folate

C. Vitamin B6

D. Vitamin E

See Answer in Part B → Q55

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Q56: What is the pattern of inheritance for hemophilia A? (Q856955)

A. X linked recessive

B. X linked Dominant

C. Autosomal dominant

D. Autosomal recessive

See Answer in Part B → Q56

@Neet_pg_bot

Q57: Which of the following enzymes is deficient in the autosomal recessive type of severe combined immunodeficiency? (Q856971)

A. Ornithine transcarbamylase

B. Phosphomannose isomerase

C. Adenosine deaminase

D. Hypoxanthine-guanine phosphoribosyl transferase

See Answer in Part B → Q57

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Q58: Which of the following molecular analysis methods is used to detect RNA? (Q855892)

A. Sanger’s method

B. G banding

C. Western blot

D. RT-PCR

See Answer in Part B → Q58

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Q59: Which of the following is the best test for quantitative analysis of an RNA sample? PCR: polymerase chain reaction RT PCR: Reverse transcriptase polymerase chain reaction (Q855940)

A. Real-time PCR

B. Reverse transcriptase PCR

C. PCR-restriction fragment length polymorphism

D. RTPCR followed by real-time PCR

See Answer in Part B → Q59

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Q60: Which of the following statements are true regarding mitochondrial DNA? Higher rate of mutations as compared to the nuclear genome Double-stranded circular DNA with about 16,000 base pairs A slight change in nucleotide sequence as compared to cellular DNA Encodes all electron transport chain proteins (Q855945)

A. 1 and 3

B. 1 and 2

C. 2 and 4

D. 2 and 3

See Answer in Part B → Q60

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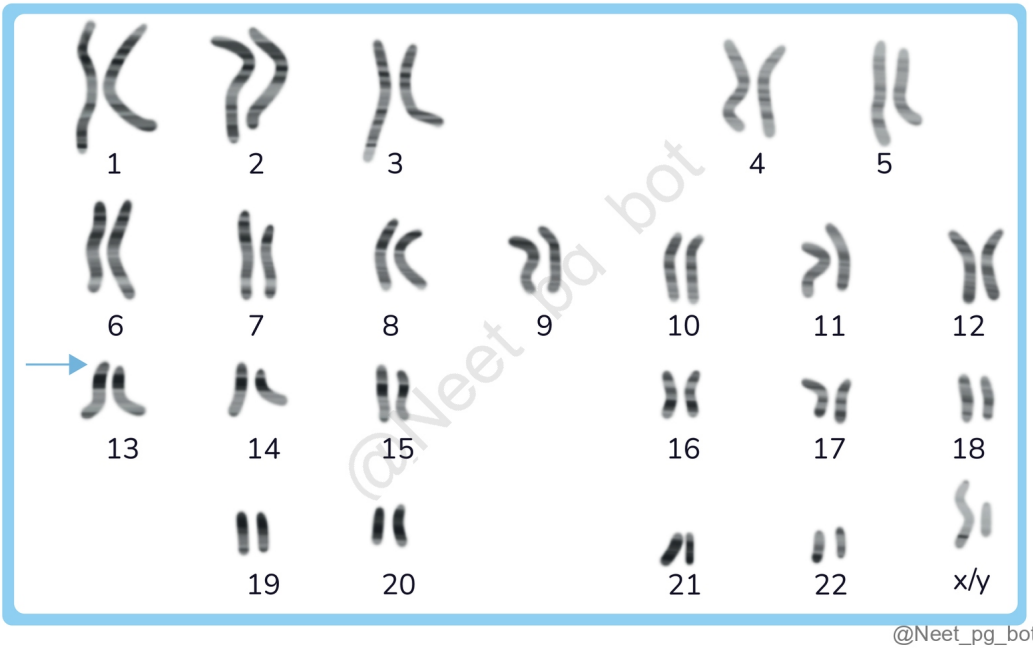
Q61: Identify the type of marked chromosome in the given karyotype. (Q856566)

A. Acrocentric

B. Submetacentric

C. Metacentric

D. Telocentric



See Answer in Part B → Q61

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Q62: How does RNAi impact gene expression? (Q856571)

A. Knock out

B. Knockdown

C. Knock up

D. Knock in

See Answer in Part B → Q62

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Q63: miRNA binds to which part of the mRNA to inhibit translation? (Q856579)

A. Gene body

B. 3'UTR

C. Gene promoter

D. 5'UTR

See Answer in Part B → Q63

@Neet_pg_bot

Q64: What is the repair mechanism associated with CRISPR-cas9? (Q856605)

A. Non-homologous end joining

B. Base excision repair

C. Nucleotide excision repair

D. Mismatch repair

See Answer in Part B → Q64

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• [08_Lipid Metabolism] 026 Previous Year Questions

« Back to Folder Directory

[08_Lipid Metabolism] 026 Previous Year Questions — Quick Jump

Q1 (p. 334)	*Q2 (p. 334)*	*Q3 (p. 335)*	*Q4 (p. 335)*	*Q5 (p. 335)*	*Q6 (p. 336)*
Q7 (p. 336)	*Q8 (p. 336)*	*Q9 (p. 337)*	*Q10 (p. 337)*	*Q11 (p. 337)*	*Q12 (p. 338)*
Q13 (p. 338)	*Q14 (p. 339)*	*Q15 (p. 340)*	*Q16 (p. 340)*	*Q17 (p. 340)*	*Q18 (p. 341)*
Q19 (p. 341)	*Q20 (p. 341)*				

Q1: The enzyme that is not used in cholesterol synthesis is: (Q856879)

A. Thiolase

B. HMG CoA reductase

C. HMG CoA synthase

D. HMG CoA lyase

See Answer in Part B → Q1

@Neet_pg_bot

Q2: A 6-month-old infant is brought to the emergency department with lethargy, vomiting, and poor feeding. The episode occurred after prolonged fasting. Laboratory results reveal hypoglycemia, low ketone levels (hypoketosis), and mild hepatomegaly. These findings suggest a defect in fat metabolism. Which of the following is the most likely underlying disorder? (Q866670)

A. Her’s disease

B. Medium-chain acyl-CoA dehydrogenase (MCAD) deficiency

C. Hereditary fructose intolerance

D. Glycogen storage disease type I (Von Gierke disease)

See Answer in Part B → Q2

@Neet_pg_bot

Q3: A patient presents with elevated total cholesterol, subcutaneous xanthomas, and a positive family history of similar findings. Triglyceride levels are normal (<140 mg/dL). What is the most likely type of familial dyslipidemia? (Q866333)

A. Type I

B. Type IIa

C. Type IIb

D. Type II

See Answer in Part B → Q3

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Q4: A patient presents with orange-colored tonsils. Laboratory investigations reveal triglyceride level of 140 mg/dL and HDL cholesterol of 5 mg/dL. What is the most likely diagnosis? (Q866332)

A. Familial hypercholesterolemia

B. Tangier disease

C. Type I hyperlipoproteinemia

D. Abetalipoproteinemia

See Answer in Part B → Q4

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Q5: Hormone-sensitive lipase is activated by all of the following EXCEPT? (Q857872)

A. Glucagon

B. Epinephrine

C. ACTH

D. Insulin

See Answer in Part B → Q5

@Neet_pg_bot

Q6: A 5-year-old girl is brought to the clinic by her mother due to the abnormalities in the genital area. On examination, there is the presence of pubic and axillary hair, indicating early androgen effects. Which of the following enzyme deficiencies is the least likely cause of her symptoms? (Q857557)

A. 17α-hydroxylase deficiency

B. 21-hydroxylase deficiency

C. 11β-hydroxylase deficiency

D. 3β-hydroxy steroid dehydrogenase deficiency

See Answer in Part B → Q6

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Q7: Enzyme deficient in Gaucher disease : (Q857482)

A. Betagalacto cerebrosidase

B. Glucose-6-phosphate

C. Betaglucocerebrosidase

D. Alpha glucocerebrosidase

See Answer in Part B → Q7

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Q8: What is the primary source of cholesterol synthesis? (Q856980)

A. Malonyl-CoA

B. Pyruvate

C. Oxaloacetate

D. Acetyl-CoA

See Answer in Part B → Q8

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Q9: Which of the cycles listed below is absent in muscle tissue? (Q856932)

A. Glycogenolysis

B. Ketogenesis

C. Glycogenesis

D. Lipolysis

See Answer in Part B → Q9

@Neet_pg_bot

Q10: What is the primary source of reducing equivalents for liver fatty acid synthesis? (Q856924)

A. Hexose monophosphate shunt pathway

B. Glycolysis

C. Tricarboxylic acid cycle

D. Uronic acid pathway

See Answer in Part B → Q10

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Q11: Which gene defect causes familial hypercholesterolemia? (Q839947)

A. Apo E

B. Apo C II

C. LDL receptor

D. Apo B48

See Answer in Part B → Q11

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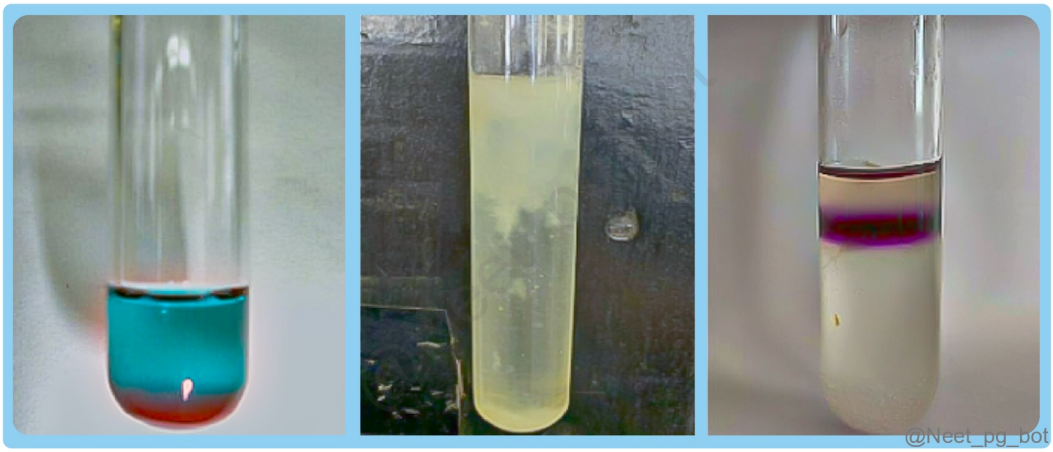
Q12: Which of the following tests will be positive in the urine of a patient who has undergone prolonged starvation? (Q855964)

A. 1 and 3

B. Only 3

C. 1, 2 and 3

D. 1 and 2



See Answer in Part B → Q12

@Neet_pg_bot

Q13: Insulin inhibits which of the following lipase enzymes? (Q855896)

A. Lipoprotein lipase

B. Pancreatic lipase

C. Gastric lipase

D. Hormone sensitive lipase

See Answer in Part B → Q13

@Neet_pg_bot

Q14: A patient presented with reduced levels of high-density lipoprotein and an ABCA1 mutation. On examination, tonsils appeared as shown in the image. What is the diagnosis? (Q855549)

A. Tangier's disease

B. Abetalipoproteinemia

C. Hyperlipoproteinemia

D. Wolman's disease



See Answer in Part B → Q14

@Neet_pg_bot

Q15: What is the enzyme defect if a child with anemia, thrombocytopenia, bony pain and hepatosplenomegaly shows a "crumpled tissue paper" appearance on light microscopy of bone marrow aspirate? (Q855548)

A. Beta- glucosidase

B. Sphingomyelinase

C. Hexosaminidase

D. Glucose6-phosphatase

See Answer in Part B → Q15

@Neet_pg_bot

Q16: Which of the options aids in the transportation of fatty acids through the inner mitochondria membrane? (Q855530)

A. Acyl carrier protein

B. Carnitine

C. Lecithin-Cholesterol acyltransferase

D. Albumin

See Answer in Part B → Q16

@Neet_pg_bot

Q17: The process of apolipoprotein B48 synthesis involves which of the following? (Q854877)

A. RNA alternate splicing

B. RNA editing

C. DNA editing

D. RNA interference

See Answer in Part B → Q17

@Neet_pg_bot

Q18: A patient with multiple tendon xanthomas is found to have a serum cholesterol level of 398 mg/dL and an LDL level of 220 mg/dL. What is the most likely defect? (Q844263)

A. Lipoprotein lipase deficiency

B. LDL receptor defect

C. Defective Apo B-100

D. PCSK9 gain-of-function mutation

See Answer in Part B → Q18

@Neet_pg_bot

Q19: A patient presents to the emergency room with severe abdominal pain, and serum triglyceride levels are 1500 mg/dL. What is the most likely underlying defect? (Q844261)

A. Apo B-48 defect

B. Apo B-100 defect

C. Apo C-II defect

D. LDL receptor defect

See Answer in Part B → Q19

@Neet_pg_bot

Q20: A patient presents with xanthomas on the Achilles tendon. Which of the following is the most likely diagnosis? (Q844257)

A. Familial hypercholesterolemia

B. Tangier disease

C. Familial hyperchylomicronemia

D. Type III hyperlipoproteinemia

See Answer in Part B → Q20

@Neet_pg_bot

• [09_Amino Acids Metabolism] 027 Previous Year Questions

« Back to Folder Directory

[09_Amino Acids Metabolism] 027 Previous Year Questions — Quick Jump

Q1 (p. 343)	*Q2 (p. 343)*	*Q3 (p. 344)*	*Q4 (p. 344)*	*Q5 (p. 345)*	*Q6 (p. 345)*
Q7 (p. 346)	*Q8 (p. 346)*	*Q9 (p. 346)*	*Q10 (p. 347)*	*Q11 (p. 348)*	*Q12 (p. 348)*
Q13 (p. 348)	*Q14 (p. 349)*	*Q15 (p. 349)*	*Q16 (p. 349)*	*Q17 (p. 350)*	*Q18 (p. 350)*
Q19 (p. 350)	*Q20 (p. 351)*	*Q21 (p. 351)*	*Q22 (p. 352)*		

Q1: Ammonia in the brain is detoxified to form (Q856874)

A. Urea

B. Glutamine

C. Ammonium ions

D. Alanine

See Answer in Part B → Q1

@Neet_pg_bot

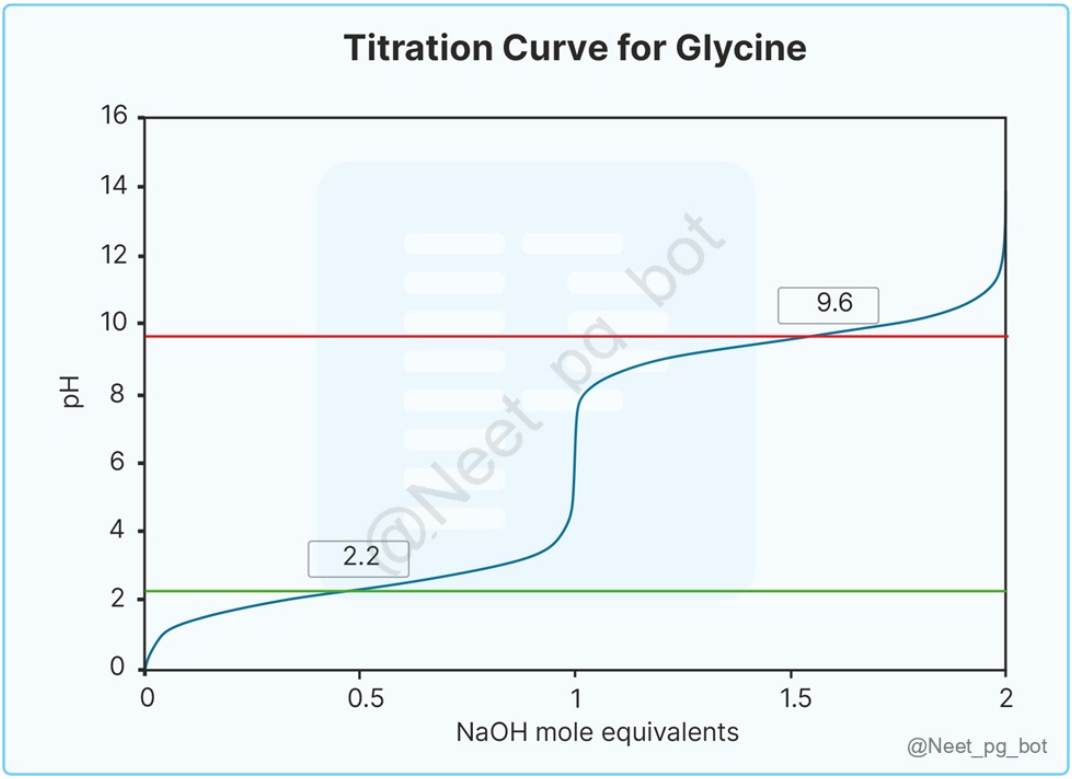
Q2: In the given glycine titration curve, calculate or identify the pH at which molecules exist in a zwitter ion form? (Q865428)

A. 4.4

B. 6

C. 9

D. 2.2



See Answer in Part B → Q2

@Neet_pg_bot

Q3: All of the following are lipotropic factors except? (Q859698)

A. Choline

B. Lecithine

C. Arginine

D. Methionine

See Answer in Part B → Q3

@Neet_pg_bot

Q4: A patient came to the hospital with a complaint of abdominal pain. Routine investigations were unremarkable. On observation, the urine turned black on standing. Which of the following enzymes is likely to be defective in this patient?

(Q858459)

A. Homogentisate oxidase

B. Xanthine oxidase

C. Phenylalanine hydroxylase

D. Dihydric orotate dehydrogenase

See Answer in Part B → Q4

@Neet_pg_bot

Q5: A 62-year-old male with CAD comes to the physician for a follow-up examination. His blood pressure is 130/80 mm Hg, height of 182 cm, weight of 85 kg, and a BMI of 25 kg/m². Serum studies show a glucose concentration of 95 mg/dL and homocysteine concentration of 19.3 μmol/L (N=5–15). Serum lipid concentrations are within the normal range. Which of the following enzyme deficiencies is most likely to be found in this patient? (Q857892)

A. Homogentisate oxidase

B. Cystathionine beta synthase

C. Cystathionine gamma synthase

D. HGPRTase

See Answer in Part B → Q5

@Neet_pg_bot

Q6: Which enzyme is likely to be deficient in a 4-year-old child who presents with a habit of biting their lips and fingers, and has a missing portion of fingers on their left hand? Further evaluation shows delayed developmental milestones. The parents report that two male cousins on the mother's side have similar symptoms and have been diagnosed with a genetic disorder. Laboratory reports indicate hyperuricemia. (Q857544)

A. Purine nucleoside phosphorylase

B. Hypoxanthine -guanine phosphoribosyl transferase.

C. Adenine phosphoribosyl transferase

D. Adenosine deaminase

See Answer in Part B → Q6

@Neet_pg_bot

Q7: Which of the following does not contribute to the repair of DNA double-stranded breaks? (Q857361)

A. ATP

B. RAD-51

C. ATM

D. Ku 70

See Answer in Part B → Q7

@Neet_pg_bot

Q8: Which of the following is considered a universal methyl group donor in various biochemical reactions? (Q856961)

A. Glucose-6-phosphate

B. Acetyl CoA

C. S-Adenosyl Methionine

D. S-Adenosyl Serine

See Answer in Part B → Q8

@Neet_pg_bot

Q9: What is the enzyme that is responsible for the defect causing Maple syrup urine disease? (Q856942)

A. Branched-chain a-ketoacid dehydrogenase

B. Isovaleryl CoA dehydrogenase

C. Pyruvate carboxylase

D. Acetyl CoA carboxylase

See Answer in Part B → Q9

@Neet_pg_bot

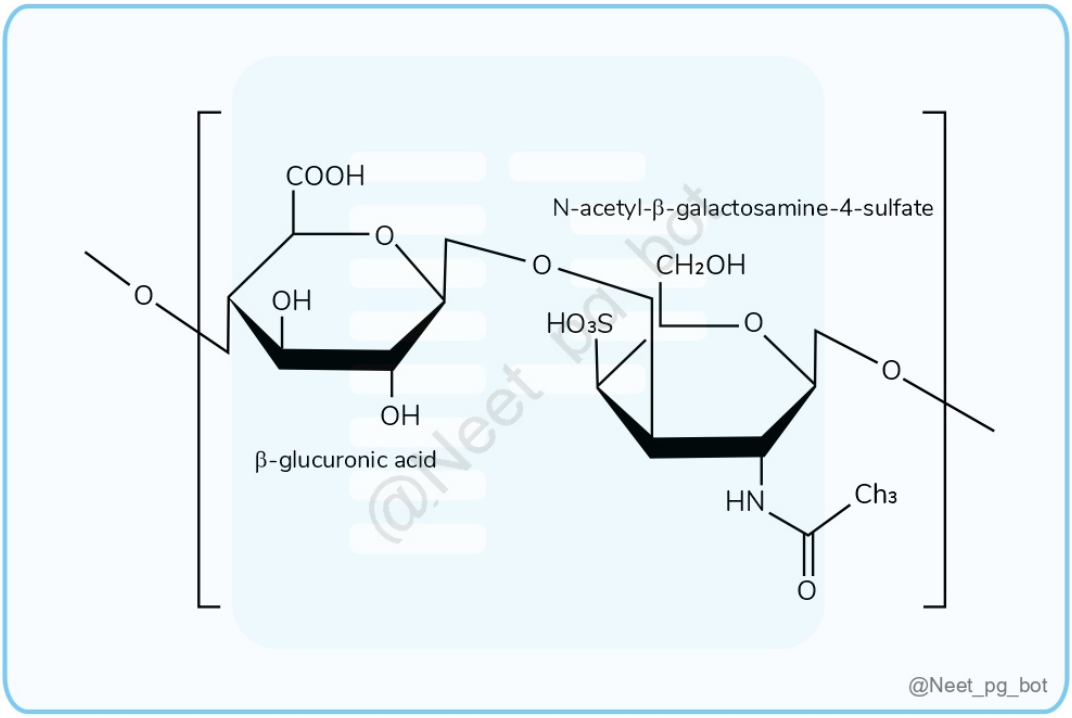
Q10: The given chemical structure is of: (Q856923)

A. Keratan sulfate

B. Chondroitin sulfate

C. Heparin

D. Dermatan sulfate



See Answer in Part B → Q10

@Neet_pg_bot

Q11: Which of the following is not a true statement regarding telomerase? (Q856880)

A. Has reverse transcriptase activity

B. Present only in Eukaryotes

C. Maintain the chromosome length

D. Involved in DNA repair

See Answer in Part B → Q11

@Neet_pg_bot

Q12: Nitric oxide is synthesized from: (Q854869)

A. L-Arginine

B. L-Citrulline

C. Glycine

D. Lysine

See Answer in Part B → Q12

@Neet_pg_bot

Q13: Which of the following options is false in a patient with advanced liver disease? (Q856619)

A. Hyperammonemia

B. Decreased ATP synthesis

C. Oxidative carboxylation of alpha-ketoglutarate

D. Increased synthesis of glutamine and glutamine from alpha-ketoglutarate

See Answer in Part B → Q13

@Neet_pg_bot

Q14: The maximum thermic effect is seen with a meal that has (Q856500)

A. Protein

B. Fat

C. Carbohydrates

D. Mixed meal having all 3 in equal amount

See Answer in Part B → Q14

@Neet_pg_bot

Q15: A young girl presented to the OPD for an examination. She is tall and thin with slender fingers, and dislocated lenses. Workup revealed cystathione beta-synthase deficiency. Which amino acid supplement is required in this case? (Q855999)

A. Cysteine

B. Methionine

C. Serine

D. Tyrosine

See Answer in Part B → Q15

@Neet_pg_bot

Q16: A patient presented with joint pain and on evaluation, urine turns black on standing. What is the likely enzyme deficiency? (Q855951)

A. Homogentisic acid 1,2-dioxygenase

B. Tyrosine transaminase

C. Fumarylacetoacetase hydrolase

D. 4-hydroxy-phenyl pyruvate dioxygenase

See Answer in Part B → Q16

@Neet_pg_bot

Q17: Which of the following consumes the highest energy during assimilation? (Q855919)

A. Proteins

B. Carbohydrates

C. Mono-unsaturated fatty acids

D. Polyunsaturated fatty acids

See Answer in Part B → Q17

@Neet_pg_bot

Q18: Which of the following options best describes the mutation seen in patients with cystic fibrosis? (Q855554)

A. Tryptophan at 708 position, chloride channel

B. Phenylalanine at 708 position, calcium channel

C. Phenylalanine at 508 position, chloride channel

D. Tryptophan at 508 position, calcium channel

See Answer in Part B → Q18

@Neet_pg_bot

Q19: An infant is brought by his parents with complaints that his urine turns black on standing. Which of the following metabolic disorders is most likely? (Q855263)

A. Phenylketonuria

B. Alkaptonuria

C. Homocystinuria

D. Maple syrup urine disease

See Answer in Part B → Q19

@Neet_pg_bot

Q20: A chronic alcoholic patient is diagnosed with gouty arthritis. What biochemical changes will be seen in this patient? (Q855230)

A. Decreased NADH/NAD+ ratio

B. Increased urea and urate levels

C. Increased urate and lactate levels

D. Increased level of alkaline phosphatase

See Answer in Part B → Q20

@Neet_pg_bot

Q21: In a patient with Maple Syrup Urine Disease (MSUD), all of the following amino acids should be restricted in diet except? (Q855044)

A. Methionine

B. Valine

C. Leucine

D. Isoleucine

See Answer in Part B → Q21

@Neet_pg_bot

Q22: In a patient with obstructive jaundice, which of the following outcomes may be observed in Van der Bergh's reaction? (Q854880)

A. Direct positive

B. Indirect positive

C. Both positive

D. Coproporphyrin

See Answer in Part B → Q22

@Neet_pg_bot

• [10_Heme Metabolism] 028 Previous Year Questions

« Back to Folder Directory

[10_Heme Metabolism] 028 Previous Year Questions — Quick Jump

Q1 (p. 353) *Q2 (p. 353)* *Q3 (p. 353)* *Q4 (p. 354)* *Q5 (p. 354)* *Q6 (p. 355)* *Q7 (p. 355)*

Q1: Decreased % transferrin saturation is seen in? (Q854613)

A. Hemochromatosis

B. Iron deficiency anemia

C. Megaloblastic anemia

D. Hemosiderosis

See Answer in Part B → Q1

@Neet_pg_bot

Q2: Which part of the haemoglobin molecule does 2,3-Bisphosphoglycerate (2,3-BPG) bind to in order to regulate oxygen release? (Q854619)

A. Alpha chains of haemoglobin

B. Heme iron centre

C. Beta chains of haemoglobin

D. Globin-heme interface

See Answer in Part B → Q2

@Neet_pg_bot

Q3: What does iron deficiency anemia (IDA) stand for? (Q854624)

A. Low serum ferritin

B. Low transferrin saturation

C. Increased TIBC

D. All of the above

See Answer in Part B → Q3

@Neet_pg_bot

Q4: Which of the following is the rate-limiting enzyme of heme synthesis pathway? (Q857891)

A. HMB synthase

B. δ Amino-Levulinic Acid dehydratase

C. δ Amino-Levulinic Acid synthase

D. Ferrochelatase

See Answer in Part B → Q4

@Neet_pg_bot

Q5: A female is diagnosed with lead poisoning . Which of the following enzyme defects is primarily responsible for the hematologic abnormalities observed in this condition? (Q857898)

A. ALA synthase

B. Uroporphyrinogen decarboxylase

C. Heme synthase

D. Coproporphyrinogen oxidase

See Answer in Part B → Q5

@Neet_pg_bot

Q6: Heme is derived from which of the following amino acids? (Q860419)

A. Glutamine

B. Glycine

C. Glutamate

D. Valine

See Answer in Part B → Q6

@Neet_pg_bot

Q7: Which of the following amino acids are necessary for purine ring formation? (Q860424)

A. Glycine, aspartate, valine

B. Glycine, Aspartate, Glutamine

C. Glycine, Aspartate, Glutamate

D. Glycine, Asparagine, Glutamine

See Answer in Part B → Q7

@Neet_pg_bot

• [11_Vitamins] 029 Fat-Soluble Vitamins - I

« Back to Folder Directory

[11_Vitamins] 029 Fat-Soluble Vitamins - I — Quick Jump

Q1 (p. 356)	*Q2 (p. 356)*	*Q3 (p. 357)*	*Q4 (p. 357)*	*Q5 (p. 357)*	*Q6 (p. 358)*
Q7 (p. 358)	*Q8 (p. 359)*	*Q9 (p. 359)*	*Q10 (p. 360)*	*Q11 (p. 360)*	*Q12 (p. 361)*
Q13 (p. 361)	*Q14 (p. 361)*	*Q15 (p. 362)*	*Q16 (p. 362)*	*Q17 (p. 363)*	*Q18 (p. 363)*
Q19 (p. 363)	*Q20 (p. 364)*	*Q21 (p. 364)*			

Q1: A 62-year-old male on warfarin therapy for atrial fibrillation presents with uncontrolled bleeding. Laboratory tests show prolonged PT and INR. Which of the following mechanisms best explains the effect of warfarin on coagulation? (Q844939)

A. Inhibition of Vitamin K epoxide reductase

B. Inhibition of thrombin activity directly

C. Binding to calcium ions

D. Direct inhibition of clotting factors

See Answer in Part B → Q1

@Neet_pg_bot

Q2: A 40 year old woman presents with complaints of persistent nausea, vomiting, and frequent urination over the past few weeks. She has a history of osteomalacia and has been taking supplements for several months without proper medical supervision. Her blood tests reveal elevated serum calcium levels. What is the most likely diagnosis? (Q844949)

A. Hyperparathyroidism

B. Vitamin D deficiency

C. Vitamin D toxicity

D. Hypercalcemia of malignancy

See Answer in Part B → Q2

@Neet_pg_bot

Q3: Which of the following is not a commonly associated symptom with vitamin E deficiency? (Q844948)

A. Spinocerebellar ataxia

B. Megaloblastic anemia

C. Peripheral neuropathy

D. Hemolytic anemia

See Answer in Part B → Q3

@Neet_pg_bot

Q4: A 45-year-old man presents with fatigue, frequent urination, constipation, and confusion. His laboratory results show hypercalcemia and low serum parathyroid hormone level. He reports taking high-dose, over-the-counter vitamin D supplements for the past year. Which of the following conditions is most likely associated with his current presentation? (Q844947)

A. Increased risk of hypertension

B. Increased risk of colon cancer

C. Increased risk of breast cancer

D. Increased risk of insulin resistance

See Answer in Part B → Q4

@Neet_pg_bot

Q5: A 38-year-old female with a history of chronic kidney disease presents with persistent fatigue, bone pain, and muscle weakness. Blood work reveals elevated

alkaline phosphatase (ALP), high serum phosphorus, and low serum calcium. What will the result of 1,25 dihydroxy vitamin D likely reveal? (Q844946)

A. Elevated 1, 25 dihydroxy vitamin D levels.

B. Estimating 1, 25 dihydroxy vitamin D is unnecessary.

C. Decreased 1, 25 dihydroxy vitamin D.

D. Normal 1, 25 dihydroxy vitamin D.

See Answer in Part B → Q5

@Neet_pg_bot

Q6: A 2-year-old child presents with progressive bowing of the legs and tenderness in the lower limbs. The child has a history of limited outdoor play and a diet low in dairy products. Which of the following limb deformities is most likely present in this child? (Q844945)

A. Rickets

B. Blount's disease

C. Osteomalacia

D. Osteogenesis imperfecta

See Answer in Part B → Q6

@Neet_pg_bot

Q7: A 35-year-old woman presents with fatigue, muscle weakness, and bone pain for the past several months. She has a history of hypothyroidism and is currently taking levothyroxine. She lives in North India and avoids dairy products. Physical examination reveals mild tenderness over her shins. Laboratory tests reveal: Serum calcium 8.2 mg/dL (normal range: 8.5-10.5 mg/dL) Serum phosphorus 2.0 mg/dL (normal range: 2.5-4.5 mg/dL) Parathyroid hormone (PTH) Elevated Which of the following is the most likely diagnosis? (Q844944)

A. Vitamin D deficiency

B. Primary hyperparathyroidism

C. Hypoparathyroidism

D. Chronic kidney disease

See Answer in Part B → Q7

@Neet_pg_bot

Q8: A 52-year-old woman presents with muscle cramps and tingling in her hands and feet. She follows a strict vegetarian diet high in whole grains and has a history of fat malabsorption due to IBS. Lab tests reveal hypocalcemia and low vitamin D levels. What is the most likely cause of her hypocalcemia? (Q844943)

A. Low intake of oxalate

B. Low fatty acid in intestine

C. High phytate intake from cereals

D. Increased calbindin level

See Answer in Part B → Q8

@Neet_pg_bot

Q9: Which of the following factors does not inhibit 1α-hydroxylase activity? (Q844942)

A. Hyperphosphatemia

B. Hypercalcemia

C. High parathormone

D. 1,25-dihydroxycholecalciferol

See Answer in Part B → Q9

@Neet_pg_bot

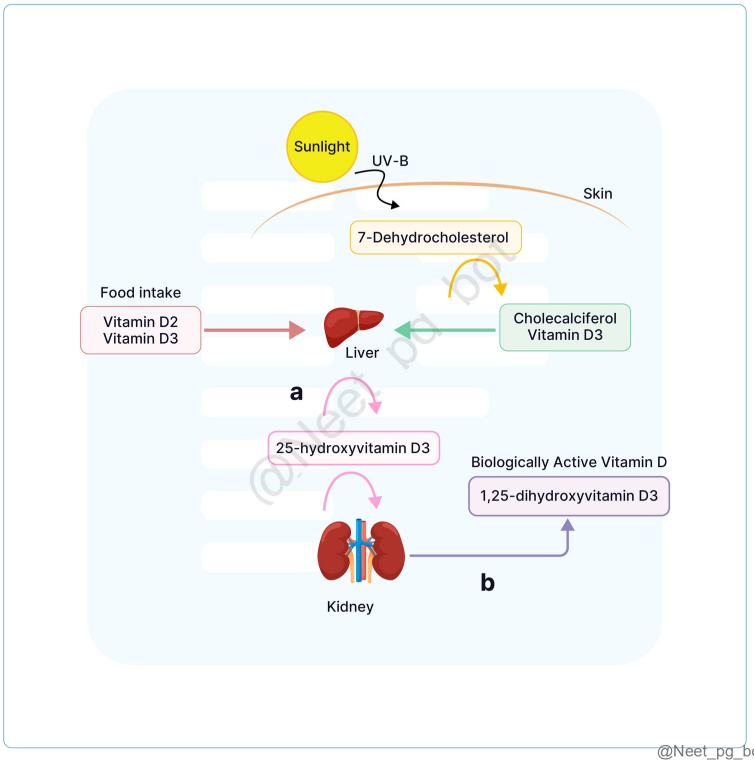
Q10: Identify the enzymes marked as a and b respectively in the flow chart, as related to vitamin D synthesis: (Q844941)

A. α-25 alpha hydroxylase, liver; β-1 alpha hydroxylase, kidney

B. α-1 alpha hydroxylase, kidney; β-25 alpha hydroxylase, liver

C. α-1 alpha hydroxylase, liver; β-25 alpha hydroxylase, kidney

D. α-25 alpha hydroxylase, kidney; β-1 alpha-hydroxylase, liver



See Answer in Part B → Q10

@Neet_pg_bot

Q11: Which of the following features is NOT associated with a deficiency of Vitamin K? (Q844940)

A. Hemorrhagic disease of the newborn

B. Prolonged prothrombin time

C. Hyperbilirubinemia

D. Mucous membrane hemorrhage

See Answer in Part B → Q11

@Neet_pg_bot

Q12: Which vitamins are least likely to accumulate in the body's adipose tissue due to its solubility characteristics? (Q844929)

A. Retinol

B. Ergocalciferol

C. Tocopherol

D. Pantothenic acid

See Answer in Part B → Q12

@Neet_pg_bot

Q13: Which of the following statements about Vitamin K is incorrect? (Q844938)

A. Vitamin K is crucial for the activation of blood clotting factors.

B. Vitamin K2 is a lipid-soluble form produced by intestinal bacteria.

C. Menadione is a natural water-soluble form of Vitamin K.

D. Vitamin K aids in the carboxylation of bone proteins.

See Answer in Part B → Q13

@Neet_pg_bot

Q14: All of the following are deficiency manifestations of Vitamin E except: (Q844937)

A. Spinocerebellar ataxia

B. Ophthalmoplegia

C. Hemorrhagic tendency

D. Peripheral neuropathy

See Answer in Part B → Q14

@Neet_pg_bot

Q15: Which of the following is NOT a chain-breaking antioxidant? (Q844936)

A. α-Tocopherol

B. β-Carotene

C. Superoxide dismutase

D. Glutathione Peroxidase

See Answer in Part B → Q15

@Neet_pg_bot

Q16: A 17-year-old girl presents with severe acne on her face and back. She was previously treated with benzoyl peroxide and oral doxycycline and found no improvement. Physical examination shows comedones, pustules, inflammatory papules, and facial scarring. Which of the following is the next best treatment? (Q844935)

A. All trans-retinoic acid

B. Tocopherol

C. Phylloquinone

D. 13 cis-retinoic acid

See Answer in Part B → Q16

@Neet_pg_bot

Q17: Which of the following is most likely observed in patients with vitamin A toxicity? (Q844934)

A. Decreased CSF pressure

B. Decreased bone mineral density

C. Thinning of long bones

D. Increased appetite

See Answer in Part B → Q17

@Neet_pg_bot

Q18: Which of the following statements about Vitamin A deficiency is not correct? (Q844933)

A. Causes rough skin and atrophy of epithelial tissues

B. Prolonged deficiency causes keratomalacia

C. Preventable cause of blindness in children under 5

D. Increased retinol-binding protein synthesis is a cause

See Answer in Part B → Q18

@Neet_pg_bot

Q19: A 35-year-old woman reports difficulty seeing in low light. She has a poor diet, lacking in fruits and vegetables. Dark adaptation is delayed on testing. What is the most likely cause of her symptoms? (Q844932)

A. Impaired retinol storage

B. Reduced 11-cis-retinal regeneration

C. Defective retinoic acid transport

D. Accumulation of all-trans-retinal

See Answer in Part B → Q19

@Neet_pg_bot

Q20: Which enzyme is responsible for the isomerization of all-trans retinyl ester to 11-cis retinol in the retinal pigment epithelium (RPE)? (Q844931)

A. Lecithin-retinol acyltransferase

B. All-trans-retinol dehydrogenase

C. Retinoid isomerohydrolase

D. 11-cis-retinol dehydrogenase

See Answer in Part B → Q20

@Neet_pg_bot

Q21: Which form of vitamin A is stored in the liver and must bind to retinol-binding protein (RBP) for transport to other tissues? (Q844930)

A. Retinoic acid

B. Retinal

C. Retinol

D. β-carotene

See Answer in Part B → Q21

@Neet_pg_bot

• [11_Vitamins] 030 Water-Soluble Vitamins - I

« Back to Folder Directory

[11_Vitamins] 030 Water-Soluble Vitamins - I — Quick Jump

Q1 (p. 365) *Q2 (p. 365)* *Q3 (p. 366)* *Q4 (p. 366)* *Q5 (p. 366)* *Q6 (p. 367)*
Q7 (p. 367) *Q8 (p. 368)* *Q9 (p. 368)* *Q10 (p. 368)* *Q11 (p. 369)* *Q12 (p. 369)*
Q13 (p. 370) *Q14 (p. 370)* *Q15 (p. 370)* *Q16 (p. 371)* *Q17 (p. 371)* *Q18 (p. 372)*
Q19 (p. 372) *Q20 (p. 372)* *Q21 (p. 373)*

Q1: Which of the following statements about vitamin B12 absorption is true? (Q843177)

A. Intrinsic factor is secreted by the pancreas and binds to inactive forms of vitamin B12.

B. Cobalophilin in the saliva binds vitamin B12 initially and is hydrolyzed in the duodenum.

C. Gastric acid and pepsin are not necessary for Vitamin B12 absorption

D. Ileal receptors bind directly to vitamin B12 without the need for intrinsic factors.

See Answer in Part B → Q1

@Neet_pg_bot

Q2: A 35-year-old woman presents with fatigue, bleeding gums, easy bruising, and poor wound healing. Laboratory tests confirm scurvy. She reports a diet low in fruits and vegetables for several months. Which nutrient deficiency can mimic these symptoms? (Q845064)

A. Iron Deficiency

B. Zinc Deficiency

C. Folate Deficiency

D. Vitamin B12 Deficiency

See Answer in Part B → Q2

@Neet_pg_bot

Q3: Which of the following statements regarding the avidin-biotin system is not true? (Q845063)

A. Avidin has a higher affinity for biotin than typical antigen-antibody interactions

B. Avidin can bind up to four molecules of biotin, enhancing assay sensitivity

C. Consumption of raw egg whites may lead to biotin deficiency due to avidin-binding biotin

D. Avidin is heat-stable and retains its binding capacity after boiling eggs

See Answer in Part B → Q3

@Neet_pg_bot

Q4: Which of the following enzymes is not classified as a biotin-dependent carboxylase? (Q843396)

A. Acetyl CoA carboxylase

B. Pyruvate carboxylase

C. Propionyl CoA carboxylase

D. Malic enzyme

See Answer in Part B → Q4

@Neet_pg_bot

Q5: A 55-year-old man on high-dose niacin for hyperlipidemia develops blurred vision and macular edema. What is the mechanism behind niacin-induced macular edema? (Q843395)

A. Increased prostaglandin synthesis

B. Inhibition of retinal blood flow

C. Direct toxicity to retinal pigment epithelium

D. Enhanced inflammation in the retina

See Answer in Part B → Q5

@Neet_pg_bot

Q6: A 28 year old pregnant woman presents with complaints of fatigue and weakness. Her dietary history reveals she follows a strict vegetarian diet. Blood tests reveal megaloblastic anaemia. Which of the following complications is most likely associated with her condition if left untreated? (Q843393)

A. Increased risk of gestational diabetes

B. Neurological problems in the mother and cognitive impairments in the foetus

C. Higher incidence of hypertension during pregnancy

D. Increased risk of maternal infections

See Answer in Part B → Q6

@Neet_pg_bot

Q7: After consuming illicit homemade alcohol at a local event, numerous individuals were hospitalized with severe abdominal pain, vomiting, malaise and vision changes. Which of the following cannot be used as an antidote for methanol poisoning? (Q843186)

A. Ethanol

B. Fomepizole

C. Folinic acid

D. Formic acid

See Answer in Part B → Q7

@Neet_pg_bot

Q8: A 50-year-old chronic alcoholic develops agitation, tremors, hallucinations, and autonomic instability three days after stopping alcohol. What is the most appropriate management? (Q843185)

A. Initiate antipsychotic medication

B. Prescribe selective serotonin reuptake inhibitors (SSRIs)

C. Administer benzodiazepines for sedation

D. Begin opioid replacement therapy

See Answer in Part B → Q8

@Neet_pg_bot

Q9: A 45-year-old man with a history of heavy alcohol consumption for the past 10 years, presents with fatigue, abdominal discomfort, weight gain, and hepatomegaly. Laboratory tests show elevated liver enzymes, and a liver ultrasound reveals fatty liver. What commonly causes fatty liver in chronic alcohol consumption? (Q843183)

A. Decreased acetyl CoA levels

B. Increased pyruvate oxidation

C. Accumulation of acetaldehyde

D. Increased fatty acid oxidation

See Answer in Part B → Q9

@Neet_pg_bot

Q10: A 45-year-old chronic alcoholic presents with confusion, tremors, diaphoresis, and hypoglycemia (50 mg/dL). He has been drinking heavily with poor nutrition. Which of the following statements about the effects of chronic alcoholism on nutritional deficiencies and glucose metabolism is false? (Q843181)

A. Chronic alcohol use can cause vitamin B12 deficiency despite normal blood levels

B. Folate deficiency in alcoholics leads to megaloblastic anemia

C. Chronic alcohol use causes hypoglycemia by increasing the NADH/NAD+ ratio

D. Chronic alcohol use causes hypoglycemia by decreasing the NADH/NAD+ ratio

See Answer in Part B → Q10

@Neet_pg_bot

Q11: A 50-year-old patient presents with symptoms of fatigue and pallor. The physician suspects a vitamin B12 absorption issue and decides to perform the Schilling test. Which of the following statements about the Schilling test is correct? (Q843179)

A. The Schilling test involves administering one dose of vitamin B12 to assess absorption.

B. An abnormal result in stage 1 and a normal result in Stage 2 of the Schilling test typically suggests a lack of intrinsic factor.

C. Radioactive vitamin B12 injection is given to saturate body stores.

D. Abnormal Stage 2 suggests intrinsic factor deficiency.

See Answer in Part B → Q11

@Neet_pg_bot

Q12: A 35-year-old woman with a white rice–based diet presents with breathlessness, palpitations, and edema. Examination shows distended neck veins and elevated lactate and pyruvate levels, suggesting thiamine deficiency. What is the likely diagnosis, and which enzyme activity confirms it? (Q843152)

A. Dry beriberi; pyruvate dehydrogenase

B. Maple Syrup Urine Disease (MSUD); branched-chain ketoacid dehydrogenase

C. Wet beriberi; transketolase

D. Lactic acidosis; lactate dehydrogenase

See Answer in Part B → Q12

@Neet_pg_bot

Q13: Match the following vitamins with their respective functional assessment tests: 1. Vitamin B1 (Thiamine) a) Measure urinary methylmalonic acid 2. Vitamin B2 (Riboflavin) b) Measure transketolase activity in red blood cells 3. Vitamin B6 (Pyridoxine) c) Measure glutathione reductase activity in red blood cells 4. Vitamin B12 (Cobalamin) d) Measure activation of red blood cell transaminases with pyridoxal phosphate (Q843176)

A. 1-b, 2-c, 3-d, 4-a

B. 1-a, 2-b, 3-c, 4-d

C. 1-b, 2-d, 3-c, 4-a

D. 1-a, 2-c, 3-b, 4-d

See Answer in Part B → Q13

@Neet_pg_bot

Q14: Match the following deficiencies with their respective impacts: 1) Adenosylcobalamin Deficiency a) Elevated homocysteine levels 2) Methylcobalamin Deficiency b) Impaired energy production and chronic fatigue 3) Methyl Malonyl-CoA Mutase Deficiency c) Accumulation of methylmalonyl-CoA 4) Methionine Synthase Deficiency d) Pernicious anaemia with immature red blood cells (Q843173)

A. 1-b, 2-a, 3-c, 4-d

B. 1-a, 2-d, 3-b, 4-c

C. 1-b, 2-d, 3-c, 4-a

D. 1-c, 2-b, 3-d, 4-a

See Answer in Part B → Q14

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Q15: Which of the following consequences of vitamin B6 deficiency directly contributes to an increased risk of seizures in a neonate with refractory seizures? (Q843171)

A. Decreased GABA synthesis

B. Elevated glutamate levels

C. Increased serotonin production

D. Enhanced myelin formation

See Answer in Part B → Q15

@Neet_pg_bot

Q16: Which of the following metabolic processes is not directly supported by vitamin B6 in its active form, pyridoxal 5'-phosphate (PLP)? (Q843169)

A. Transamination of amino acids

B. Decarboxylation of amino acids

C. Breakdown of glycogen to glucose-1-phosphate

D. Conversion of glucose to glycogen

See Answer in Part B → Q16

@Neet_pg_bot

Q17: A 15 year old child presents with skin rashes, gastrointestinal issues, aminoaciduria and cognitive disturbances. Which of the following mechanisms explains the symptoms observed? (Q843166)

A. Hartnup disease causes a direct deficiency in niacin, leading to pellagra symptoms.

B. Impaired tryptophan absorption → ↓production of niacin → pellagra-like symptoms.

C. Tryptophan excess → disrupts niacin metabolism → pellagra-like symptoms

D. Increases the metabolism of tryptophan to serotonin, depleting niacin stores

See Answer in Part B → Q17

@Neet_pg_bot

Q18: A 45-year-old woman consuming mainly maize and sorghum develops bright red rash on her feet, ankles, and face that worsens with sun exposure. She also reports frequent diarrhea and recent episodes of irritability and difficulty concentrating. What is the likely diagnosis? (Q843163)

A. Vitamin B12 deficiency

B. Pellagra

C. Rickets

D. Iron deficiency anemia

See Answer in Part B → Q18

@Neet_pg_bot

Q19: Which of the following is the earliest sign of riboflavin (Vitamin B2) deficiency? (Q843158)

A. Cheilosis

B. Glossitis

C. Seborrheic dermatitis

D. Proliferation of the bulbar conjunctival capillaries

See Answer in Part B → Q19

@Neet_pg_bot

Q20: Which of the following enzymes does not utilize FAD as a coenzyme? (Q843156)

A. Succinate Dehydrogenase

B. NADH Dehydrogenase

C. Xanthine Oxidase

D. Pyruvate Dehydrogenase

See Answer in Part B → Q20

@Neet_pg_bot

Q21: A 52-year-old alcoholic with poor nutrition presents with confusion, ataxia, and eye movement abnormalities. What is the most likely diagnosis? (Q843155)

A. Wernicke encephalopathy

B. Korsakoff psychosis

C. Lactic acidosis

D. Dry beri-beri

See Answer in Part B → Q21

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• [11_Vitamins] 031 Previous Year Questions

« Back to Folder Directory

[11_Vitamins] 031 Previous Year Questions — Quick Jump

Q1 (p. 374)	*Q2 (p. 374)*	*Q3 (p. 375)*	*Q4 (p. 376)*	*Q5 (p. 376)*	*Q6 (p. 376)*
Q7 (p. 377)	*Q8 (p. 377)*	*Q9 (p. 378)*	*Q10 (p. 378)*	*Q11 (p. 379)*	*Q12 (p. 379)*
Q13 (p. 379)	*Q14 (p. 380)*	*Q15 (p. 380)*	*Q16 (p. 381)*	*Q17 (p. 381)*	*Q18 (p. 381)*
Q19 (p. 382)	*Q20 (p. 382)*	*Q21 (p. 382)*	*Q22 (p. 383)*	*Q23 (p. 384)*	*Q24 (p. 385)*
Q25 (p. 385)	*Q26 (p. 386)*	*Q27 (p. 387)*	*Q28 (p. 387)*	*Q29 (p. 387)*	*Q30 (p. 388)*
Q31 (p. 388)	*Q32 (p. 388)*	*Q33 (p. 389)*	*Q34 (p. 389)*	*Q35 (p. 390)*	*Q36 (p. 390)*
Q37 (p. 390)	*Q38 (p. 391)*	*Q39 (p. 391)*	*Q40 (p. 392)*	*Q41 (p. 392)*	*Q42 (p. 392)*
Q43 (p. 393)	*Q44 (p. 393)*	*Q45 (p. 394)*	*Q46 (p. 395)*	*Q47 (p. 395)*	*Q48 (p. 396)*
Q49 (p. 396)	*Q50 (p. 397)*	*Q51 (p. 397)*	*Q52 (p. 397)*	*Q53 (p. 398)*	*Q54 (p. 398)*
Q55 (p. 399)	*Q56 (p. 399)*	*Q57 (p. 400)*	*Q58 (p. 400)*	*Q59 (p. 400)*	*Q60 (p. 401)*
Q61 (p. 401)	*Q62 (p. 401)*	*Q63 (p. 402)*	*Q64 (p. 402)*	*Q65 (p. 403)*	*Q66 (p. 403)*

Q1: To evaluate a deficiency of vitamin B2 (riboflavin), one can assess the activity of: (Q857548)

A. Transketolase

B. Pyruvate dehydrogenase

C. Glutathione reductase

D. Isocitrate dehydrogenase

See Answer in Part B → Q1

@Neet_pg_bot

Q2: A 34-year-old male was on maize as a staple diet for a prolonged period. He presented with diarrhea and lesion over his neck as shown in the image below. The condition occurs due to deficiency of: (Q854888)

A. Niacin

B. Vitamin B12

C. Vitamin D

D. Iron



See Answer in Part B → Q2

@Neet_pg_bot

Q3: Which vitamin, when infused in supraphysiological doses, causes macular edema and macular cysts? (Q854868)

A. Vitamin A

B. Vitamin D

C. Vitamin E

D. Vitamin B3

See Answer in Part B → Q3

@Neet_pg_bot

Q4: Dicoumarol inhibits the action of vitamin K by? (Q856506)

A. Suicide inhibition

B. Competitive inhibition

C. Non-competitive inhibition

D. Uncompetitive inhibition

See Answer in Part B → Q4

@Neet_pg_bot

Q5: The sequence of vitamin D synthesis is? (Q856503)

A. Liver-skin-kidney

B. Skin-liver-kidney

C. Skin-kidney-liver

D. Liver-kidney-skin

See Answer in Part B → Q5

@Neet_pg_bot

Q6: Which of the following are vitamin B12-dependent enzymes? Methyl malonyl CoA mutase Homocysteine synthase Folate reductase Methionine synthase (Q856920)

A. 1,2,3 and 4

B. 1 and 4 only

C. 1 and 3 only

D. 1,2 and 3

See Answer in Part B → Q6

@Neet_pg_bot

Q7: An alcoholic patient presented with itchy lesions as shown in the image. He also has a red-colored beefy tongue. Which of the following histories would you like to ask/elicit in this patient additionally? (Q855973)

A. Ask for diarrhea and a history of memory loss

B. Ask for diarrhea, thiamine deficiency features, and detailed dietary history

C. History of allergy

D. Ask for diarrhea and conjunctivitis



See Answer in Part B → Q7

@Neet_pg_bot

Q8: A farmer presented with loose stools and rashes over the sun-exposed areas. Careful diet history reveals a diet rich in maize. Which of the following deficiencies

would you suspect? (Q855968)

A. Niacin

B. Pyridoxine

C. Thiamine

D. Biotin

See Answer in Part B → Q8

@Neet_pg_bot

Q9: A patient presented with symptoms of delayed adaptation to vision in dark places, gritty sensation in the eyes, dry eyes, and softening of the cornea. What is the likely deficiency in this case? (Q855948)

A. Retinoic acid

B. Riboflavin

C. Niacin

D. Pantothenic acid

See Answer in Part B → Q9

@Neet_pg_bot

Q10: What is the most likely cause of the worsening neurological symptoms in a 30-year-old woman who presented with complaints of fatigue, difficulty breathing during exertion, weight loss, and frequent falls, and was found to have bilateral decrease in vibration sense, a hemoglobin level of 8.2 g% with high MCV, and was initially treated with folate which improved her anemia? (Q854890)

A. Folate is not well absorbed

B. Folic acid therapy unmasked pyridoxine deficiency.

C. Deficiency of folate reductase in CNS.

D. Folate therapy caused rapid use of vitamin B12 stores aggravating symptoms.

See Answer in Part B → Q10

@Neet_pg_bot

Q11: Which antioxidant deficiency can cause the symptoms of iron-deficiency anemia, painful and tender joints, and bleeding manifestations in a 34-year-old woman who primarily consumes packaged and reheated meals? (Q857546)

A. Glutathione

B. ß-carotene

C. Vitamin C

D. Zinc

See Answer in Part B → Q11

@Neet_pg_bot

Q12: What is the underlying cause of the presenting symptoms in a 21-year-old individual with chronic alcoholism who is admitted to the emergency department with confusion, tremors, ataxia, and ophthalmoplegia? (Q857545)

A. Vitamin B12

B. Vitamin B9

C. Vitamin B1

D. Vitamin B3

See Answer in Part B → Q12

@Neet_pg_bot

Q13: A young boy presented with fissured tongue, lips, and lesions in the angle of the mouth.On investigation, he had low RBC glutathione reductase activity. Which

vitamin is deficient here? (Q857487)

A. Vitamin B12

B. Vitamin B3

C. Biotin

D. Riboflavin

See Answer in Part B → Q13

@Neet_pg_bot

Q14: Which of the following substance, facilitate the absorption of dietary iron? (Q857479)

A. Calcium

B. Vitamin C

C. Vitamin D

D. Vitamin K

See Answer in Part B → Q14

@Neet_pg_bot

Q15: Which of the subsequent stages in the vitamin D metabolism necessitates exposure to sunlight? (Q857003)

A. 1,25 dihydroxy cholecalciferol to 7-dehydrocholesterol

B. Cholecalciferol to 25-hydroxycholecalciferol

C. 7-dehydrocholesterol to cholecalciferol

D. 25-hydroxycholecalciferol to 1,25 dihydroxycholecalciferol

See Answer in Part B → Q15

@Neet_pg_bot

Q16: What could be the potential diagnosis for a 34-year-old male who has been consuming maize as his main diet for an extended period and is now experiencing symptoms such as abdominal pain, decreased appetite, and diarrhea? Additionally, upon examination, glossitis (inflammation of the tongue) and a pigmented rash in sun-exposed areas were observed, along with a lesion on his neck. (Q856983)

A. Pellagra

B. Scurvy

C. Megaloblastic anemia

D. Beriberi

See Answer in Part B → Q16

@Neet_pg_bot

Q17: Which of the following vitamins is incorrectly paired with their respective enzymes? (Q856939)

A. Vitamin B1 (Thiamine): RBC Trans-ketolase

B. Vitamin B2 (Riboflavin): RBC glutathione reductase

C. Vitamin B9 (Folic acid): FIGLU excretion/Histidine load test

D. Vitamin C: Serum methylmalonate level

See Answer in Part B → Q17

@Neet_pg_bot

Q18: A family consumes a diet predominantly of rice and pulses developed signs of muscle weakness and movement. Which of the following should be given for prophylaxis? (Q866669)

A. Calcium

B. Thiamine

C. Vitamin D

D. Iron

See Answer in Part B → Q18

@Neet_pg_bot

Q19: Which of the following contains the maximum amount of vitamin B12? (Q858455)

A. Animal products

B. Green leafy vegetables

C. Roots and tubers

D. Vegetarian diet

See Answer in Part B → Q19

@Neet_pg_bot

Q20: What is the probable reason for the worsening of neurologic symptoms in a patient who initially showed improvement in blood picture after receiving a dose of 5mg folate for peripheral neuropathy and macrocytic anemia? (Q858458)

A. Malabsorption of folate

B. Treatment with folate unmasking pyridoxine deficiency

C. Deficiency of folate reductase in CNS

D. Folate therapy using the B12 stores

See Answer in Part B → Q20

@Neet_pg_bot

Q21: What is the recommended treatment for a 45-year-old male patient with a history of chronic alcoholism, who is currently experiencing symptoms of confusion, ataxia, and diplopia? (Q859571)

A. Vitamin B1 injection followed by glucose infusion

B. Glucose infusion followed thiamine injection

C. Glucose infusion alone

D. Thiamine infusion alone

See Answer in Part B → Q21

@Neet_pg_bot

Q22: A patient has come in with bleeding gums and the examination results are as follows. This particular condition can be observed in the case of a deficiency in (Q859575)

A. Vitamin A

B. Vitamin B12

C. Vitamin C

D. Vitamin D



See Answer in Part B → Q22

@Neet_pg_bot

Q23: What is the probable vitamin deficiency in a patient who displays symptoms of diarrhea, memory impairment, and a distinctive rash as described? (Q859712)

A. Thiamine

B. Niacin

C. Cyanocobalamin

D. Ascorbic acid



See Answer in Part B → Q23

@Neet_pg_bot

Q24: Which of the following enzyme activities can be estimated in red blood cells to diagnose vitamin B2 deficiency? (Q855541)

A. Transketolase

B. Glutathione reductase

C. Kynureninase

D. Pyruvate dehydrogenase

See Answer in Part B → Q24

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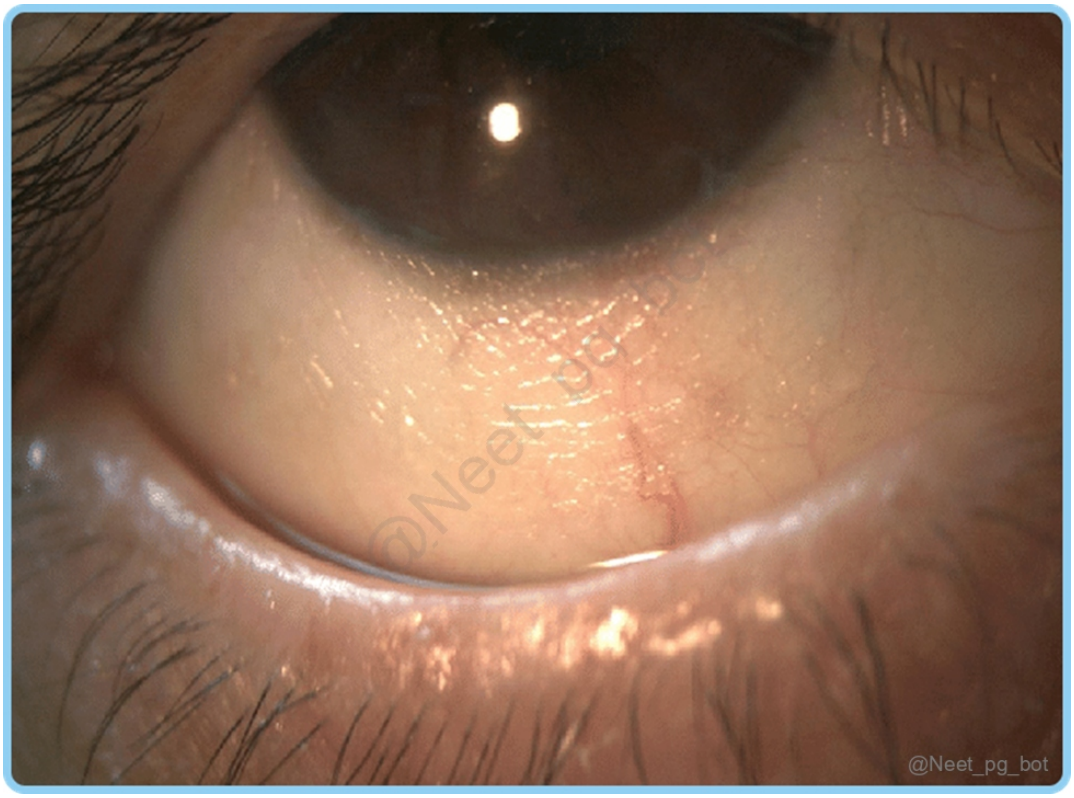
Q25: What is the cause of following condition due to? (Q855542)

A. Vitamin C

B. Vitamin A

C. Vitamin B

D. Vitamin E



See Answer in Part B → Q25

@Neet_pg_bot

Q26: A 15-year-old boy presents with tremors, difficulty in speech, and behavioral changes. On examination, he has hepatomegaly and a golden-brown ring at the limbus of the cornea (Kayser-Fleischer ring). Wilson's disease is suspected. Which of the following is the best investigation to support the diagnosis? (Q866667)

A. Serum ceruloplasmin

B. Liver biopsy for copper estimation

C. Serum copper

D. Copper excretion in urine

See Answer in Part B → Q26

@Neet_pg_bot

Q27: A child presents with erythematous scaly patches in the perioral region, mucosal ulcers, and impaired epithelial wound healing. The most likely mineral deficiency associated with this condition is (Q855233)

A. Iron deficiency

B. Zinc deficiency

C. Calcium deficiency

D. Copper deficiency

See Answer in Part B → Q27

@Neet_pg_bot

Q28: Which of the following minerals is best known for insulin secretion stabilisation? (Q857866)

A. Cadmium

B. Copper

C. Iron

D. Zinc

See Answer in Part B → Q28

@Neet_pg_bot

Q29: Menke's disease is due to a deficiency of: (Q857490)

A. Zinc

B. Copper

C. Iron

D. Magnesium

See Answer in Part B → Q29

@Neet_pg_bot

Q30: The co-factor for glutathione peroxidase is: (Q857493)

A. Manganese

B. Selenium

C. Copper

D. Magnesium

See Answer in Part B → Q30

@Neet_pg_bot

Q31: Which one of the following is not classified as a micromineral? (Q859713)

A. Calcium

B. Selenium

C. lodine

D. Copper

See Answer in Part B → Q31

@Neet_pg_bot

Q32: A patient with megaloblastic anemia is mistakenly treated with folate alone, after which the neurological symptoms worsen rather than improve. Which of the following best explains this phenomenon? (Q879002)

A. Folate trap

B. Homocysteine accumulation

C. Masking of vitamin B12 deficiency by folate therapy

D. Pemicious anemia due to intrinsic factor deficiency

See Answer in Part B → Q32

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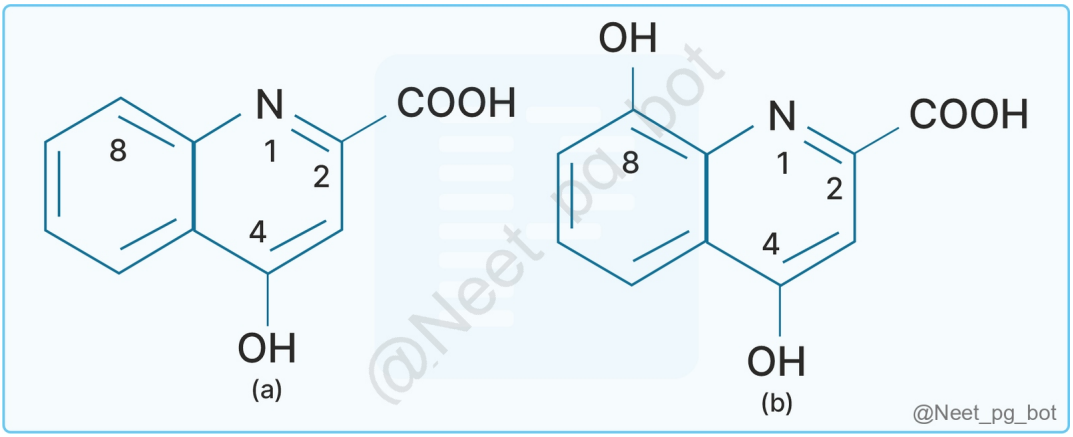
Q33: The vitamin deficiency that impairs the conversion of 3-hydroxykynurenine to xanthurenic acid is (Q879284)

A. Pyridoxal phosphate (PLP)

B. Biotin

C. Thiamine

D. B12



See Answer in Part B → Q33

@Neet_pg_bot

Q34: A patient presents with gum bleeding, petechiae, poor wound healing, and other related symptoms. Which of the following vitamin deficiencies is most likely responsible for this presentation? (Q852545)

A. Vitamin A

B. Vitamin B6

C. Vitamin B3

D. Vitamin C

See Answer in Part B → Q34

@Neet_pg_bot

Q35: Which vitamin acts as a coenzyme required for Glucose-6-Phosphate Dehydrogenase (G6PD)? (Q860364)

A. Riboflavin

B. Thiamine

C. Niacin

D. Biotin

See Answer in Part B → Q35

@Neet_pg_bot

Q36: Which of the following are metabolites of methanol poisoning? (Q844278)

A. Formic acid + Oxalic acid

B. Formic acid + Lactic acid

C. Glycolic acid + Oxalic acid

D. Lactic acid + Glycolic acid

See Answer in Part B → Q36

@Neet_pg_bot

Q37: A child presents with deformities, normal serum calcium, normal PTH, low phosphate, and elevated ALP. Which is the most likely diagnosis? (Q844271)

A. Hypophosphatemic rickets

B. Vitamin D Dependent Rickets Type 1 (VDDR type 1)

C. Vitamin D Dependent Rickets Type 2 (VDDR type 2)

D. Renal osteodystrophy

See Answer in Part B → Q37

@Neet_pg_bot

Q38: A patient presents with anemia, neutropenia, and hypopigmentation of skin and hair. Which micronutrient deficiency is most likely? (Q844267)

A. Zinc

B. Fluoride

C. Copper

D. Selenium

See Answer in Part B → Q38

@Neet_pg_bot

Q39: A patient presents with follicular hyperkeratosis on the extensor aspect of the forearm and has a dietary history of Vitamin A and C deficiency. What is the diagnosis? (Q844247)

A. Phrynoderma

B. Scurvy

C. Pellagra

D. Keratosis pilaris

See Answer in Part B → Q39

@Neet_pg_bot

Q40: Which micronutrient deficiency causes anemia, bleeding gums, and Frenkel's white line on X-ray? (Q844253)

A. Vitamin K

B. Vitamin A

C. Vitamin C

D. Vitamin E

See Answer in Part B → Q40

@Neet_pg_bot

Q41: A 45-year-old patient with a known case of homocystinuria presents with joint pain and weakness. Which vitamin is required for the treatment? (Q844246)

A. Vitamin B6

B. Vitamin B12

C. Vitamin B1

D. Vitamin B7

See Answer in Part B → Q41

@Neet_pg_bot

Q42: Which deficiency is most likely in a patient with delayed wound healing and alopecia? (Q852568)

A. Zinc

B. Copper

C. Calcium

D. Vitamin D

See Answer in Part B → Q42

@Neet_pg_bot

Q43: Which of the following is a mitochondrial matrix enzyme? (Q860380)

A. Citrate synthase

B. Monoamine oxidase

C. ATP synthase

D. Catalase

See Answer in Part B → Q43

@Neet_pg_bot

Q44: A baby presents with the clinical features shown in the image, including characteristic perioral and perianal rash along with a history of diarrhea. Which of the following is the most likely deficiency responsible for this condition? (Q852463)

A. Zinc Deficiency

B. Niacin Deficiency

C. Selenium Deficiency

D. Magnesium Deficiency



See Answer in Part B → Q44

@Neet_pg_bot

Q45: In a patient who presents with Phrynoderma as shown. What is the next best step in management of this patient? (Q866335)

A. Ocular examination

B. Check vitamin D levels

C. Check vitamin B12 levels

D. Check for DTR



See Answer in Part B → Q45

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Q46: A patient on long-term hydrochlorothiazide therapy presents with features of neuropathy, heart failure, and symmetrical tingling sensations. Which of the following nutrient deficiencies is most likely responsible? (Q866326)

A. Selenium

B. Thiamine

C. Vitamin B12

D. Zinc

See Answer in Part B → Q46

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Q47: A patient presents with anemia, positive Romberg sign, and other neurological findings suggestive of vitamin B12 deficiency. Laboratory findings show elevated

homocysteine levels. Which amino acid is likely to be deficient in this patient? (Q866321)

A. Cysteine

B. Methionine

C. Tyrosine

D. Glutamate

See Answer in Part B → Q47

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Q48: A person with jaundice presented with a history of bleeding, and symptoms normalised after giving a Vitamin K injection. The probable cause is? (Q866316)

A. Biliary obstruction

B. Alpha antitrypsin deficiency

C. Autoimmune hepatitis

D. Factor XI deficiency

See Answer in Part B → Q48

@Neet_pg_bot

Q49: A child presents with a history of fractures, multiple petechiae, perifollicular hemorrhages, and gum bleeding. Which of the following enzyme defects is involved? (Q866315)

A. Lysyl oxidase

B. Prolyl hydroxylase

C. Tyrosinase

D. Alkaline phosphatase

See Answer in Part B → Q49

@Neet_pg_bot

Q50: Which vitamin deficiency can lead to lactic acidosis? (Q866652)

A. Thiamine

B. Riboflavin

C. Niacin

D. Biotin

See Answer in Part B → Q50

@Neet_pg_bot

Q51: Features of Korsakoff Psychosis are all except Amnesia Confabulation Opthalmoplegia Polyneuropathy (Q839944)

A. 1, 2,3

B. 2,3,4

C. 3

D. 3,4

See Answer in Part B → Q51

@Neet_pg_bot

Q52: Which of the following statements is not true regarding folate deficiency? (Q839953)

A. Folate supplementation in pregnancy prevents NTD

B. High Homocysteine and Normal Methylmalonic acid

C. Hemolytic anemia is a feature

D. Treat coexisting B12 deficiency before initiating folate therapy

See Answer in Part B → Q52

@Neet_pg_bot

Q53: The image illustrates which vitamin deficiency? (Q860071)

A. Vitamin A

B. Vitamin E

C. Vitamin C

D. Niacin (Vitamin B3) Deficiency



See Answer in Part B → Q53

@Neet_pg_bot

Q54: A chronic alcoholic patient presents with agitation, confusion, and gait abnormalities. What vitamin deficiency is most likely responsible for these symptoms? (Q860084)

A. Nicotinamide (Vitamin B3) deficiency

B. Folate (Vitamin B9) deficiency

C. Methylcobalamin (Vitamin B12) deficiency

D. Tyrosinase

See Answer in Part B → Q54

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Q55: Which of the following manifestations is associated with Vitamin A deficiency? (Q860087)

A. Vogt's striae

B. Bitot's spots

C. Arlt's line

D. Horner's dots

See Answer in Part B → Q55

@Neet_pg_bot

Q56: A 50-year-old man with a history of chronic alcoholism presents with skin lesions. His diet primarily consists of polished rice. Based on his symptoms and dietary history, what is the most likely diagnosis? (Q860102)

A. Phrynoderma

B. Beriberi

C. Psoriasis

D. Eczema

See Answer in Part B → Q56

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Q57: Which of the following zymogens requires gamma-glutamyl carboxylase? (Q855186)

A. Factors II, VIII, IX, X

B. Factors II, VII, IX, and X

C. Factors II, VII, IX, XI

D. Factors II, VIII, X, XI

See Answer in Part B → Q57

@Neet_pg_bot

Q58: A patient presented with diarrhea, dermatitis, and dementia. Which of the following vitamin deficiencies would you suspect in this patient? (Q855190)

A. Vitamin B3 deficiency

B. Vitamin B1 deficiency

C. Vitamin B6 deficiency

D. Vitamin B12 deficiency

See Answer in Part B → Q58

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Q59: A family consumes only polished rice. Which of the following combinations of vitamin deficiency and enzymatic defect will be present in this family? (Q855197)

A. Riboflavin-glutathione reductase

B. Thiamine-transketolase

C. Thiamine-transaminase

D. Riboflavin-transketolase

See Answer in Part B → Q59

@Neet_pg_bot

Q60: A child was brought with pedal edema and cheilosis.Cardiomegaly was present. What is the vitamin deficiency associated with this clinical presentation? (Q855227)

A. Riboflavin

B. Thiamine

C. Pyridoxine

D. Niacin

See Answer in Part B → Q60

@Neet_pg_bot

Q61: A patient presenting with bleeding gums and easy bruisability was diagnosed with scurvy. This is due to (Q855238)

A. Low calcium

B. Defective collagen formation

C. Inhibition of clotting factors

D. Increased keratinization of epithelium

See Answer in Part B → Q61

@Neet_pg_bot

Q62: A child presents with thinning of hair and scaling of the scalp. On taking further history, it is found that she consumes raw eggs in her diet regularly. The given

manifestations are due to the deficiency of which of the following vitamins? (Q855283)

A. Niacin

B. Pyridoxine

C. Biotin

D. Thiamine

See Answer in Part B → Q62

@Neet_pg_bot

Q63: What are the consequences of a deficiency in Vitamin A? (Q857843)

A. Beriberi

B. Pellagra

C. Xerophthalmia

D. Neuropathy

See Answer in Part B → Q63

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Q64: A 42-year-old female came to the emergency department with a bleeding condition and dental caries. Which vitamin supplementation from the list below would be best for treating this condition? (Q857896)

A. Vitamin B1

B. Vitamin B9

C. Vitamin D

D. Vitamin K

See Answer in Part B → Q64

@Neet_pg_bot

Q65: Due to a prolonged period of consuming maize as a main food source, a 34-year-old man developed diarrhoea and exhibited lesions on his neck, as depicted in the accompanying image. This condition is caused by a deficiency in: (Q793361)

A. Niacin

B. Zinc

C. Thiamine

D. Iron



See Answer in Part B → Q65

@Neet_pg_bot

Q66: A 42-year-old female came to the emergency department with a bleeding condition and dental caries. Which vitamin supplementation from the list below would be best for treating this condition? (Q820421)

A. Vitamin B1

B. Vitamin B9

C. Vitamin D

D. Vitamin K

See Answer in Part B → Q66

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• [12_Heme Synthesis] 032 Heme Synthesis And Porphyrias

« Back to Folder Directory

[12_Heme Synthesis] 032 Heme Synthesis And Porphyrias — Quick Jump

Q1 (p. 405)	*Q2 (p. 405)*	*Q3 (p. 406)*	*Q4 (p. 407)*	*Q5 (p. 407)*	*Q6 (p. 408)*
Q7 (p. 408)	*Q8 (p. 408)*	*Q9 (p. 409)*	*Q10 (p. 409)*	*Q11 (p. 410)*	*Q12 (p. 411)*
Q13 (p. 411)	*Q14 (p. 411)*	*Q15 (p. 412)*	*Q16 (p. 412)*	*Q17 (p. 412)*	*Q18 (p. 413)*
Q19 (p. 413)	*Q20 (p. 413)*	*Q21 (p. 414)*			

Q1: A 7-year-old boy presents with photosensitivity, blistering skin lesions on sun exposure, and dark red urine. The parents are paint factory workers living in an industrial complex. Labs show elevated porphyrins. Which of the following statements about the diagnosis is incorrect? (Q844899)

A. The condition can occur due to exposure to lead, leading to decreased activity of ALA synthase.

B. The red fluorescence of the teeth under UV light is referred to as erythrodontia, due to emission of 600 nm wavelength by porphyrin.

C. Porphyrin has a specific absorption band called Soret band corresponding to 400 nm.

D. Photosensitivity due to the accumulation of porphyrins in the skin which release superoxide radicals causing photooxidative damage.

See Answer in Part B → Q1

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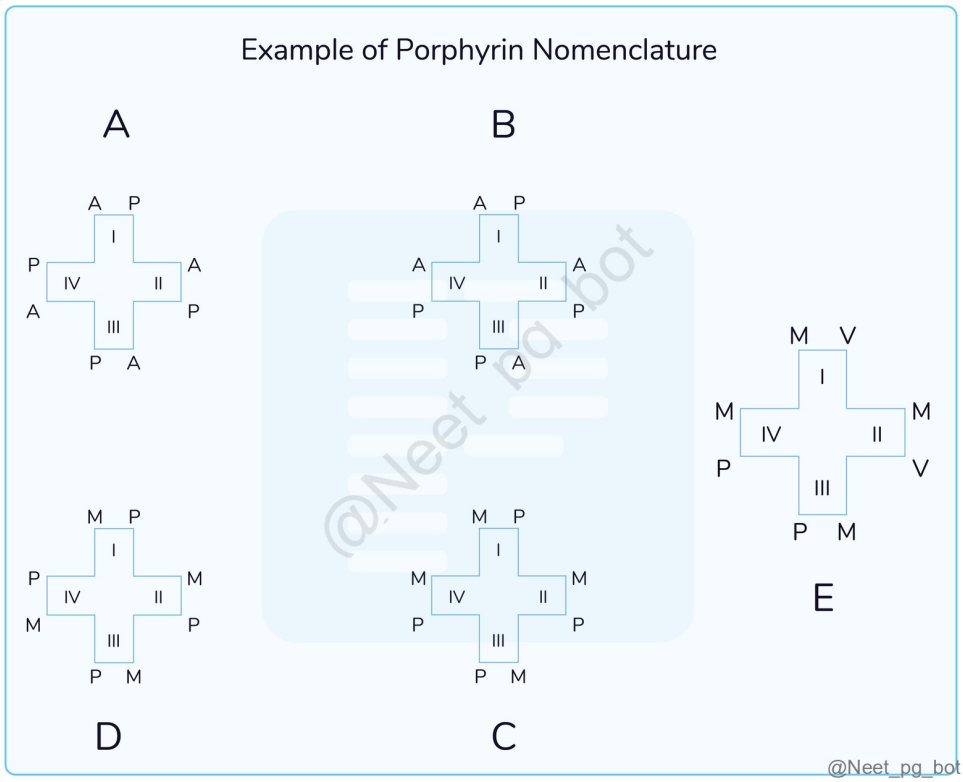
Q2: Which of the following structures correctly represents the type of porphyrin? A= acetyl, P=propionyl, M=methyl, V=vinyl (Q844901)

A. A- Uroporphyrinogen I, B- Uroporphyrinogen III, E- Protoporphyrinogen I

B. D- Coproporphyrinogen III, C- Coproporphyrinogen I, E- Protoporphyrinogen I

C. A- Uroporphyrinogen III, B- Uroporphyrinogen I, E- Protoporphyrinogen III

D. D- Coproporphyrinogen I, C- Coproporphyrinogen III, E- Protoporphyrinogen III



See Answer in Part B → Q2

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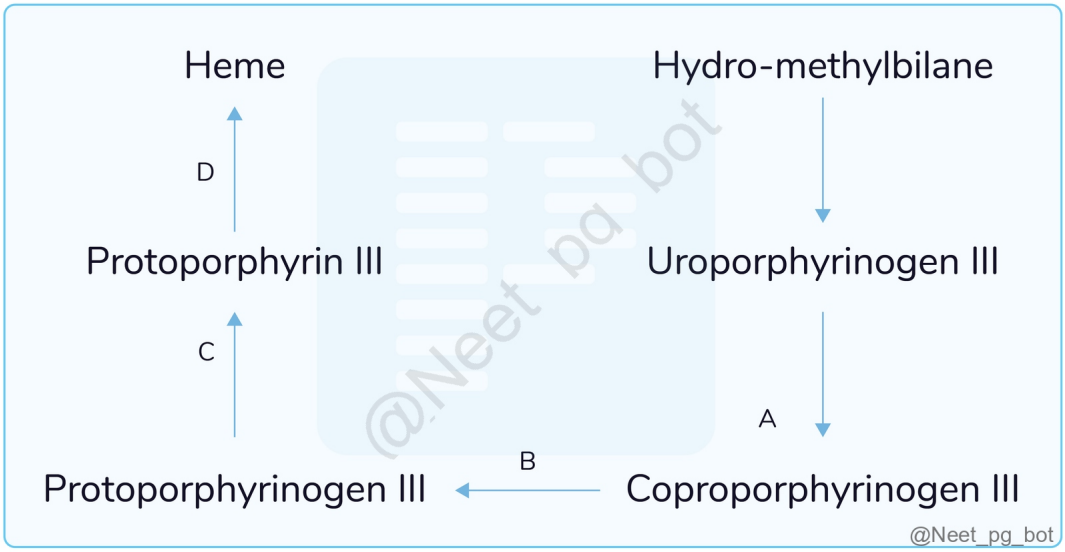
Q3: According to the given image of heme synthesis, which of the following statements is incorrect? (Q844903)

A. Steps C and D occur in Mitochondria, while steps A and B occur in the cytosol.

B. The enzyme involved in step A is Uroporphyrinogen decarboxylase

C. 4 molecules of CO2 are liberated at step A

D. Ferrochelatase is the enzyme involved in step D



See Answer in Part B → Q3

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Q4: Which of the following statements about heme synthesis is incorrect? (Q844904)

A. ALA synthase requires pyridoxal phosphate as a cofactor.

B. ALA dehydratase contains zinc as a cofactor and is inhibited by lead.

C. Pyridoxal phosphate deficiency can lead to anemia due to impaired ALA synthase activity.

D. ALA dehydratase is the rate-limiting step of the heme synthesis pathway.

See Answer in Part B → Q4

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Q5: A 35-year-old male presents with microcytic anemia and elevated delta-aminolevulinic acid (ALA). Which statement correctly describes the regulation of ALA synthase I (ALAS I) and ALA synthase II (ALAS II)? (Q844905)

A. ALAS I is primarily expressed in erythroid tissues and regulated by iron.

B. ALAS II is ubiquitously expressed and inhibited by heme.

C. ALAS I is inhibited by heme and primarily expressed in the liver.

D. ALAS II is regulated by heme and is key in hepatic heme synthesis.

See Answer in Part B → Q5

@Neet_pg_bot

Q6: A 25-year-old man with microcytic anemia, high iron and ferritin, and ringed sideroblasts has an ALAS2 gene mutation. What is the most likely diagnosis? (Q844906)

A. Iron deficiency anemia

B. Thalassemia

C. X-linked sideroblastic anemia

D. Autosomal recessive sideroblastic anemia

See Answer in Part B → Q6

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Q7: Match the following disorders with the enzyme defects in context to relation synthesis. Enzyme defect Disorder A. Uroporphyrinogen III synthase i) Porphyria cutanea tarda B. Uroporphyrinogen decarboxylase defect ii) Congenital erythropoietic porphyria C. ALA synthase II iii) Acute intermittent porphyria D. Porphobilinogen deaminase iv) X-linked sideroblastic anemia (Q844907)

A. A-ii, B-i, C-iv, D-iii

B. A-iii, B-iv, C-ii, D-i

C. A-iv, B-iii, C-i, D-ii

D. A-i, B-ii, C-iv, D-iii

See Answer in Part B → Q7

@Neet_pg_bot

Q8: Match the following disorder with its clinical classification: Disorder Clinical Manifestations A.Acute intermittent porphyria i) Neuropsychiatric manifestations B.Porphyria Cutanea Tarda ii) Photosensitivity C.Hereditary coproporphyria iii) Both Neuropsychiatric manifestation and photosensitivity D.Congenital erythropoietic porphyria (Q844908)

A. A-i, B-ii, C-iii, D-ii

B. A-i, B-ii, C-ii, D-iii

C. A-ii, B-i, C-iii, D-iii

D. A-ii, B-i, C-ii, D-iii

See Answer in Part B → Q8

@Neet_pg_bot

Q9: Lead poisoning can cause acquired porphyria by inhibiting several enzymes in the heme synthesis pathway. Which of the following enzymes is not affected by lead poisoning? (Q844909)

A. ALA dehydratase

B. Coproporphyrinogen oxidase

C. Ferrochelatase

D. ALA synthase

See Answer in Part B → Q9

@Neet_pg_bot

Q10: Which of the following statements about the protoporphyrin ring and its associated metals is incorrect? (Q844898)

A. Heme is a derivative of porphyrin, a cyclic compound formed by the fusion of 4 pyrrole rings with a metallic group in the center

B. Heme-containing proteins such as myoglobin have Fe3+ as the metallic group.

C. Heme-containing enzymes are catalase, peroxidase, and nitric oxide synthase.

D. The metallic group in Chlorophyll is magnesium, while in cytochrome a/a3 is copper.

See Answer in Part B → Q10

@Neet_pg_bot

Q11: An infant presents with severe photosensitivity and recurrent blistering of sun-exposed skin. Parents also report pink-colored urine since birth. On examination, the teeth appear as shown in the image. The most likely diagnosis is: (Q891737)

A. Congenital erythropoietic porphyria (Gunther disease)

B. Erythropoietic protoporphyria

C. Porphyria cutanea tarda

D. Acute intermittent porphyria



See Answer in Part B → Q11

@Neet_pg_bot

Q12: Which porphyria is not classified as a hepatic porphyria? (Q891734)

A. Acute intermittent porphyria

B. Porphyria cutanea tarda

C. Hereditary coproporphyria

D. Erythropoietic protoporphyria

See Answer in Part B → Q12

@Neet_pg_bot

Q13: An adult female presents with recurrent episodes of abdominal pain, nausea, constipation, tachycardia, hypertension, and peripheral neuropathy. Urine darkens on standing. Elevated urinary ALA and PBG are found. The most likely diagnosis is: (Q891733)

A. Hereditary coproporphyria

B. Variegate porphyria

C. Porphyria cutanea tarda

D. Acute intermittent porphyria (AIP)

See Answer in Part B → Q13

@Neet_pg_bot

Q14: Which porphyria is not inherited in an autosomal dominant pattern? (Q891731)

A. Acute intermittent porphyria

B. Variegate porphyria

C. Hereditary coproporphyria

D. Erythropoietic protoporphyria

See Answer in Part B → Q14

@Neet_pg_bot

Q15: Cyclisation of hydroxymethylbilane to the first porphyrinogen (uroporphyrinogen III) is catalysed by which enzyme? (Q891729)

A. Uroporphyrinogen III synthase

B. Uroporphyrinogen decarboxylase

C. Coproporphyrinogen oxidase

D. ALA dehydratase

See Answer in Part B → Q15

@Neet_pg_bot

Q16: The by-product released during the condensation of porphobilinogen to hydroxymethylbilane by hydroxymethylbilane synthase (PBG deaminase) is: (Q891728)

A. CO■

B. NH■

C. H■O

D. Glutamate

See Answer in Part B → Q16

@Neet_pg_bot

Q17: What is the mechanism by which barbiturates worsen porphyria? (Q891719)

A. Inhibition of ferrochelatase

B. Activation of ALA dehydratase

C. Induction (de-repression) of ALA synthase

D. Inhibition of uroporphyrinogen decarboxylase

See Answer in Part B → Q17

@Neet_pg_bot

Q18: The conversion of succinyl-CoA and glycine to α-amino-β-ketoadipate requires which cofactor? (Q891718)

A. Thiamine pyrophosphate

B. Pyridoxal phosphate (Vitamin B■)

C. Biotin

D. FAD

See Answer in Part B → Q18

@Neet_pg_bot

Q19: Succinyl-CoA, an essential substrate for heme biosynthesis, is derived from which pathway? (Q891717)

A. Glycolysis

B. Fatty acid oxidation

C. TCA cycle (Krebs cycle)

D. Urea cycle

See Answer in Part B → Q19

@Neet_pg_bot

Q20: Which of the following is not a site for heme biosynthesis? (Q891715)

A. Erythroid precursor cells in bone marrow

B. Hepatocytes

C. Mature erythrocytes

D. Mitochondria

See Answer in Part B → Q20

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Q21: A patient with anaemia has elevated levels of 2,3-Bisphosphoglycerate (2,3-BPG) in their red blood cells. How does this increased 2,3-BPG level affect the patient's haemoglobin in terms of oxygen delivery? (Q875968)

A. Forms salt bridges with positively charged residues forming a relaxed structure

B. Disrupts salt bridges formed in the taut structure, decreasing oxygen affinity

C. Forms salt bridges with positively charged residues stabilising taut structure

D. Binds to the heme directly, decreasing the oxygen affinity

See Answer in Part B → Q21

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• [12_Heme Synthesis] 033 Previous Year Questions

« Back to Folder Directory

[12_Heme Synthesis] 033 Previous Year Questions — Quick Jump

Q1 (p. 415) *Q2 (p. 416)* *Q3 (p. 416)* *Q4 (p. 417)* *Q5 (p. 417)* *Q6 (p. 418)*
Q7 (p. 418) *Q8 (p. 418)* *Q9 (p. 419)* *Q10 (p. 419)* *Q11 (p. 419)* *Q12 (p. 420)*
Q13 (p. 420)

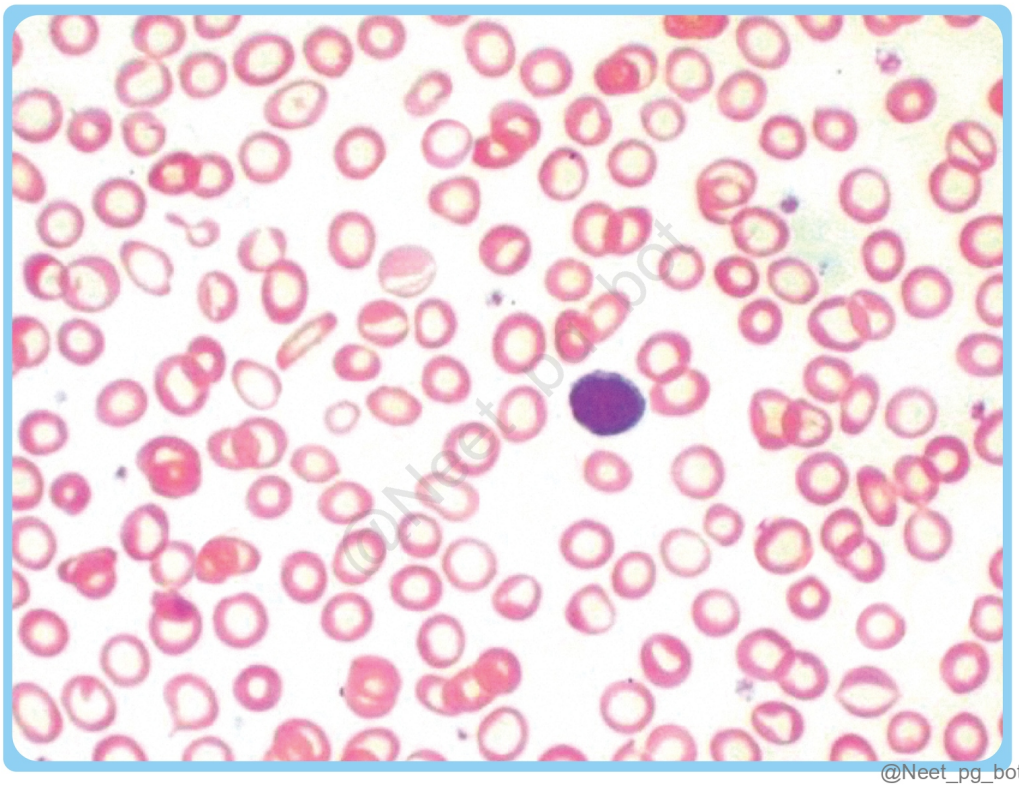
Q1: A 7-year-old child is brought to the clinic with complaints of fatigue and poor concentration. The mother reports that the child has been eating non-food items such as chalk and soil for the past few months. A peripheral blood smear image shows microcytic, hypochromic red blood cells. Which of the following is the most likely diagnosis? (Q866661)

A. Vitamin B12 deficiency

B. Iron deficiency anemia

C. Thalassemia major

D. Lead poisoning



See Answer in Part B → Q1

@Neet_pg_bot

Q2: A patient's relatives sent a message on social media to the consulting doctor. He is unaware of the other investigations. However, he mentioned that urinary Coproporphyrin is positive. What is the probable cause? (Q844265)

A. Lead poisoning

B. Asbestosis

C. Silicosis

D. Mercury poisoning

See Answer in Part B → Q2

@Neet_pg_bot

Q3: Which of the following is not a product of heme breakdown? (Q839941)

A. Biliverdin

B. Carbon monoxide

C. Ferric Ion

D. Aminolevulinic acid

See Answer in Part B → Q3

@Neet_pg_bot

Q4: Which of the following is the rate-limiting enzyme of heme synthesis pathway? (Q820401)

A. HMB synthase

B. δ Amino-Levulinic Acid dehydratase

C. δ Amino-Levulinic Acid synthase

D. Ferrochelatase

See Answer in Part B → Q4

@Neet_pg_bot

Q5: What plasma protein is found in the highest concentration? (Q793458)

A. Bence jones protein

B. Globulin

C. Fibrinogen

D. Albumin

See Answer in Part B → Q5

@Neet_pg_bot

Q6: What is the most appropriate dietary recommendation for a middle-aged man diagnosed with a rare variant of pyruvate dehydrogenase deficiency, resulting in high anion gap metabolic acidosis? (Q793470)

A. High protein and low carbohydrate intake

B. High carbohydrate and low fat intake

C. High fat and low carbohydrate intake

D. Only carbohydrate intake henceforth

See Answer in Part B → Q6

@Neet_pg_bot

Q7: Heme is derived from which of the following amino acids? (Q860419)

A. Glutamine

B. Glycine

C. Glutamate

D. Valine

See Answer in Part B → Q7

@Neet_pg_bot

Q8: Which of the following amino acids are necessary for purine ring formation? (Q860424)

A. Glycine, aspartate, valine

B. Glycine, Aspartate, Glutamine

C. Glycine, Aspartate, Glutamate

D. Glycine, Asparagine, Glutamine

See Answer in Part B → Q8

@Neet_pg_bot

Q9: Decreased % transferrin saturation is seen in? (Q854613)

A. Hemochromatosis

B. Iron deficiency anemia

C. Megaloblastic anemia

D. Hemosiderosis

See Answer in Part B → Q9

@Neet_pg_bot

Q10: Which part of the haemoglobin molecule does 2,3-Bisphosphoglycerate (2,3-BPG) bind to in order to regulate oxygen release? (Q854619)

A. Alpha chains of haemoglobin

B. Heme iron centre

C. Beta chains of haemoglobin

D. Globin-heme interface

See Answer in Part B → Q10

@Neet_pg_bot

Q11: What does iron deficiency anemia (IDA) stand for? (Q854624)

A. Low serum ferritin

B. Low transferrin saturation

C. Increased TIBC

D. All of the above

See Answer in Part B → Q11

@Neet_pg_bot

Q12: Which of the following is the rate-limiting enzyme of heme synthesis pathway? (Q857891)

A. HMB synthase

B. δ Amino-Levulinic Acid dehydratase

C. δ Amino-Levulinic Acid synthase

D. Ferrochelatase

See Answer in Part B → Q12

@Neet_pg_bot

Q13: A female is diagnosed with lead poisoning . Which of the following enzyme defects is primarily responsible for the hematologic abnormalities observed in this condition? (Q857898)

A. ALA synthase

B. Uroporphyrinogen decarboxylase

C. Heme synthase

D. Coproporphyrinogen oxidase

See Answer in Part B → Q13

@Neet_pg_bot

• [13_Amino Acid and Protein Metabolism] 034 Previous Year Questions

« Back to Folder Directory

[13_Amino Acid and Protein Metabolism] 034 Previous Year Questions — Quick Jump

Q1 (p. 421) *Q2 (p. 421)* *Q3 (p. 421)* *Q4 (p. 422)* *Q5 (p. 422)* *Q6 (p. 423)*

Q1: Which of the following is deficient in a patient with phenylketonuria with normal phenyl alanine hydroxylase levels? (Q844228)

A. Tyrosine transaminase

B. Dopa decarboxylase

C. Tyrosine hydroxylase

D. Dihydrobiopterine reductase

See Answer in Part B → Q1

@Neet_pg_bot

Q2: A patient presents with dermatitis, dementia, and cognitive decline. Which micronutrient deficiency is most likely responsible? (Q844235)

A. Niacin

B. Thiamine

C. Tryptophan

D. Riboflavin

See Answer in Part B → Q2

@Neet_pg_bot

Q3: What is true about the Hayflick limit? (Q856475)

A. Human cells can divide a finite number of times only

B. Free radical oxidation level is unrestricted

C. Telomerase activity is unrestricted

D. RNA polymerase can function for a finite number of time only

See Answer in Part B → Q3

@Neet_pg_bot

Q4: A female patient presented with pain and redness in the great toe. The serum uric acid level is 9.4mg/dl. The physician prescribed a drug for the treatment. Which of the following enzyme is likely to be inhibited by this drug? (Q859691)

A. Phosphoribosyl transferase

B. Thymidylate synthase

C. Xanthine oxidase

D. Dihydrofolate reductase

See Answer in Part B → Q4

@Neet_pg_bot

Q5: Which of the following post-translational modifications is common to collagen, hemoglobin, and immunoglobulins? (Q860100)

A. Hydroxylation

B. Subunit aggregation

C. Poly A tail addition

D. DNA mismatch repair

See Answer in Part B → Q5

@Neet_pg_bot

Q6: A 2-month-old infant presents with poor feeding, vomiting, and lethargy. Laboratory studies show hyperammonemia and elevated levels of orotic acid in the urine. Which of the following is the most likely enzyme deficiency? (Q866671)

A. Carbamoyl phosphate synthetase I deficiency

B. Ornithine transcarbamylase (OTC) deficiency

C. Orotidine 5'-phosphate decarboxylase deficiency

D. Argininosuccinate lyase deficiency

See Answer in Part B → Q6

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• [14_Clinical Biochemistry] 035 Previous Year Questions

« Back to Folder Directory

[14_Clinical Biochemistry] 035 Previous Year Questions — Quick Jump

Q1 (p. 424)	*Q2 (p. 424)*	*Q3 (p. 425)*	*Q4 (p. 425)*	*Q5 (p. 425)*	*Q6 (p. 426)*
Q7 (p. 426)	*Q8 (p. 426)*	*Q9 (p. 427)*	*Q10 (p. 427)*	*Q11 (p. 428)*	*Q12 (p. 428)*
Q13 (p. 428)	*Q14 (p. 429)*	*Q15 (p. 429)*	*Q16 (p. 430)*	*Q17 (p. 430)*	*Q18 (p. 430)*
Q19 (p. 431)	*Q20 (p. 431)*	*Q21 (p. 431)*	*Q22 (p. 432)*	*Q23 (p. 432)*	*Q24 (p. 432)*
Q25 (p. 433)	*Q26 (p. 434)*				

Q1: Which of the following uses CRISPR-Cas9 technology? (Q860081)

A. DNA Editing

B. Post-transcriptional Modification

C. DNA Induction

D. DNA Editing Solution

See Answer in Part B → Q1

@Neet_pg_bot

Q2: A child presents with abdominal distension and an enlarged liver by 8 cm below the costal margin. The liver is smooth on palpation. Which of the following is the most likely diagnosis? (Q866675)

A. Glycogen Storage Disease (GSD)

B. Lysosomal Storage Disease (LSD)

C. Hepatocellular Carcinoma (HCC)

D. Autoimmune Hepatitis

See Answer in Part B → Q2

@Neet_pg_bot

Q3: A patient presents with fatigue, anemia, and hepatomegaly. Hemoglobin electrophoresis reveals the presence of HbS. What will be the appropriate treatment? (Q866656)

A. Hydroxyurea

B. Blood transfusion

C. Iron supplementation

D. Vitamin B12 supplementation

See Answer in Part B → Q3

@Neet_pg_bot

Q4: A neonate presents with seizures and is found to have a cherry red spot on fundus examination. Enzyme assay reveals deficiency of hexosaminidase A. Which of the following substances is most likely to be accumulated in this patient? (Q866323)

A. GM1 ganglioside

B. GM2 ganglioside

C. Sphingomyelin

D. Glucocerebroside

See Answer in Part B → Q4

@Neet_pg_bot

Q5: In uncontrolled diabetes, in which of the following cells there is decreased glucose uptake due to insulin deficiency: (Q865446)

A. RBC

B. Skeletal muscle

C. Liver

D. Brain

See Answer in Part B → Q5

@Neet_pg_bot

Q6: For mycobacterium species differentiation, technique used is/are? Line probe assay MIRU (Mycobacterium Interspersed Rapatitive Units) Spoligotyping RFLP (Q865405)

A. 1, 2, 3

B. 2, 3, 4

C. 1, 2, 4

D. 1, 2, 3, 4

See Answer in Part B → Q6

@Neet_pg_bot

Q7: Which of the following is caused by the deficiency of the enzyme tyrosine transaminase? (Q860430)

A. Type I Tyrosinemia

B. Type II Tyrosinemia

C. Alkaptonuria

D. Type III Tyrosinemia

See Answer in Part B → Q7

@Neet_pg_bot

Q8: An infant presents to the Pediatrics OPD with a distended abdomen and jaundice. The mother reports that the baby has shown an increased appetite in recent months. During the clinical examination, the physician notes hepatomegaly. Which of the

following enzyme deficiencies is most likely responsible for this condition? (Q860357)

A. Glycogen Synthase

B. Glycogen phosphorylase

C. Glucose-6-Phosphatase

D. Galactose-1-Phosphate Uridyl Transferase

See Answer in Part B → Q8

@Neet_pg_bot

Q9: A 1-month-old infant presents with recurrent vomiting, diarrhea, jaundice, and failure to thrive shortly after breastfeeding began. Examination shows hepatomegaly, and urine tests are positive for reducing substances but negative for glucose. What is the most likely diagnosis? (Q860334)

A. Lactose Intolerance

B. Duodenal Atresia

C. Galactosemia

D. Pyloric Stenosis

See Answer in Part B → Q9

@Neet_pg_bot

Q10: An infant presents with distended abdomen, jaundice, increased appetite, and hepatomegaly. Which enzyme deficiency is most likely responsible for this condition? (Q860329)

A. Glucose-6-phosphatase

B. Glycogen phosphorylase

C. Galactose-1-phosphate uridyl transferase

D. Glycogen synthase

See Answer in Part B → Q10

@Neet_pg_bot

Q11: A 7-year-old child of short stature presents with skin patches and learning difficulties. Diagnostic workup indicates a defect in DNA repair specifically related to double-strand breaks. What is the most likely diagnosis? (Q860325)

A. Bloom Syndrome

B. Xeroderma Pigmentosum

C. Cockayne Syndrome

D. Lynch Syndrome

See Answer in Part B → Q11

@Neet_pg_bot

Q12: A 16-year-old patient presents with pellagra-like skin eruptions during the spring season for the past 5 years. Laboratory tests reveal normal levels of 5-Hydroxyindoleacetic Acid (HIAA), high aminoaciduria, and normal urinary indole levels. What is the most likely diagnosis? (Q860099)

A. Carcinoid tumor

B. Nutritional pellagra

C. Hartnup disorder

D. Essential fructosuria

See Answer in Part B → Q12

@Neet_pg_bot

Q13: Which of the following substances is used in a tube for collecting blood samples specifically for measuring alcohol concentration? (Q860092)

A. Heparin + EDTA

B. Sodium fluoride + Potassium dichromate

C. Heparin

D. Sodium citrate

See Answer in Part B → Q13

@Neet_pg_bot

Q14: A 45-year-old man presents with deep labored breathing and altered sensorium. His arterial blood gas shows a metabolic acidosis. The following serum electrolyte values are obtained: Na■: 145 mmol/L K■: 4 mmol/L Cl■: 90 mmol/L HCO■■: 15 mmol/L Calculate the anion gap (AG) and interpret the finding. (Q854606)

A. 16 mmol/L – Normal anion gap metabolic acidosis

B. 40mmol/L – High anion gap metabolic acidosis

C. 14 mmol/L – Normal anion gap metabolic acidosis

D. 32 mmol/L – Mixed metabolic acidosis

See Answer in Part B → Q14

@Neet_pg_bot

Q15: Which of the following disorders is characterized by the presence of a cherry red spot on the retina? (Q860068)

A. Tay-Sachs disease

B. Fabry's disease

C. Gaucher's disease

D. Von Gierke's disease

See Answer in Part B → Q15

@Neet_pg_bot

Q16: A 40-year-old female working as a senior executive leading a sedentary lifestyle presents to the OPD with palmoplantar pigmentation. X-ray spine shows calcification of intervertebral discs. On adding Benedict's reagent to the urine sample, it gives a greenish-brown precipitate and comes positive. What is the most likely enzyme deficiency in this patient? (Q860064)

A. Phenylalanine hydroxylase

B. Tyrosine hydroxylase

C. Homogentisic acid oxidase

D. PHPP dioxygenase

See Answer in Part B → Q16

@Neet_pg_bot

Q17: A 3-month-old baby presents with severe hepatomegaly, cataracts in both eyes, lethargy, and hypotonia. Based on these symptoms, which enzyme deficiency is most likely involved? (Q860054)

A. Galactokinase

B. Hepatic Kinase

C. Hepatic Glucose-6-Phosphatase

D. Galactose-1-Phosphate Uridyl Transferase

See Answer in Part B → Q17

@Neet_pg_bot

Q18: A couple brings their 4-year-old child to the outpatient department (OPD) due to self-mutilating behavior and poor growth. Which of the following associated enzyme deficiencies could explain these symptoms? (Q860052)

A. HGPRT deficiency

B. Homogentisic Aciduria

C. Phenylalanine Hydroxylase deficiency

D. Adenosine deaminase deficiency

See Answer in Part B → Q18

@Neet_pg_bot

Q19: Zellweger syndrome is associated with which cellular organelle? (Q860043)

A. Peroxisomes

B. Nucleosomes

C. Lysosomes

D. Ribosomes

See Answer in Part B → Q19

@Neet_pg_bot

Q20: On centrifugation of a cell, LDH will get deposited in: (Q859706)

A. Mitochondria

B. Lysosome

C. Cytoplasm

D. Golgi complex

See Answer in Part B → Q20

@Neet_pg_bot

Q21: Which of the following enzyme defects leads to Pompe’s disease? (Q857351)

A. Lysosomal alpha glucosidase

B. β-hexosaminidase -A

C. β-galactocerebrosidase

D. β-glucocerebrosidase

See Answer in Part B → Q21

@Neet_pg_bot

Q22: Xanthurenic aciduria may be treated with high doses of which vitamin? (Q857350)

A. Vitamin B6

B. Vitamin B12

C. Vitamin B5

D. Vitamin B7

See Answer in Part B → Q22

@Neet_pg_bot

Q23: Deficiency of which of the following causes Barth’s syndrome? (Q857325)

A. Cardiolipin

B. Sphingomyelin

C. Glycolipids

D. Cerebroside

See Answer in Part B → Q23

@Neet_pg_bot

Q24: Liver produces ketones but cannot use it due to the deficiency of which of the following? (Q857321)

A. Thiophorase

B. Alkaline phosphatase

C. Alanine transaminase

D. Thoiolase

See Answer in Part B → Q24

@Neet_pg_bot

Q25: Identify the test: (Q854626)

A. Rothera’s test

B. Benedict’s test

C. Fehling’s test

D. Barfoed’s test



See Answer in Part B → Q25

@Neet_pg_bot

Q26: What happens to chloride levels in sweat and pancreatic secretions in cystic fibrosis? (Q854625)

A. Chloride is raised in sweat

B. Chloride is decreased in sweat

C. Chloride is decreased in pancreatic secretions

D. None of the above

See Answer in Part B → Q26

@Neet_pg_bot

• [15_Biochemistry] 036 Previous Year Questions

« Back to Folder Directory

[15_Biochemistry] 036 Previous Year Questions — Quick Jump

Q1 (p. 435) *Q2 (p. 435)*

Q1: In a case presented in the outpatient department (OPD), a patient reported experiencing abdominal pain and diarrhea. Upon further examination, the patient exhibited symptoms of cutaneous flushing, extensive wheezing, and tricuspid insufficiency. Subsequent contrast enhanced MRI scans indicated the presence of endocardial fibrosis. As a result of this condition, which substance is likely to be excreted excessively by the patient? (Q793465)

A. Vanillylmandelic acid

B. Imidazoleacetic acid

C. 5-hydroxy indole acetic acid

D. Choline

See Answer in Part B → Q1

@Neet_pg_bot

Q2: The best method for the detection of mutations with low allele frequency is: (Q855560)

A. Droplet digital PCR

B. FISH

C. Nested PCR

D. Sanger sequencing

See Answer in Part B → Q2

@Neet_pg_bot

• [16_Concepts] 037 Previous Year Questions

« Back to Folder Directory

[16_Concepts] 037 Previous Year Questions — Quick Jump

Q1 (p. 436) *Q2 (p. 436)* *Q3 (p. 436)* *Q4 (p. 437)* *Q5 (p. 437)* *Q6 (p. 438)* *Q7 (p. 438)*

Q1: Cholera is known to cause diarrhea through its interaction with specific receptors in the human body. Which of the following is known to make it easier for cholera toxin to enter the body through the intestinal mucosa? (Q820337)

A. GLUT 1

B. GM2 ganglioside

C. Cephalin

D. GM1 ganglioside

See Answer in Part B → Q1

@Neet_pg_bot

Q2: Which of the following minerals is best known for insulin secretion stabilisation? (Q820373)

A. Cadmium

B. Copper

C. Iron

D. Zinc

See Answer in Part B → Q2

@Neet_pg_bot

Q3: A 13-year-old boy who accompanied his father to a fish farming region in his hometown accidentally consumes a white odourless chemical. He was under some medication and post-meal he developed toxicity of the drug. Which of the the following toxin is most likely to cause this condition? (Q820415)

A. Alkyl guanides

B. BAL

C. Antimycin A

D. Strychnine

See Answer in Part B → Q3

@Neet_pg_bot

Q4: In which of the following locations can cardiolipin be found? (Q830710)

A. Outer mitochondrial membrane

B. Inner mitochondrial membrane

C. Cytosol

D. Mitochondrial matrix

See Answer in Part B → Q4

@Neet_pg_bot

Q5: What is the primary mechanism behind tissue damage in long-standing diabetes? (Q852558)

A. Insulin resistance

B. Sorbitol accumulation

C. Hyperglycemia

D. Decreased insulin secretion

See Answer in Part B → Q5

@Neet_pg_bot

Q6: A 13-year-old boy who accompanied his father to a fish farming region in his hometown accidentally consumes a white odourless chemical. He was under some medication and post-meal he developed toxicity of the drug. Which of the following toxin is most likely to cause this condition? (Q857895)

A. Alkyl guanides

B. BAL

C. Antimycin A

D. Strychnine

See Answer in Part B → Q6

@Neet_pg_bot

Q7: Complex IV is inhibited by --? (Q859423)

A. Rotenone

B. Cyanide

C. Carboxin

D. Antimycin A

See Answer in Part B → Q7

@Neet_pg_bot

• [17_Miscellaneous] 038 Previous Year Questions

« Back to Folder Directory

[17_Miscellaneous] 038 Previous Year Questions — Quick Jump

Q1 (p. 439)	*Q2 (p. 439)*	*Q3 (p. 439)*	*Q4 (p. 440)*	*Q5 (p. 441)*	*Q6 (p. 441)*
Q7 (p. 441)	*Q8 (p. 442)*	*Q9 (p. 442)*	*Q10 (p. 443)*	*Q11 (p. 443)*	*Q12 (p. 444)*
Q13 (p. 444)	*Q14 (p. 445)*	*Q15 (p. 445)*	*Q16 (p. 445)*	*Q17 (p. 446)*	

Q1: Nitrogenous waste is excreted from the body in the form of? (Q859710)

A. Urea

B. Nitric oxide

C. Ammonia

D. Glutamine

See Answer in Part B → Q1

@Neet_pg_bot

Q2: Which of the following substances, at the given concentration, makes water unsuitable for human consumption according to safety standards? (Q866677)

A. Fluoride – 0.8 mg/L

B. Cadmium – 0.3 mg/L

C. Chloride - 200 mg/L

D. Calcium – 7 mg/L

See Answer in Part B → Q2

@Neet_pg_bot

Q3: In a village of Assam, a man is observed walking with the help of a stick and has genu valgum. Many people in the same village present with similar symptoms. What is the most probable cause of this condition? (Q866664)

A. Calcium deficiency

B. Vitamin C deficiency

C. Skeletal fluorosis

D. Vitamin D deficiency (Osteomalacia)



See Answer in Part B → Q3

@Neet_pg_bot

Q4: A 5-year-old child is brought to the clinic with abdominal pain, irritability, and developmental delay. On examination, the child has pallor and a bluish line on the gums. Laboratory tests reveal microcytic anemia, and a peripheral blood smear shows red blood cells with basophilic stippling. Which of the following is the most likely diagnosis? (Q866662)

A. Iron deficiency anemia

B. Sideroblastic anemia

C. Lead poisoning

D. Thalassemia minor

See Answer in Part B → Q4

@Neet_pg_bot

Q5: If you need to diagnose a syndromic illness such as meningitis, which of the following diagnostic techniques is most appropriate? (Q866328)

A. Multiplex PCR

B. Uniplex PCR

C. Attributed PCR

D. Nested PCR

See Answer in Part B → Q5

@Neet_pg_bot

Q6: Cyanide and carbon monoxide act on which of the heme proteins: (Q865454)

A. Cytochrome c oxidase

B. Myoglobin

C. Catalase

D. NADH dehydrogenase

See Answer in Part B → Q6

@Neet_pg_bot

Q7: Ascent to high altitude causes ? (Q865438)

A. Metabolic acidosis

B. Metabolic alkalosis

C. Respiratory acidosis

D. Respiratory alkalosis

See Answer in Part B → Q7

@Neet_pg_bot

Q8: A child presents with hepatomegaly and a cherry-red spot on the macula. Neurological regression is also noted. These findings are suggestive of a lysosomal storage disorder. Which of the following enzyme deficiencies is most likely responsible? (Q865412)

A. Arylsulfatase A

B. Hexosaminidase A

C. Sphingomyelinase

D. Galactocerebrosidase

See Answer in Part B → Q8

@Neet_pg_bot

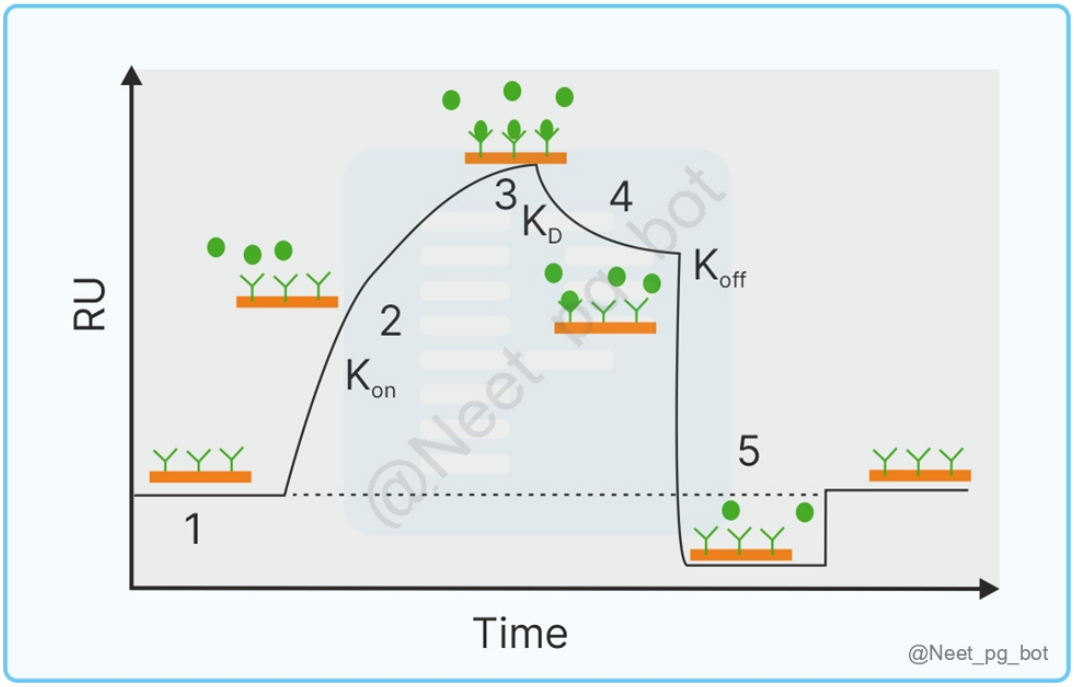
Q9: Name the graph and mention its application. (Q865396)

A. ELISA, Antibody concentration

B. ELISA, Antibody affinity

C. Plasma resonance, Ab concentration

D. Plasma resonance, Ab affinity



See Answer in Part B → Q9

@Neet_pg_bot

Q10: Which test should be done for detecting metabolite levels in inborn errors of metabolism in neonates? (Q839926)

A. Tandem mass spectroscopy

B. Complete blood count (CBC)

C. Urinalysis

D. Genetic testing

See Answer in Part B → Q10

@Neet_pg_bot

Q11: In a 14-year-old male, a medical condition has been identified as a result of a defect in a gene responsible for DNA repair. The individual is experiencing increased pigmentation on the skin and is at a heightened risk of developing skin cancers, according to the physician. Which of the following mechanisms is most likely to be

impaired in this particular condition? (Q859573)

A. Nonhomologous end joining repair defect

B. Nucleotide excision repair defect

C. DNA base excision repair defect

D. DNA mismatch repair defect

See Answer in Part B → Q11

@Neet_pg_bot

Q12: A child with mental retardation, blindness, and muscle weakness was investigated and was found to be deficient for Hexosaminidase-A enzyme. She is most likely suffering from .. (Q859572)

A. Krabbe's disease

B. Fabry's disease

C. Tay-sachs disease

D. Gaucher's disease

See Answer in Part B → Q12

@Neet_pg_bot

Q13: Nitric oxide is synthesized from (Q859428)

A. L-arginine

B. L-citrulline

C. Glycine

D. Lysine

See Answer in Part B → Q13

@Neet_pg_bot

Q14: What is the order of the band in the sarcomere? (Q854616)

A. I-Z-M-A-H

B. Z-H-M-A-I

C. Z-I-A-H-M

D. Z-H-I-A-M

See Answer in Part B → Q14

@Neet_pg_bot

Q15: The development of cataracts in patients with Diabetes Mellitus is primarily due to the accumulation of which of the following substances in the lens? (Q852565)

A. Sorbitol

B. Dulcitol

C. Mannitol

D. Galactitol

See Answer in Part B → Q15

@Neet_pg_bot

Q16: Which of the following enzymes is typically elevated in prostate cancer? (Q852562)

A. Acid phosphatase

B. Alkaline phosphatase

C. Lactate dehydrogenase

D. Alpha-fetoprotein

See Answer in Part B → Q16

@Neet_pg_bot

Q17: A patient presents with a history of vomiting. Arterial blood gas analysis reveals the following: pH : 7.5 pCO■ : 48 mm Hg HCO■■ : 30 mm Hg What is the most likely acid-base abnormality? (Q852561)

A. Metabolic alkalosis

B. Metabolic acidosis

C. Respiratory acidosis

D. Respiratory alkalosis

See Answer in Part B → Q17

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Part B — All Answers & Explanations

• [01_Introduction] 001 Previous Year Questions

« Back to Folder Directory

[01_Introduction] 001 Previous Year Questions — Quick Jump

Q1 (p. 447) *Q2 (p. 448)* *Q3 (p. 448)* *Q4 (p. 449)* *Q5 (p. 449)* *Q6 (p. 451)*
Q7 (p. 452) *Q8 (p. 452)* *Q9 (p. 453)* *Q10 (p. 454)* *Q11 (p. 455)* *Q12 (p. 456)*
Q13 (p. 457) *Q14 (p. 458)*

Q1: In which organelle is DNA present? (Q820383)

A. Mitochondria

B. Lysosome

C. Endoplasmic reticulum

D. Cytoplasm

Correct Answer: A. Mitochondria

Explanation: DNA is present in the mitochondria, which are double-membraned organelles found in the cells of most eukaryotic organisms. Mitochondrial DNA (mtDNA) is a small circular molecule separate from the nuclear DNA. It carries genetic information essential for the functioning of mitochondria and plays a crucial role in energy production through oxidative phosphorylation. Lysosome (Option B): Lysosomes are membrane-bound organelles containing enzymes responsible for breaking down various cellular waste materials and substances. They do not contain DNA; their primary function is related to digestion and waste disposal within the cell. Endoplasmic Reticulum (Option C): The endoplasmic reticulum (ER) is an organelle involved in the synthesis, folding, modification, and transport of proteins and lipids. It does not contain DNA. The ER is composed of membranous structures and is mainly responsible for the manufacturing and transportation of molecules within the cell. Cytoplasm (Option D): The cytoplasm is the gel-like substance found inside the cell, which includes various organelles and is the site of many cellular processes. While the cytoplasm contains various molecules and structures necessary for cell function, DNA itself is not typically present in the cytoplasm. DNA is primarily found within the nucleus of eukaryotic cells, where it houses the genetic information required for cell function and development.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: Zellweger syndrome results from abnormality in formation of: (Q855055)

A. Peroxisomes

B. Lysosomes

C. Mitochondria

D. Nucleus

Correct Answer: A. Peroxisomes

Explanation: Zellweger syndrome/Cerebrohepatorenal syndrome results from an abnormality in the formation of peroxisomes , which are cellular organelles responsible for various metabolic functions, including: Defective Peroxisomal Biogenesis: Mutations in PEX genes disrupt the assembly and function of peroxisomes. Key Consequence: Impaired breakdown of very long-chain fatty acids (VLCFAs), branched-chain fatty acids and other metabolic substrates leads to severe metabolic dysfunction. It is part of the peroxisome biogenesis disorder (PBD) spectrum. Accumulates C26-C38 polyenoic acids in brain tissue and exhibit generalised loss of peroxisomal functions. Management: Docosahexaenoic acid, Lorenzo oil and Cholic acid.

References:

www.ncbi.nlm.nih.gov/books/NBK560676/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q3: Refsum disease is caused due to absence of which of the following enzymes? (Q855061)

A. Absence of alpha oxidation in peroxisomes.

B. Absence of beta oxidation in peroxisomes.

C. Absence of alpha oxidation in lysosomes.

D. Absence of beta oxidation in lysosomes.

Correct Answer: A. Absence of alpha oxidation in peroxisomes.

Explanation: Refsum disease is a rare inherited disorder caused by mutation in PHYH gene , leading to deficiency of the enzyme phytanoyl-CoA hydroxylase , which is involved in the alpha-oxidation of phytanic acid (a fatty acid). Alpha oxidation occurs in peroxisomes and aids in fatty acid breakdown. Triad : Cerebellar Ataxia Retinitis pigmentosa Chronic polyneuropathy

References:

pubmed.ncbi.nlm.nih.gov/32809453/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q4: What is the cause of elevated H+ ions in the intermembrane space of mitochondria? (Q855538)

A. Decreased oxidative phosphorylation

B. Increased ATP production

C. Reduced chemiosmosis

D. Water retention in inner mitochondrial membrane

Correct Answer: A. Decreased oxidative phosphorylation

Explanation: Decreased oxidative phosphorylation Elevated H■ ions in the intermembrane space of mitochondria occur due to active proton pumping during oxidative phosphorylation . In this process: Electrons from NADH and FADH■ pass through the electron transport chain (Complexes I, III, and IV) . The released energy drives H■ ions from the mitochondrial matrix into the intermembrane space , creating a proton gradient (electrochemical potential). This proton motive force is later used by ATP synthase (Complex V) to synthesize ATP as H■ flows back into the matrix via chemiosmosis . Thus, oxidative phosphorylation is the primary cause of H■ accumulation in the intermembrane space. Increased ATP production (Option B): This is a result of proton gradient utilization, not its cause. Reduced chemiosmosis (Option C): Would decrease H■ re-entry, stalling ATP synthesis but not explaining the normal physiological gradient formation. Water retention in inner mitochondrial membrane (Option D): Unrelated to proton concentration or electron transport.

References:

pmc.ncbi.nlm.nih.gov/articles/PMC2712118/

↑ *Back to this question in Part A*

@Neet_pg_bot

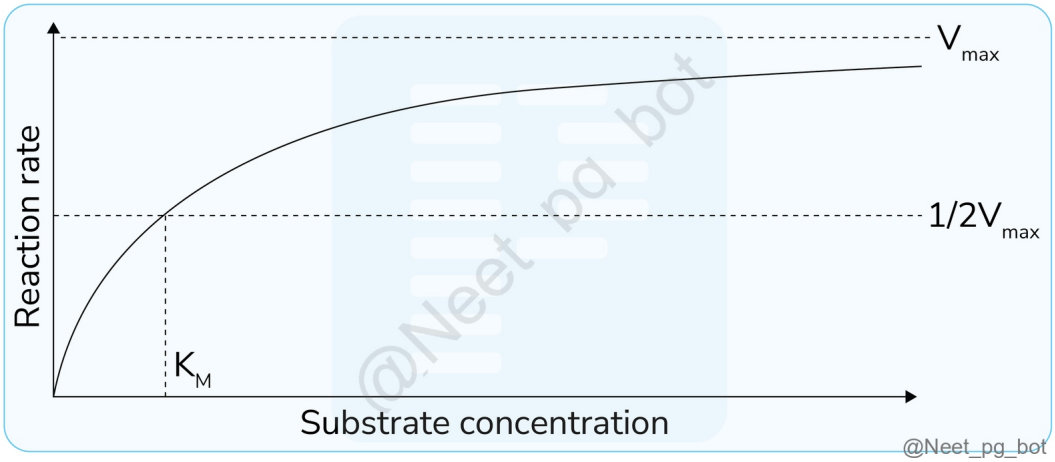
Q5: The enzyme kinetics depicted in the graph is suggestive of (Q855904)

A. Allosteric activation

B. Cooperative binding

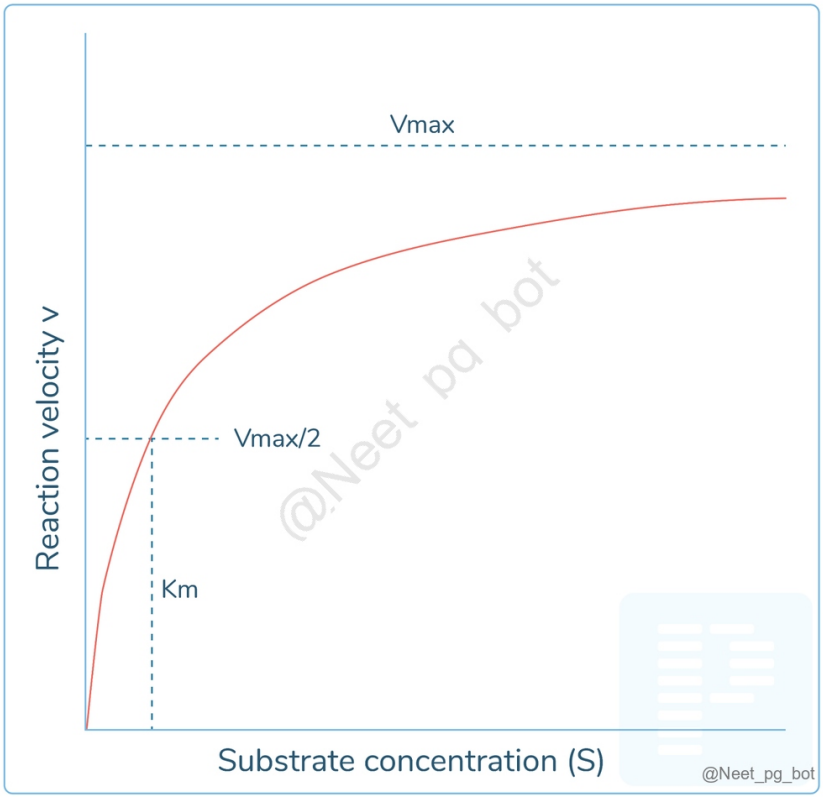
C. Michaelis Menten kinetics

D. Sigmoid curve



Correct Answer: C. Michaelis Menten kinetics

Explanation: The graph depicted is of Michaelis Menten's kinetics. Michaelis Menten's kinetics is intended to explain the broad mechanism and rate of enzyme-catalyzed reactions. The Michaelis-Menten equation is widely used to analyze enzyme kinetics and determine important parameters such as Vmax and Km. Michalis Menten equation: V = Vmax x S / (Km + S) Where, V = Velocity of reaction Vmax = Maximum velocity of the reaction S = Substrate concentration Km = Michaelis constant = Substrate concentration at which the velocity of the reaction is half the maximal velocity (Vmax/2) Allosteric activation (Option A) They are regulatory enzymes /multi-subunit enzymes. The activator shifts the curve to the left while the inhibitor shifts the cure to right Cooperative binding (Option B) One substrate binding to an enzyme increases the affinity for other substrates for enzymes. Sigmoid curve (Option D) In the presence of allosteric modifiers the graph of enzyme–catalyzed reaction obtained is a sigmoid / S-shaped graph.



References:

Harper, 32nd Edition, Page 76-77

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: Which of the following enzymes has the highest catalytic efficiency? (Q856482)

A. Km of an enzyme = 10 micromole and Kcat = 20 per sec

B. Km of an enzyme = 2000 nanomole and Kcat = 50 per sec

C. Km of an enzyme = 2 micromole and Kcat = 200 per sec

D. Km of an enzyme = 4 micromole and Kcat = 200 per sec

Correct Answer: C. Km of an enzyme = 2 micromole and Kcat = 200 per sec

Explanation: Km of an enzyme = 2 micromole and Kcat = 200 per sec Catalytic efficiency, also known as specificity constant, is a measure of how effectively an enzyme converts substrate to product. It is calculated as the ratio of the catalytic rate constant (Kcat) to the Michaelis constant (Km). Km of an enzyme = 2 micromole and Kcat = 200 per sec : Catalytic efficiency = Kcat / Km = 200 / 2 = 100 per micromole per sec. Catalytic efficiency

refers to the measure of how effectively an enzyme catalyzes a specific reaction. It is a measure of the enzyme's ability to convert substrate molecules into products within a given time frame. Catalytic efficiency is commonly expressed using the Michaelis-Menten equation, which relates the rate of the enzymatic reaction to the concentration of the substrate. The two important parameters used to evaluate catalytic efficiency are the turnover number (kcat) and the Michaelis constant (Km).

References:

Catalytic efficiency

↑ *Back to this question in Part A*

@Neet_pg_bot

Q7: Which mechanism is responsible for the inhibition of the acetylcholinesterase enzyme by organophosphorus compounds? (Q856597)

A. Non-competitive inhibition

B. Reversible competitive inhibition

C. Irreversibleinhibition

D. Suicidal inhibition

Correct Answer: C. Irreversibleinhibition

Explanation: Organophosphorus compounds, such as certain pesticides and nerve agents, can irreversibly inhibit the activity of acetylcholinesterase (AChE The inhibition caused by organophosphorus compounds is considered irreversible because these compounds form strong covalent bonds with the active site of the enzyme . They covalently bind to the serine residue within the active site of AChE, forming a stable complex that cannot be easily reversed or dissociated. Reversible competitive inhibition (Option A) occurs when an inhibitor molecule competes with the substrate for binding to the active site of the enzyme. Non-competitive inhibition (Option B) occurs when an inhibitor binds to a site on the enzyme other than the active site, altering its conformation and reducing its activity. Suicidal inhibition (Option D) is another term used to describe the irreversible nature of inhibition caused by organophosphorus compounds. It refers to the fact that the inhibitor "kills" the enzyme by permanently modifying it, leading to its inactivation.

References:

Textbook of Biochemistry by DM Vasudevan, 7th edition, pg 59-62

↑ *Back to this question in Part A*

@Neet_pg_bot

Q8: A TPN bag consists of 100g glucose, 30g amino acids, and 40g lipids. What is the amount of calories delivered? (Q856864)

A. 840 KCal

B. 880 KCal

C. 640 KCal

D. 680 KCal

Correct Answer: B. 880 KCal

Explanation: To calculate the number of calories delivered in the TPN (Total Parenteral Nutrition) bag, we need to know the caloric value of each macronutrient. The caloric values per gram for each macronutrient are as follows: Glucose: 4 calories per gram Amino acids: 4 calories per gram Lipids (fats): 9 calories per gram Now let's calculate the calories for each component of the TPN bag: Glucose: 100g of glucose * 4 calories/gram = 400 calories. Amino acids: 30g of amino acids * 4 calories/gram = 120 calories. Lipids: 40g of lipids * 9 calories/gram = 360 calories. Now, let's add up the calories from each component: 400 calories (glucose) + 120 calories (amino acids) + 360 calories (lipids) = 880 calories

References:

Calories

↑ *Back to this question in Part A*

@Neet_pg_bot

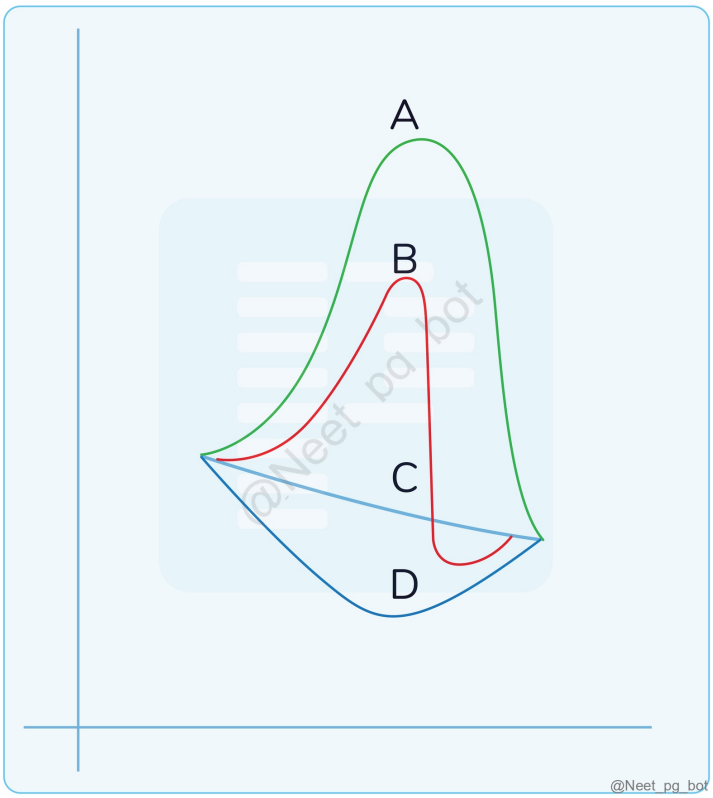
Q9: The Gibbs free energy curve of an enzyme-catalysed reaction is given by: (Q856930)

A. Curve B

B. Curve A

C. Curve D

D. Curve C



Correct Answer: A. Curve B

Explanation: The enzyme-catalyzed reaction corresponds to Curve B , which shows a lower activation energy than the uncatalyzed reaction (Curve A) , while both have the same ΔG (C → D) . Curve A – Represents the uncatalyzed reaction , which has a higher activation energy (Ea) . Curve B – Represents the enzyme-catalyzed reaction , which has a lower activation energy , hence proceeds faster. C → D – Represent the reactants and products , respectively. Enzymes lower the activation energy barrier by stabilizing the transition state, without altering the overall Gibbs free energy (ΔG) or the reaction equilibrium.

References:

Gibbs free energy

↑ *Back to this question in Part A*

@Neet_pg_bot

Q10: The active site of an enzyme is most complementary to? (Q857356)

A. Allosteric activator

B. Substrate

C. Product

D. Transition state

Correct Answer: B. Substrate

Explanation: Properties of Enzyme Nature of Enzymes Enzymes are proteins, heat-sensitive, water-soluble Efficiency Enzymes can accelerate reactions by factors 10^6 to 10^9 or more. Specificity Enzymes act on specific substrates and can distinguish stereoisomers. Cofactors/Coenzymes Some enzymes need non-protein molecules (cofactors or coenzymes) to be active. Substrate Binding Enzymes bind substrates at multiple points, creating chiral products from non-chiral substrates. Transition State Enzymes stabilize the transition state, lowering activation energy. Reversibility Enzymes can catalyze both forward and reverse reactions. Regulation Activity is regulated by pH, temperature, inhibitors, and activators. Active site The region where the substrate binds and undergoes a chemical reaction It is most complimentary to enzyme-substrate

References:

Harper 32nd edition, page 59,60

https://pubmed.ncbi.nlm.nih.gov/35534116/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: The subcellular organelle which is associated with the killing of viruses and bacteria is? (Q857485)

A. Peroxisome

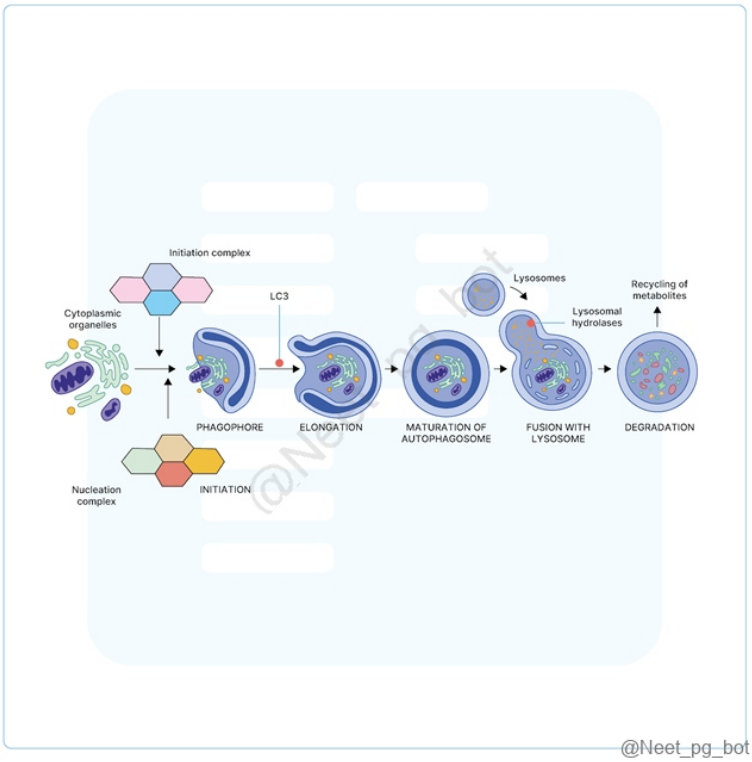
B. Mitochondria

C. Lysosome

D. Golgi apparatus

Correct Answer: C. Lysosome

Explanation: Lysosomes are membrane-bound organelles containing hydrolytic enzymes (like proteases, lipases, and nucleases) that digest and destroy pathogens, including viruses and bacteria, through a process called phagocytosis. After engulfment by phagocytes (macrophages), the pathogen is enclosed in a phagosome, which fuses with a lysosome to form a phagolysosome, where enzymatic degradation occurs. {{caption_text}} Peroxisome (Option A): Involved in fatty acid oxidation and reactive oxygen species detoxification. Mitochondria (Option B): Powerhouse of the cell, involved in ATP production. Golgi apparatus (Option D): Involved in protein modification, packaging, and transport.



References:

Lysosome

↑ *Back to this question in Part A*

@Neet_pg_bot

Q12: In which organelle is DNA present? (Q857869)

A. Mitochondria

B. Lysosome

C. Endoplasmic reticulum

D. Cytoplasm

Correct Answer: A. Mitochondria

Explanation: DNA is present in the mitochondria, which are double-membraned organelles found in the cells of most eukaryotic organisms. Mitochondrial DNA (mtDNA) is a small circular molecule separate from the nuclear DNA. It carries genetic information essential for the functioning of mitochondria and plays a crucial role in energy production through oxidative phosphorylation. Lysosome (Option B) are membrane-bound organelles containing

enzymes responsible for breaking down various cellular waste materials and substances. Their primary function is related to digestion and waste disposal within the cell. Endoplasmic Reticulum (Option C) is an organelle involved in the synthesis, folding, modification, and transport of proteins and lipids. The ER is composed of membranous structures and is mainly responsible for the manufacturing and transportation of molecules within the cell. Cytoplasm (Option D) is the gel-like substance found inside the cell, which includes various organelles and is the site of many cellular processes. The cytoplasm contains various molecules and structures necessary for cell function, DNA is not present in the cytoplasm. DNA is primarily found within the nucleus of eukaryotic cells, where it houses the genetic information required for cell function and development.

References:

Vasudevan Edition 7 page no:10-23.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q13: What is the process of converting unconjugated substances into conjugated forms called? (Q860059)

A. Glycation

B. Glucuronidation

C. Glycosylation

D. Oxidation

Correct Answer: B. Glucuronidation

Explanation: Glucuronidation is a biochemical process in which a glucuronic acid molecule is attached to a substance, making it more water-soluble and easier to excrete. This process is essential for the detoxification and elimination of substances like bilirubin, drugs, and hormones in the liver. Synthetic Reaction Description Examples Glucuronidation Conjugation with glucuronic acid via UDP-glucuronosyl transferases (UGTs). Chloramphenicol, aspirin, paracetamol, morphine, bilirubin, steroid hormones, thyroxine. Acetylation Conjugation with acetyl CoA, mediated by N-acetyl transferases. Isoniazid, sulfonamides, hydralazine, dapsone, clonazepam. Methylation Methylation of amines and phenols by methyl transferases using methionine or cysteine as donors. Adrenaline, histamine, methyldopa, captopril, mercaptopurine. Sulfation Conjugation of phenols and steroids with sulfate by sulfotransferases (SULTs). Chloramphenicol, methyldopa, adrenal steroids. Glycine Conjugation Conjugation with glycine for drugs with carboxylic acid groups. Salicylates, nicotinic acid. Glutathione Conjugation Conjugation of reactive intermediates with glutathione via glutathione-S-transferases. Paracetamol (reactive intermediates), quinones, epoxides. Ribonucleoside Synthesis Conjugation for activation of purine and pyrimidine antimetabolites. Cancer chemotherapeutic agents. Glycation (Option A): Glycation is the non-enzymatic attachment of sugars to proteins or lipids, typically seen in conditions like diabetes. It does not involve the conjugation of substances in the way glucuronidation does. Glycosylation (Option C): Glycosylation involves the enzymatic attachment of a sugar molecule (often glucose or mannose) to proteins or lipids, typically

in the endoplasmic reticulum and Golgi apparatus. It is distinct from glucuronidation and is not primarily involved in detoxification. Oxidation (Option D): Oxidation refers to the chemical reaction where a molecule loses electrons, often resulting in the addition of oxygen or the removal of hydrogen. While oxidation can modify molecules, it does not specifically refer to the conjugation process like glucuronidation.

References:

KDT- Essentials of Medical Pharmacology, 8th Ed, Pg: 31

↑ *Back to this question in Part A*

@Neet_pg_bot

Q14: Which substance is involved in the conjugation process in the liver? (Q860320)

A. Hyaluronic Acid

B. Glucuronic Acid

C. Gluconic Acid

D. Glycolic Acid

Correct Answer: B. Glucuronic Acid

Explanation: Glucuronidation is a biochemical process in which a glucuronic acid molecule is attached to a substance, making it more water-soluble and easier to excrete. This process is essential for the detoxification and elimination of substances like bilirubin, drugs, and hormones in the liver. Synthetic Reaction Description Examples Glucuronidation Conjugation with glucuronic acid via UDP-glucuronosyl transferases (UGTs). Chloramphenicol, aspirin, paracetamol, morphine, bilirubin, steroid hormones, thyroxine. Acetylation Conjugation with acetyl CoA, mediated by N-acetyl transferases. Isoniazid, sulfonamides, hydralazine, dapsone, clonazepam. Methylation Methylation of amines and phenols by methyl transferases using methionine or cysteine as donors. Adrenaline, histamine, methyldopa, captopril, mercaptopurine. Sulfation Conjugation of phenols and steroids with sulfate by sulfotransferases (SULTs). Chloramphenicol, methyldopa, adrenal steroids. Glycine Conjugation Conjugation with glycine for drugs with carboxylic acid groups. Salicylates, nicotinic acid. Glutathione Conjugation Conjugation of reactive intermediates with glutathione via glutathione-S-transferases. Paracetamol (reactive intermediates), quinones, epoxides. Ribonucleoside Synthesis Conjugation for activation of purine and pyrimidine antimetabolites. Cancer chemotherapeutic agents. Hyaluronic Acid (Option A): Hyaluronic acid is a glycosaminoglycan involved in extracellular matrix formation and lubrication of joints. It does not play a role in hepatic conjugation processes. Gluconic Acid (Option C): Gluconic acid is a product of glucose oxidation and is not involved in the conjugation process in the liver. Glycolic Acid (Option D): Glycolic acid is an alpha-hydroxy acid commonly associated with skincare and is not involved in hepatic conjugation.

References:

KDT- Essentials of Medical Pharmacology, 8th Ed, Pg: 31

↑ *Back to this question in Part A*

@Neet_pg_bot

• [02_Carbohydrate Chemistry] 002 Carbohydrates

« Back to Folder Directory

[02_Carbohydrate Chemistry] 002 Carbohydrates — Quick Jump

Q1 (p. 460)	*Q2 (p. 460)*	*Q3 (p. 461)*	*Q4 (p. 463)*	*Q5 (p. 464)*	*Q6 (p. 465)*
Q7 (p. 466)	*Q8 (p. 467)*	*Q9 (p. 468)*	*Q10 (p. 469)*	*Q11 (p. 470)*	*Q12 (p. 471)*
Q13 (p. 472)	*Q14 (p. 473)*	*Q15 (p. 473)*	*Q16 (p. 474)*	*Q17 (p. 475)*	*Q18 (p. 476)*
Q19 (p. 477)	*Q20 (p. 478)*	*Q21 (p. 478)*	*Q22 (p. 479)*	*Q23 (p. 480)*	*Q24 (p. 481)*

Q1: Which of the following statements is correct? (Q844975)

A. Glycoproteins contain long mucopolysaccharide chains

B. Proteoglycans have long mucopolysaccharide chains and high carbohydrate content.

C. Glycoproteins have long mucopolysaccharide chains and higher carbohydrate content

D. Proteoglycans contain short oligosaccharide chains and lower carbohydrate content

Correct Answer: B. Proteoglycans have long mucopolysaccharide chains and high carbohydrate content.

■ *Listen to Audio 1*

Explanation: Proteoglycans : Long mucopolysaccharide chains Negatively charged due to sulfated or acetylated groups Higher carbohydrate content Location - Extracellular matrix Glycoproteins : Oligosaccharide chain Not negatively charged Location - Plasma proteins, Cellular receptors – except albumin

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page. 612.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: A 15-year-old girl presents with fatigue and excessive thirst. Urine tests like Benedict’s, Barfoed’s, and Glucose Oxidase Peroxidase tests are positive. What is the most likely diagnosis? (Q844986)

A. Diabetes Mellitus

B. Essential Fructosuria

C. Lactose due to pregnancy and lactation

D. Classical Galactosemia

Correct Answer: A. Diabetes Mellitus

■ *Listen to Audio 1*

Explanation: The patient’s fatigue and polydipsia along with positive urine glucose tests (Benedict’s, Barfoed’s, Glucose Oxidase Peroxidase) indicate hyperglycemia with glycosuria , which is characteristic of type 1 diabetes , commonly presenting in adolescents. Tests for detecting sugars in urine: Benedict’s Test → Detects reducing sugars in urine (glucose, galactose, fructose). Barfoed’s Test → Differentiates monosaccharides (fast reaction) from disaccharides (slower). Glucose Oxidase Peroxidase (GOD-POD) Test → Specific for glucose, confirming glycosuria. Seliwanoff Test → Detects ketoses like fructose (positive in essential fructosuria) .(Option B) Mucic Acid Test → Detects galactose (positive in classical galactosemia). (Option D) Bial’s Test → Detects pentoses (used in essential pentosuria). Lactose due to pregnancy and lactation (Option C) would show a negative Barfoed's test, and generally would have history and features of pregnancy.

References:

Vasudevan’s Textbook of Biochemistry for Medical Students, 7th Edition, Page 316, 317.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q3: A laboratory technician performs an Osazone test on a patient’s urine sample and observes fluffy, hedgehog-shaped crystals. Which sugar is most likely present in the urine? (Q844985)

A. Glucose

B. Lactose

C. Maltose

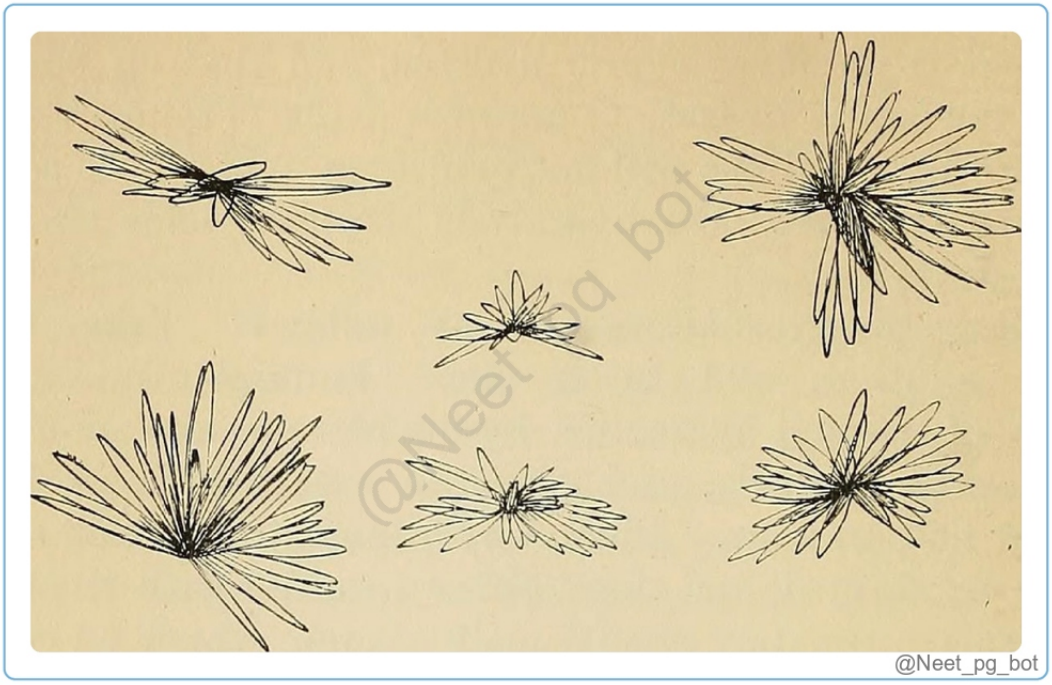
D. Sucrose

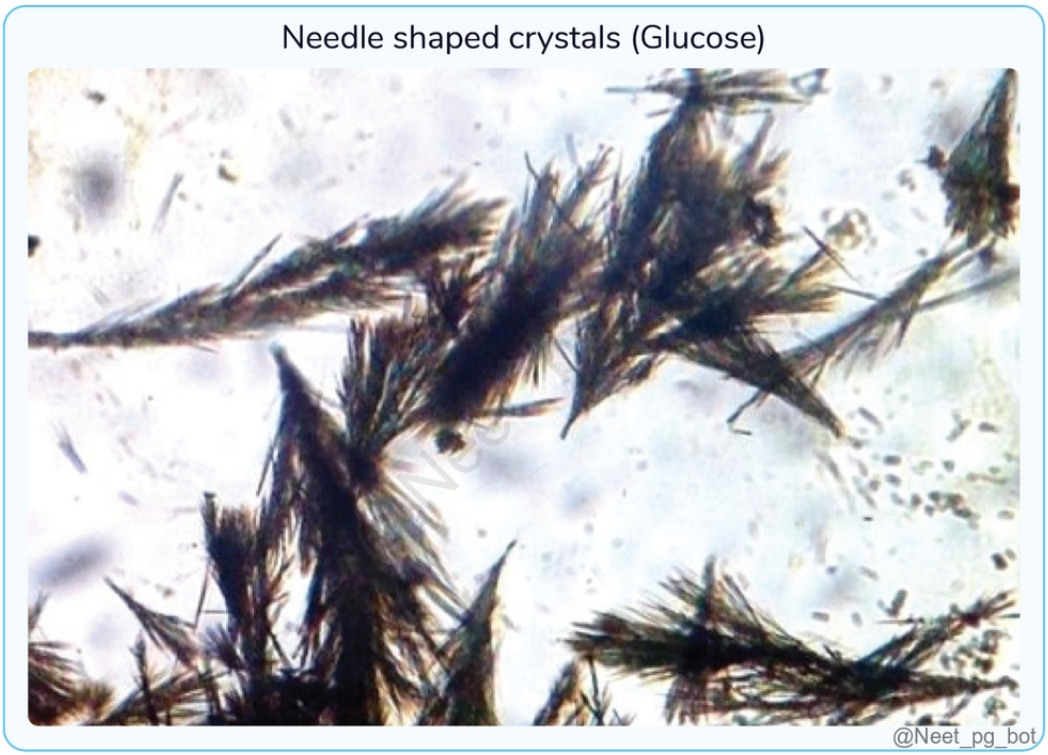
Correct Answer: B. Lactose

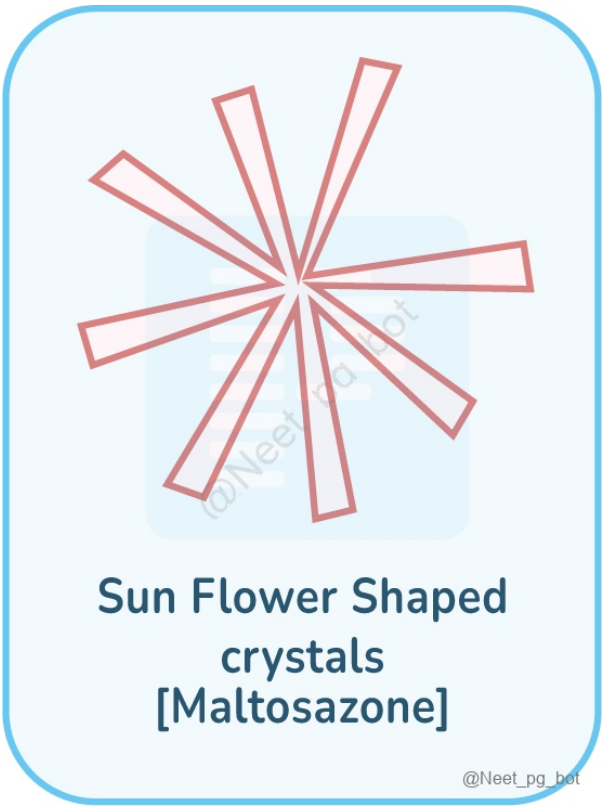
■ *Listen to Audio 1*

Explanation: Osazone test is used to differentiate reducing sugars based on the unique shapes and formation times of crystals. Lactose forms Hedgehog or “Pincushion with pins” shaped crystals on Osazone test. Sugar Crystal Shape Notes Lactose (Option B) Hedgehog or “Pincushion with pins” or “flower of touch-me-not plant” Indicates lactose presence, as seen in galactosemia. Glucose (Option A) Fructose Needle-shaped crystals arranged like a

broom Often associated with conditions like Diabetes Mellitus. Maltose (Option C) Sunflower-shaped or Petal-shaped Takes longer time to form, associated with menopausal status in women. Sucrose (Option D) No crystals Sucrose is a non-reducing sugar and does not form crystals in the Osazone test.







References:

Vasudevan’s Textbook of Biochemistry for Medical Students, 7th Edition, Page. 73.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q4: What is the reagent used in Barfoed’s test to detect monosaccharides? (Q844984)

A. Sodium carbonate, copper sulfate, and sodium citrate

B. Cupric acetate in acetic acid solution

C. Nitric acid

D. Phenolphthalein blue

Correct Answer: B. Cupric acetate in acetic acid solution

■ *Listen to Audio 1*

Explanation: Barfoed’s test is used to distinguish between monosaccharides and disaccharides. The reagent used is cupric acetate in an acetic acid solution. It is weakly acidic and is reduced by only monosaccharides. A false

positive result can occur with prolonged boiling of disaccharides. Monosaccharides react with this reagent within 5 minutes to give a brick-red precipitate of Cu■O Disaccharides take a longer time to react because the aldehyde function is masked in the acetal linkage and gives an orange-red color. Sodium carbonate, copper sulfate, sodium citrate(Option A): Benedict's reagent used to detect the presence of reducing sugars and other reducing substances in a sample Nitric acid, phenolphthalein blue(Option C,D) Not used in sugar tests

References:

Vasudevan's Textbook of Biochemistry for Medical Students, 7th Edition, Page 65

↑ *Back to this question in Part A*

@Neet_pg_bot

Q5: A new mother comes to the pediatric OPD with her infant as she notices dark staining in diapers after the baby urinates. What is the possible enzyme defect here? (Q844983)

A. Fumarylacetoacetase hydrolase

B. Homogentisic acid oxidase

C. Cystathionine β-synthase

D. Phenylalanine hydroxylase

Correct Answer: B. Homogentisic acid oxidase

■ *Listen to Audio 1*

Explanation: The dark staining of diapers is due to oxidation of homogentisic acid in urine on standing, turning it black . This occurs in Alkaptonuria , an autosomal recessive disorder caused by deficiency of homogentisic acid oxidase , an enzyme in the tyrosine degradation pathway . Accumulation of homogentisic acid leads to: Dark urine on standing Ochronosis (bluish-black pigmentation of connective tissues, sclera, cartilage) Arthropathy in adulthood Fumarylacetoacetase hydrolase(Option A): Deficiency causes Tyrosinemia type I. Cystathionine β-synthase(Option C): Deficiency causes Homocystinuria. Phenylalanine hydroxylase (Option D): Deficiency causes Phenylketonuria (PKU).

References:

Lippincott illustrated reviews Biochemistry, 7th Edition, Page. 779,787-790

Vasudevan textbook of biochemistry, Page. 237, 238

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: A urine sample of a diabetes patient was tested to see the presence of reducing sugars. Initial color of solution is blue. What would be the color of the solution after the test? (Q844982)

A. Blue

B. Orange

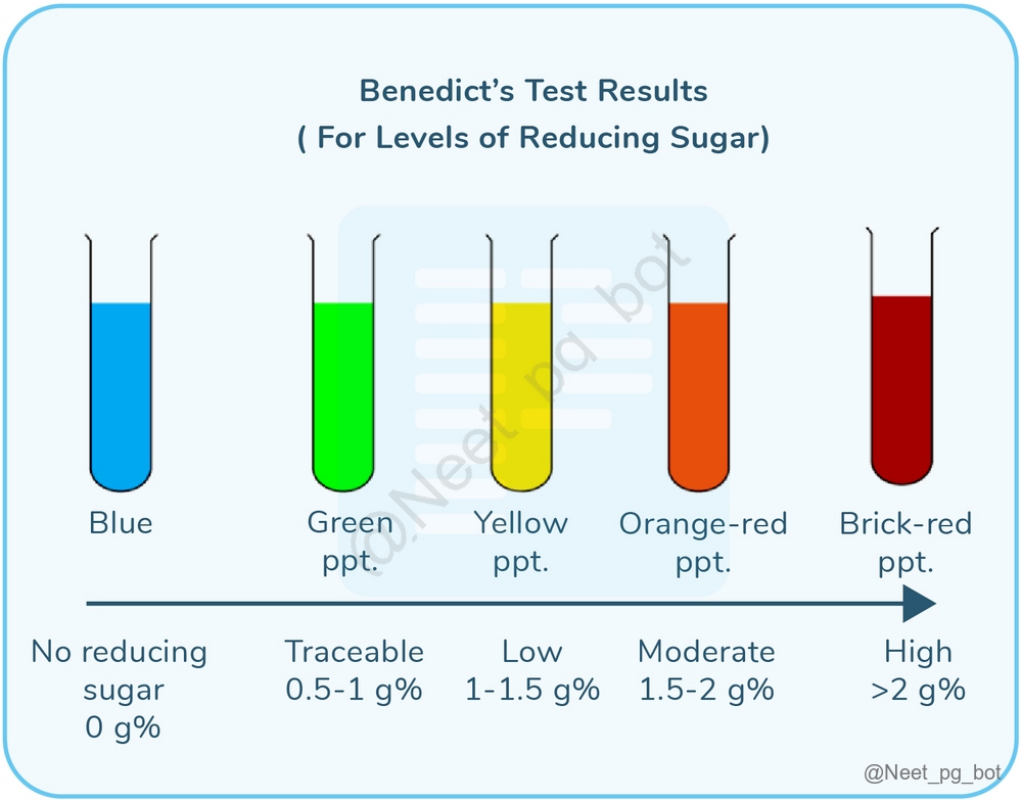
C. Colourless

D. Black

Correct Answer: B. Orange

■ *Listen to Audio 1*

Explanation: In Benedict's test, blue solution changes its color in the range from green to yellow to orange to red according to the concentration of the reducing sugars. A negative Benedict's test : no change in color. Any sugar with a free aldehyde or keto group will reduce the Benedict's reagent and hence, this is not a reliable test for detecting glucose in urine. Interpretation of Benedict's test: Reagents of Benedict's Solution: Reagent Function Copper(II) sulfate (CuSO■) Provides Cu^{2}■ ions that are reduced to Cu■O (cuprous oxide) by reducing sugars Sodium carbonate (Na■CO■) Creates a strong alkaline medium required for the redox reaction Sodium citrate Chelates copper ions and prevents precipitation of copper carbonate in alkaline solution



References:

Lippincott illustrated reviews of Biochemistry, 7th Edition, Page 264

Vasudevan textbook of biochemistry, Page 73, 76

Image: www.careerpower.in/school/chemistry/benedicts-test

↑ *Back to this question in Part A*

@Neet_pg_bot

Q7: A 55-year-old woman presents with polydipsia, polyuria, and weight loss. Diabetes mellitus is suspected, and her urine is tested for reducing sugars Image A: Benedict's Test Results Image B: Fehling's Test Results Based on the images provided, which of the following statements is correct? (Q844981)

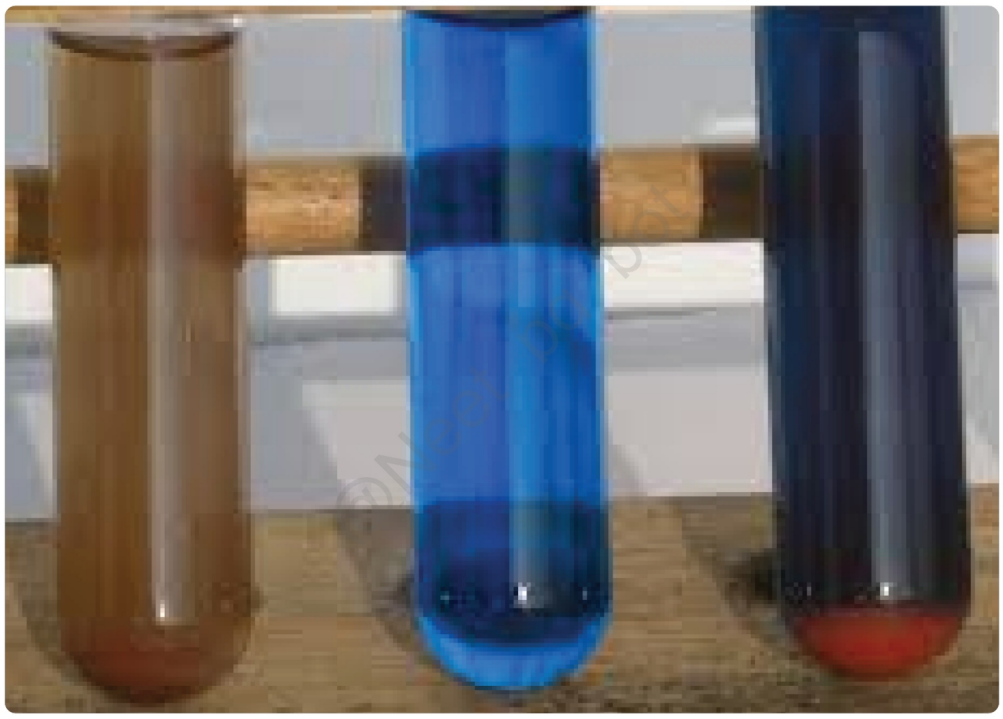
A. Both tests indicate absence of reducing sugars

B. Both tests indicate presence of reducing sugars

C. Benedict's test indicates presence, while Fehling's test indicates absence of reducing sugars.

D. Benedict's test indicates absence, while Fehling's test indicates presence of reducing sugars.





Correct Answer: B. Both tests indicate presence of reducing sugars

■ *Listen to Audio 1*

Explanation: Image A (Benedict's Test): The color changes from blue to green, yellow, and red, indicating the presence of reducing substances in the urine. Image B (Fehling's Test) : The color changes from blue to brick-red, indicating the presence of reducing substances in the urine sample. Therefore, both tests confirm the presence of reducing sugars , which may suggest diabetes mellitus in the patient.

References:

Lippincott illustrated biochemistry, 7th Edition, Page. 789

↑ *Back to this question in Part A*

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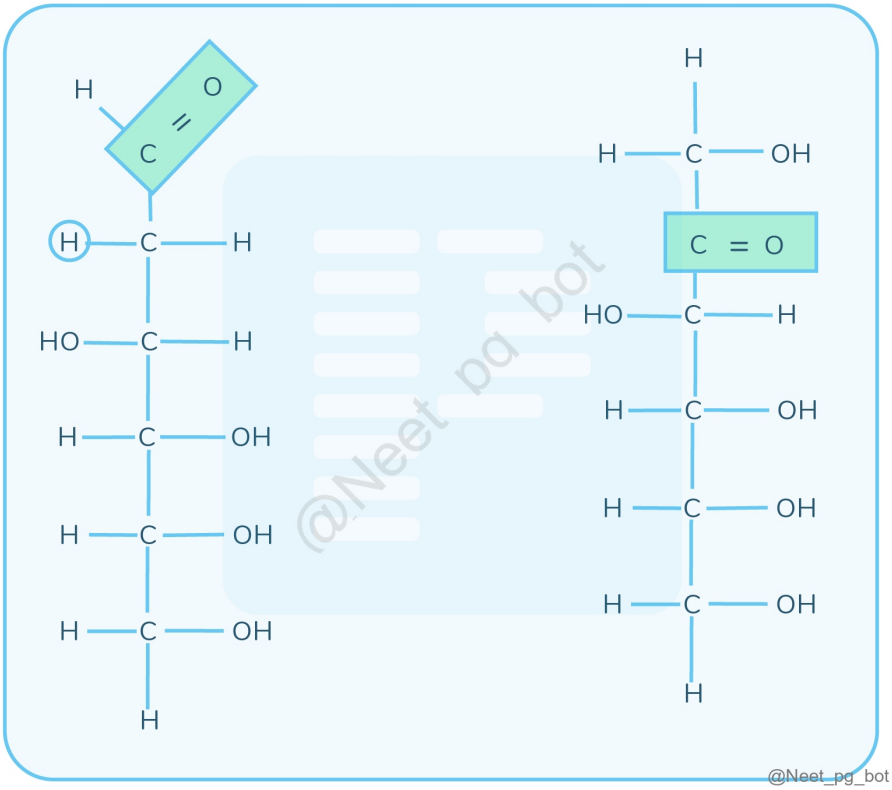
Q8: Which type of isomerism is exhibited by these two compounds? (Q844980)

A. Functional Isomerism

B. Epimerism

C. Enantiomerism

D. Anomerism



Correct Answer: A. Functional Isomerism

■ *Listen to Audio 1*

Explanation: The given structures are glucose (aldohexose) and fructose (ketohexose) They are examples of functional isomers because they have the same molecular formula but differ in their functional groups. Epimerism (Option B) : Differ in configuration around one chiral carbon atom. e.g D-Glucose and D-Mannose Enantiomerism (Option C) Compounds that are non-superimposable mirror images , differing at all chiral centers. e.g L-Glucose and D-Glucose are enantiomers Anomerism (Option D) Compounds differing at the anomeric carbon formed during the ring closure of carbohydrates. α-D-Glucose and β-D-Glucose differ in configuration at the anomeric carbon (C-1)

References:

Harper’s Textbook of Biochemistry, 32nd Edition, Pages 149, 150

↑ *Back to this question in Part A*

@Neet_pg_bot

Q9: Xylose and arabinose are examples of? (Q844979)

A. Optical isomerism

B. Diastereoisomerism

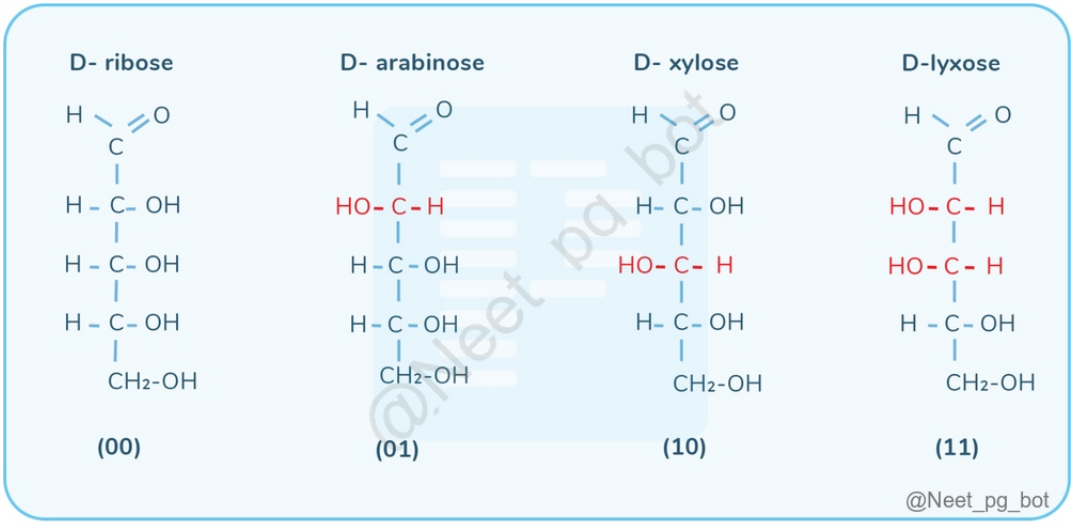
C. Enantiomerism

D. Epimerism

Correct Answer: B. Diastereoisomerism

■ *Listen to Audio 1*

Explanation: Xylose and Arabinose are both aldopentoses (five-carbon sugars with an aldehyde group). They differ at two stereocentres : typically C-2 and C-3 . Since diastereomers differ at one or more (but not all) chiral centres and are not mirror images , xylose and arabinose qualify as diastereoisomers , not epimers . Optical Isomerism (Option A) : Two molecules with same molecular formula, but differ in the way they turn the plain polarised lights . Enantiomerism (Option C) : Exact mirror image of the carbon atoms. Epimerism (Option D): Epimers differ at only one chiral centre.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page. 610

↑ *Back to this question in Part A*

@Neet_pg_bot

Q10: What type of isomerism occurs when two molecules have the same molecular and structural formula but differ in the three-dimensional arrangement of their groups? (Q844978)

A. Optical Isomerism

B. Functional Isomerism

C. Stereoisomerism

D. All of the above

Correct Answer: C. Stereoisomerism

■ *Listen to Audio 1*

Explanation: Stereoisomerism refers to compounds that have: The same molecular formula The same order of bonding (connectivity) But differ in the three-dimensional spatial arrangement of atoms Optical Isomerism: (Option A) It is a type of stereoisomerism , not a separate category Optical isomers differ in their ability to rotate plane-polarized light The isomer that turns the light to the right side is called Dextrorotatory (d/ +) and that turns the light to the left side is called Levorotatory (l / -) Example: L- and D-lactic acid . Functional Isomerism: (Option B) Occurs when two compounds have the same formula but different functional groups Example: Ethanol (CH■–CH■–OH) Dimethyl ether (CH■–O–CH■)

References:

Harper’s Biochemistry, 32nd Edition, Page. 611,612,613.

↑ *Back to this question in Part A*

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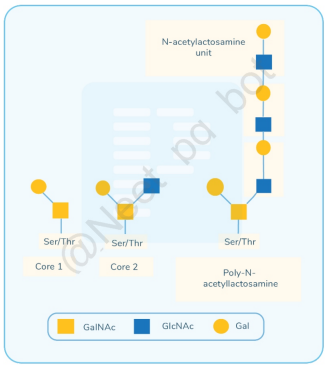
Q11: Identify the type of linkage in the given Structure. (Q844977)

A. O-linked glycoproteins

B. N- N-linked glycoproteins

C. GPI anchored Glycoproteins

D. None of the above



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Correct Answer: A. O-linked glycoproteins

■ *Listen to Audio 1*

Explanation: O-linked glycoproteins (post-translation modification) Carbohydrate chains are attached to the oxygen atom of serine or threonine residues. Mucins: Major components of mucus, providing lubrication and protection to epithelial surfaces. Glycoproteins in collagen N-linked glycoproteins (Option B) Carbohydrate chains are attached to the nitrogen atom of asparagine residues. Immunoglobulins (IgG) & Erythropoietin GPI-anchored Glycoproteins (Option C) Carbohydrates are not attached to any amino acid Anchor consists of a lipid tail (phosphatidylinositol) and a glycan chain, which covalently links the protein to the membrane.

References:

Harper's Biochemistry, 32nd Edition, Page. 611

↑ *Back to this question in Part A*

@Neet_pg_bot

Q12: A 45-year-old with fatigue, abdominal pain, and morning dark urine shows hemoglobinuria. Which test confirms Paroxysmal Nocturnal Hemoglobinuria (PNH)? (Q844976)

A. Bone Marrow Biopsy

B. Flow Cytometry

C. Serum Ferritin Test

D. Haemoglobin Electrophoresis

Correct Answer: B. Flow Cytometry

Listen to Audio 1

Explanation: PNH is caused by a somatic mutation in the PIGA gene , leading to absence of GPI-anchored complement regulatory proteins (CD55 and CD59) . This makes RBCs susceptible to complement-mediated intravascular hemolysis , diagnosed definitively by flow cytometry . Flow cytometry is the gold standard diagnostic test for PNH . It detects the absence or deficiency of GPI-anchored proteins on blood cells. Target Proteins: CD55 (decay-accelerating factor) CD59 (membrane inhibitor of reactive lysis) A deficiency of these proteins leads to complement-mediated hemolysis , which causes the hemoglobinuria characteristic of PNH. Bone Marrow Biopsy (Option A) : It is used to diagnose various bone marrow disorders, but is not specific for PNH. Serum Ferritin Test (Option C) : This test measures iron storage levels and is not specific to PNH. Haemoglobin Electrophoresis (Option D) : This test separates different types of haemoglobin but does not diagnose PNH.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 611

↑ *Back to this question in Part A*

@Neet_pg_bot

Q13: Which class of carbohydrates cannot be further hydrolyzed into simpler sugar units? (Q844962)

A. Monosaccharides

B. Disaccharides

C. Oligosaccharides

D. Polysaccharides

Correct Answer: A. Monosaccharides

Listen to Audio 1

Explanation: Monosaccharides are simple carbohydrates made up of single carbohydrate units and hence cannot be hydrolysed. Carbohydrates are divided into simple and complex carbohydrates. Simple carbohydrates can further be classified as follows: Monosaccharides : Single carbohydrate unit Ex: glucose, fructose Disaccharides : Two monosaccharide units Ex: sucrose, maltose, isomaltose, trehalose, lactose Oligosaccharides : 3- 10 monosaccharide units Ex: Maltotriose Polysaccharides : More than 10 monosaccharide units Ex: starch, cellulose,

glycogen

References:

Lippincott illustrated reviews: Biochemistry, 7th edition, Page. 256 Vasudevan textbook of biochemistry, Page. 69, 76

↑ *Back to this question in Part A*

@Neet_pg_bot

Q14: Which of the following is a distinguishing feature between Hunter syndrome and Hurler syndrome? (Q844974)

A. Hunter is associated with corneal clouding; Hurler is not.

B. Hurler is due to a deficiency in iduronate-2-sulfatase; Hunter is due to a deficiency in alpha-L-iduronidase.

C. Both result in the accumulation of heparan and dermatan sulfate.

D. Hunter is X-linked ; Hurler is autosomal recessive.

Correct Answer: D. Hunter is X-linked ; Hurler is autosomal recessive.

■ *Listen to Audio 1*

Explanation: Differentiating Features of Hurler and Hunter Syndromes Feature Hurler Syndrome (MPS I) Hunter Syndrome (MPS II) Deficient Enzyme (Option B) L-Iduronidase Iduronate sulfatase Inheritance Autosomal recessive X-linked recessive Mental Retardation Severe (MR+++) Mild to moderate (MR+) Corneal Opacity (Option A) Present (Corneal opacity++) Absent (No corneal clouding) Both syndromes do accumulate heparan sulfate and dermatan sulfate, but this does not distinguish them (Option C)

References:

Harper’s Biochemistry, 32nd Edition, Page. 611

↑ *Back to this question in Part A*

@Neet_pg_bot

Q15: A 4-year-old child shows developmental delay, behavioral problems, and progressive cognitive decline. Examination reveals coarse facial features and hepatosplenomegaly, but normal ocular findings. Which type of mucopolysaccharidosis is most likely? (Q844973)

A. MPS I (Hurler syndrome)

B. MPS IX (Natowicz syndrome)

C. MPS III (Sanfilippo syndrome)

D. MPS IV (Morquio syndrome)

Correct Answer: C. MPS III (Sanfilippo syndrome)

■ *Listen to Audio 1*

Explanation: MPS III (Sanfilippo syndrome) is the most common type of mucopolysaccharidosis. In this syndrome neurological symptoms predominate , while corneal clouding is absent. Clinical Features and Investigations of Mucopolysaccharidoses : Disease Name Abbreviation Enzyme Defective GAG(s) Affected Symptoms Hurler, Scheie, Hurler-Scheie syndrome (Option A) MPS I α-L-Iduronidase Dermatan sulfate, heparan sulfate Mental retardation, coarse facial features, hepatosplenomegaly, cloudy cornea Hunter syndrome MPS II Iduronate sulfatase Dermatan sulfate, heparan sulfate Mental retardation Sanfilippo syndrome A (Option C) MPS IIIA Heparan sulfate-N-sulfatase Heparan sulfate Delay in development, motor dysfunction Sanfilippo syndrome B MPS IIIB α-N-Acetylglucosaminidase Heparan sulfate As MPS IIIA Sanfilippo syndrome C MPS IIIC α-Glucosaminide N-acetyltransferase Heparan sulfate As MPS IIIA Sanfilippo syndrome D MPS IIID N-Acetylglucosamine 6-sulfatase Heparan sulfate As MPS IIIA Morquio syndrome A (Option D) MPS IVA Galactosamine 6-sulfatase Keratan sulfate, chondroitin 6-sulfate Skeletal dysplasia, short stature Cognition preserved Morquio syndrome B MPS IVB β-Galactosidase Keratan sulfate Skeletal dysplasia, short stature Maroteaux-Lamy syndrome MPS VI N-Acetylgalactosamine 4-sulfatase Dermatan sulfate Curvature of the spine, short stature, skeletal dysplasia, cardiac defects Sly syndrome MPS VII β-Glucuronidase Dermatan sulfate, heparan sulfate, chondroitin 4-sulfate, chondroitin 6-sulfate Skeletal dysplasia, short stature, hepatosplenomegaly, cloudy cornea Natowicz syndrome (Option B) MPS IX Hyaluronidase Hyaluronic acid Joint pain , short stature

References:

Harper’s Biochemistry, 32nd Edition, Page. 611.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q16: A patient with joint pain and restricted movement is found to have reduced levels of a key mucopolysaccharide in their synovial fluid responsible for lubrication. Which glycosaminoglycan is most likely deficient? (Q844972)

A. Heparin

B. Chondroitin Sulfate

C. Dermatan Sulfate

D. Hyaluronic Acid

Correct Answer: D. Hyaluronic Acid

■ *Listen to Audio 1*

Explanation: Hyaluronic acid : Predominantly found in connective tissues, tendons, synovial fluid, and the vitreous humor of the eye. Lubricant in joint cavities, helping to maintain joint health and mobility. Deficiency leads to joint pain and reduced mobility. Mucopolysaccharides : Glycosaminoglycan Locations Key Functions Hyaluronic acid (Option D) Synovial fluid, vitreous humor, connective tissue, and umbilical cord Serves as a lubricant in joint cavities and contributes to the structure of connective tissues Chondroitin sulfate (Option B) Cartilage, bone, tendons, ligaments, aorta Provides structural support to connective tissues and contributes to their resilience and elasticity Dermatan sulfate (Option C) Skin, blood vessels, heart valves Contributes to the structure of the skin, blood vessels, and heart valves Keratan sulfate Cornea, cartilage, intervertebral discs Maintains corneal transparency and cartilage structure Heparan sulfate Basement membranes, cell surfaces Cell signaling, filtration barrier Heparin (Option A) Mast cells (liver, lung) Acts as an anticoagulant by activating antithrombin III, which inactivates thrombin and other clotting factors

References:

Harper’s Biochemistry, 32nd Edition, Page 608

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: A patient presents with hyperextensible skin and fragile blood vessels due to a rare genetic disorder affecting connective tissue. Which glycosaminoglycan (mucopolysaccharide) found in the skin and vascular tissue is most likely implicated in this condition?? (Q844971)

A. Hyaluronic Acid

B. Heparin

C. Chondroitin Sulfate

D. Dermatan Sulfate

Correct Answer: D. Dermatan Sulfate

■ *Listen to Audio 1*

Explanation: Dermatan sulfate supports the structure of skin, blood vessels, and heart valves; its deficiency or dysfunction can cause hyperextensible skin and fragile blood vessels. Locations of Various Mucopolysaccharides: Mucopolysaccharide Primary Locations Hyaluronic Acid (Option A) Connective tissues, tendons, synovial fluid, vitreous humor Heparin (Option B) Liver, lungs, spleen, monocytes Chondroitin Sulfate (Option C) Cartilage,

bone, tendons, cornea, skin Keratan Sulfate Cornea, cartilage Dermatan Sulfate (Option D) Skin, blood vessels, heart valves

References:

Harper’s Biochemistry Edition 32; Page 610.

↑ *Back to this question in Part A*

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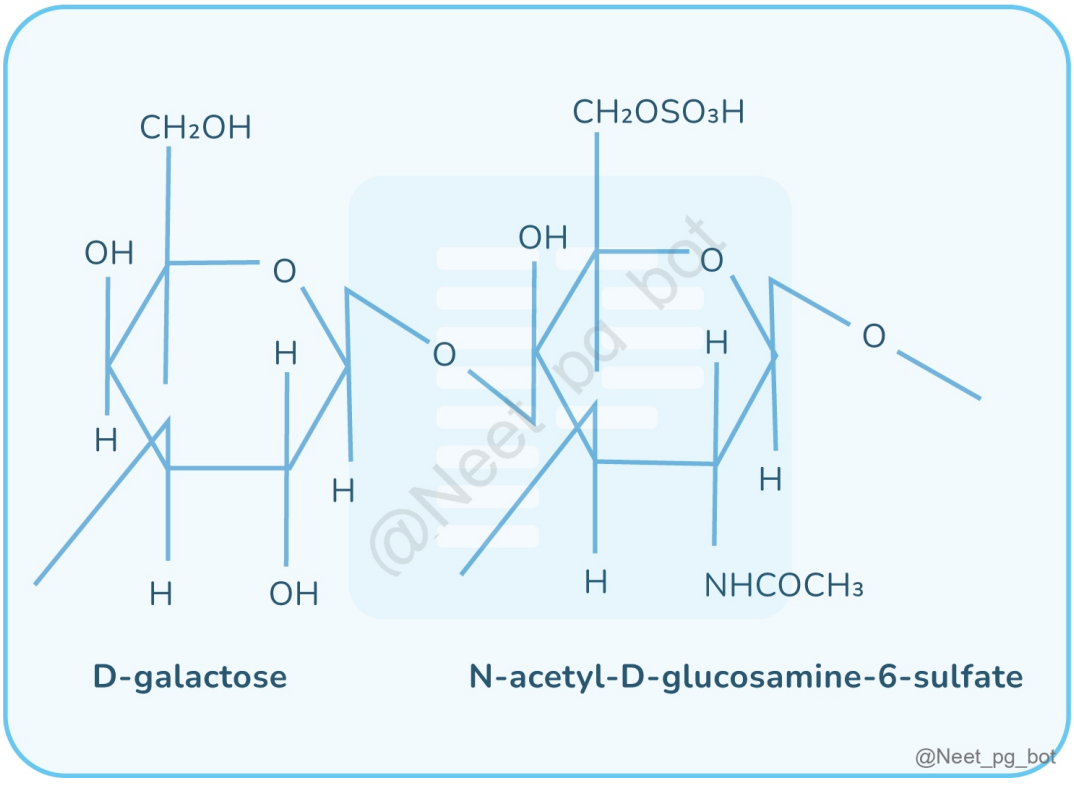
Q18: Analyse the structure of the mucopolysaccharide shown below: Which mucopolysaccharide is represented in this image? (Q844970)

A. Heparan sulfate

B. Keratan sulfate

C. Chondroitin sulfate

D. Dermatan sulfate



Correct Answer: B. Keratan sulfate

■ *Listen to Audio 1*

Explanation: Identification tips : Look for the presence of galactose as one of the primary sugars in the disaccharide unit. N-acetylglucosamine can be identified by the presence of the N-acetyl group (CH■CONH). Sulfate groups may be attached to either of the sugar moieties, contributing to the molecule's negative charge. The structure given above is of Keratan sulfate. It lacks uronic acid■ and is primarily found in the cornea and cartilage. Mucopolysaccharide (MPS) Components Keratan Sulfate Galactose and N-acetylglucosamine Heparan Sulfate (Option A) Glucuronic acid (or iduronic acid) and N-sulfated glucosamine Dermatan Sulfate (Option D) Iduronic acid and N-acetylgalactosamine Chondroitin Sulfate (Option C) Glucuronic acid and N-acetylgalactosamine Hyaluronic Acid Glucuronic acid and N-acetylglucosamine

References:

Harper’s Biochemistry 32nd Edition, Page 608

↑ *Back to this question in Part A*

@Neet_pg_bot

Q19: Mucopolysaccharides are heteropolysaccharides containing uronic acid and amino sugars. Which among the following mucopolysaccharides contradicts the above statement? (Q844969)

A. Keratan sulfate

B. Chondroitin sulfate

C. Dermatan sulfate

D. Heparin

Correct Answer: A. Keratan sulfate

■ *Listen to Audio 1*

Explanation: Keratan sulfate is unique among mucopolysaccharides as it is the only GAG that does not contain any uronic acid. Keratan sulfate (Option A) Repeating units are galactose and N-acetyl glucosamine in beta linkage. Chondroitin sulfate (Option B) Repeating units of glucuronic acid → beta-1,3-N-acetyl galactosamine sulfate → beta-1,4 and so on. Dermatan sulfate (Option C) It Contains L-iduronic acid and N-acetyl galactosamine in beta-1, 3 linkages. Heparin (Option D) An anticoagulant widely used Repeating units of sulphated glucosamine → alpha-1, 4-L-iduronic acid → and so on Iduronic acid is the oxidized form of idose.

References:

Lippincott Illustrated Reviews: Biochemistry, 7th Edition, Pages 470, 471

Vasudevan textbook of biochemistry, Pages 80, 81

↑ *Back to this question in Part A*

@Neet_pg_bot

Q20: Which of the following is accurate about dietary fibres? (Q844968)

A. Insoluble fibre is fermented in the large intestine

B. Dietary fibre provides a significant source of energy

C. Soluble fibre helps lower LDL cholesterol

D. Soluble fibre promotes bowel movements

Correct Answer: C. Soluble fibre helps lower LDL cholesterol

■ *Listen to Audio 1*

Explanation: Dietary fiber is the non-digestible, non-starch carbohydrate and lignin present intact in plants. It does not provide much energy, but provides other benefits. Soluble dietary fiber : Lowers LDL cholesterol levels by preventing absorption of cholesterol and bile acids into the bloodstream. This forces liver to pull more cholesterol from the blood to produce new bile acids, which, in turn, reduces the circulating LDL levels. Stabilizes blood sugar Fermented in the large intestine to short-chain fatty acids Ex: Oats, barley, psyllium, brussels sprouts, etc Energy provided by dietary fiber ~2 kcal/g (via short-chain fatty acids) (Option B) Recommended daily intake (RDA): 25–40 g/day (adults) Source of energy: Fermentation by gut microbiota → SCFAs (acetate, propionate, butyrate) Insoluble dietary fiber : Adds bulk and promotes bowel movements. (Option D) Prevents constipation, lowers the risk of diverticular disease and haemorrhoids. Passes through the digestive tract largely unchanged (Option A) Ex: Whole wheat, walnuts, chickpeas, etc

References:

Lippincott illustrated reviews: Biochemistry, 7th Edition, Pages 1045, 1046

↑ *Back to this question in Part A*

@Neet_pg_bot

Q21: Which of the following pairs correctly distinguishes between homoglycans and heteroglycans? (Q844967)

A. Homoglycans - repeated units of the same monosaccharide; Heteroglycans - two or more different types of monosaccharide units.

B. Homoglycans - used for structural purposes; Heteroglycans - used forenergy storage.

C. Homoglycans- typically soluble in water; Heteroglycans - insoluble in water

D. Homoglycans - only in plant cells; Heteroglycans - only in animal cells.

Correct Answer: A. Homoglycans - repeated units of the same monosaccharide; Heteroglycans - two or more different types of monosaccharide units.

■ *Listen to Audio 1*

Explanation: Polysaccharides are classified into : Homoglycans/ homopolysaccharides Heteroglycans/ heteropolysaccharides Made of a single kind of monosaccharide unit. Made of two or more different monosaccharides. Used for both structural and energy storage. Used for both structural and energy storage. Variable solubility (dextran, cellulose) Variable solubility (agar, hyaluronic acid, keratin sulphate) Found in both plants and animals (starch & glycogen) Found in both plants & animals (agar & keratan sulfate) Ex: starch, glycogen and cellulose. Ex: hyaluronic acid, chondroitin sulfate.

References:

Lippincott illustrated reviews: Biochemistry, 7th Edition, Page. 1045 Vasudevan textbook of Biochemistry, Page. 78 to 80.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q22: A patient comes with complaints of abdominal pain, bloating, flatulence and frothy stools. It is noticed that these symptoms arise only when he consumes milk or other dairy products. Which of the following is the IOC in this situation to diagnose him? (Q844966)

A. Lactose tolerance test

B. Hydrogen breath test

C. Stool osmolarity test

D. Serum calcium test

Correct Answer: B. Hydrogen breath test

■ *Listen to Audio 1*

Explanation: The diagnosis here points to lactose intolerance , caused by the enzyme lactase deficiency . It presents with symptoms like osmotic diarrhoea, flatulence, bloating, frothy stools, and perianal excoriation. IOC is the Hydrogen breath test and the methane breath test. Other tests include: Stool acidity test : Unabsorbed lactose is fermented by colonic bacteria into lactic acid, lowering stool pH. Dietary elimination : Elimination of certain foods reduces the symptoms. Milk tolerance test : 500ml milk to be consumed, and the blood glucose level is to be noted to see malabsorption Small intestine biopsy : Done to find secondary causes only. Genotyping Lactose tolerance test: Checks lactose absorption after consuming a lactose-containing liquid. Less preferred because it is invasive and less sensitive than the hydrogen breath test. (Option A) Stool osmolarity test(Option C): Mainly used in infants

and young children to detect osmotic diarrhea. Serum calcium test(Option D): Measures calcium levels; not related to diagnosing lactose intolerance.

References:

Lippincott illustrated reviews: Biochemistry, 7th Edition, Page 274

www.ncbi.nlm.nih.gov/books/NBK532285/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q23: Which of the following is non-reducing sugar? (Q844964)

A. Maltose

B. Glucose

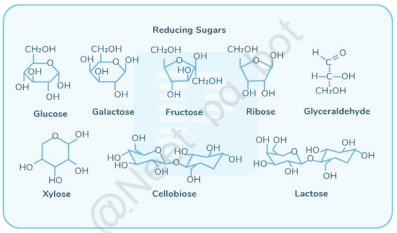
C. Sucrose

D. Fructose

Correct Answer: C. Sucrose

■ *Listen to Audio 1*

Explanation: Non-reducing sugars are carbohydrates that lack a free aldehyde or ketone group , preventing them from acting as reducing agents. Ex. Sucrose and Trehalose . They do not react with Benedict’s or Fehling’s solutions because they lack a free anomeric carbon. After acid hydrolysis, they break down into their constituent monosaccharides, which are reducing sugars , giving a positive reaction. Maltose , Glucose and Fructose are reducing sugars.



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References:

Lippincott illustrated reviews of biochemistry, 7th Edition, Page. 788.

↑ *Back to this question in Part A*

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Q24: Which monosaccharides combine to form the disaccharide lactose? (Q844963)

A. Galactose and Glucose

B. Glucose and Fructose

C. Galactose and Sucrose

D. Glucose and Glucose

Correct Answer: A. Galactose and Glucose

■ *Listen to Audio 1*

Explanation: Lactose is a disaccharide formed by the combination of galactose and glucose . Disaccharide Sugars present Linkage Maltose Glucose + Glucose alpha(1,4) Isomaltose Glucose + Glucose alpha(1,6) Trehalose

Glucose + Glucose alpha(1,1) Sucrose Glucose + Fructose alpha(1,2) Lactose Galactose + Glucose beta(1,4)

References:

Lippincott illustrated reviews: Biochemistry, 7th Edition, Page. 787,788.

↑ *Back to this question in Part A*

@Neet_pg_bot

• [02_Carbohydrate Chemistry] 003 Fructose And Galactose Metabolism Hmp Pathway

« Back to Folder Directory

[02_Carbohydrate Chemistry] 003 Fructose And Galactose Metabolism Hmp Pathway — Quick Jump

Q1 (p. 483)	*Q2 (p. 484)*	*Q3 (p. 485)*	*Q4 (p. 486)*	*Q5 (p. 487)*	*Q6 (p. 488)*
Q7 (p. 490)	*Q8 (p. 491)*	*Q9 (p. 492)*	*Q10 (p. 493)*	*Q11 (p. 494)*	*Q12 (p. 494)*
Q13 (p. 495)	*Q14 (p. 496)*	*Q15 (p. 497)*	*Q16 (p. 497)*	*Q17 (p. 499)*	

Q1: How many ATPs are produced from the HMP shunt pathway? (Q875991)

A. Zero

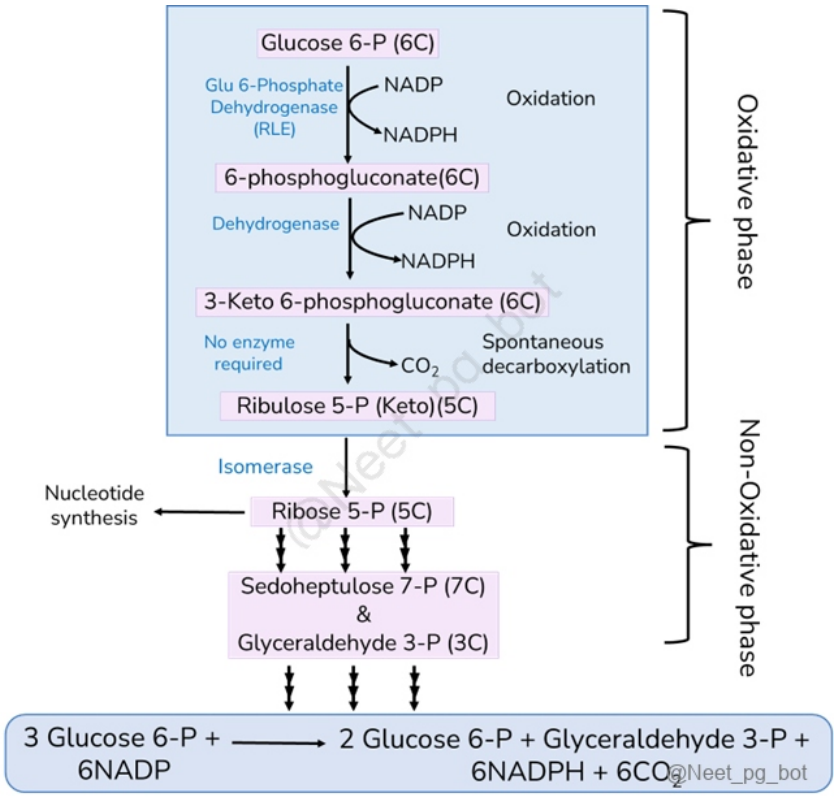
B. One

C. Two

D. Four

Correct Answer: A. Zero

Explanation: of A



References:

Harper 30th Ed., pg. 199

↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: Which of the following enzymes is not involved in the reversible phase of the pentose phosphate pathway? (Q845033)

A. Ribulose-5-phosphate epimerase

B. Transketolase

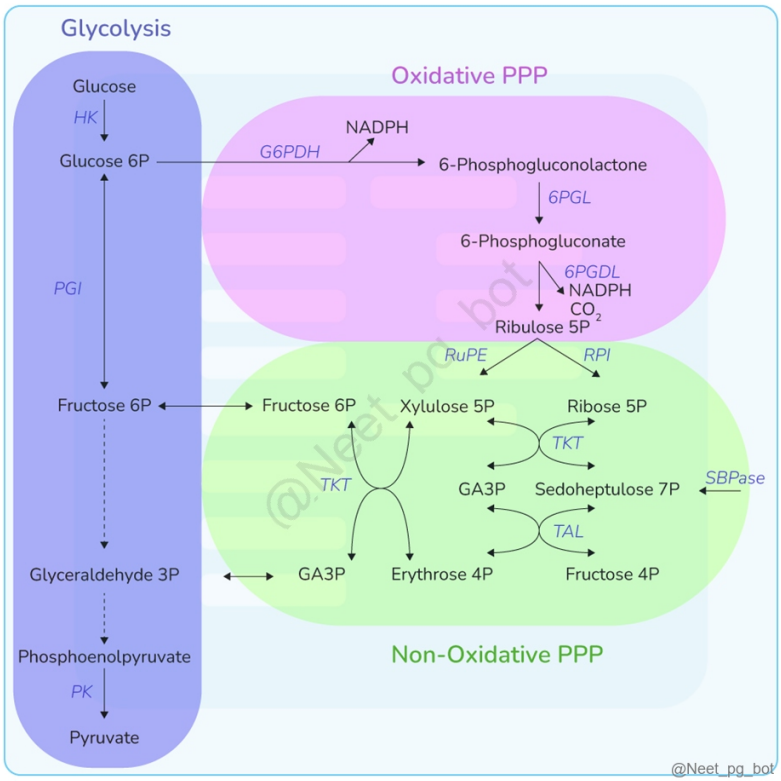
C. 6-phosphogluconate dehydrogenase

D. Transaldolase

Correct Answer: C. 6-phosphogluconate dehydrogenase

■ *Listen to Audio 1*

Explanation: Enzymes of the HMP (Hexose Monophosphate) Shunt / Pentose Phosphate Pathway (PPP) are divided into the two phases: Oxidative (Irreversible) Phase – Produces NADPH Glucose-6-phosphate dehydrogenase (G6PD) 6-Phosphogluconolactonase 6-phosphogluconate dehydrogenase Non-Oxidative (Reversible) Phase – Interconversion of sugars Ribulose-5-phosphate isomerase Ribulose-5-phosphate epimerase Transketolase Transaldolase Pentose Phosphate Pathway :



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 194 Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Page 131

↑ *Back to this question in Part A*

@Neet_pg_bot

Q3: Which of the following statements about the pentose phosphate (HMP) pathway in skeletal muscle is most accurate? (Q891569)

A. It is completely absent because muscle lacks glucose-6-phosphate dehydrogenase

B. It is highly active because muscle requires large amounts of NADPH for lipogenesis

C. It is present but has low activity because muscle has low demand for NADPH

D. It functions only through the non-oxidative phase and cannot generate NADPH

Correct Answer: C. It is present but has low activity because muscle has low demand for NADPH

Explanation: The pentose phosphate (HMP) pathway is present in most tissues , including skeletal muscle , but its activity depends on the tissue's metabolic role . The major functions of the HMP shunt are: Production of NADPH for reductive biosynthesis and maintenance of reduced glutathione Production of ribose-5-phosphate for nucleotide and nucleic acid synthesis Tissues specialized in reductive synthesis (e.g., fatty acids, steroids) or antioxidant defense show high activity of this pathway. Skeletal muscle has low demand for NADPH because: It performs minimal fatty acid or steroid synthesis Its primary role is ATP generation via glycolysis and oxidative phosphorylation Therefore, in muscle, the HMP shunt is present but operates at a low rate . (Option A and B ruled out) Muscle contains low activity of the oxidative enzymes (glucose-6-phosphate dehydrogenase and 6-phosphogluconate dehydrogenase) (Option D ruled out) , but: It can still synthesize ribose-5-phosphate by reversal of the non-oxidative phase using glycolytic intermediates (fructose-6-phosphate and glyceraldehyde-3-phosphate). In contrast, tissues like liver, adipose tissue, adrenal cortex, testis, lactating mammary gland, and erythrocytes have high HMP activity because they require large amounts of NADPH (for lipid/steroid synthesis or for maintaining reduced glutathione).

References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 194 - 195

↑ *Back to this question in Part A*

@Neet_pg_bot

Q4: The HMP shunt pathway occurs in which cellular compartment? (Q891568)

A. Mitochondria

B. Nucleus

C. Cytosol

D. Endoplasmic Reticulum

Correct Answer: C. Cytosol

Explanation: All enzymes of the pentose phosphate pathway (HMP shunt) are located in the cytosol . The pathway produces NADPH (for reductive biosynthesis and maintenance of reduced glutathione) and ribose-5-phosphate (for nucleotide synthesis), both of which are utilised in the cytosolic metabolic network. Both phases the oxidative (irreversible) and non-oxidative (reversible) occur entirely in the cytosol . This localisation is logical because glycolysis and fatty acid synthesis also occur in the cytosol and are metabolically linked to the HMP shunt. The pathway is especially active in tissues with high NADPH demand such as liver, adrenal cortex, and mammary glands . The HMP shunt does not produce or consume ATP ; its main outputs are NADPH and ribose-5-phosphate . Mitochondria (Option A): Mitochondria are the site of the TCA cycle, oxidative phosphorylation, and β-oxidation , but the enzymes of the HMP shunt are not located here . Nucleus (Option B): Although

ribose-5-phosphate is used for nucleotide synthesis in the nucleus, the enzymatic reactions of the HMP shunt occur in the cytosol , not in the nucleus. Endoplasmic Reticulum (Option D): The ER is involved in protein synthesis, lipid synthesis, and detoxification , but it does not contain the enzymes of the pentose phosphate pathway.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 192

↑ *Back to this question in Part A*

@Neet_pg_bot

Q5: Which phase of the hexose monophosphate (HMP) shunt pathway is reversible? (Q891567)

A. Oxidative phase

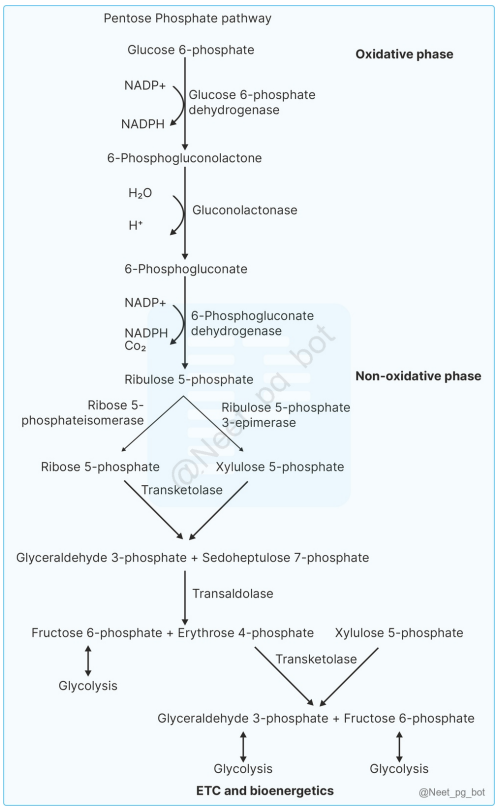
B. Non-oxidative phase

C. Both phases

D. Neither phase

Correct Answer: B. Non-oxidative phase

Explanation: The HMP shunt (pentose phosphate pathway) has two phases : Oxidative phase (Irreversible): Converts glucose-6-phosphate → ribulose-5-phosphate Produces NADPH and CO■ The rate-limiting enzyme is glucose-6-phosphate dehydrogenase (G6PD) Because CO■ is released and the reactions have a large negative ΔG, this phase is irreversible Non-oxidative phase (Reversible): Involves interconversion of sugars (ribose-5-phosphate, xylulose-5-phosphate, etc.) Uses transketolase (TPP-dependent) and transaldolase Converts 3 pentoses → 2 fructose-6-phosphate + 1 glyceraldehyde-3-phosphate Contains unique intermediates like sedoheptulose-7-phosphate (C7) and erythrose-4-phosphate (C4) Because these reactions are near-equilibrium , the pathway can run in either direction depending on whether the cell needs ribose-5-phosphate (for nucleotide synthesis) or glycolytic intermediates No ATP is produced in the HMP shunt Main products: NADPH (for reductive biosynthesis & antioxidant defence) and ribose-5-phosphate (for nucleotides) Clinical link: Thiamine (TPP) deficiency impairs transketolase , seen in Wernicke-Korsakoff syndrome



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 192

↑ *Back to this question in Part A*

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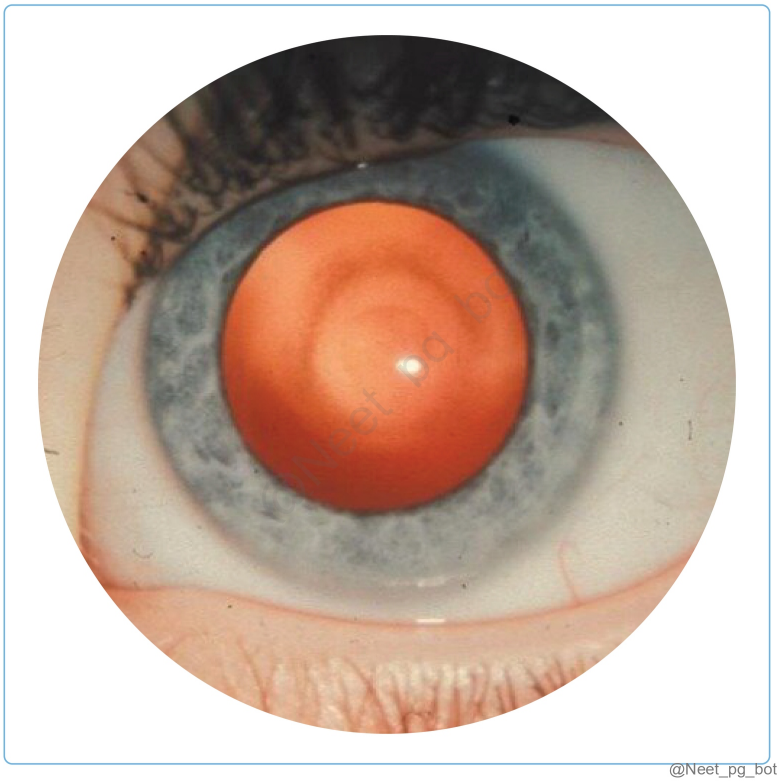
Q6: A newborn presents with bilateral lens opacities showing an “oil droplet” appearance on retroillumination. Which metabolic disorder is classically associated with this finding? (Q845037)

A. Phenylketonuria

B. Galactosemia

C. Maple Syrup Urine Disease

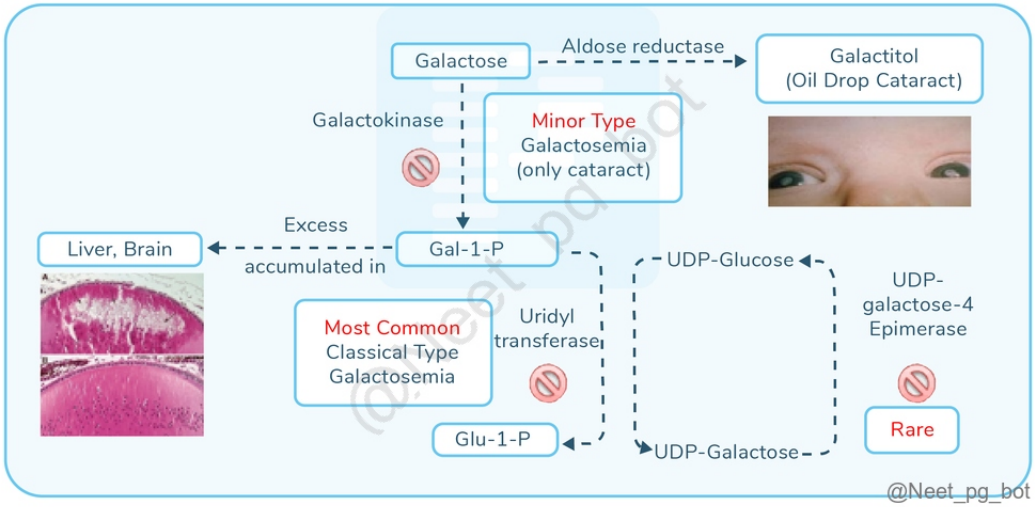
D. Homocystinuria



Correct Answer: B. Galactosemia

■ *Listen to Audio 1*

Explanation: Galactosemia Autosomal recessive Enzyme deficiency: Galactose-1-phosphate uridyltransferase (GALT) Impaired conversion of galactose to glucose leads to the buildup of galactitol leads to oil-droplet cataract shortly after birth Phenylketonuria (Option A) is a metabolic disorder affecting phenylalanine metabolism, leading to intellectual disability if untreated. Maple Syrup Urine Disease (Option C) is a metabolic disorder affecting branched-chain amino acids, characterized by sweet-smelling urine. Homocystinuria (Option D) is a disorder affecting methionine metabolism, leading to symptoms such as skeletal abnormalities and lens dislocation.



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 200
https://www.aao.org/education/bcscsnippetdetail.aspx?id=e3168532-be6c-41e3-8ea5- 47c8317832df
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1310285/pdf/taos00034-0671.pdf

↑ *Back to this question in Part A*

@Neet_pg_bot

Q7: A diabetic patient develops snowflake cataracts. Sorbitol accumulation in the lens fibers is responsible. Deficiency or low activity of which enzyme in the lens leads to this complication? (Q845036)

A. Aldose Reductase

B. Glucose-6-Phosphate Dehydrogenase

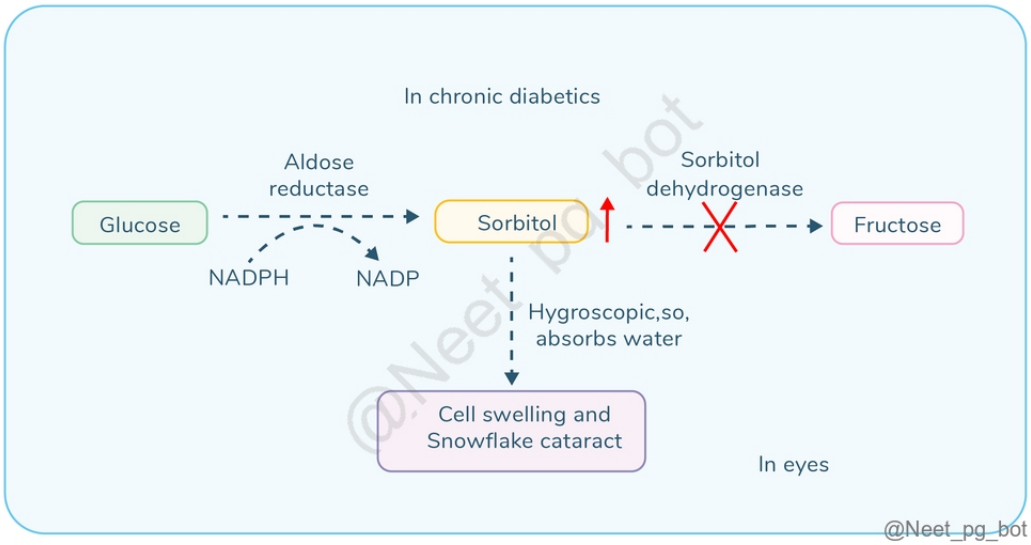
C. Sorbitol Dehydrogenase

D. Hexokinase

Correct Answer: C. Sorbitol Dehydrogenase

■ *Listen to Audio 1*

Explanation: Hyperglycemia and Sorbitol Accumulation: Excess glucose enters insulin-independent cells (retina, lens, kidneys, peripheral nerves). Aldose reductase activity increases, leading to more sorbitol production. Sorbitol dehydrogenase enzyme is absent in the eye hence sorbitol cannot be broken down further. Sorbitol gets trapped inside cells, causing water influx and cell swelling and cataract Formation. Retina, lens, kidneys and peripheral nerves have aldose reductase but lack sorbitol dehydrogenase, leading to sorbitol accumulation. Aldose Reductase (Option A) converts glucose to sorbitol. Glucose-6-phosphate dehydrogenase (Option B) is a part of the pentose phosphate pathway and does not play a direct role in the sorbitol pathway. Hexokinase (Option D): Catalyses the first step of glycolysis and is not involved in the sorbitol pathway.



References:

Lippincott Illustrated Reviews Biochemistry, 7th Edition, Chapter 12, II. Fructose Metabolism

↑ *Back to this question in Part A*

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Q8: What is the role of inosine monophosphate in stored blood? (Q845035)

A. It increases plasma Na and K levels.

B. It stimulates 2,3-DPG synthesis.

C. It prevents lactic acid buildup.

D. It stabilizes hemoglobin concentration.

Correct Answer: B. It stimulates 2,3-DPG synthesis.

■ *Listen to Audio 1*

Explanation: Inosine and 2,3-DPG in Stored Blood : 2,3-DPG in RBCs binds hemoglobin, reducing O■ affinity and promoting oxygen release to tissues. In stored blood, 2,3-DPG declines → hemoglobin binds O■ more tightly → impaired tissue oxygen delivery. IMP in stored blood is metabolized by RBCs to produce ribose-1-phosphate, which enters the pentose phosphate pathway and is ultimately converted to 1,3-DPG → 2,3-DPG. This helps to maintain adequate 2,3-DPG levels, ensuring that transfused red blood cells can effectively deliver oxygen to tissues. Rapoport–Luebering Cycle: A shunt pathway of glycolysis in RBCs that regulates 2,3-BPG levels . Location: Only in RBCs Purpose: Regulates oxygen delivery Step Enzyme Product 1,3-Bisphosphoglycerate Bisphosphoglycerate mutase 2,3-BPG 2,3-Bisphosphoglycerate 2,3-Bisphosphoglycerate phosphatase

3-Phosphoglycerate It increases plasma Na and K levels (Option A): IMP does not affect plasma electrolytes It prevents lactic acid buildup (Option C): Lactic acid accumulates due to anaerobic glycolysis . IMP helps RBC metabolism, but does not prevent lactic acid formation . It stabilizes hemoglobin concentration (Option D): Hemoglobin concentration depends on initial blood collection, not IMP

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 561

www.sciencedirect.com/science/article/pii/S0006

www.ncbi.nlm.nih.gov/pmc/articles/PMC1062830/pdf/jcinvest00347-0031.pdf

↑ *Back to this question in Part A*

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Q9: A 30-year-old woman with G6PD deficiency presents with fatigue, jaundice, dark urine, and hemolytic anemia after eating fava beans. How does G6PD deficiency lead to these symptoms? (Q845034)

A. Reduced NADPH impairs glutathione regeneration, causing hemolytic anemia.

B. Increased ribulose-5-phosphate overloads nucleotides, leading to renal failure.

C. Impaired glucose-6-phosphate conversion disrupts glycolysis, causing hypoglycemia.

D. Decreased ATP production affects muscle function, causing myopathy.

Correct Answer: A. Reduced NADPH impairs glutathione regeneration, causing hemolytic anemia.

■ *Listen to Audio 1*

Explanation: In G6PD deficiency, RBCs have insufficient NADPH due to impaired pentose phosphate pathway activity. NADPH is needed to regenerate reduced glutathione (GSH) , which protects RBCs from oxidative damage. Oxidative triggers (e.g., fava beans, infections, certain drugs) cause RBC membrane and hemoglobin damage → hemolysis . Hemolysis leads to fatigue, jaundice, and dark urine due to hemoglobin and bilirubin release. Increased ribulose-5-phosphate overloads nucleotides, leading to renal failure (Option B): While ribulose-5-phosphate is a product of the PPP, its accumulation does not directly lead to renal failure. Impaired glucose-6-phosphate conversion disrupts glycolysis, causing hypoglycemia (Option C): While G6PD deficiency affects glucose-6-phosphate, it does not significantly disrupt glycolysis to cause hypoglycemia. The primary issue is oxidative stress, not energy deficiency. Decreased ATP production affects muscle function, causing myopathy (Option D): While ATP is important, the PPP is not a major ATP-producing pathway in RBCs.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 198, 657

Lippincott Illustrated Reviews Biochemistry, 7th Edition, V. G6PD Deficiency

↑ *Back to this question in Part A*

@Neet_pg_bot

Q10: Which is the rate-limiting enzyme in galactose metabolism? (Q845025)

A. Galactokinase

B. Galactose-1-phosphate uridyltransferase

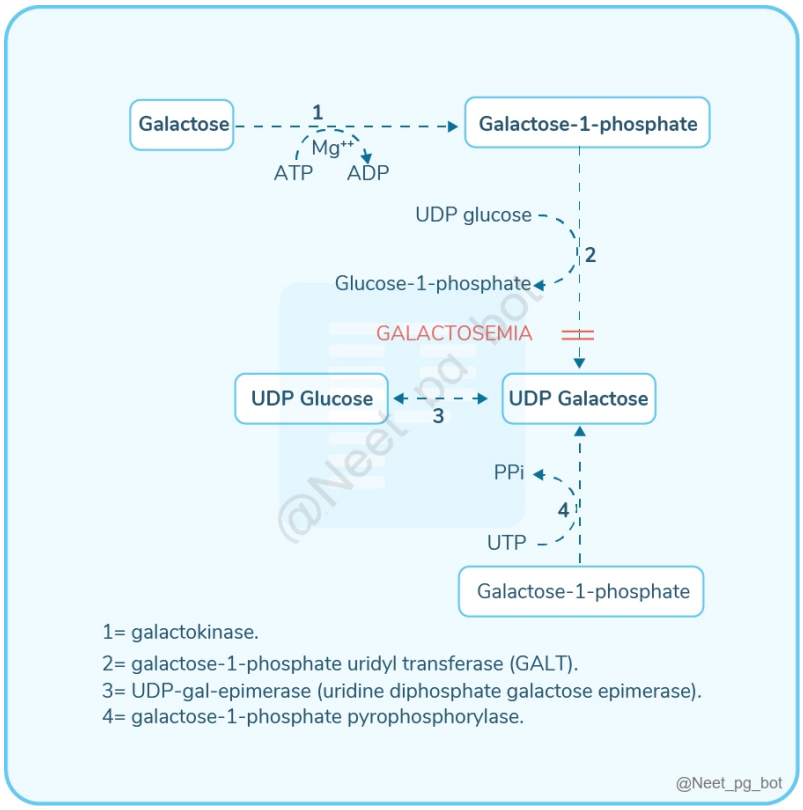
C. UDP-hexose-4-epimerase

D. Aldolase Reductase

Correct Answer: B. Galactose-1-phosphate uridyltransferase

■ *Listen to Audio 1*

Explanation: Galactose-1-phosphate uridyltransferase (GALT) is the rate-limiting enzyme in galactose metabolism. Deficiency in GALT leads to classic galactosemia Galactose Metabolism : Aldolase reductase (Option D)- reduces galactose to galactitol and glucose to sorbitol.



References:

Lippincott Illustrated Biochemistry, 7th Edition, Pages 427–430 Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Pages 138–139

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: All the following enzymes catalyze oxidative decarboxylation reactions except? (Q845032)

A. α-Ketoglutarate dehydrogenase

B. Malate dehydrogenase

C. 6-Phosphogluconate dehydrogenase

D. Isocitrate dehydrogenase

Correct Answer: B. Malate dehydrogenase

■ *Listen to Audio 1*

Explanation: Malate Dehydrogenase Catalyzes the oxidation of malate to oxaloacetate in the citric acid cycle, producing NADH but not releasing CO2. This is an oxidation reaction, not an oxidative decarboxylation. Malate + NAD + → Oxaloacetate + NADH + H + Enzymes that catalyse oxidative decarboxylation reaction: Enzyme Reaction (Oxidative decarboxylation of) Cofactors Pyruvate Dehydrogenase (PDH) Pyruvate to acetyl-CoA Thiamin diphosphate, lipoate, NAD+, FAD, CoA α-Ketoglutarate Dehydrogenase (αKGDH) (Option A ruled out) α-ketoglutarate to succinyl-CoA Isocitrate Dehydrogenase (IDH) (Option D ruled out) Isocitrate to α-ketoglutarate Mg 2+ or Mn 2+ ions 6-Phosphogluconate Dehydrogenase (6-PGDH) (Option C ruled out) 6-phosphogluconate to ribulose-5-phosphate NADP + Branched-Chain Ketoacid Dehydrogenase (BCKADH) Decarboxylation of branched-chain keto acids Thiamin pyrophosphate (TPP), lipoate, NAD + , FAD, CoA

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 159, 168, 193, 298

↑ *Back to this question in Part A*

@Neet_pg_bot

Q12: Which statement is true regarding NADH and NADPH? (Q845031)

A. Both are produced in the Pentose Phosphate Pathway.

B. NADH is used in lipid biosynthesis, while NADPH is not.

C. NADPH is used for anabolic reactions, while NADH is primarily for catabolic reactions.

D. Both serve same function in the cell.

Correct Answer: C. NADPH is used for anabolic reactions, while NADH is primarily for catabolic reactions.

■ *Listen to Audio 1*

Explanation: NADH vs NADPH : Feature NADH NADPH Pathways (Option A) Primarily generated in catabolic pathways: Citric Acid Cycle, Glycolysis, Fatty Acid oxidation. Primarily generated in anabolic pathways: Pentose Phosphate Pathway, Malic Enzyme Reaction, Oxidative Decarboxylation - Isoforms of Isocitrate Dehydrogenase. Key Functions (Option B, D) Donates electrons to the Electron Transport Chain (ETC) for ATP production. The ETC uses NADH for ATP production, yielding 2.5 ATPs. Donates electrons & provides reducing power for biosynthetic reactions like lipid and nucleotide synthesis. Maintains reduced glutathione (G-SH) in RBCs Regenerates reduced glutathione (G-SH) from its oxidized form. Production Site Mitochondria (mainly), Cytosol Cytosol (mainly), some in mitochondria and plastids

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 87, 117, 194–195, 228, 342 Lippincott Illustrated Reviews Biochemistry, 7th Edition, Page 129

↑ *Back to this question in Part A*

@Neet_pg_bot

Q13: Which sequence correctly represents the steps of the oxidative phase of the pentose phosphate pathway (PPP)? (Q845030)

A. 6-Phosphogluconolactone → Glucose-6-phosphate → 6-Phosphogluconate → Ribulose-5-phosphate

B. Glucose-6-phosphate → 6-Phosphogluconate → 6-Phosphogluconolactone → Ribulose-5-phosphate

C. Glucose-6-phosphate → 6-Phosphogluconolactone → 6-Phosphogluconate → Ribulose-5-phosphate

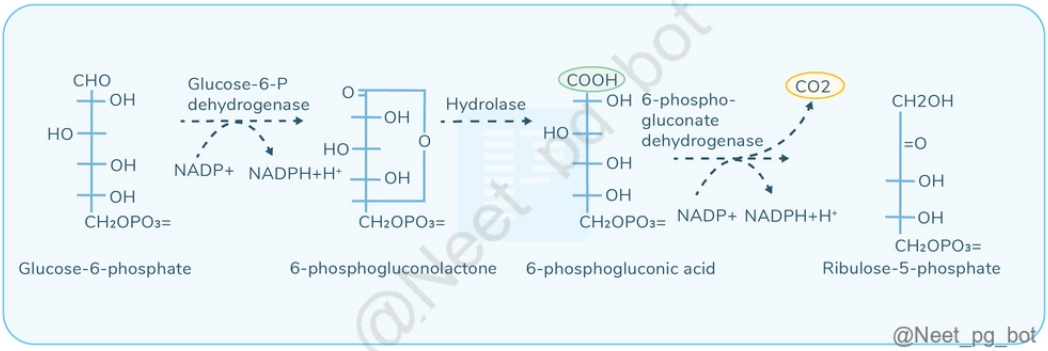
D. 6-Phosphogluconate → 6-Phosphogluconolactone → Glucose-6-phosphate → Ribulose-5-phosphate

Correct Answer: C. Glucose-6-phosphate → 6-Phosphogluconolactone → 6-Phosphogluconate → Ribulose-5-phosphate

■ *Listen to Audio 1*

Explanation: Oxidative Phase of the Pentose Phosphate Pathway (PPP): Irreversible process producing NADPH and converting glucose-6-phosphate to ribulose-5-phosphate. Fate of products: NADPH: The two molecules of NADPH produced in this phase are essential for various biosynthetic pathways, such as fatty acid synthesis and steroid synthesis. Ribulose-5-Phosphate: Used for nucleotide synthesis or converted to other sugars for glycolysis

or gluconeogenesis.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 193–194

↑ *Back to this question in Part A*

@Neet_pg_bot

Q14: A 45-year-old man with hemolytic anemia shows RBCs susceptible to oxidative damage. What is the main function of the pentose phosphate pathway in RBCs? (Q845029)

A. To produce ATP for energy metabolism

B. To provide NADPH for fatty acid and cholesterol synthesis

C. To provide NADPH for maintaining reduced glutathione levels

D. To synthesize hemoglobin

Correct Answer: C. To provide NADPH for maintaining reduced glutathione levels

■ *Listen to Audio 1*

Explanation: Pentose phosphate pathway i s essential in erythrocytes because it generates NADPH which keeps glutathione in its reduced form. Reduced glutathione (GSH) acts as a key antioxidant that donates electrons to neutralize reactive oxygen species. In the process, GSH gets oxidized to glutathione disulfide (GSSG) . NADPH reduces GSSG back to GSH via glutathione reductase . In G6PD deficiency , NADPH production is impaired → GSH cannot be maintained → RBCs become vulnerable to oxidative stress → hemolysis occurs. To produce ATP for energy metabolism (Option A): While RBCs do require ATP, their primary energy source is glycolysis. To provide NADPH for fatty acid and cholesterol synthesis(Option B): PPP produce NADPH , which can be used for anabolic reactions. However, mature RBCs lack organelles for lipid synthesis, so this is not the primary role in RBCs. To synthesize haemoglobin (Option D): Haemoglobin synthesis occurs during RBC development in the

bone marrow.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 191, 194–196

↑ *Back to this question in Part A*

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Q15: Which of the following statements is true regarding Hereditary Fructose Intolerance (HFI) and Classical Galactosemia? (Q845028)

A. Both present with hyperglycemia.

B. Both require lifelong avoidance of lactose.

C. Both present with hypophosphatemia.

D. Both have symptoms that appear at birth.

Correct Answer: C. Both present with hypophosphatemia.

■ *Listen to Audio 1*

Explanation: In HFI, accumulated fructose-1-phosphate and in Classical Galactosemia, accumulated galactose-1-phosphate both interferes with phosphate metabolism, leading to a decrease in available phosphate, resulting in hypophosphatemia Feature Hereditary Fructose Intolerance Classical Galactosemia Enzyme Deficiency Aldolase B Galactose-1-phosphate uridyltransferase (GALT) Onset (Option D ruled out) Symptoms appear when sucrose/ fructose is introduced into the diet (around 6 months of age) Initial signs usually present in the first few weeks of life Accumulated Metabolite Fructose-1-phosphate Galactose-1-phosphate and galactose Symptoms (Option A ruled out) (Option C) Hypoglycemia, h ypophosphatemia, hyperuricemia, lactic acidosis Hepatomegaly, jaundice, Vomiting, Loss of appetite, Failure to thrive. Aldose reductase cannot act on fructose (very low risk of cataract) Hypoglycemia, hypophosphatemia, hyperuricemia, lactic acidosis, Hepatomegaly, jaundice, Severe mental retardation, Galactosuria, Cataracts Dietary Management (Option B ruled out) Sucrose, sorbitol and fructose must be removed from the diet Galactose and lactose must be removed from the diet

References:

Lippincott Illustrated Reviews Biochemistry, 7th Edition, Pages 423, 424, 430 Harper’s Illustrated Biochemistry, 32nd Edition, Pages 199, 200 Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Pages 138, 139, 140 https://www.childrenshospital.org/conditions/galactosemia https://www.ncbi.nlm.nih.gov/books/NBK1518/

↑ *Back to this question in Part A*

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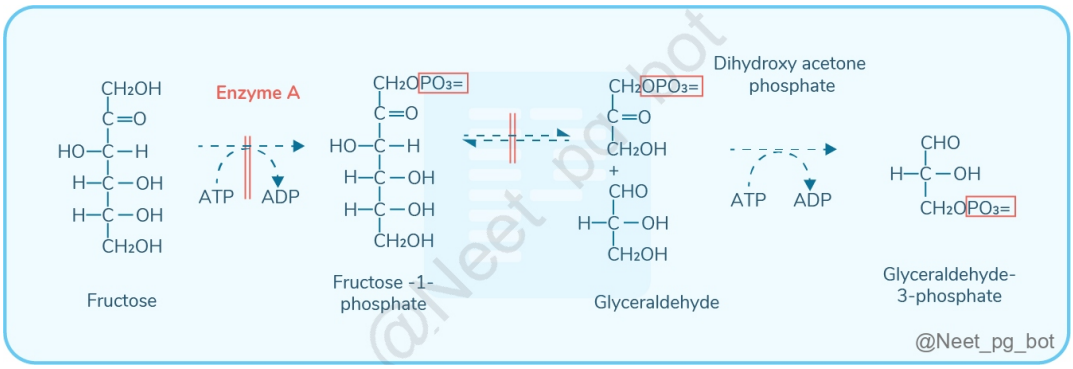
Q16: A child is diagnosed with essential fructosuria during a routine health check. Name the enzyme A responsible for this condition. (Q845027)

A. Aldolase B

B. Hexokinase

C. Fructokinase

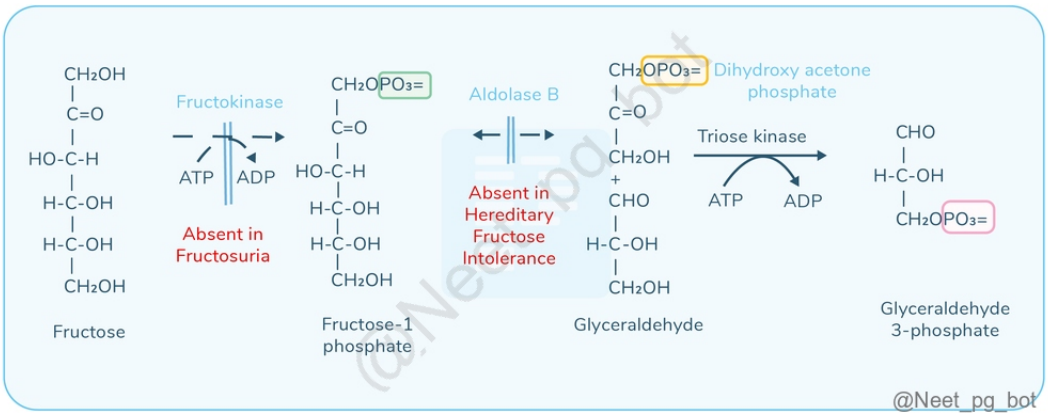
D. Glucokinase



Correct Answer: C. Fructokinase

■ *Listen to Audio 1*

Explanation: Fructose Metabolism : Fructokinase is the primary enzyme for fructose phosphorylation. It converts fructose to fructose-1-phosphate using ATP. Aldolase B c leaves fructose 1-phosphate to dihydroxyacetone phosphate (DHAP) and glyceraldehyde. DHAP enters glycolysis or gluconeogenesis. Disorders of Fructose metabolism: 1) Essential Fructosuria: Caused by fructokinase deficiency 2) Hereditary Fructose Intolerance (HFI): Caused by aldolase B deficiency . (Option A ruled out) Hexokinase (Option B) – It is involved in the initial phosphorylation of glucose, not fructose. Hexokinase can phosphorylate fructose to fructose-6-phosphate, but it does not cause the specific symptoms seen in essential fructosuria. Glucokinase (Option D) – It is involved in glucose metabolism.



References:

Lippincott Illustrated Reviews Biochemistry, 7th Edition, Pages 420–424 Harper’s Illustrated Biochemistry, 32nd Edition, Pages 199, 200 Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Pages 137, 138

↑ *Back to this question in Part A*

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Q17: An infant is diagnosed with classic galactosemia. Which enzyme deficiency is responsible for this condition? (Q845026)

A. Galactokinase

B. Galactose-1-phosphate uridyltransferase

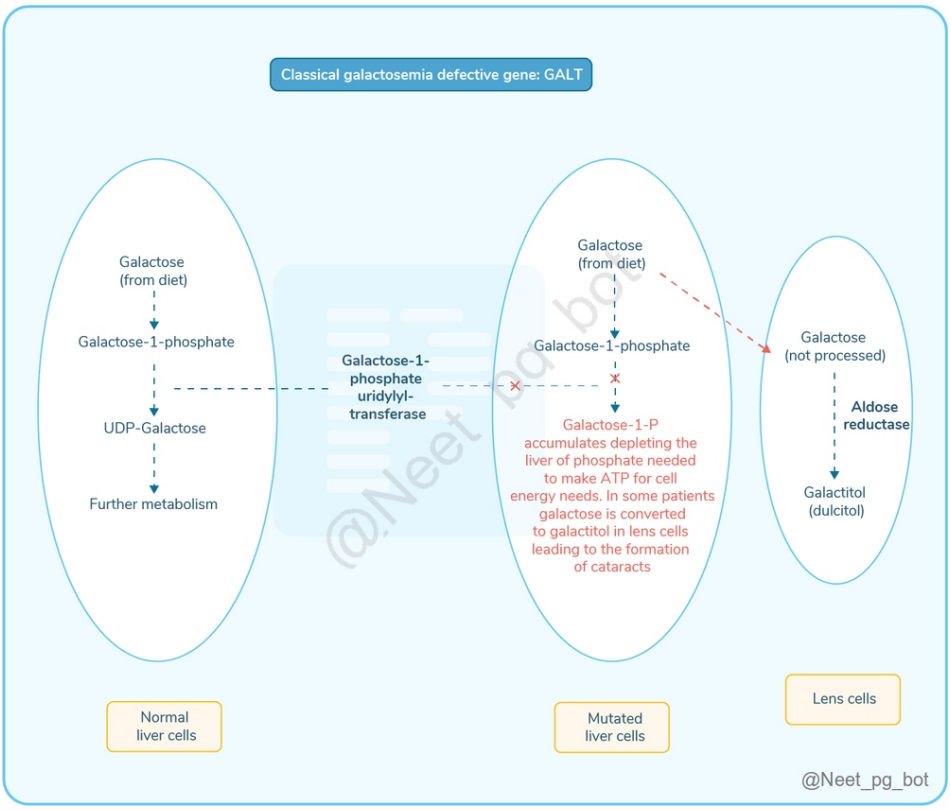
C. UDP-galactose 4-epimerase

D. Aldose reductase

Correct Answer: B. Galactose-1-phosphate uridyltransferase

■ *Listen to Audio 1*

Explanation: Galactose-1-phosphate uridylyltransferase (GALT) deficiency is seen in classic g alactosemia, in which galactose-1-phosphate and, therefore, galactose accumulate. Accumulation of galactose-1-phosphate → inhibits galactokinase & glycogen phosphorylase → hypoglycemia . Excess galactose → converted to galactitol by aldose reductase → cataracts GALT deficiency is a part of the newborn screening panel Galactokinase (Option A) phosphorylate galactose to form galactose 1-phosphate. Its deficiency leads to less severe disorder of galactose metabolism (Benign Galactosemia). UDP-hexose-4-epimerase (Option C) isomerises UDP-galactose to UDP-glucose. Its deficiency leads to a less severe disorder of galactose metabolism. Aldolase reductase (Option D) reduces galactose to galactitol and glucose to sorbitol.



References:

Lippincott Illustrated Reviews Biochemistry, 7th Edition, Page 430 Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Page 139

↑ *Back to this question in Part A*

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• [02_Carbohydrate Chemistry] 004 Fates Of Pyruvate Pdh Complex Electron Transport Chain

« Back to Folder Directory

[02_Carbohydrate Chemistry] 004 Fates Of Pyruvate Pdh Complex Electron Transport Chain — Quick Jump

Q1 (p. 501)	*Q2 (p. 502)*	*Q3 (p. 503)*	*Q4 (p. 504)*	*Q5 (p. 504)*	*Q6 (p. 505)*
Q7 (p. 506)	*Q8 (p. 507)*	*Q9 (p. 508)*	*Q10 (p. 509)*	*Q11 (p. 510)*	*Q12 (p. 511)*
Q13 (p. 513)	*Q14 (p. 513)*	*Q15 (p. 517)*	*Q16 (p. 518)*	*Q17 (p. 519)*	*Q18 (p. 520)*
Q19 (p. 521)	*Q20 (p. 522)*				

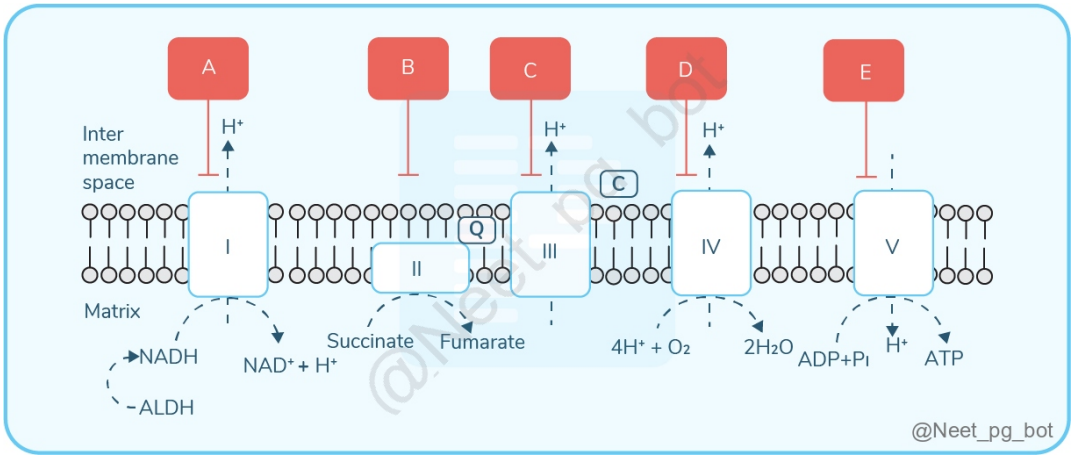
Q1: The image below shows the sites of action for different ETC inhibitors. Based on the image, which of the following is the site of action of Cyanide? (Q845065)

A. A

B. B

C. C

D. D

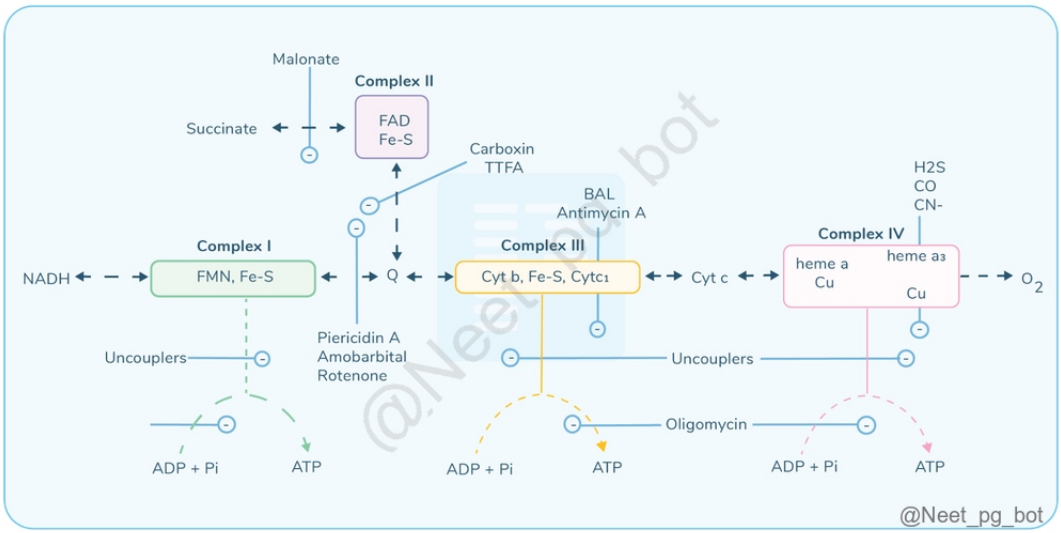


Correct Answer: D. D

■ *Listen to Audio 1*

Explanation: Cyanide is a strong inhibitor of Complex IV (cytochrome c oxidase). It blocks electron transfer to oxygen, disrupting the electron transport chain and reducing ATP production, potentially causing cellular damage . Inhibitors of the Electron Transport Chain (ETC): Complex I (Option A) : Rotenone, Amobarbital,Piericidin Complex II (Option B) : TTFA,Carboxin , Malonate (competitive inhibition.) Complex III (Option C) :

Antimycin A, BAL Complex IV : Cyanide, Carbon monoxide, Hydrogen Sulfide Complex V(ATP Synthase) : Oligomycin, Atractyloside



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 127 https://www.researchgate.net/figure/Inhibitors-of-oxidative-phosphorylation-OXPHOS-The-OXPHOS-metabolic-pathway-generates_fig1_342929817

↑ *Back to this question in Part A*

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Q2: Oligomycin inhibits oxidative phosphorylation by: (Q891582)

A. Blocking electron transport at Complex I

B. Binding to ATP synthase (F■ domain), blocking proton flow

C. Uncoupling proton gradient

D. Inhibiting ADP-ATP translocase

Correct Answer: B. Binding to ATP synthase (F■ domain), blocking proton flow

Explanation: Oligomycin binds to the F■ domain of ATP synthase , which forms the proton channel in the inner mitochondrial membrane. Blocking H■ re-entry into the mitochondrial matrix , it prevents proton flow through ATP synthase and thereby halts ATP synthesis . Because proton flow through F■ is required to drive rotation of the central stalk (γ subunit) and the conformational changes in the F■ catalytic domain, ATP formation from ADP + Pi is completely arrested. Since the proton gradient can no longer be dissipated, the proton-motive force builds up , creating back-pressure that slows or stops the electron transport chain (ETC) . As a result, oxygen consumption decreases , a classic laboratory finding with oligomycin. Oligomycin is therefore widely used

experimentally to distinguish ATP-linked respiration from proton leak respiration . Blocking electron transport at Complex I (Option A): This is the mechanism of rotenone and amobarbital , which inhibit electron transfer from NADH at Complex I. They reduce ATP synthesis by blocking electron flow, not by acting on ATP synthase . Uncoupling proton gradient (Option C): This describes the action of uncouplers such as 2,4-dinitrophenol (DNP) and thermogenin , which increase proton permeability of the inner mitochondrial membrane and dissipate the proton gradient as heat rather than blocking ATP synthase. Inhibiting ADP-ATP translocase (Option D): This is the mechanism of atractyloside and bongkrekic acid , which block the exchange of ADP and ATP across the inner mitochondrial membrane. This indirectly halts oxidative phosphorylation due to lack of substrate, but does not involve the F■ domain of ATP synthase .

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 127

↑ *Back to this question in Part A*

@Neet_pg_bot

Q3: According to the chemiosmotic theory, ATP synthesis is driven by: (Q891580)

A. Substrate-level phosphorylation

B. Direct transfer of phosphate from NADH

C. Proton gradient across inner mitochondrial membrane

D. Electron transfer without gradient

Correct Answer: C. Proton gradient across inner mitochondrial membrane

Explanation: The chemiosmotic theory proposed by Peter Mitchell states that electron transport through the respiratory chain generates an electrochemical proton gradient (proton-motive force) across the inner mitochondrial membrane. Complexes I, III, and IV pump H■ ions from the mitochondrial matrix into the intermembrane space , creating both a concentration gradient and an electrical gradient . The inner mitochondrial membrane is largely impermeable to protons , so H■ can return to the matrix mainly through the ATP synthase (FoF■) complex . The flow of protons through the Fo subunit drives rotational catalysis in the F■ subunit, leading to conformational changes that catalyse the reaction: ADP + Pi → ATP . Thus, ATP synthesis in oxidative phosphorylation is driven by the proton-motive force , not by direct electron transfer or substrate-level phosphorylation. This represents the final energy-conserving step of the electron transport chain. Substrate-level phosphorylation (Option A): This is the direct transfer of a phosphate group from a high-energy intermediate to ADP (as in glycolysis and the succinyl-CoA step of the TCA cycle). It does not require the electron transport chain or a proton gradient , so it is not the mechanism described by chemiosmotic theory. Direct transfer of phosphate from NADH (Option B): NADH carries high-energy electrons, not phosphate groups . It donates electrons to Complex I of the ETC to help generate the proton gradient, but it does not directly provide phosphate for ATP synthesis . Electron transfer without gradient (Option D): Electron transfer through the ETC provides the energy to pump protons and create the gradient , but electron flow alone cannot drive ATP synthesis .

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 124

↑ *Back to this question in Part A*

@Neet_pg_bot

Q4: Which metal ion is a component of cytochrome oxidase (Complex IV)? (Q891577)

A. Iron only

B. Magnesium

C. Zinc

D. Copper

Correct Answer: D. Copper

Explanation: Cytochrome c oxidase (Complex IV) contains heme a, heme a■ (iron-containing heme groups) and copper centres (CuA and CuB) . The copper ions cycle between Cu■ and Cu²■ during electron transfer. Complex IV catalyses the final step of the electron transport chain , transferring electrons to molecular oxygen and reducing it to water (H■O) . The redox reactions involving the heme and copper centres provide the free energy required for proton pumping across the inner mitochondrial membrane , contributing to the electrochemical gradient used for ATP synthesis. Copper is also essential for the proper assembly and stability of Complex IV; copper deficiency impairs its structure and activity. Clinically, disorders of copper metabolism (e.g., Menkes disease) lead to reduced cytochrome c oxidase activity , resulting in severe neurological and developmental impairment. Complex IV is classically inhibited by CN■, CO, and H■S , which block electron transfer to oxygen. Iron only (Option A): Complex IV does contain iron in heme a and heme a■ , but it also requires copper centers (CuA and CuB) for electron transfer to oxygen. Magnesium (Option B): Magnesium is a cofactor mainly for ATP-dependent enzymes and kinases and stabilizes ATP, but it does not participate in the electron transport chain Zinc (Option C): Zinc is a structural or catalytic ion in enzymes such as carbonic anhydrase and alcohol dehydrogenase and in zinc-finger protein

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 124

↑ *Back to this question in Part A*

@Neet_pg_bot

Q5: Succinate-Q reductase belongs to which ETC complex? (Q891575)

A. Complex I

B. Complex II

C. Complex III

D. Complex IV

Correct Answer: B. Complex II

Explanation: Complex II is also known as succinate dehydrogenase or succinate-Q reductase . It catalyses the oxidation of succinate to fumarate in the TCA cycle. During this reaction, electrons are transferred to a covalently bound FAD , forming FADH■ . Electrons then pass through iron-sulfur (Fe-S) centers to ubiquinone (Coenzyme Q) , reducing it to ubiquinol (QH■) . Complex II does not pump protons , unlike Complexes I, III, and IV , and therefore contributes less to the proton motive force and yields less ATP per electron pair . Because electrons enter the ETC at the level of Complex II (via FADH■), they bypass Complex I . Complex I (Option A): This is NADH-Q oxidoreductase , which accepts electrons from NADH and pumps protons across the inner mitochondrial membrane. Complex III (Option C): This is Q-cytochrome c oxidoreductase , which transfers electrons from ubiquinol (QH■) to cytochrome c and pumps protons via the Q cycle. Complex IV (Option D): This is cytochrome c oxidase , the terminal complex of the ETC, which transfers electrons from cytochrome c to oxygen , forming water , and pumps protons .

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 122

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: Complex I of the electron transport chain is also known as: (Q891574)

A. Succinate-Q reductase

B. Cytochrome c oxidase

C. NADH-Q oxidoreductase

D. Q-cytochrome c oxidoreductase

Correct Answer: C. NADH-Q oxidoreductase

Explanation: Complex I is also called NADH dehydrogenase or NADH-CoQ (Q) oxidoreductase . It accepts electrons from NADH and transfers them to ubiquinone (Coenzyme Q) . It contains FMN and multiple iron-sulfur (Fe-S) clusters as redox centers. During electron transfer, it pumps 4 H■ from the mitochondrial matrix to the intermembrane space , contributing to the proton motive force . Structurally, it is a large L-shaped complex , with one arm embedded in the inner mitochondrial membrane and the other projecting into the matrix. Inhibitors such

as rotenone, amytal, and piericidin A block electron transfer through Complex I and increase reactive oxygen species (ROS) formation. Succinate-Q reductase (Option A): This is Complex II (succinate dehydrogenase) , which transfers electrons from FADH■ to coenzyme Q and does not pump protons . Cytochrome c oxidase (Option B): This is Complex IV , the terminal enzyme of the ETC, which transfers electrons from cytochrome c to oxygen , forming water . Q-cytochrome c oxidoreductase (Option D): This is Complex III , which transfers electrons from ubiquinol (QH■) to cytochrome c via the Q cycle .

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 122

↑ *Back to this question in Part A*

@Neet_pg_bot

Q7: Where is ATP synthase (Complex V) is located? (Q891572)

A. Mitochondrial matrix

B. Outer mitochondrial membrane

C. Inner mitochondrial membrane

D. Intermembrane space

Correct Answer: C. Inner mitochondrial membrane

Explanation: ATP synthase (Complex V) is embedded in the inner mitochondrial membrane (IMM) . The enzyme spans the inner membrane , connecting the intermembrane space and the mitochondrial matrix . It consists of two major parts: F■ : The membrane-embedded proton channel F■ : The catalytic head that projects into the matrix and synthesises ATP Protons (H■) flow from the intermembrane space to the matrix through the F■ portion, driven by the proton motive force created by ETC Complexes I, III, and IV . This proton flow causes rotation of the central stalk , leading to conformational changes in F■ that catalyse the formation of ATP from ADP + Pi . Although its main role is ATP synthesis, under certain conditions (e.g., ischemia), ATP synthase can operate in reverse , hydrolysing ATP to help maintain the mitochondrial membrane potential. Mitochondrial matrix (Option A): The F■ subunit projects into the matrix and performs catalysis, but the ATP synthase complex itself is embedded in the inner mitochondrial membrane , not located freely in the matrix. Outer mitochondrial membrane (Option B): The outer membrane contains porins and is relatively permeable; it does not maintain the proton gradient and does not contain ATP synthase . Intermembrane space (Option D): This space serves as the proton reservoir generated by the ETC, but ATP synthase is not located within this space ; it is embedded in the inner mitochondrial membrane .

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 125

↑ *Back to this question in Part A*

Q8: The electron transport chain (ETC) carriers are located on which structure? (Q891571)

A. Inner mitochondrial membrane

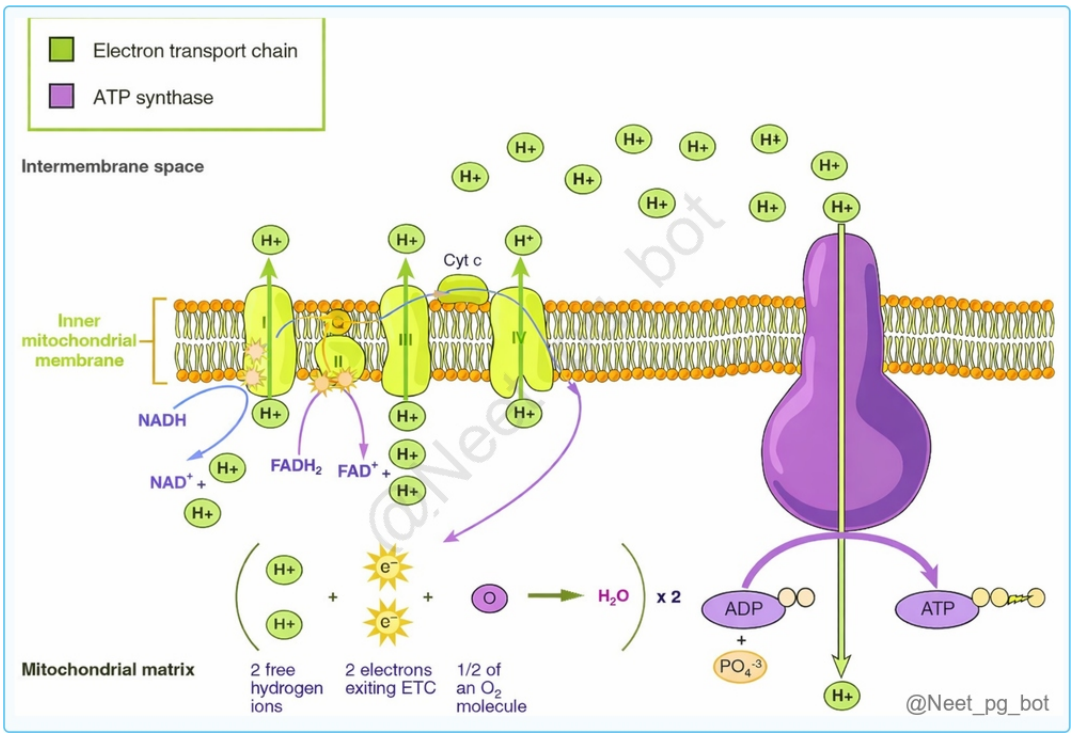
B. Outer mitochondrial membrane

C. Mitochondrial matrix

D. Cytosol

Correct Answer: A. Inner mitochondrial membrane

Explanation: The electron transport chain (ETC) is located in the inner mitochondrial membrane (IMM) . Complexes I, II, III, and IV are embedded in the inner mitochondrial membrane . The mobile electron carriers are: Ubiquinone (Coenzyme Q) , which moves within the membrane Cytochrome c , which is loosely attached to the outer surface of the inner membrane in the intermembrane space The inner membrane folds into cristae , greatly increasing surface area and thereby increasing the capacity for ATP synthesis . The IMM is highly impermeable to ions, which is essential for maintaining the proton gradient . During electron transport, protons (H■) are pumped from the matrix to the intermembrane space by Complexes I, III, and IV, creating the proton motive force . ATP synthase (Complex V) , also located in the inner mitochondrial membrane, uses this gradient to synthesise ATP. The final electron acceptor is oxygen , which is reduced to water . Outer mitochondrial membrane (Option B): The outer membrane contains porins and is freely permeable to small molecules, but it does not contain the ETC complexes or ATP synthase . Mitochondrial matrix (Option C): The matrix is the site of the TCA cycle and β-oxidation and contains NADH and FADH■, but the ETC carriers themselves are membrane-bound , not in the matrix. Cytosol (Option D): The cytosol is the site of glycolysis ; although NADH is produced here, electrons must be transferred into mitochondria via shuttles because the ETC is not located in the cytoplasm .



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 122

↑ *Back to this question in Part A*

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Q9: A 28-year-old woman presents with recurrent episodes of neurologic disturbances and lactic acidosis, particularly after carbohydrate-rich meals. Genetic testing reveals a mutation affecting one of the components of the pyruvate dehydrogenase complex. Which of the following is most likely to be directly affected by this mutation? (Q845067)

A. Flavin adenine dinucleotide (FAD)

B. Dihydrolipoyl transacetylase

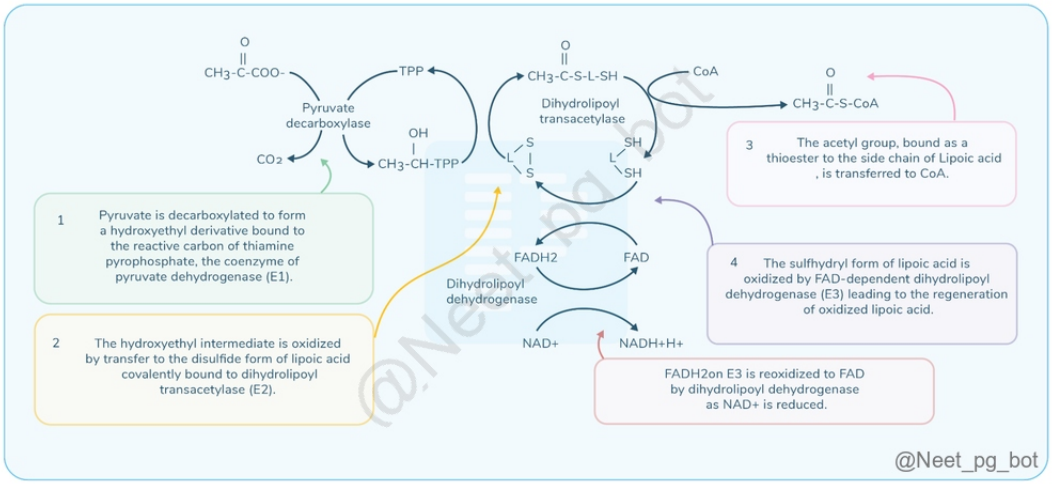
C. Thiamine diphosphate

D. Coenzyme A

Correct Answer: B. Dihydrolipoyl transacetylase

■ *Listen to Audio 1*

Explanation: Pyruvate Dehydrogenase (PDH) Complex Deficiency PDH complex enzymes: Component Enzyme Name Function Cofactor E1 Pyruvate dehydrogenase Decarboxylation of pyruvate TPP (Vit B1) E2 Dihydrolipoyl transacetylase Transfers acetyl group to CoA Lipoic acid, CoA (Vit B5) E3 Dihydrolipoyl dehydrogenase Regenerates oxidized lipoic acid FAD (Vit B2), NAD■ (Vit B3) Mutation in any of these enzymes → PDH deficiency Pathophysiology: Impaired pyruvate → acetyl-CoA conversion ↑ Pyruvate shunted to lactate → lactic acidosis , especially after glucose load Brain energy deficit → neurological impairments Flavin adenine dinucleotide (FAD) (Option A) is a cofactor for E3 (dihydrolipoyl dehydrogenase) It is a coenzyme, not a protein component of the complex. Thiamin diphosphate (Option C) is a cofactor for E1 (pyruvate dehydrogenase). It is a cofactor, not a protein enzyme. Coenzyme A (Option D) accepts the acetyl group from E2 to form acetyl-CoA



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 168

↑ *Back to this question in Part A*

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Q10: Which of the following is a physiological uncoupler? (Q845066)

A. 2,4-dinitrophenol

B. Uncoupling protein 1 [UCP1]

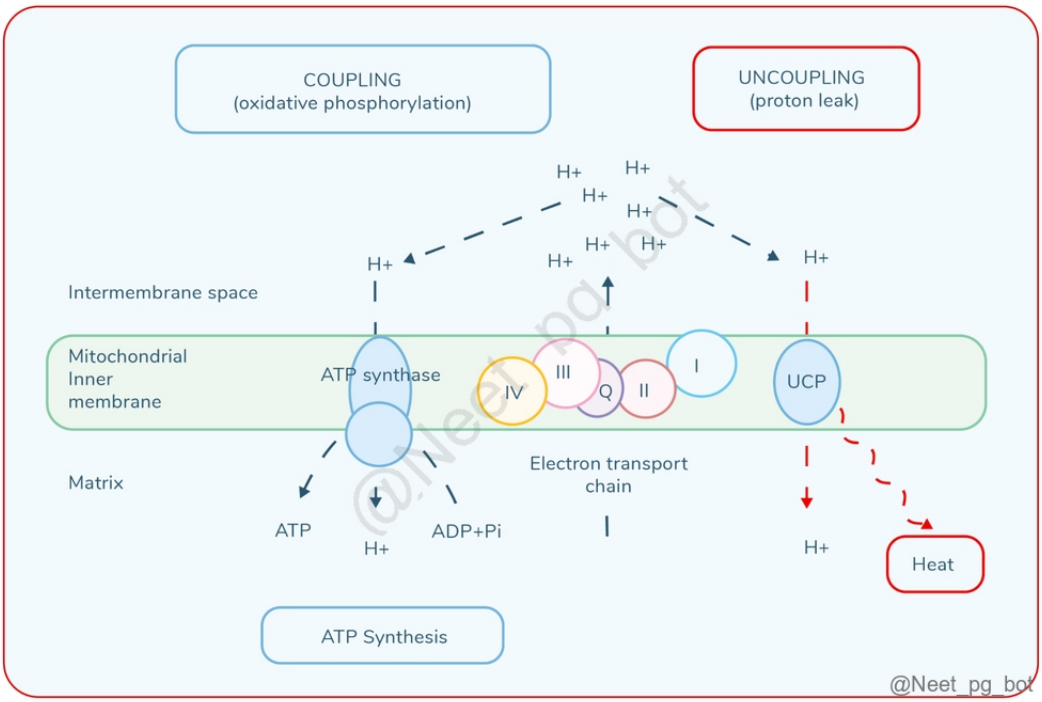
C. Valinomycin

D. Nigericin

Correct Answer: B. Uncoupling protein 1 [UCP1]

■ *Listen to Audio 1*

Explanation: UCP1, also known as thermogenin , is the physiological uncoupler found in brown adipose tissue. Mechanism of Uncoupling: Uncouplers act as proton carriers across the inner mitochondrial membrane. Protons bypass ATP synthase , releasing energy as heat. Energy from proton movement is released as heat rather than being used for ATP synthesis which leads to no ATP production, increased heat production, and increased basal metabolic rate. Physiological uncouplers Artificial uncouplers Present in brown adipose tissue Aid in thermogenesis in newborns Stimulated by hormones such as thyroxine Ex: Thermogenin (UCP1) Thyroxine Free fatty acids (long chain) These were once used as anti-obesity drugs. Ex: 2,4 Dinitrophenol (Option A) Valinomycin (Option C) Nigericin (Option D)



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 127, 128 Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Page 19 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC316328/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: In red blood cells under low energy states, which of the following best describes the fate of pyruvate? (Q845038)

A. Converted to acetyl-CoA to enter the citric acid cycle.

B. Converted to oxaloacetate to replenish the citric acid cycle.

C. Converted to lactate by lactate dehydrogenase to regenerate NAD + .

D. Converted to alanine via transamination.

Correct Answer: C. Converted to lactate by lactate dehydrogenase to regenerate NAD + .

■ *Listen to Audio 1*

Explanation: In RBCs, pyruvate is reduced to lactate by lactate dehydrogenase . RBCs lack mitochondria , so they cannot perform oxidative phosphorylation. Under anaerobic or low-oxygen conditions, pyruvate cannot enter the TCA cycle, so pyruvate is converted to lactate , regenerating NAD■ for continued glycolysis. In low-energy state , the fate of pyruvate depends on the oxygenation of the cell Pyruvate can become lactate, acetyl CoA, alanine and oxaloacetate based on the energy state of the cell. In anaerobic conditions: Pyruvate is converted to lactate by lactate dehydrogenase, using NADH to regenerate NAD + , which is necessary for ATP production. It takes place in cells lacking mitochondria (e.g., RBCs) and in muscle cells during intense exercise. In aerobic conditions: Pyruvate combines with coenzyme A to produce Acetyl CoA, which enters the citric acid pathway (Option A ruled out). It takes place in mitochondria. Converted to oxaloacetate to replenish the citric acid cycle (Option B): Oxaloacetate conversion by pyruvate carboxylase mainly occurs in liver and kidney cells, replenishing the citric acid cycle under in high energy state (not in anaerobic conditions). Converted to alanine via transamination (Option D): Pyruvate can be converted to alanine via transamination, which is seen in the high-energy state of cells.

References:

Lippincott Illustrated Reviews Biochemistry, 7th Edition, Pages 326–328

↑ *Back to this question in Part A*

@Neet_pg_bot

Q12: In the electron transport chain, how many ATP molecules are produced per NADH and FADH 2 , respectively? How many protons (H+) are translocated across the inner mitochondrial membrane for each? (Q845047)

A. 2.5 ATP per NADH and 1.5 ATP per FADH 2 ; with 10 H + and 6 H + translocated, respectively.

B. 3 ATP per NADH and 2 ATP per FADH 2 ; with 12 H + and 8 H + translocated, respectively.

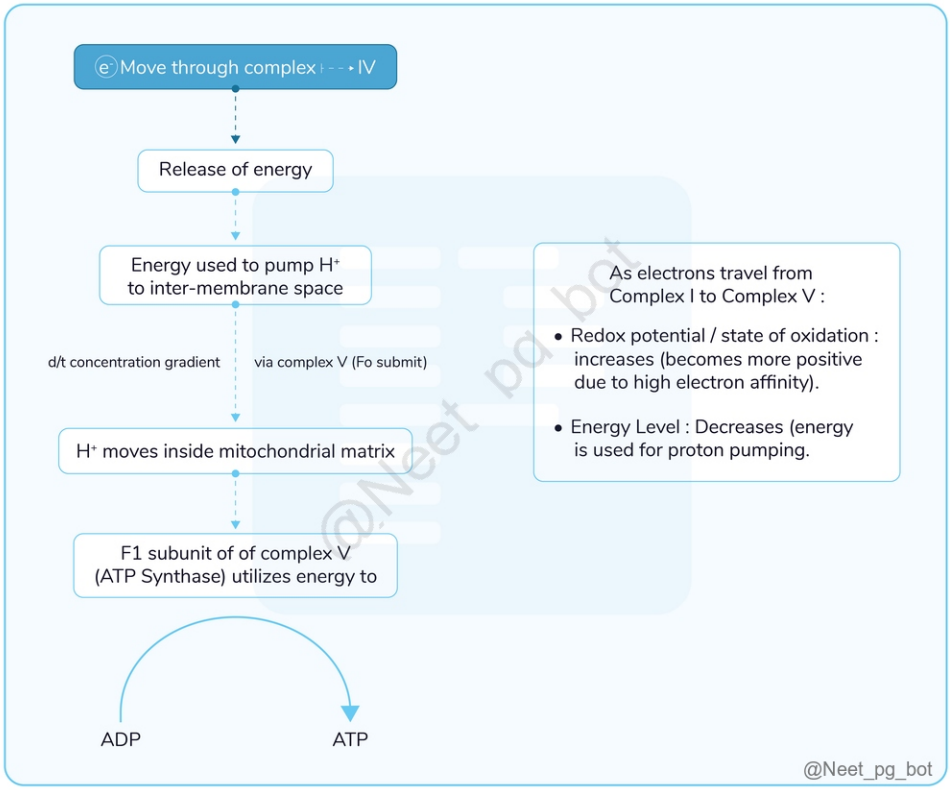
C. 2 ATP per NADH and 1 ATP per FADH 2 ; with 8 H + and 4 H + translocated, respectively.

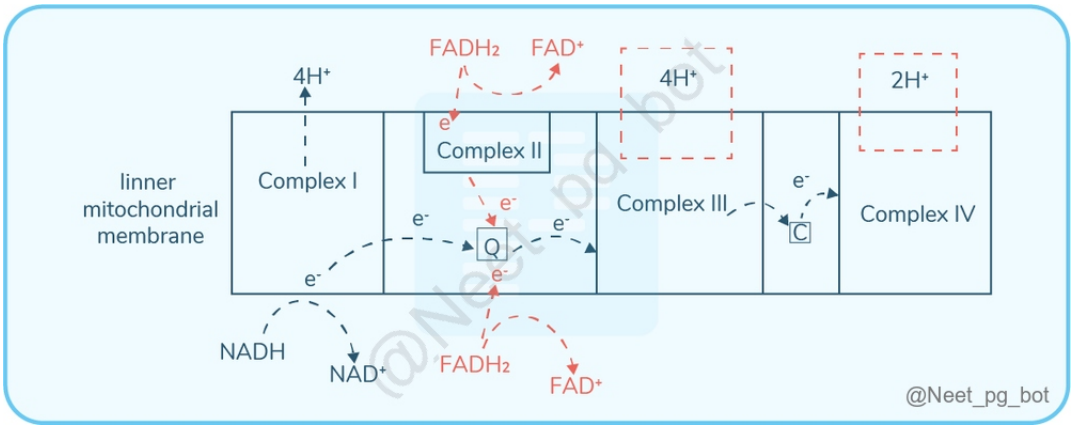
D. 1 ATP per NADH and 0.5 ATP per FADH 2 ; with 6 H + and 3 H + translocated, respectively.

Correct Answer: A. 2.5 ATP per NADH and 1.5 ATP per FADH 2 ; with 10 H + and 6 H + translocated, respectively.

■ *Listen to Audio 1*

Explanation: In oxidative phosphorylation , electrons from NADH and FADH■ enter the electron transport chain (ETC) at different complexes: NADH donates electrons to Complex I , passing through Complexes III and IV , translocating 10 H■ total across the inner mitochondrial membrane. FADH■ donates electrons to Complex II (succinate dehydrogenase) , bypassing Complex I, so only Complexes III and IV pump protons, translocating 6 H■ . Each 4 H■ passing through ATP synthase (Complex V) generates 1 ATP molecule: 10 H■ → 2.5 ATP per NADH 6 H■ → 1.5 ATP per FADH■





References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 126

↑ *Back to this question in Part A*

@Neet_pg_bot

Q13: A patient, with a history of attempting weight loss, presents with fatigue, muscle weakness, and vision problems. Genetic testing confirms MELAS (mitochondrial encephalopathy, lactic acidosis, and stroke). Which electron transport chain complex is most likely affected in this patient (Q845046)

A. Complex I

B. Complex II

C. Complex III

D. Complex V

Correct Answer: A. Complex I

■ *Listen to Audio 1*

Explanation: MELAS (Mitochondrial Encephalopathy, Lactic Acidosis, Stroke-like episodes) Mutations in mitochondrial DNA , primarily affecting Complex I of the ETC Dysfunction in Complex I leads to impaired oxidative phosphorylation , decreased ATP generation , and increased lactic acid levels. Clinical features: Fatigue, muscle weakness, n eurological issues: vision problems, stroke-like episodes Energy deficiency affects high-demand tissues like muscle and brain . Electron Transport Chain Defects in Mitochondrial Disorders : Complex Association with Disease Complex I (NADH dehydrogenase) MELAS, Leber's Hereditary Optic Neuropathy (LHON) Complex II (Succinate dehydrogenase) Rarely affected in mitochondrial diseases Complex III (Cytochrome bc■ complex) Rare disorders (e.g., Mitochondrial Complex III deficiency) Complex IV (Cytochrome c oxidase) Mitochondrial disorders like Leigh syndrome Complex V (ATP synthase) Neuromuscular disorders, Leigh syndrome

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 127, 130

↑ *Back to this question in Part A*

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Q14: Which of the following statements regarding the electron transport chain (ETC) is incorrect? (Q845045)

A. Electrons from NADH enter the ETC through Complex I

B. Complex II does not participate in electron transfer

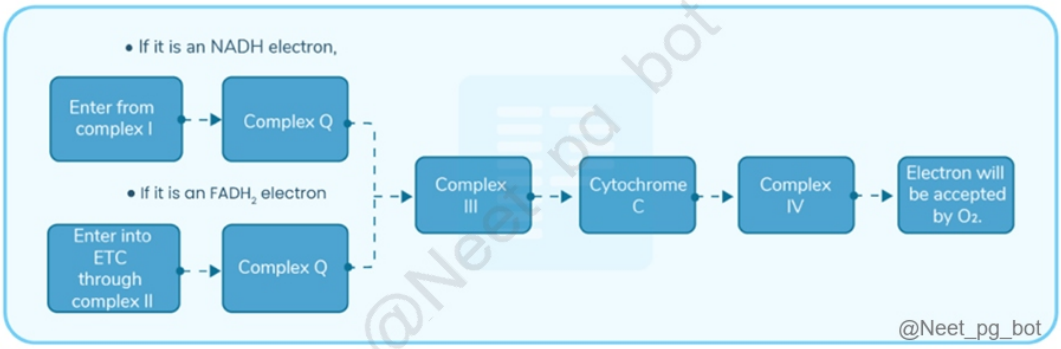
C. Coenzyme Q transfers electrons between Complex I/II and III

D. Cytochrome c transfers electrons between Complex III and IV

Correct Answer: B. Complex II does not participate in electron transfer

■ *Listen to Audio 1*

Explanation: Complex II (succinate dehydrogenase) does participate in electron transfer Transfers electrons from FADH■ → Coenzyme Q. However, it does not pump protons. ■ ■ ■ ■ Complex Name Electron Donor Electron Acceptor Protons Pumped Key Function Important Inhibitors Complex I NADH dehydrogenase NADH Coenzyme Q (Ubiquinone) 4 Entry of electrons from NADH Rotenone, Amytal, Piericidin A Complex II Succinate dehydrogenase FADH■ (from succinate) Coenzyme Q 0 Links TCA cycle to ETC Malonate, TTFA Coenzyme Q (Ubiquinone) Mobile carrier From Complex I & II Complex III — Lipid-soluble electron carrier — Complex III Cytochrome bc■ complex Coenzyme Q (QH■) Cytochrome c 4 Transfers electrons to Cyt c Antimycin A Cytochrome c Mobile protein Complex III Complex IV — Transfers electrons — Complex IV Cytochrome c oxidase Cytochrome c O■ → H■O 2 Final electron transfer Cyanide, CO, Azide, H■S Complex V ATP synthase Proton gradient ATP — ATP production Oligomycin ■ Electrons from NADH enter the ETC through Complex I (Option A) Correct. NADH donates electrons to Complex I (NADH:ubiquinone oxidoreductase) Coenzyme Q transfers electrons between Complex I/II and III (Option C) Correct. Coenzyme Q (ubiquinone) shuttles electrons from Complex I & II → Complex III. Cytochrome c transfers electrons between Complex III and IV (Option A) Correct. Cytochrome c carries electrons from Complex III → Complex IV in the intermembrane space.


• If it is an NADH electron,
Enter from complex I
Complex Q
• If it is an $FADH_2$ electron
Enter into ETC through complex II
Complex Q
Complex III
Cytochrome C
Complex IV
Electron will be accepted by O_2.
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References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 122–123

↑ *Back to this question in Part A*

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Q15: A 25-year-old athlete experiences exercise intolerance and muscle fatigue during high-intensity activity. Genetic testing shows a deficiency in the enzyme that transfers electrons from cytosolic NADH to mitochondrial FADH■ in skeletal muscle.Which mitochondrial shuttle system is most likely impaired? (Q845044)

A. Malate shuttle

B. Glycerol phosphate shuttle

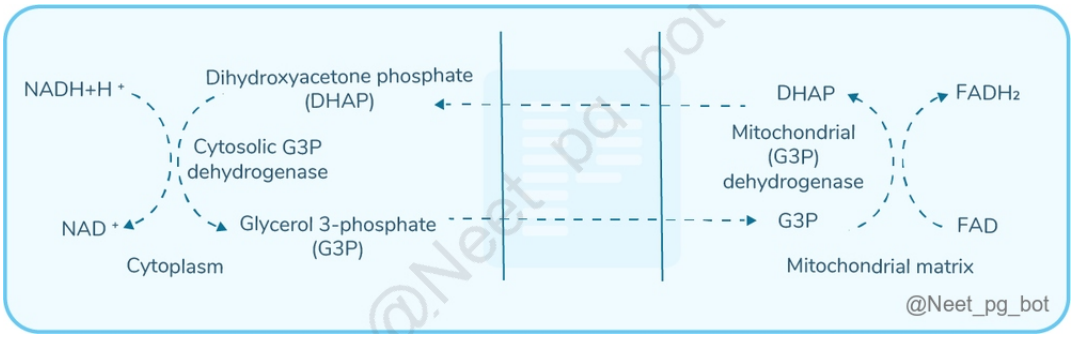
C. Citrate shuttle

D. Aspartate shuttle

Correct Answer: B. Glycerol phosphate shuttle

■ *Listen to Audio 1*

Explanation: Glycerol phosphate shuttle : Found primarily in skeletal muscle and brain . Transfers electrons from cytosolic NADH to mitochondrial FAD , forming FADH■ in the inner mitochondrial membrane. Feature Glycerol-3-Phosphate Shuttle (Option B) Malate-Aspartate Shuttle (Option A and D) Citrate Shuttle (Option C) Main function Transfers electrons from cytosolic NADH → FADH■ Transfers electrons from cytosolic NADH → mitochondrial NADH Transfers acetyl-CoA from mitochondria → cytosol Location Skeletal muscle, brain Liver, heart, kidney Liver, adipose tissue Electron acceptor FAD → FADH■ NAD■ → NADH Not an electron shuttle ATP yield per NADH ~1.5 ATP (via FADH■) ~2.5 ATP (via NADH) Key intermediates DHAP ↔ Glycerol-3-phosphate Malate ↔ Oxaloacetate ↔ Aspartate Citrate ↔ Acetyl-CoA Key enzymes Glycerol-3-P dehydrogenase (cytosolic + mitochondrial) Malate dehydrogenase, AST (transaminase) Citrate lyase Speed vs efficiency Fast but less efficient Slower but more efficient Not for ATP, for lipid synthesis Reversibility Irreversible (functional) Reversible Reversible



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 129, 130

Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Pages 260, 261, 262, 265

↑ *Back to this question in Part A*

@Neet_pg_bot

Q16: Which of the following regulatory effects best explains the anti-neoplastic effect of the ketogenic diet? (Q845043)

A. Acetyl CoA stimulates pyruvate carboxylase.

B. Malonyl CoA inhibits carnitine acyltransferase-1.

C. Citrate inhibits PFK1.

D. Acyl CoA stimulates carnitine acyltransferase-1.

Correct Answer: C. Citrate inhibits PFK1.

■ *Listen to Audio 1*

Explanation: Antineoplastic effect of ketogenic diet's is explained by the Pasteur effect : Tumor cells rely mainly on glycolysis for energy (Warburg effect) even in the presence of oxygen. A ketogenic diet is low in carbohydrates , leading to decreased glycolysis and increased fatty acid oxidation and ketone body production . Citrate , produced from increased fatty acid oxidation, inhibits phosphofructokinase-1 (PFK-1) — the rate-limiting enzyme of glycolysis . This inhibition reduces glycolytic flux , starving tumor cells of energy and growth intermediates. Acetyl-CoA stimulates pyruvate carboxylase (Option A): True, but this occurs in gluconeogenesis (liver), not directly related to the anti-cancer mechanism of the ketogenic diet. Malonyl-CoA inhibits carnitine acyltransferase-1 (Option B): This is related to fatty acid oxidation but is not directly involved in the antineoplastic effect of a ketogenic diet. Acyl CoA stimulates carnitine acyltransferase-1 (Option D): This is related to fatty acid oxidation but is not directly involved in the antineoplastic effect of a ketogenic diet.

References:

www.mdpi.com/1422-0067/24/3/2606

www.ncbi.nlm.nih.gov/pmc/articles/PMC4783224/

www.ncbi.nlm.nih.gov/pmc/articles/PMC3078804/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: A 5-year-old boy with lethargy, vomiting, and ataxia shows lactic acidosis and elevated pyruvate levels. Genetic testing confirms pyruvate dehydrogenase complex deficiency. What is the underlying mechanism for his metabolic derangements? (Q845042)

A. Impaired conversion of pyruvate to acetyl-CoA.

B. Increased conversion of pyruvate to ribose-5-phosphate.

C. Reduced activity of the electron transport chain.

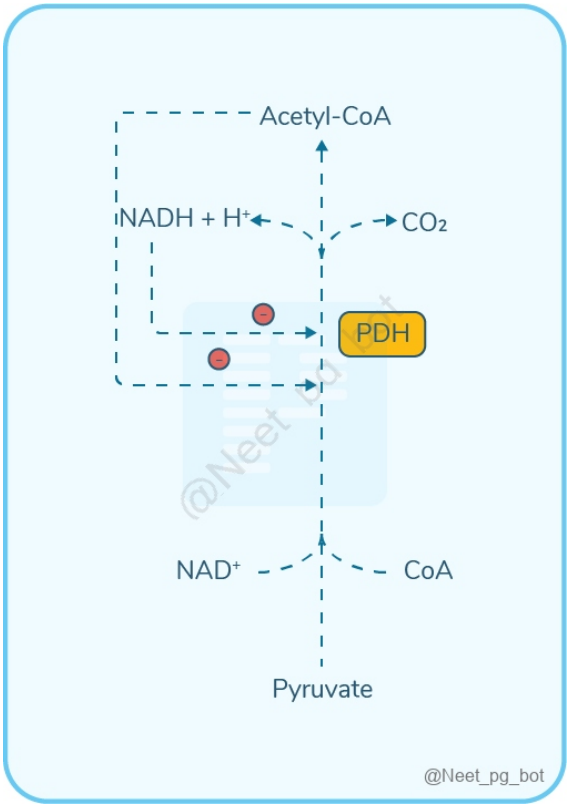
D. Inhibition of gluconeogenesis in the liver.

Correct Answer: A. Impaired conversion of pyruvate to acetyl-CoA.

■ *Listen to Audio 1*

Explanation: Pyruvate dehydrogenase complex is responsible for the irreversible conversion of pyruvate to acetyl-CoA, a crucial step in aerobic glucose metabolism. Deficiency in this complex leads to the accumulation of pyruvate , which is then shunted towards lactate production, resulting in lactic acidosis. Clinical Features: Lethargy, vomiting, ataxia, neurological deficits in children Lab: Elevated lactate and pyruvate , metabolic acidosis Increased conversion of pyruvate to ribose-5-phosphate (Option B): Pyruvate does not convert to ribose-5-phosphate (R-5-P), which is produced through the pentose phosphate pathway, and it does not involve the

PDH complex. Reduced activity of the electron transport chain (Option C) is a potential cause of metabolic acidosis. Inhibition of gluconeogenesis in the liver (Option D) would result in hypoglycemia but would not explain the accumulation of lactate and pyruvate.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 168–170

↑ *Back to this question in Part A*

@Neet_pg_bot

Q18: Which of the following is the primary site for peripheral lipolysis? (Q845041)

A. Liver

B. Skeletal muscle

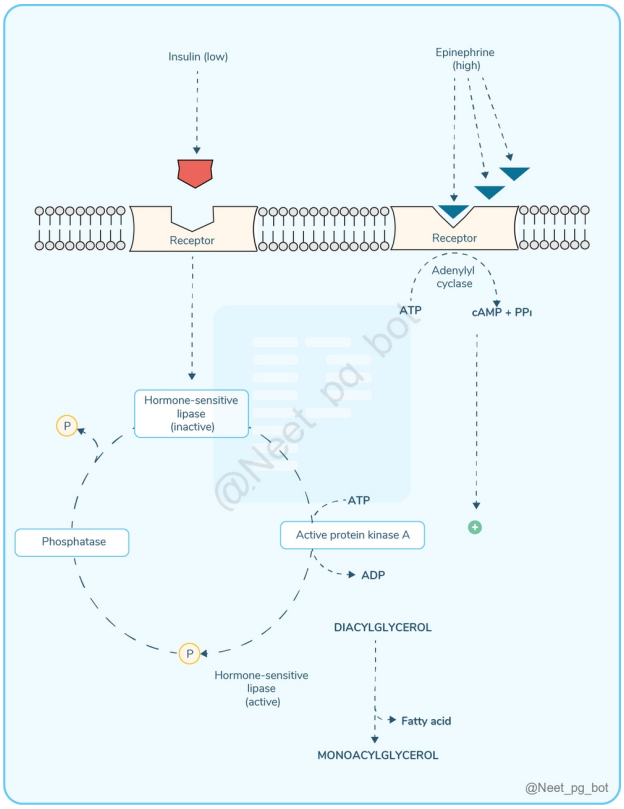
C. Adipose tissue

D. Pancreas

Correct Answer: C. Adipose tissue

■ *Listen to Audio 1*

Explanation: Peripheral Lipolysis Key enzyme: Hormone-sensitive lipase (HSL) Lipase first converts Triacylglycerol → diacylglycerol (preferred HSL substrate) Diacylglycerol is converted to free fatty acids (FFA) + glycerol FFA: bind albumin → transported to muscles → oxidized for ATP Glycerol: transported to liver → used in gluconeogenesis or glycolysis Regulation of HSL: Activation : Catecholamines (epinephrine, norepinephrine), Glucagon, Cortisol → cAMP → phosphorylated with protein kinase A → HSL activated Insulin dephosphorylates HSL and inactivates it. Liver (Option A): Liver processes fatty acids but is not the primary site for peripheral lipolysis. Skeletal muscle (Option B): Muscle utilizes fatty acids but does not primarily break down triglycerides. Pancreas (Option D): Pancreas regulates metabolism via insulin and glucagon but does not carry out lipolysis.



References:

Lippincott Illustrated Reviews Biochemistry, 7th Edition, Pages 553–556

↑ *Back to this question in Part A*

@Neet_pg_bot

Q19: During fasting, after how many hours does gluconeogenesis become the primary source of blood glucose? (Q845040)

A. 8 to 12 hours

B. 12 to 24 hours

C. 16 to 48 hours

D. 48 to 72 hours

Correct Answer: C. 16 to 48 hours

■ *Listen to Audio 1*

Explanation: Gluconeogenesis becomes the primary source of blood glucose after liver glycogen stores are depleted. After 16 hours , gluconeogenesis gradually increases and becomes the dominant source by 24 to 48 hours . Major substrates include lactate, alanine, and glycerol. Duration Dominant Process Source of Glucose Organ-wise Fuel Utilization 0–4 hours (Fed state) Glycolysis Dietary glucose Brain: Glucose Muscle: Glucose Liver: Glucose (glycogenesis) Adipose: Lipogenesis 4–16 hours (Early fasting) Glycogenolysis Liver glycogen Brain: Glucose Muscle: Fatty acids + glycogen Liver: Glycogen breakdown Adipose: Lipolysis begins 16–48 hours (Intermediate fasting) Gluconeogenesis (primary) Lactate, alanine, glycerol Brain: Glucose Muscle: Fatty acids (↓ glucose use) Liver: Gluconeogenesis Adipose: ↑ Lipolysis >48 hours (Prolonged fasting) Gluconeogenesis + Ketogenesis Amino acids, glycerol Brain: Ketone bodies + glucose Muscle: Fatty acids Liver: Ketone production Adipose: High lipolysis >3–5 days (Starvation) Ketosis dominant Minimal glucose (for RBCs) Brain: Mainly ketones Muscle: Fatty acids (protein sparing) Liver: Ketogenesis RBC: Glucose only

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 180–189

Lippincott Illustrated Reviews Biochemistry, 7th Edition, Pages 376–378

↑ *Back to this question in Part A*

@Neet_pg_bot

Q20: Which of the following metabolic disturbances is most likely to be seen in a patient with pyruvate dehydrogenase deficiency? (Q845039)

A. Hypoglycemia due to impaired gluconeogenesis

B. Hyperglycemia due to increased gluconeogenesis

C. Ketoacidosis due to excessive fatty acid oxidation

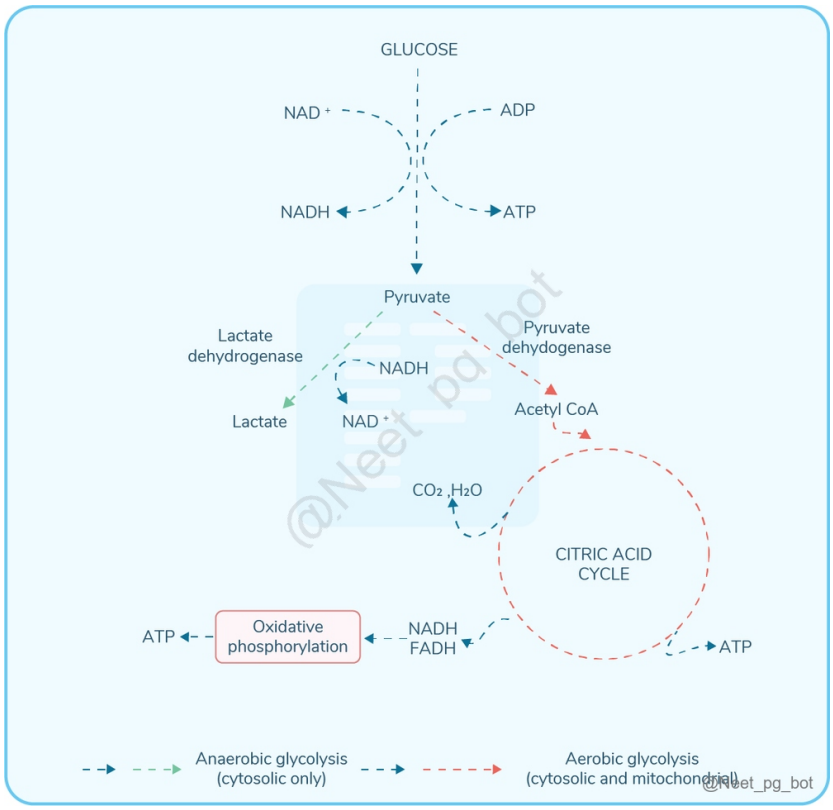
D. Lactic acidosis due to impaired conversion of pyruvate to acetyl-CoA

Correct Answer: D. Lactic acidosis due to impaired conversion of pyruvate to acetyl-CoA

■ *Listen to Audio 1*

Explanation: Pyruvate Dehydrogenase Deficiency Mutation in PDHA1 gene (X-linked) Impaired conversion of pyruvate → acetyl-CoA → pyruvate shunted to lactate → lactic acidosis (especially after glucose load) Glucose

levels are normal (Options A&B ruled out) Clinical features: Nausea, vomiting, neurologic symptoms (ataxia, seizures, hypotonia, intellectual disability, poor coordination) Treatment: Cofactor supplementation (thiamine, lipoic acid) to stimulate PDH activity Ketoacidosis due to excessive fatty acid oxidation (Option C): Ketoacidosis is more commonly associated with conditions like diabetic ketoacidosis or inborn errors of metabolism involving fatty acid oxidation, not PDH deficiency.



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 170

https://www.orpha.net/en/disease/detail/765#

https://medlineplus.gov/genetics/condition/pyruvate-dehydrogenase-deficiency/#frequency

↑ *Back to this question in Part A*

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• [02_Carbohydrate Chemistry] 005 Glut Transporters And Glycolysis

« Back to Folder Directory

[02_Carbohydrate Chemistry] 005 Glut Transporters And Glycolysis — Quick Jump

Q1 (p. 524) *Q2 (p. 524)* *Q3 (p. 525)* *Q4 (p. 526)* *Q5 (p. 527)* *Q6 (p. 527)*
Q7 (p. 528) *Q8 (p. 530)* *Q9 (p. 530)* *Q10 (p. 531)* *Q11 (p. 532)* *Q12 (p. 532)*

Q1: Match the following GLUT transporters with their primary locations: 1. GLUT1 a) Liver 2. GLUT2 b) Neurons 3. GLUT3 c) Erythrocytes 4.GLUT4 d) Skeletal Muscle (Q844950)

A. 1-c, 2-a, 3-b, 4-d

B. 1-d, 2-b, 3-c, 4-a

C. 1-b, 2-d, 3-a, 4-c

D. 1-a, 2-c, 3-d, 4-b

Correct Answer: A. 1-c, 2-a, 3-b, 4-d

■ *Listen to Audio 1*

Explanation: 1. GLUT1 c) Erythrocytes 2. GLUT2 a) Liver 3. GLUT3 b) Neurons 4.GLUT4 d) Skeletal Muscle GLUT 1-5 are facilitative bidirectional transporters SGLT 1 is a sodium-dependent unidirectional transporter.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 186, 187

↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: Which of the following mechanisms is primarily responsible for glucose uptake into enterocytes? (Q844951)

A. Facilitated diffusion via GLUT5 transporter

B. Secondary active transport via SGLT1 transporter

C. Primary active transport via Na+/K+ ATPase

D. Simple diffusion through the lipid bilayer

Correct Answer: B. Secondary active transport via SGLT1 transporter

■ *Listen to Audio 1*

Explanation: Glucose enters intestinal cells (enterocytes) via SGLT1 , a sodium-dependent glucose transporter. SGLT1 carries glucose together with sodium into the cell. Sodium moves down its concentration gradient , maintained by the Na■/K■ pump on the basolateral membrane. As sodium flows in, it pulls glucose into the cell against its concentration gradient . Facilitated diffusion via GLUT5 transporter (Option A): Transports fructose , not glucose. Primary active transport via Na+/K+ ATPase (Option C): Maintains a sodium gradient, does not directly transport glucose . Simple diffusion through the lipid bilayer (Option D): Glucose is polar and cannot passively diffuse across the lipid bilayer.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 186

www.ncbi.nlm.nih.gov/pmc/articles/PMC4728656/#

↑ *Back to this question in Part A*

@Neet_pg_bot

Q3: Which of the following tissues is unable to transport glucose independently of insulin? (Q844952)

A. Hepatocytes

B. Cardiac muscle

C. RBC

D. Neurons

Correct Answer: B. Cardiac muscle

■ *Listen to Audio 1*

Explanation: Cardiac muscle primarily relies on Insulin-dependent GLUT4 transporter for glucose uptake. Rest other tissues use GLUT transporters which are insulin-independent for glucose uptake, as given in the table below

Glut Transporters	Locations
GLUT1	● Neurons, RBCs, and Placenta
GLUT2	● Enterocytes, Hepatocytes, ßcells of pancreas, Proximal convoluted tubule
GLUT3	● Neurons, RBCs, and Placenta
GLUT4	● Insulin Dependent Glucose Uptake in Skeletal Muscles, Cardiac Muscles and Adipose Tissues
GLUT 5	● Fructose Absorption along the intestine.

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References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 187, 188

↑ *Back to this question in Part A*

@Neet_pg_bot

Q4: All of the following are true about glycolysis except: (Q844953)

A. Aerobic glycolysis produces 2 pyruvates and 7 ATPs while anaerobic glycolysis produces 2 lactates and 2 ATPs

B. Retinal and corneal cells can produce energy using glycolysis in hypoxic conditions

C. Tumor cells rely mainly on fatty acid oxidation

D. Fast twitch muscle fibers primarily rely on anaerobic glycolysis for ATP sources

Correct Answer: C. Tumor cells rely mainly on fatty acid oxidation

■ *Listen to Audio 1*

Explanation: Tumor cells rely mainly on fatty acid oxidation – this is false . Tumor cells primarily use anaerobic glycolysis for energy production even in the presence of oxygen (Warburg effect) where glucose is converted to lactate despite sufficient oxygen. Aerobic glycolysis produces 2 pyruvates and 7 ATPs while anaerobic glycolysis produces 2 lactates and 2 ATPs (Option A): This is true statement. Retinal & corneal cells can produce energy via anaerobic glycolysis under hypoxic conditions (Option B): This is true statement. RBC exclusively uses anaerobic glycolysis as the primary source of energy due to the lack of mitochondria, thereby reducing pyruvate to lactate. Fast twitch muscle fibers (white muscle fibers or Type II fibers) primarily rely on anaerobic glycolysis for ATP sources (Option D): This is true statement. Also, Slow twitch muscles (red muscle fibers or Type I fibers) have a high mitochondrial number and primarily rely on aerobic pathways for the generation of ATP.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 163, 164, 167, 704

↑ *Back to this question in Part A*

@Neet_pg_bot

Q5: Which of the following statements is false? (Q844954)

A. RBCs solely depend on glycolysis for ATP

B. White muscle fibers depend on anaerobic glycolysis during muscular contraction

C. Myoglobin is the storage form of oxygen in white muscle fibers

D. Slow-twitch muscle fibers contain high amounts of myoglobin

Correct Answer: C. Myoglobin is the storage form of oxygen in white muscle fibers

■ *Listen to Audio 1*

Explanation: Myoglobin is the storage form of oxygen in white muscle fibers -this is false statement Myoglobin is a protein that stores oxygen in red muscle fibers (slow-twitch fibers). RBCs solely depend on glycolysis for ATP (Option A): True, Mature erythrocytes do not contain mitochondria. Without mitochondria, erythrocytes cannot utilize fatty acids or ketone bodies as metabolic fuel. Therefore, glycolysis is the only pathway available for erythrocytes to generate ATP . White muscle fibers depend on anaerobic glycolysis during muscular contraction (Option B):True, White muscle fibers have few mitochondria and little myoglobin, relying on anaerobic glycolysis and creatine phosphate for rapid energy during contraction. Slow-twitch muscle fibers contain high amounts of myoglobin (Option D):True, Slow-twitch fibers are rich in myoglobin, supporting aerobic metabolism.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 628, 655

https://www.sciencedirect.com/topics/medicine-and-dentistry/kidney-ischemia#

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: Which of the following statements regarding aerobic glycolysis is true? (Q844955)

A. Each acetyl CoA molecule yields 5 ATPs by converting to pyruvate

B. Each molecule of NADPH yields 2.5 ATP

C. Aerobic glycolysis of one molecule of glucose yields a net total of 7 ATP molecules.

D. Each turn of the citric acid cycle produces 20 ATP molecules.

Correct Answer: C. Aerobic glycolysis of one molecule of glucose yields a net total of 7 ATP molecules.

■ *Listen to Audio 1*

Explanation: In aerobic glycolysis: Total ATP generated: 5 (from 2NADH oxidation) + 2 (from 1,3-BPG) + 2 (from PEP) = 9 ATP. Net ATP yield: 9 (total ATP generated) - 2 (consumed in initial steps) = 7 ATP In total, aerobic glycolysis of one molecule of glucose yields a net total of 7 ATP molecules. Each acetyl CoA molecule yields 5 ATPs by converting to pyruvate(Option A) : Incorrect because acetyl CoA does not convert to pyruvate . Pyruvate is converted into acetyl CoA before entering the citric acid cycle. Each acetyl CoA generates ~10 ATP Each molecule of NADPH yields 2.5 ATP (Option B): Incorrect because NADPH is not used in ATP production . Only NADH from glycolysis and the citric acid cycle contributes to ATP via oxidative phosphorylation. Each turn of the citric acid cycle produces 20 ATP molecule (Option B) : Incorrect. Each acetyl CoA oxidation in the TCA cycle produces ~10 ATP , not 20.

Glycolysis in Anaerobic Cells (RBCs)	Glycolysis in Aerobic Cells Neurons
Anaerobic glycolysis.	Aerobic glycolysis.
2 Lactates are formed.	2 Pyruvates are formed.
2 ATPs are generated.	32 ATPs are generated on the complete oxidation of glucose in neurons. ● 7 ATPs from Glycolysis. ● 2 NADH × 2.5 ATP = 5 ATPs on conversion of 2 Pyruvates ⟶ 2 Acetyl CoA. With the help of PDH complex. ● Acetyl CoA enters the TCA cycle to produce 10 ATPs per molecule. ● 2 Acetyl CoA 10 × ATPs=20ATPs.

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References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 126, 168

↑ *Back to this question in Part A*

@Neet_pg_bot

Q7: Which of the following enzymes catalyzes the conversion of glucose to glucose-6-phosphate in the glycolysis pathway? (Q844956)

A. Hexokinase

B. Phosphofructokinase

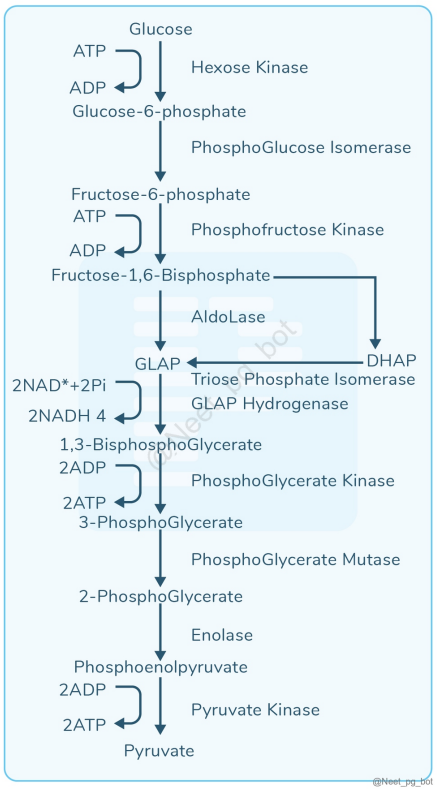
C. Aldolase

D. Enolase

Correct Answer: A. Hexokinase

■ *Listen to Audio 1*

Explanation: Hexokinase catalyzes the first step of glycolysis , converting glucose → glucose-6-phosphate using one ATP. This phosphorylation traps glucose inside the cell and prepares it for further metabolism. Rate-Limiting Enzyme of Glycolysis: Phosphofructokinase-1 (PFK-1) Converts ■■■■■■■ Fructose-6-P to Fructose-1,6-bisphosphate ■■■■■■■ Steps of Glycolysis: Phosphofructokinase (Option B): This enzyme catalyzes the third step of glycolysis, converting fructose-6-phosphate to fructose-1,6-bisphosphate. Aldolase (Option C): This enzyme catalyzes the fourth step of glycolysis, splitting fructose-1,6-bisphosphate into two three-carbon sugars: glyceraldehyde-3-phosphate and dihydroxyacetone phosphate. ■■■■■■■Enolase (Option D): This enzyme catalyzes the ninth step of glycolysis, converting 2-phosphoglycerate to phosphoenolpyruvate.



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 165

↑ *Back to this question in Part A*

@Neet_pg_bot

Q8: Which of the following steps in glycolysis is irreversible? (Q844957)

A. Conversion of glucose-6-phosphate to fructose-6-phosphate

B. Conversion of 3-phosphoglycerate to 2-phosphoglycerate

C. Conversion of phosphoenolpyruvate to pyruvate

D. Conversion of fructose-1,6-bisphosphate to glyceraldehyde-3-phosphate and dihydroxyacetone phosphate

Correct Answer: C. Conversion of phosphoenolpyruvate to pyruvate

■ *Listen to Audio 1*

Explanation: Three Major Irreversible Steps in Glycolysis: Step Enzyme Reaction Importance 1 Hexokinase / Glucokinase Glucose → Glucose-6-phosphate Consumes 1 ATP, traps glucose inside the cell 2 Phosphofructokinase-1 (PFK-1) Fructose-6-phosphate → Fructose-1,6-bisphosphate Rate-limiting step, consumes 1 ATP 3 Pyruvate kinase Phosphoenolpyruvate → Pyruvate Produces 1 ATP per molecule of PEP Replacement Steps in Gluconeogenesis Gluconeogenesis Bypass Enzymes Involved Glucose → Glucose-6-phosphate Glucose-6-phosphatase Fructose-6-phosphate → Fructose-1,6-bisphosphate Fructose-1,6-bisphosphatase Phosphoenolpyruvate → Pyruvate Pyruvate carboxylase + PEP carboxykinase (PEPCK) Option A, B, D are all reversible reactions in glycolysis.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 167

↑ *Back to this question in Part A*

@Neet_pg_bot

Q9: Which of the following statements accurately describes the difference between hexokinase and glucokinase? (Q844958)

A. Hexokinase has a higher Km and Vmax compared to glucokinase.

B. Glucokinase is inhibited by glucose-6-phosphate, whereas hexokinase is not.

C. Hexokinase is primarily found in the liver and pancreatic β-cells, while glucokinase is found in most tissues.

D. Glucokinase has a higher Km and Vmax compared to hexokinase.

Correct Answer: D. Glucokinase has a higher Km and Vmax compared to hexokinase.

■ *Listen to Audio 1*

Explanation: Glucokinase has a lower affinity (higher Km) for glucose but a higher capacity (higher Vmax) compared to hexokinase. Feature Hexokinase Glucokinase Tissue Distribution (Option C) Most tissues Liver and pancreatic β-cells Km (Affinity for Glucose) (Option A) Low Km (high affinity for glucose) High Km (low affinity for glucose) Vmax (Maximum Rate) Low Vmax High Vmax Regulation (Option B) Inhibited by glucose-6-phosphate Not inhibited by glucose-6-phosphate Role Maintains basal glucose utilization Regulates blood glucose levels Function at Low Glucose Active even at low glucose concentrations Only active when glucose levels are high Gene Regulation Constitutive expression Inducible by insulin Substrate Specificity Broad (can phosphorylate other hexoses) Specific to glucose

References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 164, 165

↑ *Back to this question in Part A*

@Neet_pg_bot

Q10: Which of the following chemical compounds inhibits glycolysis by inhibiting the enzyme enolase? (Q844959)

A. Iodoacetate

B. Fluoride

C. Arsenate

D. 2-Deoxyglucose

Correct Answer: B. Fluoride

■ *Listen to Audio 1*

Explanation: Fluoride inhibits the enzyme enolase in the glycolytic pathway, preventing the conversion of 2-phosphoglycerate to phosphoenolpyruvate. By blocking this step, fluoride effectively halts glycolysis, leading to an accumulation of glycolytic intermediates upstream of the enolase reaction. Chemical Inhibitors of Glycolysis: Chemical Inhibitor Target Enzyme Mechanism of Inhibition Fluoride Enolase Inhibits the conversion of 2-phosphoglycerate to phosphoenolpyruvate. Iodoacetate (Option A) Glyceraldehyde-3-phosphate dehydrogenase Inhibits the conversion of glyceraldehyde-3-phosphate to 1,3-bisphosphoglycerate by covalently modifying the enzyme. Arsenate (Option C) Glyceraldehyde-3-phosphate dehydrogenase Replaces phosphate, forming unstable 1-arseno-3-phosphoglycerate, bypassing ATP generation. 2-Deoxyglucose (Option D) Hexokinase Phosphorylated to 2-deoxyglucose-6-phosphate, which accumulates and inhibits hexokinase, halting glycolysis.

References:

Harper’s Illustrated Biochemistry 32nd Edition, Page 166, 167

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: What is the primary function of the Rapoport-Luebering Shunt in red blood cells? (Q844960)

A. To generate ATP from 2,3-bisphosphoglycerate

B. To produce 2,3-bisphosphoglycerate, which regulates oxygen binding to hemoglobin

C. To convert pyruvate to lactate

D. To facilitate the conversion of glucose to glucose-6-phosphate

Correct Answer: B. To produce 2,3-bisphosphoglycerate, which regulates oxygen binding to hemoglobin

■ *Listen to Audio 1*

Explanation: Rapoport–Luebering Shunt: In glycolysis, glyceraldehyde-3-phosphate is converted to 1,3-bisphosphoglycerate (1,3-BPG) by glyceraldehyde-3-phosphate dehydrogenase (using NAD■ → NADH). In this shunt, 1,3-BPG → 2,3-BPG via bisphosphoglycerate mutase , shifting the phosphate from C1 to C2. Then, 2,3-BPG → 3-phosphoglycerate by 2,3-bisphosphoglycerate phosphatase , allowing re-entry into glycolysis. To generate ATP from 2,3-bisphosphoglycerate (Option A): The Rapoport–Luebering Shunt actually bypasses ATP generation . Normally, 1,3-BPG → 3-phosphoglycerate produces ATP, but in this shunt, the step is skipped to form 2,3-BPG, sacrificing ATP for oxygen regulation. To convert pyruvate to lactate (Option C): This reaction is catalyzed by lactate dehydrogenase , not part of the Rapoport–Luebering pathway. To facilitate the conversion of glucose to glucose-6-phosphate (Option D): This step is catalyzed by hexokinase , the first enzyme of glycolysis.

References:

https://pubchem.ncbi.nlm.nih.gov/pathway/BioCyc:HUMAN_PWY-6405

↑ *Back to this question in Part A*

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Q12: Which of the following is a key positive regulator of the glycolytic enzyme phosphofructokinase-1 (PFK-1)? (Q844961)

A. ATP

B. Citrate

C. Fructose-2,6-bisphosphate

D. Acetyl-CoA

Correct Answer: C. Fructose-2,6-bisphosphate

■ *Listen to Audio 1*

Explanation: Fructose-2,6-bisphosphate is a potent activator of PFK-1 . It enhances the enzyme's affinity for its substrate (fructose-6-phosphate) and diminishes the inhibitory effects of ATP, thus promoting glycolysis. Allosteric Inhibitors of PFK-1: ATP – signals high energy, inhibits PFK-1 (Option A) Citrate – indicates abundant biosynthetic precursors, inhibits PFK-1 (Option B) H■ (low pH) – prevents excessive acidification Other Regulators: AMP – activates PFK-1 when energy is low Acetyl-CoA – indirectly reduces glycolysis by signaling a high-energy state Hormonal Control: Insulin – promotes glycolysis by increasing F2,6BP and PFK-1 activity Glucagon – inhibits glycolysis by lowering F2,6BP, reducing PFK-1 activity ■■■■■■■Acetyl-CoA (Option D): While Acetyl-CoA is not a direct regulator of PFK-1, it generally indicates a high-energy state and leads to a decrease in glycolysis through the activation of pyruvate carboxylase and the inhibition of pyruvate dehydrogenase.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 169

↑ *Back to this question in Part A*

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• [02_Carbohydrate Chemistry] 006 Glycogen Metabolism And Glycogen Storage Disorders

« Back to Folder Directory

[02_Carbohydrate Chemistry] 006 Glycogen Metabolism And Glycogen Storage Disorders — Quick Jump

Q1 (p. 534)	*Q2 (p. 535)*	*Q3 (p. 535)*	*Q4 (p. 536)*	*Q5 (p. 537)*	*Q6 (p. 538)*
Q7 (p. 539)	*Q8 (p. 540)*	*Q9 (p. 541)*	*Q10 (p. 541)*	*Q11 (p. 542)*	*Q12 (p. 543)*
Q13 (p. 544)	*Q14 (p. 544)*	*Q15 (p. 545)*	*Q16 (p. 546)*	*Q17 (p. 547)*	*Q18 (p. 548)*
Q19 (p. 548)	*Q20 (p. 549)*	*Q21 (p. 550)*	*Q22 (p. 551)*	*Q23 (p. 552)*	*Q24 (p. 553)*

Q1: A 5-year-old child presents with muscle weakness, mild hepatomegaly, and cardiomegaly. Labs show mild hypoglycemia and elevated creatine kinase. Which enzyme deficiency is most likely responsible? (Q844869)

A. Glycogen synthase

B. Glucose-6-phosphatase

C. Debranching enzyme

D. Muscle phosphorylase

Correct Answer: C. Debranching enzyme

■ *Listen to Audio 1*

Explanation: The patient’s clinical signs — hypoglycemia, hepatomegaly, and muscle weakness — suggest Cori Disease , caused by a deficiency of the debranching enzyme . Cori Disease (GSD Type III) Enzyme Deficiency: Liver and muscle debranching enzyme (α-1,6-glucosidase) Gene: AGL , Autosomal Recessive Inheritance Clinical Features: Hypoglycemia, hepatomegaly, limit dextrin accumulation , muscle weakness Management: High-protein, low-carb diet; enzyme replacement therapy in select cases Glycogen synthase (Option A): This enzyme is involved in glycogen synthesis, and its deficiency causes a Type 0 GSD and it does not present with hepatomegaly or cardiomegaly Glucose-6-phosphatase (Option B): This enzyme deficiency is associated with von Gierke disease does not present with muscle weakness Muscle phosphorylase (Option D): Deficiency in this enzyme causes McArdle disease and it does not present with hepatomegaly or hypoglycemia.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 174

Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Page 129

www.ncbi.nlm.nih.gov/gtr/conditions/C0017922/

↑ *Back to this question in Part A*

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Q2: Glycogen phosphorylase requires which vitamin-derived coenzyme? (Q891558)

A. Thiamine pyrophosphate

B. Pyridoxal phosphate (Vitamin B6)

C. Biotin

D. Coenzyme A

Correct Answer: B. Pyridoxal phosphate (Vitamin B6)

Explanation: Glycogen phosphorylase requires pyridoxal phosphate (PLP) , the active form of Vitamin B6 , as an essential coenzyme. Enzymatic role: PLP is tightly bound to glycogen phosphorylase, the rate-limiting enzyme of glycogenolysis , which cleaves α(1→4) bonds in glycogen to release glucose-1-phosphate . Mechanism: Unlike its role in transamination reactions, in glycogen phosphorylase the 5′-phosphate group of PLP acts as a general acid-base catalyst , facilitating the attack of inorganic phosphate on the glycosidic bond. The aldehyde group of PLP forms a Schiff base with a lysine residue of the enzyme to anchor the coenzyme, rather than participating directly in substrate chemistry. About 70-80% of the body’s Vitamin B6 is stored in skeletal muscle , mainly bound to glycogen phosphorylase , making this enzyme a major PLP reservoir. Clinical relevance: Vitamin B6 deficiency can impair glycogen breakdown, leading to reduced energy availability during fasting or exercise. Thiamine pyrophosphate (Option A): A Vitamin B1-derived cofactor involved in oxidative decarboxylation reactions (e.g., pyruvate dehydrogenase) and transketolase in the HMP shunt Biotin (Option C): A Vitamin B7-derived cofactor for carboxylation reactions (e.g., pyruvate carboxylase, acetyl-CoA carboxylase); it transfers CO■ , not involved in phosphorolysis of glycogen. Coenzyme A (Option D): Derived from Vitamin B5, functions as an acyl group carrier in fatty acid and TCA cycle metabolism

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 536

↑ *Back to this question in Part A*

@Neet_pg_bot

Q3: Muscle cannot release glucose into the bloodstream because it lacks which enzyme? (Q891554)

A. Glycogen phosphorylase

B. Hexokinase

C. Glucose-6-phosphatase

D. Phosphoglucomutase

Correct Answer: C. Glucose-6-phosphatase

Explanation: Glucose-6-phosphatase (G6Pase) catalyzes the final step of both glycogenolysis and gluconeogenesis, converting glucose-6-phosphate (G6P) → free glucose + Pi. This reaction occurs in the lumen of the smooth endoplasmic reticulum and is essential for releasing glucose into the bloodstream. Tissue distribution: G6Pase is present in liver and kidney (and to a lesser extent intestinal mucosa), but absent in muscle . Physiological consequence: Because muscle lacks G6Pase , the G6P formed from glycogen breakdown is trapped inside the cell and is used locally for glycolysis and ATP production . Muscle glycogen therefore cannot contribute to blood glucose . Metabolic logic: This ensures that muscle glycogen is reserved for muscle contraction , while the liver maintains systemic glucose homeostasis (via G6Pase and export through GLUT2). Clinical correlation: Deficiency of G6Pase causes Von Gierke disease (GSD type I) , characterized by severe fasting hypoglycemia, hepatomegaly, and lactic acidosis . Indirect support of blood glucose: Muscle can export lactate (Cori cycle) or alanine , which the liver converts back to glucose. Glycogen phosphorylase (Option A): This enzyme is present in muscle and catalyzes the breakdown of glycogen to glucose-1-phosphate. Its deficiency causes McArdle disease . Hexokinase (Option B): This enzyme is present in muscle and phosphorylates glucose to glucose-6-phosphate to trap it inside the cell for glycolysis. Phosphoglucomutase (Option D): This enzyme is present in muscle and catalyzes the reversible conversion of glucose-1-phosphate to glucose-6-phosphate, allowing entry into glycolysis.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 171

↑ *Back to this question in Part A*

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Q4: The correct sequence of enzymes involved in glycogenolysis (glycogen breakdown) is: (Q891549)

A. Phosphorylase → Glucose-6-phosphatase → Phosphoglucomutase → Glucan transferase

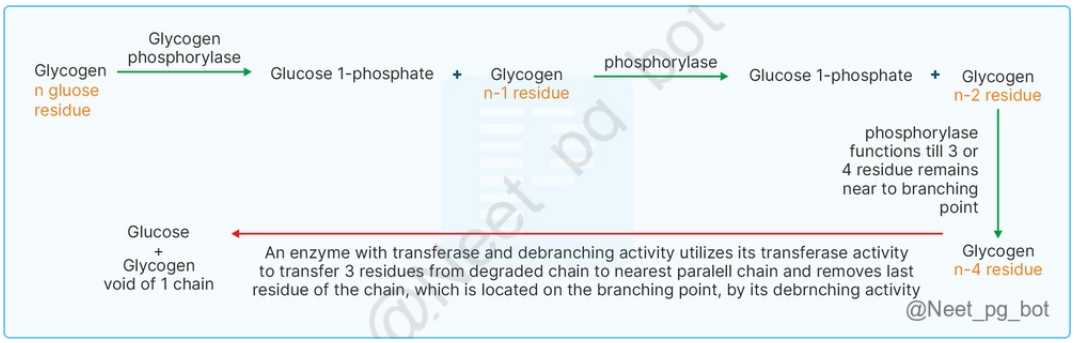
B. Glucan transferase → Phosphorylase → Amylo-1,6-glucosidase → Phosphoglucomutase

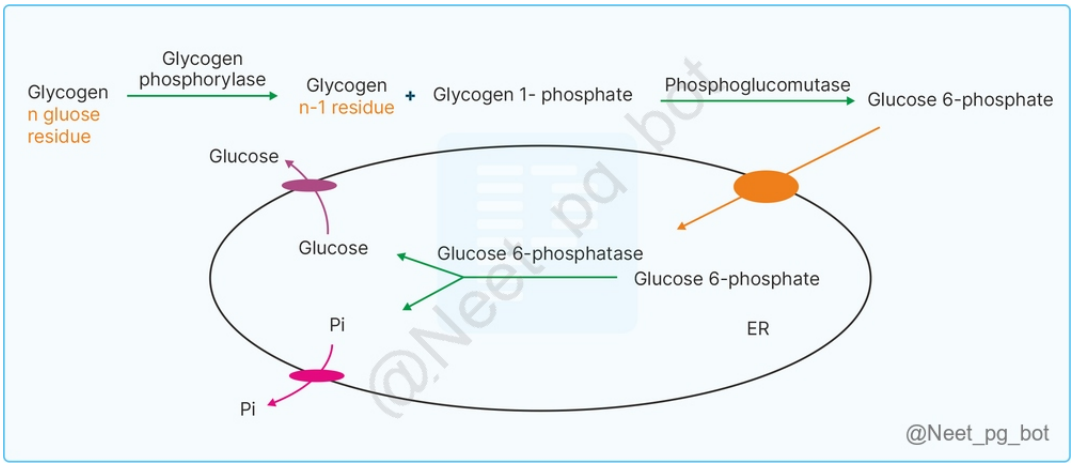
C. Phosphorylase → Phosphoglucomutase → Glucan transferase → Glucose-6-phosphatase

D. Phosphorylase → Debranching enzyme → Phosphoglucomutase → Glucose-6-phosphatase

Correct Answer: D. Phosphorylase → Debranching enzyme → Phosphoglucomutase → Glucose-6-phosphatase

Explanation: Glycogenolysis proceeds in a fixed biochemical sequence : Glycogen phosphorylase Cleaves α(1→4) bonds at non-reducing ends Releases glucose-1-phosphate (G1P) Stops 4 residues before a branch point Debranching enzyme (has two activities): Glucan transferase → transfers 3 residues to another chain Amylo-1,6-glucosidase → removes the α(1→6) branch glucose (as free glucose) Phosphoglucomutase Converts G1P → G6P Glucose-6-phosphatase (liver & kidney only) Converts G6P → free glucose for release into blood





References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 173 - 174

↑ *Back to this question in Part A*

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Q5: The activated form of glucose used for addition to the growing glycogen chain is: (Q891547)

A. Glucose-1-phosphate

B. Glucose-6-phosphate

C. UDP-glucose

D. ADP-glucose

Correct Answer: C. UDP-glucose

Explanation: UDP-glucose (uridine diphosphate glucose) is the activated nucleotide sugar donor used for glycogen synthesis. It is formed from glucose-1-phosphate (G1P) and UTP by the enzyme UDP-glucose pyrophosphorylase . Activation Mechanism: UDP-glucose is synthesized from glucose-1-phosphate and UTP in a reaction catalyzed by UDP-glucose pyrophosphorylase . The reaction is driven forward by the hydrolysis of inorganic pyrophosphate (PPi) , making the overall process energetically favourable in vivo. Enzymatic Substrate: UDP-glucose serves as the direct glucose donor for glycogen synthase , the rate-limiting enzyme of glycogen synthesis. Glycosidic Bond Formation: The glucose moiety of UDP-glucose is transferred to the non-reducing end of a growing glycogen chain, forming an α(1→4) glycosidic bond and releasing UDP . Formation of the sugar nucleotide (UDP-glucose) creates an activated glucose intermediate , allowing glycosyl transfer reactions to proceed efficiently; the true thermodynamic driving force is the PPi hydrolysis coupled to its synthesis. Beyond glycogen synthesis, UDP-glucose is an important precursor for the formation of other carbohydrates, including UDP-galactose , and participates in the synthesis of glycoproteins and glycolipids . Glucose-1-phosphate (Option A): G1P is an intermediate in the pathway but cannot directly donate glucose to glycogen. It must first be converted into UDP-glucose by UDP-glucose pyrophosphorylase before it can be used for chain elongation. Glucose-6-phosphate (Option B): G6P is a central metabolic intermediate and the precursor that is converted to G1P by phosphoglucomutase during glycogenesis, but it is not the activated donor for glycogen synthesis. ADP-glucose (Option D): ADP-glucose is the activated glucose donor used for starch synthesis in plants .

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 173

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: Which protein serves as the primer for glycogen synthesis, accepting the first glucose residues? (Q891544)

A. Glycogen synthase

B. Glycogenin

C. Branching enzyme

D. UDP-glucose pyrophosphorylase

Correct Answer: B. Glycogenin

Explanation: Glycogenin is the protein primer that autoglucosylates itself , adding approximately 7-8 glucose residues via α(1→4) linkages . This short oligosaccharide chain then serves as the substrate for glycogen synthase , the rate-limiting enzyme of glycogenesis. Primer Function: Glycogenin serves as the obligatory primer for glycogen granule formation. Without glycogenin, the cell cannot initiate the assembly of new glycogen molecules

de novo. Autocatalytic Activity: Glycogenin possesses intrinsic glucosyltransferase activity , meaning it catalyzes its own modification by attaching glucose molecules to itself. Initial Attachment: The process begins with the attachment of a glucose residue from UDP-glucose to a specific tyrosine residue on the glycogenin protein, forming the first covalent glucose-protein linkage. Chain Elongation: Glycogenin continues to add approximately 7-8 (up to ~10) glucose units to this initial residue, forming a short, linear α(1→4)-linked primer chain . Transition to Synthase: Once the primer reaches a sufficient length, glycogen synthase takes over and further elongates the chain, after which the branching enzyme introduces α(1→6) branches to build the mature glycogen structure. Core Presence: Each mature glycogen granule contains a single molecule of glycogenin at its core , which remains covalently attached to the polymer it initiated. Glycogen synthase (Option A): This is the key regulatory and rate-limiting enzyme of glycogenesis, but it cannot initiate a new glycogen chain de novo. It can only add glucose units to an existing primer chain already attached to glycogenin. Branching enzyme (Option C): Also known as amylo-(1,4→1,6)-transglycosylase , this enzyme creates α(1→6) glycosidic bonds , increasing the solubility of glycogen and the number of terminal residues. It acts only after a linear chain has already been formed. UDP-glucose pyrophosphorylase (Option D): This enzyme catalyses the formation of UDP-glucose from glucose-1-phosphate and UTP, providing the activated glucose donor for glycogen synthesis.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 173

↑ *Back to this question in Part A*

@Neet_pg_bot

Q7: Glycogen synthesis occurs in which cellular compartment? (Q891541)

A. Mitochondria

B. Nucleus

C. Cytosol

D. Endoplasmic Reticulum

Correct Answer: C. Cytosol

Explanation: Glycogen synthesis and degradation occur in the cytosol . The entire enzymatic machinery required for glycogen synthesis is located in the aqueous component of the cytoplasm and is associated with glycogen granules. This separates glycogen metabolism from mitochondrial oxidative reactions. Glucose is first “trapped” inside the cell as glucose-6-phosphate (G6P) , which is converted to glucose-1-phosphate (G1P) and then to UDP-glucose . UDP-glucose is the immediate activated donor of glucose units for glycogen synthesis. Glycogen synthase is the rate-limiting enzyme of glycogenesis. It catalyses the formation of α(1→4) glycosidic bonds to elongate the glycogen chain. Glycogen synthesis cannot start de novo; it requires glycogenin , a primer protein that acts as a scaffold for the addition of the first few glucose residues. Mitochondria (Option A): The mitochondria are the site of the TCA (Krebs) cycle and oxidative phosphorylation. Although ATP is produced here, the enzymes required for glycogen synthesis are not located in the mitochondrial matrix. Nucleus (Option B): The nucleus

houses genetic material and regulates processes such as transcription and replication. Endoplasmic Reticulum (Option D): The ER is involved in protein synthesis (rough ER) and lipid synthesis and calcium storage (smooth ER).

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 175

↑ *Back to this question in Part A*

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Q8: Glycogenesis (glycogen synthesis) occurs predominantly in which two tissues? (Q891539)

A. Liver and kidney

B. Skeletal muscle and liver

C. Brain and adipose tissue

D. Heart and pancreas

Correct Answer: B. Skeletal muscle and liver

Explanation: Glycogenesis primarily occurs in skeletal muscle (for local energy needs) and the liver (for maintenance of blood glucose homeostasis). Muscle glycogen cannot be released into the bloodstream because skeletal muscle lacks the enzyme glucose-6-phosphatase . Liver Storage: The liver stores glycogen amounting to approximately 8-10% of its mass in the fed state. The liver synthesises glycogen to help maintain blood glucose levels between meals and during early fasting, and can release free glucose into the bloodstream due to the presence of glucose-6-phosphatase . Skeletal Muscle Storage: Skeletal muscle stores glycogen at about 1-2% of muscle mass . However, because total muscle mass is much greater than liver mass, the majority of the body’s total glycogen is stored in muscle . Energy Utility: Muscle glycogen is used only for local energy production during muscle contraction. Unlike the liver, skeletal muscle lacks glucose-6-phosphatase and therefore cannot export glucose into the systemic circulation. Enzymatic Control: The rate-limiting enzyme of glycogenesis is glycogen synthase , which adds glucose from UDP-glucose (uridine diphosphate glucose) to the growing glycogen chain. This enzyme is activated by insulin in both the liver and the muscle. Liver and kidney (Option A): While both organs are capable of gluconeogenesis (creating glucose) and the liver is a major site for glycogenesis, the kidneys store very little glycogen. Its primary role in glucose metabolism is filtration, reabsorption, and minimal glucose production during prolonged fasting. Brain and adipose tissue (Option C): The brain has an extremely high metabolic demand but lacks significant glycogen storage; it relies on a continuous supply of glucose from the blood. Adipose tissue (fat) specialises in storing energy as triglycerides, not glycogen. Heart and pancreas (Option D): While the heart (cardiac muscle) does contain small glycogen stores for emergency anaerobic conditions, it is not a predominant site compared to skeletal muscle. The pancreas functions as an endocrine and exocrine gland and does not serve as a significant storage depot for glycogen.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 175

↑ *Back to this question in Part A*

@Neet_pg_bot

Q9: Which glycogen storage disease does not involve hypoglycemia or exercise intolerance? (Q844873)

A. Von Gierke Disease

B. McArdle Disease

C. Andersen’s Disease

D. Tarui Disease

Correct Answer: C. Andersen’s Disease

■ *Listen to Audio 1*

Explanation: Andersen’s Disease (GSD Type IV) : Does not involve hypoglycemia or exercise intolerance. Enzyme Deficiency: Branching enzyme (glycogen branching enzyme), leading to abnormal glycogen accumulation Gene: GBE1 , Autosomal recessive Key Features: Hepatosplenomegaly, cirrhosis, failure to thrive Prognosis: Progressive liver disease → liver failure Classification Based on Hypoglycemia and Exercise Intolerance : GSD with Hypoglycemia: Von Gierke (I), Cori (III), Hers (VI), GSD IX, Fanconi-Bickel (XI), GSD 0 (Option A ruled out) GSD with Exercise Intolerance: Pompe (II), McArdle (V), Tarui (VII), Cori (III), GSD IX (Option B and D ruled out) GSD with Both Hypoglycemia and Exercise Intolerance: Cori (III), GSD IX GSD with Neither Hypoglycemia and Exercise Intolerance: Andersen (IV)

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 174 Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Page 129 Anderson-Fabry disease management: Anderson's Disease

↑ *Back to this question in Part A*

@Neet_pg_bot

Q10: A 6-month-old infant presents with recurrent episodes of hypoglycemia and elevated ketone levels. Genetic testing was done and diagnosed as Type 0 Glycogen storage disorder. Which of the following statements about Glycogen Storage Disease Type 0 is true? (Q844872)

A. The enzyme deficiency is glycogen synthase.

B. The affected genes are GYS2 and GYS1.

C. It is an autosomal recessive disorder

D. All of the above

Correct Answer: D. All of the above

■ *Listen to Audio 1*

Explanation: Glycogen Storage Disease Type 0 (GSD 0) Enzyme Deficiency: Glycogen synthase (GYS1 – muscle, GYS2 – liver) , Autosomal recessive inheritence Pathophysiology: Inability to synthesize glycogen → Reduced glycogen stores → Fasting hypoglycemia and ketosis Clinical Features: Fasting hypoglycemia, Ketosis, Postprandial hyperglycemia, Muscle weakness (if GYS1 affected) Diagnosis: Genetic testing (GYS1, GYS2), blood glucose & ketone levels Management: Frequent meals, cornstarch supplementation, avoid prolonged fasting

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 174 Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Page 129 Management glycogen storage disease 0

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: A 1 year old child presents with failure to thrive, weak bones, and renal dysfunction. The physician suspected glycogen storage disorder and further identified a mutation in the SLC2A2 gene while genetic testing. Which among the following defects is associated with it? (Q844871)

A. GLUT 2

B. Liver phosphorylase

C. Muscle and erythrocyte phosphofructokinase 1

D. Muscle Phosphorylase

Correct Answer: A. GLUT 2

■ *Listen to Audio 1*

Explanation: The child's clinical features along with identification of an SLC2A2 gene mutation , point to Fanconi-Bickel Syndrome which is caused by a defective GLUT2 transporter. Fanconi-Bickel Syndrome (GSD Type XI) : Cause: SLC2A2 mutation → defective GLUT2 transporter Affected Organs: Liver, kidney, intestines, pancreas Pathophysiology: Impaired glucose/galactose transport → hepatic glycogen accumulation → Renal

Fanconi Syndrome Clinical Features: Failure to thrive, Hepatomegaly, Renal dysfunction (glycosuria, phosphaturia, aminoaciduria), Osteopenia / rickets, Fasting hypoglycemia Diagnosis: Genetic testing, urine analysis, fasting blood glucose Management: Supportive care, frequent small meals, vitamin D & phosphate supplementation, uncooked cornstarch to prevent hypoglycemia Liver phosphorylase (Option B): Hers' disease leads to mild hepatomegaly and hypoglycemia, not renal dysfunction. Muscle and erythrocyte phosphofructokinase 1 (Option C) : Tarui disease causing exercise intolerance and hemolysis, not liver and renal dysfunction. Muscle Phosphorylase (Option D) : McArdle disease leads to exercise-induced muscle cramps and myoglobinuria, with no hepatic or renal involvement.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 174 Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Page 129 Management Fanconi-Bickel Syndrome

↑ *Back to this question in Part A*

@Neet_pg_bot

Q12: A 6-month-old infant presents with muscle hypotonia and heart failure. Laboratory tests reveal an accumulation of glycogen in lysosomes. Which of the following is the most likely diagnosis? (Q844870)

A. McArdle Disease

B. Pompe Disease

C. Von Gierke Disease

D. Cori Disease

Correct Answer: B. Pompe Disease

■ *Listen to Audio 1*

Explanation: The infant’s clinical features, along with glycogen accumulation in lysosomes on laboratory evaluation, are characteristic of Pompe Disease . Pompe Disease (GSD Type II) Enzyme Deficiency: Lysosomal acid maltase (α-1,4 and α-1,6 glucosidase) Gene: GAA , Autosomal Recessive Clinical Features: Infantile: Muscle hypotonia (“floppy baby”), cardiomegaly, heart failure, early death Juvenile/Adult: Muscle weakness/dystrophy Laboratory Findings: Glycogen accumulation in lysosomes, elevated CK, LDH, transaminases, lactate Management: Supportive care, enzyme replacement therapy (ERT) , cardiac evaluation (echocardiogram), careful fluid management Alglucosidase alfa (Myozyme/Lumizyme): Standard, long-standing ERT Avalglucosidase alfa-ngpt (Nexviazyme): N ewer ERT with improved cellular uptake designed for better efficacy in late-onset cases Disorder Enzyme Deficiency Key Clinical Features McArdle Disease (GSD V) (Option A) Muscle phosphorylase Exercise intolerance, muscle cramps, myoglobinuria Von Gierke Disease (GSD I) (Option C) Glucose-6-phosphatase Severe fasting hypoglycemia, hepatomegaly, lactic acidosis Cori Disease (GSD III) (Option D) Debranching enzyme Hepatomegaly, muscle weakness, limit dextrin accumulation

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 174

Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Page 129

www.ncbi.nlm.nih.gov/pmc/articles/PMC3110959/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q13: Which of the following statements is true on the structure of glycogen? Arranged in 12 concentric layers Glucose residues are connected by ■-1,4 linkage Branching points formed by α-1,6 linkage (Q844857)

A. 1,2

B. 2,3

C. 1,2,3

D. 1,3

Correct Answer: C. 1,2,3

■ *Listen to Audio 1*

Explanation: All three statements are true. ■■ Glycogen is a highly branched polysaccharide of glucose . The linear chains are joined by α-1,4-glycosidic bonds .(Statement 2) The branch points occur every 8–12 residues via α-1,6 linkages .(Statement 3) A mature glycogen molecule is roughly 20–40 nm in diameter and consists of about 12 concentric layers of glucose chains radiating from a glycogenin core protein .(Statement 1) Glycogenin –Acts as a primer enzyme and anchor for glycogen synthesis. Highly branched & compact structure allows rapid release of glucose during metabolic need and dense energy storage in limited space.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 153

↑ *Back to this question in Part A*

@Neet_pg_bot

Q14: A 6-month-old infant presents with severe hypoglycemia, hepatomegaly, growth retardation, irritability, and lethargy between feedings. Labs show elevated lactate,

triglycerides, and uric acid, and the child requires frequent feedings to prevent hypoglycemia. Which condition is most likely? (Q844868)

A. Von Gierke's disease

B. McArdle's disease

C. Tarui's Disease

D. Hers' Disease

Correct Answer: A. Von Gierke's disease

■ *Listen to Audio 1*

Explanation: The infant’s clinical signs, along with elevated lactate, triglycerides, and uric acid, suggest Von Gierke’s Disease . Von Gierke’s Disease (GSD Type I) Due to a deficiency of glucose-6-phosphatase Pathophysiology: Impaired conversion of glucose-6-phosphate → glucose, which leads to accumulation of glycogen, lactate, and triglycerides Key Symptoms: Severe hypoglycemia Hepatomegaly, renomegaly Growth retardation Lactic acidosis Doll facies Hyperuricemia (Excess G6P → ↑ glycolysis → ↑ pyruvate → ↑ lactate → lactate competes with uric acid in renal tubules → ↓ uric acid excretion) Hyperlipidemia (G6P diverted to glycolysis → ↑ acetyl-CoA → ↑ fatty acid & triglyceride synthesis → ↑ VLDL) Ketosis (Hypoglycemia → ↑ glucagon → ↑ lipolysis → ↑ fatty acids → liver converts to ketone bodies) Diagnosis: Biochemical tests (hypoglycemia, lactic acidosis, hyperuricemia, hyperlipidemia) Genetic testing Management: Frequent feedings with slow-release carbs Blood glucose monitoring Medications for complications Liver transplant in severe cases Glycogen Storage Disorders (GSDs) GSD Type Enzyme Deficiency Affected Organs Key Features Type V - McArdle's Disease (Option B) Muscle phosphorylase Skeletal Muscle Exercise intolerance, muscle cramps, and myoglobinuria after exertion Type VII - Tarui's Disease (Option C) Muscle phosphofructokinase Muscle, RBCs Exercise intolerance, muscle cramps, hemolytic anemia Type VI - Hers' Disease (Option D) Liver phosphorylase Liver Hepatomegaly, mild hypoglycemia, milder symptoms than Von Gierke's

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 174 Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Pages 128, 129

↑ *Back to this question in Part A*

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Q15: Match the glycogen storage disorders with their respective enzyme defects. Glycogen Storage Disorders: Enzyme Defects: 1. Pompe Disease a. Debranching enzyme 2. McArdle Disease b. Acid maltase 3. Von Gierke Disease c. Glucose-6-phosphatase 4. Cori Disease d. Myophosphorylase (Q844867)

A. 1-b, 2-d, 3-c, 4-a

B. 1-a, 2-c, 3-b, 4-d

C. 1-d, 2-b, 3-a, 4-c

D. 1-c, 2-a, 3-d, 4-b

Correct Answer: A. 1-b, 2-d, 3-c, 4-a

■ *Listen to Audio 1*

Explanation: Glycogen Storage Disorders: Enzyme Defects: 1. Pompe Disease Acid maltase (b) 2. McArdle Disease Myophosphorylase (d) 3. Von Gierke Disease Glucose-6-phosphatase (c) 4. Cori Disease Debranching enzyme (a) Glycogen storage disorders are a group of inherited metabolic diseases characterized by enzyme deficiencies affecting glycogen metabolism. These disorders cause abnormal glycogen accumulation or impaired glycogen utilization in various tissues. GSD (Type) Enzyme Defect Affected Organs Key Features I – Von Gierke Glucose-6-phosphatase Liver, Kidney Severe fasting hypoglycemia, hepatomegaly, lactic acidosis II – Pompe Acid maltase (lysosomal α-1,4-glucosidase) Muscle, Heart Glycogen accumulation in lysosomes, cardiomegaly, hypotonia, early infantile death III – Cori Debranching enzyme (α-1,6-glucosidase) Liver, Muscle Abnormal glycogen, mild hypoglycemia, hepatomegaly, muscle weakness V – McArdle Myophosphorylase (muscle glycogen phosphorylase) Skeletal Muscle Impaired glycogen breakdown, exercise intolerance, muscle cramps, myoglobinuria

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 174 Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Page 129

↑ *Back to this question in Part A*

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Q16: A 32-year-old woman consumes a high-carbohydrate diet. You are asked to explain the impact of her dietary habits on glycogen metabolism. Which of the following statements best describes glycogen metabolism? (Q844866)

A. Increased Gluconeogenesis

B. Reduced peripheral lipolysis

C. Low rate of glycogen synthesis

D. High rate of glycogenolysis

Correct Answer: B. Reduced peripheral lipolysis

■ *Listen to Audio 1*

Explanation: A high-carbohydrate diet increases blood glucose levels, leading to insulin secretion . ↑ High blood glucose → ↑ Insulin Insulin effects: Activates glycogen synthase → promotes glycogen storage in liver and muscle Inhibits glycogen phosphorylase → suppresses glycogen breakdown Reduces peripheral lipolysis to favor glucose utilization and storage Body enters the fed state , characterized by active glycogenesis and glucose utilization. Glycogen Metabolism in Different Metabolic States : 1. Fed State and Glycogenesis (Option B ruled out) After a high-carbohydrate meal , blood glucose levels increase , stimulating insulin secretion from the pancreas. Insulin activates glycogen synthase , promoting the conversion of glucose into glycogen in the liver and muscle . Glycogenolysis is inhibited to store excess glucose. 2. Fasted State and Glycogenolysis (Option A ruled out) Low blood glucose stimulates glucagon release , which activates glycogen phosphorylase , breaking down glycogen into glucose-1-phosphate. This provides energy for vital functions, primarily in the liver to maintain blood glucose levels. 3. Starvation State and Gluconeogenesis (Option C ruled out) Glycogen stores are depleted after prolonged fasting. The body shifts to gluconeogenesis , converting amino acids and glycerol into glucose. Cortisol and glucagon drive this process. 4. Post-Exercise State and Glycogen Replenishment (Option D ruled out) After intense exercise, muscle glycogen is depleted , and the body prioritizes glycogen resynthesis . Insulin sensitivity is high , allowing rapid glucose uptake for glycogenesis in muscle cells.

References:

Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Page 101

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: Which of the following enzymes is responsible for converting the glycogen molecule from a branched structure to a linear structure during glycogenolysis? (Q844865)

A. Glycogen Phosphorylase

B. Branching Enzyme

C. Debranching Enzyme

D. Glycogen Synthase

Correct Answer: C. Debranching Enzyme

■ *Listen to Audio 1*

Explanation: Debranching Enzyme plays a crucial role in glycogenolysis by removing the branches from the glycogen molecule. Function: Removes branches from glycogen to allow further degradation. Activities: Transferase: Moves a 3-glucose segment from a branch to a nearby chain. α-1,6-Glucosidase: Hydrolyzes the remaining α-1,6 bond at the branch point → releases free glucose . Glycogen Phosphorylase (Option A) Cleaves glycogen’s linear chains into glucose-1-phosphate; does not act on branches. Branching Enzyme(Option B) Creates branch points during glycogen synthesis, not involved in glycogenolysis Glycogen Synthase(Option D) Synthesizes glycogen by forming α-1,4 bonds; does not break down glycogen or linearize branches.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 174

Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Page 124

↑ *Back to this question in Part A*

@Neet_pg_bot

Q18: A 25-year-old male athlete develops fatigue and muscle cramps after intense exercise. Blood tests show elevated lactic acid. What is the net ATP yield from one molecule of muscle glycogen converted to lactic acid during strenuous exercise? (Q844864)

A. 1 ATP

B. 2 ATP

C. 3 ATP

D. 4 ATP

Correct Answer: C. 3 ATP

■ *Listen to Audio 1*

Explanation: Anaerobic glycolysis of muscle glycogen → lactate yields more ATP than free glucose because glycogen bypasses the hexokinase step that normally consumes 1 ATP. Here’s a stepwise

References:

Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Page 125

↑ *Back to this question in Part A*

@Neet_pg_bot

Q19: Which of the following statements correctly differentiates liver glycogen phosphorylase (LGP) and muscle glycogen phosphorylase (MGP)? (Q844863)

A. LGP is activated by glucagon, while MGP is activated by epinephrine.

B. LGP releases glucose-6-phosphate into the bloodstream, while MGP releases free glucose into the muscle.

C. LGP activity is regulated by AMP, while MGP is regulated by glucagon and epinephrine.

D. MGP is highly active during fasting compared to LGP.

Correct Answer: A. LGP is activated by glucagon, while MGP is activated by epinephrine.

■ *Listen to Audio 1*

Explanation: There are two main isoforms of glycogen phosphorylase: Aspect Liver Glycogen Phosphorylase (Liver-specific isoform) Muscle Glycogen Phosphorylase (Muscle-specific isoform) Primary Role Maintains blood glucose levels by breaking down glycogen during fasting Provides glucose for ATP production during muscle contraction. Function (Option B ruled out) Converts glycogen to glucose-1-phosphate, then to glucose-6-phosphate, which is converted to free glucose by glucose-6-phosphatase and released into the bloodstream Converts glycogen to glucose-1-phosphate, then to glucose-6-phosphate for energy production within muscle cells (muscle lacks glucose-6-phosphatase) Hormonal Activation (Option C ruled out) Activated by glucagon (during fasting) and epinephrine (during stress) Activated by epinephrine and AMP Allosteric Regulation Inhibited by glucose Activated by AMP, inhibited by ATP and glucose-6-phosphate Response to Fasting (Option D ruled out) Increased activity Not significantly affected Response to Feeding Decreased activity Not significantly affected Response to Exercise No significant change Increased activity

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 173–177 Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Pages 124–127

↑ *Back to this question in Part A*

@Neet_pg_bot

Q20: Which of the following enzymes catalyzes the rate-limiting step of glycogenolysis (Q844862)

A. Phosphoglucomutase

B. Glucose-6-phosphatase

C. Glucan transferase

D. Glycogen phosphorylase

Correct Answer: D. Glycogen phosphorylase

■ *Listen to Audio 1*

Explanation: Glycogenolysis : Breakdown of glycogen to release glucose Provides glucose for energy during fasting (liver) and muscle contraction (muscle) Rate-Limiting Enzyme Glycogen phosphorylase, Requires pyridoxal phosphate (PLP) as a coenzyme. Steps of Glycogenolysis 1. Glycogen phosphorylase: Cleaves α-1,4

glycosidic bonds and releases glucose-1-phosphate . Rate-limiting enzyme , activated by phosphorylation. 2. Debranching enzyme (Glucan transferase & α-1,6-glucosidase): Transfers trisaccharide units to the main chain (glucan transferase). Hydrolyzes α-1,6 glycosidic bonds to release free glucose. 3. Phosphoglucomutase(Option A): Converts glucose-1-phosphate to glucose-6-phosphate . 4. Glucose-6-phosphatase (Liver only)(Option B): Converts glucose-6-phosphate to free glucose , which is released into the blood. Stimulators of Glycogenolysis : Glucagon (Liver), Epinephrine (Muscle & Liver), AMP (Muscle) Inhibitors of Glycogenolysis: Insulin, ATP, Glucose-6-P Glucan transferase (Option C): Incorrect because it helps in debranching glycogen by transferring trisaccharide units but does not control the rate of glycogen breakdown.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 173

↑ *Back to this question in Part A*

@Neet_pg_bot

Q21: Which of the following statements is false regarding glycogenolysis? (Q844861)

A. Glucose-6-phosphatase acts in liver

B. Glycogen phosphorylase cleaves ■-1,4 linkages

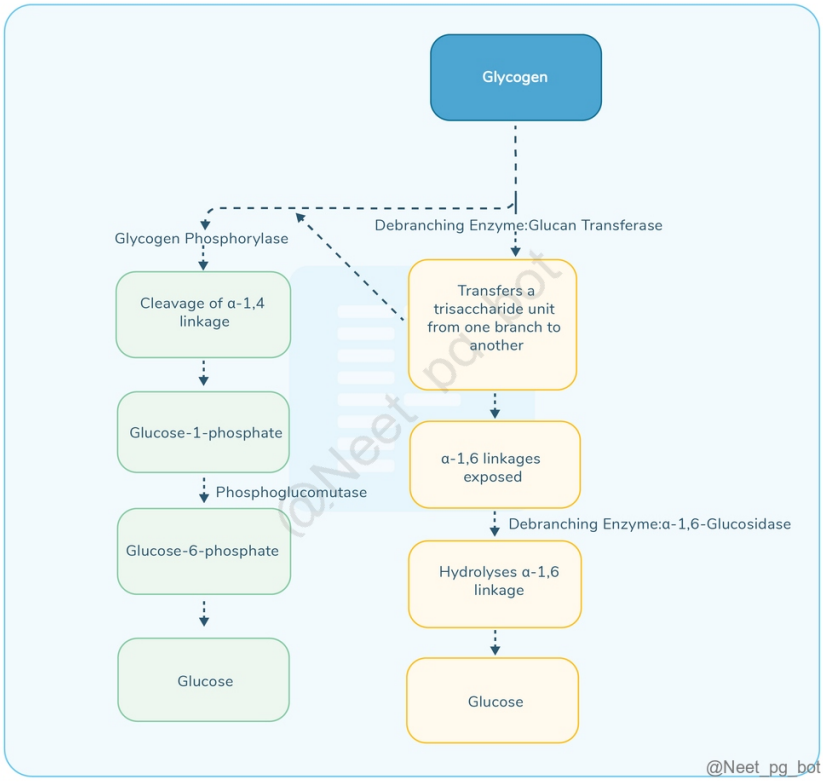
C. Phosphoglucomutase converts glucose-1-phosphate into glucose-6-phosphate

D. Glycogenolysis is the reverse process of glycogenesis.

Correct Answer: D. Glycogenolysis is the reverse process of glycogenesis.

■ *Listen to Audio 1*

Explanation: Glycogenolysis is NOT simply the reverse of glycogenesis Although related, it involves different enzymes : Glycogen phosphorylase (breaks α-1,4) Debranching enzyme (handles α-1,6 branches) Glycogenolysis involves breaking down glycogen into glucose-1-phosphate, whereas glycogenesis involves synthesizing glycogen from glucose-1-phosphate. Glucose-6-phosphatase acts in the liver (Option A): Converts Glucose-6-phosphate → Glucose for release into blood. Absent in muscle; muscle glycogen is used locally. Glycogen phosphorylase cleaves α-1,4 linkages (Option B): Removes glucose residues from non-reducing ends as glucose-1-phosphate. Phosphoglucomutase converts glucose-1-phosphate into glucose-6-phosphate (Option C) :Prepares glucose for glycolysis (muscle) or glucose release (liver).



References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 173, 174

↑ *Back to this question in Part A*

@Neet_pg_bot

Q22: Which of the following enzymes is activated by phosphorylation? (Q844860)

A. Acetyl-CoA carboxylase

B. Glycogen synthase

C. Pyruvate dehydrogenase

D. Glycogen phosphorylase

Correct Answer: D. Glycogen phosphorylase

■ *Listen to Audio 1*

Explanation: Enzymes that increase blood glucose are stimulated by phosphorylation. Enzymes that decrease blood glucose are stimulated by dephosphorylation. Activated by phosphorylation Deactivated by phosphorylation

Glycogen phosphorylase Debranching enzyme Pyruvate carboxylase PEPCK (Phosphoenolpyruvate carboxykinase). Acetyl-CoA carboxylase (Option A ruled out) Glycogen synthase (Option B ruled out) Pyruvate dehydrogenase (Option C ruled out) HMG-CoA reductase

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 93

↑ *Back to this question in Part A*

@Neet_pg_bot

Q23: What is the correct order of enzymes that act in glycogenesis? (Q844859)

A. Hexokinase→ Phosphoglucomutase → UDP-glucose pyrophosphorylase

B. Phosphoglucomutase → UDP-glucose pyrophosphorylase → Hexokinase

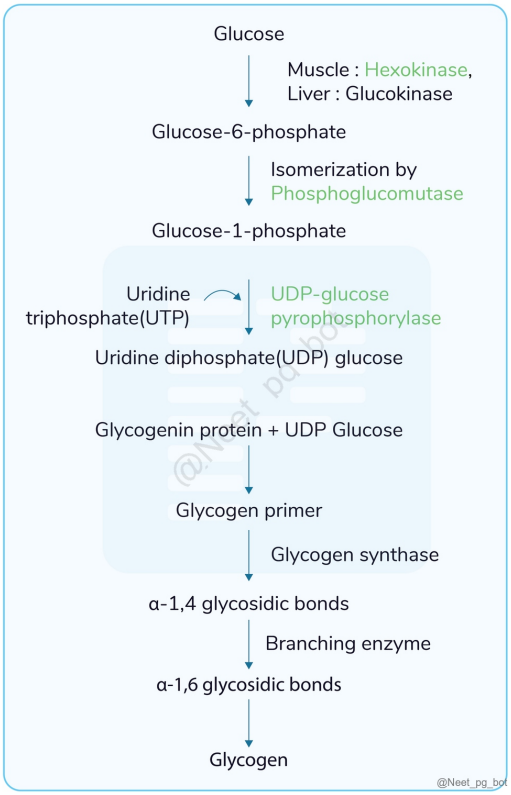
C. Hexokinase→ UDP-glucose pyrophosphorylase → Phosphoglucomutase

D. UDP-glucose pyrophosphorylase → Phosphoglucomutase → Hexokinase

Correct Answer: A. Hexokinase→ Phosphoglucomutase → UDP-glucose pyrophosphorylase

■ *Listen to Audio 1*

Explanation: Glycogenesis Process of glycogen synthesis . Occurs in liver and muscle , with liver playing a major role in maintaining blood glucose levels



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 173

↑ *Back to this question in Part A*

@Neet_pg_bot

Q24: Assertion (A): Glycogen and amylopectin are both branched polysaccharides composed of glucose units. Reason (R): Both contain α-1,4 glycosidic linkages in the linear chains and α-1,6 glycosidic linkages at the branch points. (Q844858)

A. Both the assertion and the reason are correct, and the reason is the correct explanation for the assertion.

B. Both the assertion and the reason are correct, but the reason is not the correct explanation for the assertion.

C. The assertion is correct, but the reason is incorrect.

D. The assertion is incorrect, but the reason is correct.

Correct Answer: A. Both the assertion and the reason are correct, and the reason is the correct explanation for the assertion.

■ *Listen to Audio 1*

Explanation: for the assertion.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 152, 153 Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Pages 78, 79

↑ *Back to this question in Part A*

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• [02_Carbohydrate Chemistry] 007 Citric Acid Cycle And Gluconeogenesis

« Back to Folder Directory

[02_Carbohydrate Chemistry] 007 Citric Acid Cycle And Gluconeogenesis — Quick Jump

Q1 (p. 555)	*Q2 (p. 556)*	*Q3 (p. 557)*	*Q4 (p. 558)*	*Q5 (p. 559)*	*Q6 (p. 560)*
Q7 (p. 561)	*Q8 (p. 563)*	*Q9 (p. 564)*	*Q10 (p. 565)*	*Q11 (p. 566)*	*Q12 (p. 567)*
Q13 (p. 568)	*Q14 (p. 569)*	*Q15 (p. 570)*	*Q16 (p. 570)*	*Q17 (p. 571)*	*Q18 (p. 572)*

Q1: Which enzyme is the rate-limiting enzyme of gluconeogenesis? (Q845015)

A. PEP Carboxykinase

B. Pyruvate Carboxylase

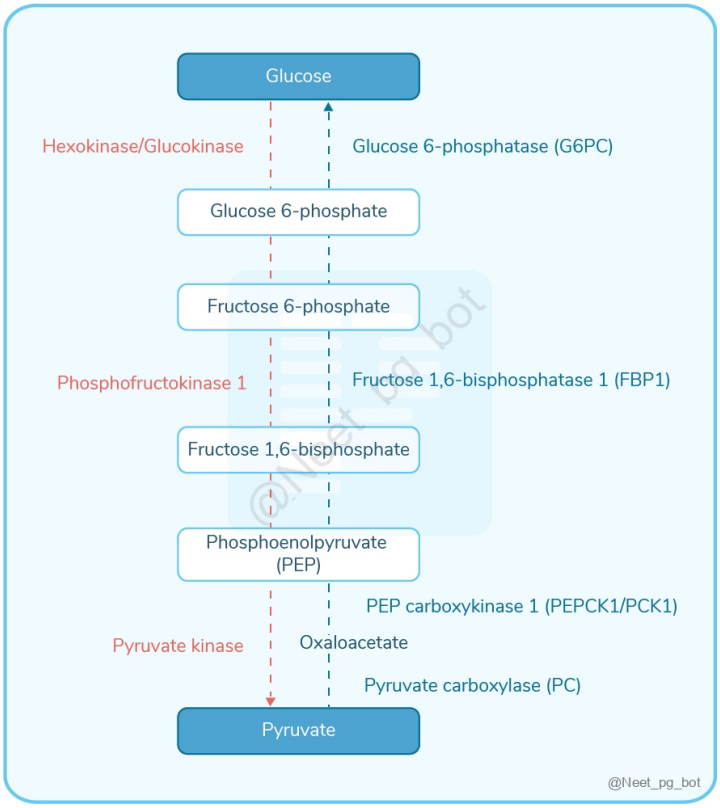
C. Glucose-6-phosphatase

D. Fructose 1,6-bisphosphatase

Correct Answer: D. Fructose 1,6-bisphosphatase

■ *Listen to Audio 1*

Explanation: The rate-limiting enzyme of gluconeogenesis is Fructose 1,6-bisphosphatase, which plays a crucial role in the regulation of gluconeogenesis, catalysing the conversion of fructose 1,6-bisphosphate to fructose 6-phosphate. Regulation: Fructose-1,6-bisphosphatase is inhibited by AMP , which indicates a low energy state , thereby suppressing gluconeogenesis. It is also inhibited by fructose-2,6-bisphosphate (F-2,6-BP) , a potent regulator that opposes gluconeogenesis and promotes glycolysis . It is activated by ATP and citrate , which signal a high-energy state , favoring glucose production. Hormonal Regulation Glucagon stimulates gluconeogenesis by decreasing levels of fructose-2,6-bisphosphate . This occurs via activation of cAMP and Protein Kinase A (PKA) , which phosphorylates the bifunctional enzyme (PFK-2/FBPase-2) . Phosphorylation inhibits PFK-2 and activates FBPase-2 , leading to ↓ F-2,6-BP levels . Reduced F-2,6-BP removes inhibition on Fructose-1,6-bisphosphatase , thereby stimulating gluconeogenesis .



References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 162,163,171

↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: In patients with uncontrolled diabetes, which of the following intermediates is depleted, suppressing the TCA cycle? (Q845024)

A. Acetyl CoA

B. Oxaloacetate

C. Succinyl-CoA

D. Malate

Correct Answer: B. Oxaloacetate

■ *Listen to Audio 1*

Explanation: In uncontrolled diabetes , gluconeogenesis is upregulated due to low insulin and high glucagon levels , leading to excessive oxaloacetate (OAA) utilization for glucose production. This depletes oxaloacetate , a key intermediate in the TCA cycle , causing suppression of the cycle despite an abundance of acetyl-CoA . Excess acetyl-CoA is then diverted to ketogenesis , leading to ketoacidosis . Role of Oxaloacetate in the TCA Cycle Function Effect in Uncontrolled Diabetes Combines with acetyl-CoA to form citrate Depleted due to increased gluconeogenesis Essential for TCA cycle continuation Suppression of the TCA cycle Deficiency leads to ketone body formation Ketogenesis → Ketoacidosis Acetyl-CoA (Option A): Incorrect because acetyl-CoA accumulates due to β-oxidation but cannot enter the TCA cycle without oxaloacetate . Succinyl-CoA (Option C): Incorrect because its levels are not significantly affected in diabetes; it plays a role in the TCA cycle but is not a primary regulator. Malate (Option D): Incorrect because malate can be converted to oxaloacetate , but oxaloacetate is preferentially diverted to gluconeogenesis , leading to depletion.

References:

Vasudevan's Textbook of Biochemistry, 6th Edition, Page 252

↑ *Back to this question in Part A*

@Neet_pg_bot

Q3: Which of the following factors regulates the expression of PEPCK in liver cells? (Q845023)

A. Glucagon

B. Insulin

C. Citrate

D. Acetyl-CoA

Correct Answer: A. Glucagon

■ *Listen to Audio 1*

Explanation: Phosphoenolpyruvate carboxykinase (PEPCK) is a key enzyme in gluconeogenesis that converts oxaloacetate to phosphoenolpyruvate (PEP) . Glucagon stimulates gluconeogenesis by increasing cAMP levels , activating protein kinase A (PKA) , and enhancing PEPCK gene transcription . This ensures glucose production during fasting states to maintain blood glucose levels. Regulation of PEPCK Expression Regulator Effect on PEPCK Mechanism Glucagon Increases ↑ cAMP → ↑ PKA → ↑ PEPCK transcription Insulin Decreases ↓ cAMP → ↓ PKA → ↓ PEPCK transcription Cortisol Increases Activates PEPCK gene transcription Glucose Decreases Inhibits gluconeogenic enzymes Acetyl-CoA Activates pyruvate carboxylase Does not directly regulate PEPCK Regulation of Gluconeogenesis Enzymes Enzyme Reaction Regulation Activators Inhibitors Hormonal Control Pyruvate Carboxylase Pyruvate → Oxaloacetate Allosteric Acetyl-CoA ↑ — Glucagon indirectly ↑ (via lipolysis → ↑ acetyl-CoA) PEP Carboxykinase (PEPCK) Oxaloacetate → PEP Gene expression (rate controlled) Glucagon, Cortisol ↑ Insulin ↓ Glucagon → ↑ cAMP → ↑ PKA → ↑ transcription Fructose-1,6-bisphosphatase (Rate-limiting) F-1,6-BP → F-6-P Allosteric ATP, Citrate ↑ AMP, F-2,6-BP ↓ Glucagon ↓ F-2,6-BP → activation

Glucose-6-phosphatase G-6-P → Glucose Substrate level + gene expression ↑ G6P — Glucagon ↑, Insulin ↓ expression Insulin (Option B): Incorrect because insulin inhibits PEPCK expression by reducing cAMP and promoting glycolysis . Citrate (Option C): Incorrect because citrate mainly activates gluconeogenesis by inhibiting PFK-1 but does not regulate PEPCK expression. Acetyl-CoA (Option D): Incorrect because it activates pyruvate carboxylase , not PEPCK.

References:

pubmed.ncbi.nlm.nih.gov/16375695/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q4: A 45-year-old woman presents with fatigue and muscle weakness. Laboratory tests reveal hypoglycemia and elevated lactate levels. The physician suspects a deficiency in a coenzyme essential for gluconeogenesis. Which coenzyme is most likely deficient in this patient? (Q845022)

A. Magnesium

B. Thiamine Pyrophosphate

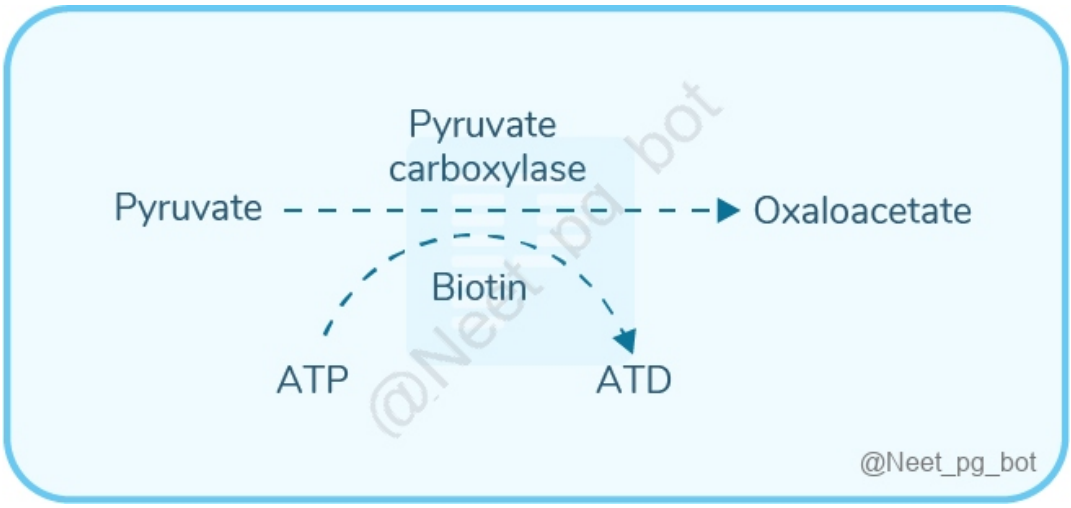
C. Biotin

D. Fluoride

Correct Answer: C. Biotin

■ *Listen to Audio 1*

Explanation: Biotin is a crucial coenzyme for pyruvate carboxylase , which catalyzes the conversion of pyruvate to oxaloacetate, the first regulatory step of gluconeogenesis. This reaction occurs in mitochondria and is essential for maintaining blood glucose levels during fasting. Biotin also functions as a coenzyme for propionyl-CoA carboxylase , which is involved in odd-chain fatty acid metabolism. Biotin deficiency leads to decreased gluconeogenesis , causing hypoglycemia and elevated lactate levels due to impaired conversion of pyruvate to glucose. Causes of biotin deficiency include: Severe malnutrition Consumption of raw eggs (contain avidin , which binds biotin and prevents its absorption) Inherited metabolic disorders (e.g., autosomal recessive biotinidase deficiency or holo-carboxylase synthetase deficiency) Gluconeogenesis & Coenzymes Step Enzyme Required Coenzyme Location Pyruvate → Oxaloacetate Pyruvate carboxylase Biotin Mitochondria Oxaloacetate → PEP PEPCK GTP Cytoplasm/Mitochondria F-1,6-BP → F-6-P Fructose-1,6-bisphosphatase Mg^{2}■ Cytoplasm G-6-P → Glucose Glucose-6-phosphatase Mg^{2}■ Endoplasmic Reticulum Magnesium (Option A): Required for ATP-dependent reactions but does not function as a coenzyme for pyruvate carboxylase. Thiamine Pyrophosphate (Option B): Cofactor for pyruvate dehydrogenase , not for gluconeogenesis. Fluoride (Option D): Inhibits enolase in glycolysis but has no role in gluconeogenesis.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 182

pubmed.ncbi.nlm.nih.gov/10846444/

↑ *Back to this question in Part A*

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Q5: Which of the following enzymes in gluconeogenesis is considered a "tandem enzyme" with fructose-2,6-bisphosphatase, functioning in the opposite direction? (Q845021)

A. Pyruvate carboxylase

B. Phosphoenolpyruvate carboxykinase

C. Phosphofructokinase I

D. Phosphofructokinase-2

Correct Answer: D. Phosphofructokinase-2

■ *Listen to Audio 1*

Explanation: Tandem enzymes refer specifically to a single bifunctional enzyme with both kinase and phosphatase activity, such as PFK-2/Fructose-2,6-bisphosphatase , rather than two separate enzymes working in opposing pathways. Phosphofructokinase-2 (PFK-2) and Fructose-2,6-bisphosphatase (FBPase-2) are two opposing activities within a single bifunctional enzyme , making it a tandem enzyme . PFK-2 activity: Converts fructose-6-phosphate to fructose-2,6-bisphosphate , which stimulates glycolysis by activating PFK-1 . FBPase-2 activity: Converts fructose-2,6-bisphosphate back to fructose-6-phosphate , reducing glycolysis and promoting

gluconeogenesis. This regulation allows for reciprocal control of glycolysis and gluconeogenesis in response to insulin and glucagon. Tandem Enzymes in Glucose Metabolism Tandem enzymes are pairs of enzymes that catalyze opposite reactions in glycolysis and gluconeogenesis, ensuring precise regulation of glucose metabolism. These enzyme pairs prevent futile cycling and allow metabolic control in response to hormonal signals. Tandem Enzyme Pairs Tandem Enzyme Pair Glycolysis Enzyme Gluconeogenesis Enzyme Function & Regulation PFK-2 / FBPase-2 Phosphofructokinase-2 (PFK-2) Fructose-2,6-bisphosphatase (FBPase-2) Regulates fructose-2,6-bisphosphate levels , controlling glycolysis & gluconeogenesis. Hexokinase / Glucose-6-phosphatase Hexokinase/Glucokinase Glucose-6-phosphatase Controls phosphorylation and dephosphorylation of glucose. Pyruvate kinase / PEPCK Pyruvate kinase Phosphoenolpyruvate carboxykinase (PEPCK) Controls conversion between PEP and pyruvate, linking glycolysis and gluconeogenesis. PFK-2 / FBPase-2: The Classic Tandem Enzyme PFK-2 and FBPase-2 are two opposing activities within a single bifunctional enzyme , which regulates glycolysis and gluconeogenesis. Condition PFK-2 Activity FBPase-2 Activity Metabolic Effect Fed state (Insulin ↑) Active Inhibited ↑ Fructose-2,6-bisphosphate → ↑ Glycolysis Fasting state (Glucagon ↑, cAMP ↑, PKA ↑) Inhibited Active ↓ Fructose-2,6-bisphosphate → ↑ Gluconeogenesis PFK-2 Activity: Converts fructose-6-phosphate into fructose-2,6-bisphosphate, which activates PFK-1 , promoting glycolysis. FBPase-2 Activity: Converts fructose-2,6-bisphosphate back to fructose-6-phosphate, inhibiting glycolysis and promoting gluconeogenesis.

References:

Textbook of Biochemistry, Vasudevan, 7th Edition, Page 113

Harper’s Biochemistry, 32nd Edition, Page 181

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: A patient presents with symptoms of hypoglycemia shortly after consuming a high-carbohydrate meal. Laboratory tests reveal elevated insulin levels. Which of the following enzymes in the gluconeogenesis pathway is most likely to be strongly inhibited in this scenario? (Q845020)

A. Pyruvate carboxylase

B. Phosphoenolpyruvate carboxykinase (PEPCK)

C. Fructose-1,6-bisphosphatase

D. Glucose-6-phosphatase

Correct Answer: C. Fructose-1,6-bisphosphatase

■ *Listen to Audio 1*

Explanation: Fructose-1,6-bisphosphatase is a key enzyme in gluconeogenesis that converts fructose-1,6-bisphosphate to fructose-6-phosphate . Elevated insulin levels after a high-carbohydrate meal lead to

increased fructose-2,6-bisphosphate , which strongly inhibits fructose-1,6-bisphosphatase , suppressing gluconeogenesis and contributing to hypoglycemia . Regulation of Gluconeogenesis Regulatory Mechanism Effect on Gluconeogenesis Key Enzymes Affected Hormonal Regulation Insulin (Fed State) Inhibits gluconeogenesis by increasing fructose-2,6-bisphosphate , which inhibits fructose-1,6-bisphosphatase. Fructose-1,6-bisphosphatase , PEPCK , Glucose-6-phosphatase Glucagon (Fasting State) Stimulates gluconeogenesis by decreasing fructose-2,6-bisphosphate . Fructose-1,6-bisphosphatase , PEPCK , Glucose-6-phosphatase Allosteric Regulation Fructose-2,6-bisphosphate Strongly inhibits fructose-1,6-bisphosphatase , suppressing gluconeogenesis. Fructose-1,6-bisphosphatase AMP (Low Energy State) Inhibits gluconeogenesis, signaling low ATP levels. Fructose-1,6-bisphosphatase Acetyl-CoA (High Energy State) Activates pyruvate carboxylase , promoting gluconeogenesis. Pyruvate carboxylase Substrate Availability Lactate Enters gluconeogenesis via pyruvate. Pyruvate carboxylase , PEPCK Glycerol Converted to DHAP , feeding into gluconeogenesis. Glycerol kinase , G3P dehydrogenase Alanine Converted to pyruvate for gluconeogenesis. Alanine aminotransferase , PEPCK Energy Status High ATP/Low AMP Promotes gluconeogenesis. Fructose-1,6-bisphosphatase , PEPCK Low ATP/High AMP Inhibits gluconeogenesis. Fructose-1,6-bisphosphatase Pyruvate carboxylase (Option A): Incorrect because insulin does not directly inhibit this enzyme; it is activated by acetyl-CoA during fasting. Phosphoenolpyruvate carboxykinase (PEPCK) (Option B): Incorrect because insulin suppresses its gene transcription, but it is not the most strongly inhibited enzyme in this acute setting. Glucose-6-phosphatase (Option D): Incorrect because it is regulated at the transcriptional level by insulin but is not directly inhibited by fructose-2,6-bisphosphate.

References:

Harper’s Biochemistry, 32nd Edition, Page 181, 182

↑ *Back to this question in Part A*

@Neet_pg_bot

Q7: Which of the following statements about the malate shuttle in gluconeogenesis is not true? (Q845018)

A. Helps transfer reducing equivalents from the cytoplasm to mitochondria

B. Involves the interconversion of oxaloacetate and malate

C. Requires the enzyme malate dehydrogenase in both mitochondria and cytosol

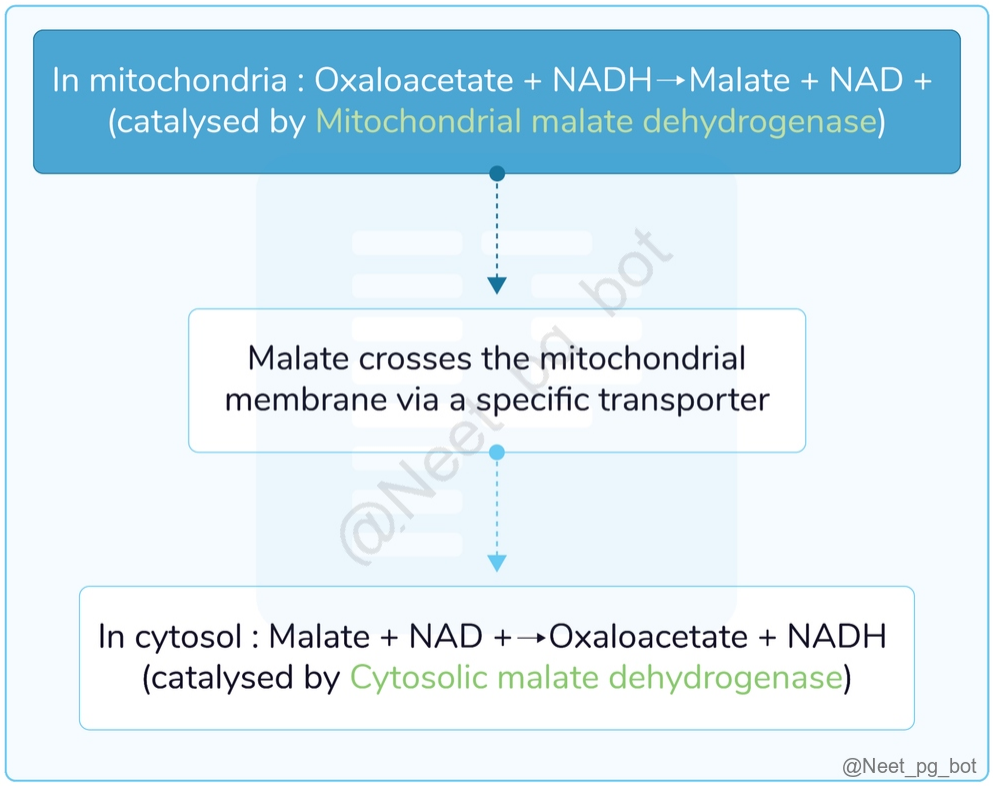
D. Directly transports NADH across the mitochondrial membrane

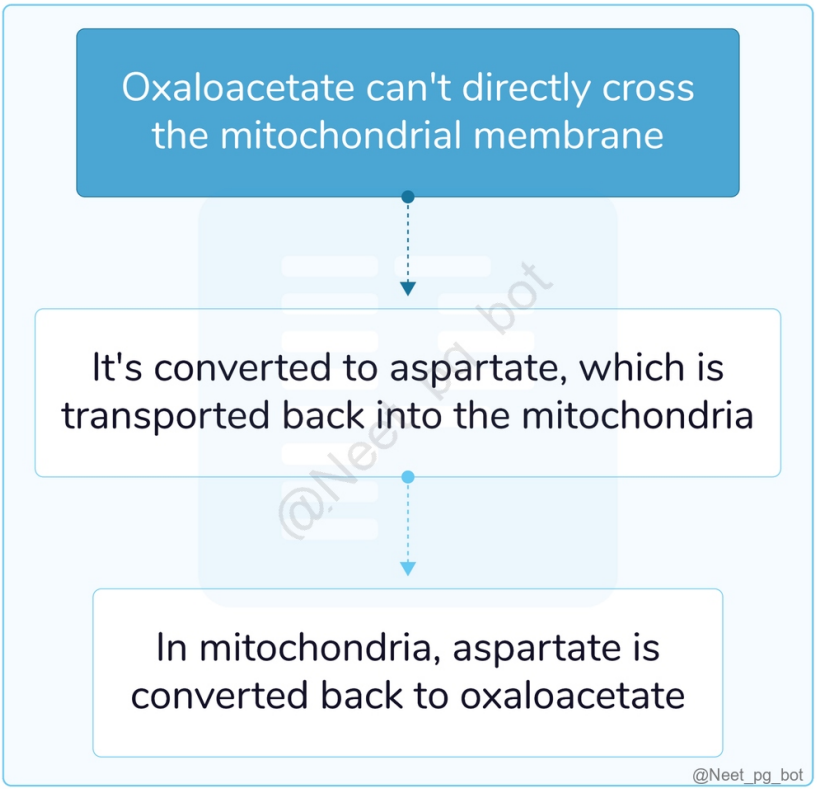
Correct Answer: D. Directly transports NADH across the mitochondrial membrane

■ *Listen to Audio 1*

Explanation: The malate shuttle does not directly transport NADH across the mitochondrial membrane. Instead, it indirectly transfers reducing equivalents by converting oxaloacetate to malate in the cytoplasm, allowing malate to cross the membrane, and then converting malate back to oxaloacetate in the mitochondria. This is necessary because the inner mitochondrial membrane is impermeable to NADH , preventing its direct transport. Malate

Shuttle: The malate shuttle, or malate-aspartate shuttle , is a biochemical system that facilitates the transport of electrons from the cytosol to the mitochondria. It is crucial for oxidative phosphorylation, as the mitochondrial inner membrane is impermeable to NADH, the primary reducing equivalent of the electron transport chain. Malate Shuttle in Gluconeogenesis Step Process Cytosol Oxaloacetate → Malate (via cytosolic malate dehydrogenase, using NADH) Transport Malate crosses mitochondrial membrane via malate-α-ketoglutarate antiporter Mitochondria Malate → Oxaloacetate (via mitochondrial malate dehydrogenase, regenerating NADH) Return Pathway Oxaloacetate → Aspartate (via transamination), which exits mitochondria via the glutamate-aspartate antiporter In mitochondria: Oxaloacetate return: The malate shuttle is important in both gluconeogenesis and glycolysis





References:

Harper's Biochemistry, 32nd Edition, Page 129

↑ *Back to this question in Part A*

@Neet_pg_bot

Q8: Which of the following enzymes involved in gluconeogenesis is located in the endoplasmic reticulum? (Q845017)

A. Pyruvate carboxylase

B. Phosphoenolpyruvate carboxykinase (PEPCK)

C. Fructose-1,6-bisphosphatase

D. Glucose-6-phosphatase

Correct Answer: D. Glucose-6-phosphatase

■ *Listen to Audio 1*

Explanation: Glucose-6-phosphatase is the only gluconeogenic enzyme located in the endoplasmic reticulum (ER) . It catalyzes the final step of gluconeogenesis: Glucose-6-phosphate→Glucose+P i This reaction occurs in the ER lumen , and glucose is then transported into the cytoplasm and released into the bloodstream. Enzymes of Gluconeogenesis, Their Locations, and Cofactors Enzyme Location Function Cofactors Pyruvate Carboxylase (Option A ruled out) Mitochondria Converts pyruvate to oxaloacetate Biotin, ATP, CO■ Phosphoenolpyruvate Carboxykinase (PEPCK) (Option B ruled out) Cytoplasm & Mitochondria Converts oxaloacetate to PEP GTP (as a phosphate donor) Fructose-1,6-bisphosphatase (Option C ruled out) Cytoplasm Converts F-1,6-BP to F-6-P Mg^{2}■ Glucose-6-phosphatase Endoplasmic Reticulum Converts G-6-P to free glucose Mg^{2}

References:

Harper’s Biochemistry, 32nd Edition, Page 184, 185

↑ *Back to this question in Part A*

@Neet_pg_bot

Q9: Which of the following is NOT a substrate for gluconeogenesis? (Q845016)

A. Lactate

B. Glycerol

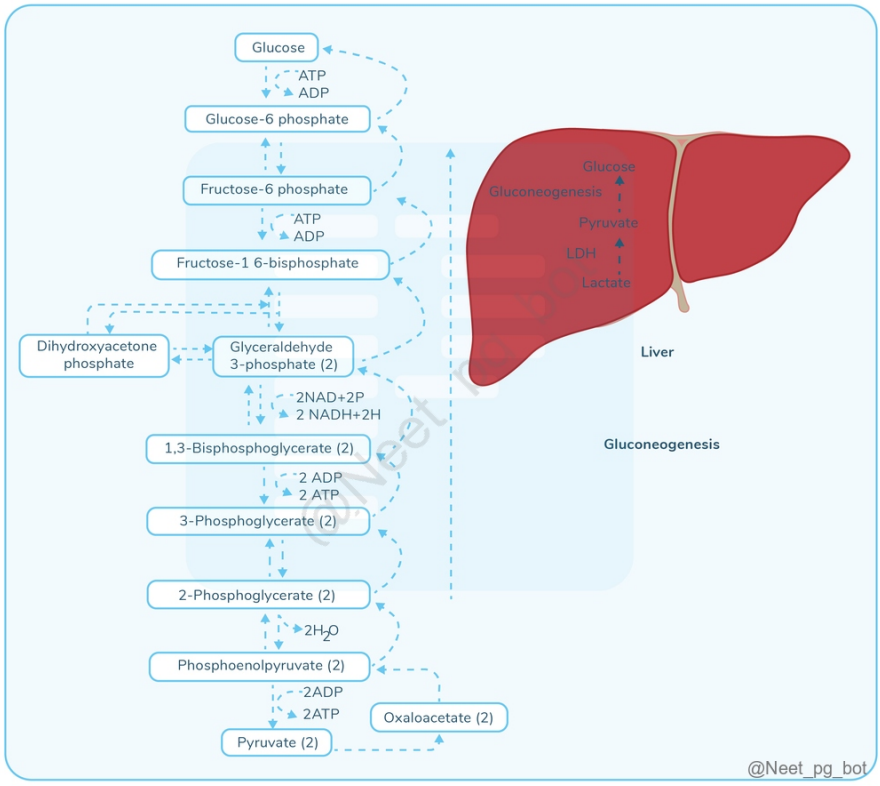
C. Alanine

D. Acetyl-CoA

Correct Answer: D. Acetyl-CoA

■ *Listen to Audio 1*

Explanation: Acetyl-CoA is not a substrate for gluconeogenesis. Although acetyl-CoA is an important metabolic intermediate, it cannot be converted to glucose because the reaction catalyzed by pyruvate dehydrogenase is irreversible. Substrates for Gluconeogenesis Substrate Source Entry Point in Gluconeogenesis Lactate Anaerobic glycolysis (muscles, RBCs) Converted to pyruvate via the Cori cycle Glycerol Lipolysis (adipose tissue) Converted to DHAP Alanine Muscle protein breakdown Converted to pyruvate via transamination Propionate Odd-chain fatty acid oxidation Converted to succinyl-CoA (TCA cycle) Alanine is converted to pyruvate, which enters the gluconeogenesis pathway as part of the glucose-alanine (Cahill) cycle. Propionate : Obtained from the metabolism of odd-chain fatty acids. Lactate (Option A): Incorrect because it is a major gluconeogenic substrate via the Cori's cycle. Glycerol (Option B): Incorrect because it enters gluconeogenesis via DHAP formation. Alanine (Option C): Incorrect because it contributes to gluconeogenesis via pyruvate formation.



References:

Harper's Biochemistry, 32nd Edition, Page 142, 186, 187

↑ *Back to this question in Part A*

@Neet_pg_bot

Q10: All of the following metabolic pathways occur in the cytoplasm, except? (Q844990)

A. Glycolysis

B. Pentose Phosphate Pathway

C. Fatty Acid Synthesis

D. Citric Acid Cycle

Correct Answer: D. Citric Acid Cycle

■ *Listen to Audio 1*

Explanation: The Krebs Cycle, also known as the Citric Acid Cycle, primarily occurs in the mitochondria, not the cytoplasm. The other pathways listed (Glycolysis, Pentose Phosphate Pathway, and Fatty Acid Synthesis) primarily occur in the cytoplasm. Krebs Cycle: It's a mitochondrial pathway and a common pathway for carbohydrates, lipids, and protein. Strictly aerobic and produces 10 ATP per cycle It is an Amphibolic pathway and consists of Anaplerotic reactions that replenish intermediates of the Krebs cycle. Cellular Metabolism Pathways based on their locations: Cytoplasm Mitochondria Glycolysis (Option A ruled out) Pentose Phosphate Pathway (Option B ruled out) Fatty Acid Synthesis (Option C ruled out) Urea Cycle (partial- Cytoplasm & Mitochondria) HMP Shunt Krebs Cycle (Citric Acid Cycle) Electron Transport Chain Fatty Acid Oxidation Ketogenesis Gluconeogenesis occurs in the cytoplasm, mitochondria, and endoplasmic reticulum.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 156

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: Which of the following best describes the amphibolic nature of the citric acid cycle? (Q845014)

A. It generates ATP and produces citrate for fatty acid synthesis.

B. It exclusively produces NADH for oxidative phosphorylation.

C. It breaks down fatty acids and synthesises only cholesterol.

D. It provides intermediates solely for glucose synthesis.

Correct Answer: A. It generates ATP and produces citrate for fatty acid synthesis.

■ *Listen to Audio 1*

Explanation: The citric acid cycle is amphibolic because it both produces energy (catabolic function) and provides intermediates for biosynthesis, such as amino acids, glucose, and fatty acids (anabolic function). Amphibolic nature of TCA cycle: 1. Catabolic Role (Energy Production) Oxidation of Acetyl-CoA: Acetyl-CoA, derived from the breakdown of carbohydrates, fats, and proteins, enters the TCA cycle and is oxidized to produce NADH, FADH■, and GTP (converted to ATP). . Generation of Reducing Equivalents: The cycle generates NADH and FADH2, which are essential for the electron transport chain and subsequent ATP production. 2. Anabolic Role (Biosynthesis) Citrate for Fatty Acid Synthesis : Citrate from the TCA cycle is exported to the cytoplasm and cleaved to provide acetyl-CoA for fatty acid synthesis. α-Ketoglutarate for Amino Acid Synthesis : α-Ketoglutarate is transaminated to form glutamate, which can be converted to glutamine, proline, and arginine. Succinyl-CoA for Heme Synthesis: Succinyl-CoA and glycine participate in the synthesis of δ-aminolevulinic acid (ALA) , the first step in heme biosynthesis. Oxaloacetate for Gluconeogenesis: Oxaloacetate can be converted to phosphoenolpyruvate (PEP), which is a key intermediate in gluconeogenesis, the pathway that produces glucose from non-carbohydrate sources. It exclusively produces NADH for oxidative phosphorylation (Option B): While the citric acid cycle produces NADH, its amphibolic nature also involves generating intermediates for

biosynthesis, not just ATP production. It directly synthesises fatty acids within the cycle (Option C) : The citric acid cycle does not directly synthesise fatty acids. Instead, it produces citrate, which can be transported out of the mitochondria and used in the cytoplasm for fatty acid synthesis. It provides intermediates solely for glucose synthesis (Option D) : The citric acid cycle provides intermediates for various biosynthetic pathways, not just glucose synthesis.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 164,165,171

↑ *Back to this question in Part A*

@Neet_pg_bot

Q12: Which of the following is correct regarding anaplerotic reactions? (Q845013)

A. Produce ATP

B. Replenish TCA intermediates

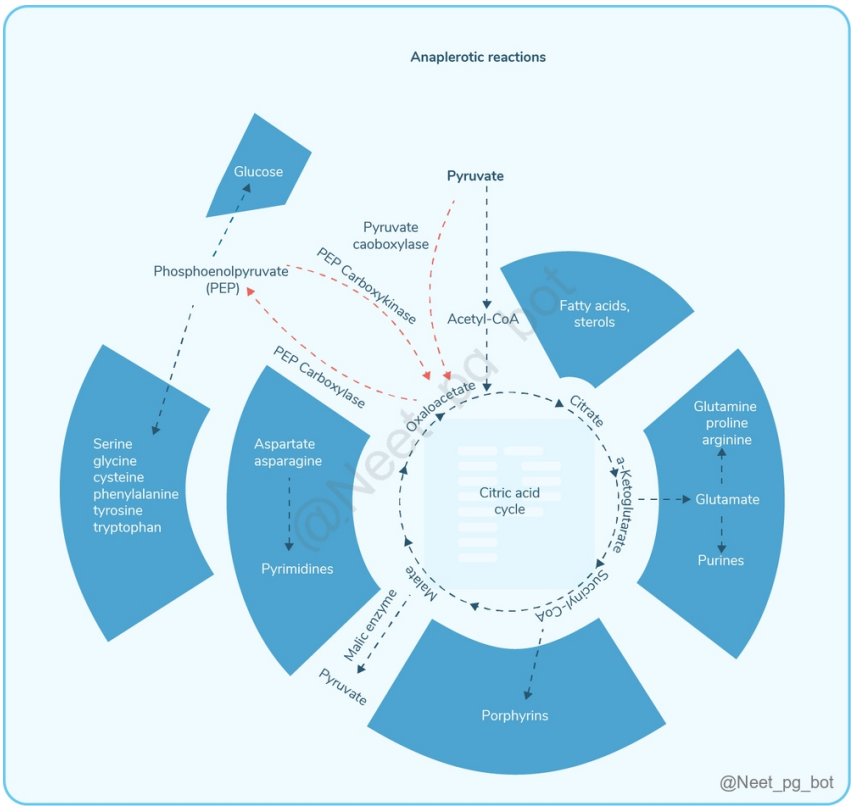
C. Reduction of NAD+ to NADH

D. Breakdown of fatty acids

Correct Answer: B. Replenish TCA intermediates

■ *Listen to Audio 1*

Explanation: Anaplerotic reactions are biochemical pathways that replenish intermediates of the TCA cycle , ensuring its continuous function. These reactions compensate for the loss of intermediates used in biosynthetic pathways (e.g., amino acid synthesis, gluconeogenesis). Anaplerotic Reactions Reaction Enzyme Intermediate Replenished Source Pyruvate → Oxaloacetate Pyruvate carboxylase Oxaloacetate Pyruvate (from glycolysis) Glutamate → α-Ketoglutarate Glutamate dehydrogenase α-Ketoglutarate Glutamate (from amino acid metabolism) Propionyl-CoA → Succinyl-CoA Propionyl-CoA carboxylase Succinyl-CoA Odd-chain fatty acids, Valine, Isoleucine, Methionine Aspartate → Oxaloacetate Aspartate transaminase Oxaloacetate Aspartate (from protein metabolism) Produce ATP (Option A) : Incorrect because anaplerotic reactions do not generate ATP directly. ATP is primarily produced through substrate-level phosphorylation and oxidative phosphorylation in the TCA cycle. Reduction of NAD+ to NADH (Option C) : Incorrect because anaplerotic reactions do not involve NADH production. NADH is generated during oxidative pathways like glycolysis and the TCA cycle. Break down fatty acids (Option D) : Incorrect because fatty acid β-oxidation generates acetyl-CoA , which enters the TCA cycle but does not serve as an anaplerotic reaction.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 162,171

↑ *Back to this question in Part A*

@Neet_pg_bot

Q13: ATP inhibits all of the following enzymes in the citric acid cycle except: (Q845012)

A. Citrate synthase

B. Isocitrate dehydrogenase

C. α-Ketoglutarate dehydrogenase

D. Succinate dehydrogenase

Correct Answer: D. Succinate dehydrogenase

■ *Listen to Audio 1*

Explanation: Succinate dehydrogenase is not regulated by ATP. Its regulation is primarily by the availability of its substrate (succinate) and competitive inhibition by oxaloacetate. Regulation of Enzymes in the TCA Cycle Enzyme Activators Inhibitors Citrate Synthase (Option A ruled out) ADP ATP, Citrate, NADH, Succinyl-CoA Isocitrate Dehydrogenase (Option B ruled out) ADP, NAD+, Ca²■ ATP, NADH α-Ketoglutarate Dehydrogenase (Option C ruled out) ADP, Ca²■ ATP, NADH, Succinyl-CoA Succinate Dehydrogenase Succinate availability Oxaloacetate

References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 82,158-159,165

↑ *Back to this question in Part A*

@Neet_pg_bot

Q14: Which of the following toxins directly inhibits the activity of the enzyme aconitase in the Citric Acid Cycle? (Q845011)

A. Cyanide

B. Fluoroacetate

C. Rotenone

D. Antimycin A

Correct Answer: B. Fluoroacetate

■ *Listen to Audio 1*

Explanation: Fluoroacetate itself does not directly inhibit aconitase. Instead, fluoroacetate is converted to fluoroacetyl-CoA , which then forms fluorocitrate . Fluorocitrate inhibits aconitase , leading to citrate accumulation. Fluoroacetyl-CoA condenses with oxaloacetate to form fluorocitrate, which inhibits the aconitase enzyme, causing citrate accumulation. Inhibitors of enzymes involved in the citric acid cycle: Inhibitor Enzyme Result Fluoroacetate Aconitase Accumulation of citrate Arsenite Alpha-ketoglutarate dehydrogenase complex Accumulation of alpha-ketoglutarate Malonate Succinate dehydrogenase Accumulation of succinate Cyanide (Option A) is a very toxic chemical that inhibits cytochrome c oxidase (Complex IV) in the electron transport chain, leading to the failure of aerobic respiration and histotoxic hypoxia. Rotenone (Option C) is an inhibitor of Complex I (NADH dehydrogenase) of the electron transport chain, and it is not directly associated with the inhibition of the TCA cycle Antimycin A (Option D) is a direct inhibitor of Complex III (cytochrome bc1 complex) of the electron transport chain, and it is not directly associated with the inhibition of the TCA cycle

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 127 and 158

↑ *Back to this question in Part A*

@Neet_pg_bot

Q15: Calculate the total amount of ATP produced from the Citric Acid Cycle alone for one glucose molecule. (Q845008)

A. 30 ATP

B. 9 ATP

C. 18 ATP

D. 20 ATP

Correct Answer: D. 20 ATP

■ *Listen to Audio 1*

Explanation: Each glucose molecule produces 2 Acetyl-CoA , and each Acetyl-CoA completes one turn of the TCA cycle . Substrate ATP Yield (Approx.) 1 NADH 2.5 ATP 1 FADH■ 1.5 ATP 1 GTP 1 ATP 1 Acetyl-CoA (TCA cycle) 10 ATP 1 Glucose (TCA only) 20 ATP 1 Pyruvate → Acetyl-CoA (PDH) 2.5 ATP Glycolysis (aerobic) ~7 ATP Glycolysis (anaerobic) 2 ATP So, Each Acetyl-CoA generates 3 NADH, 1 FADH■, and 1 ATP Molecule Produced Total ATP for 2 Cycles NADH (3 per cycle) 3 × 2.5 × 2 = 15 ATP FADH■ (1 per cycle) 1 × 1.5 × 2 = 3 ATP GTP (Direct ATP) (1 per cycle) 1 × 2 = 2 ATP Total ATP 20 ATP Since one glucose produces 2 Acetyl-CoA, we double the ATP yield Total ATP from Citric Acid Cycle per glucose = 20 ATP

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 159

↑ *Back to this question in Part A*

@Neet_pg_bot

Q16: Which of the following by-products from the Krebs Cycle is involved in heme synthesis? (Q845007)

A. Citrate

B. α-Ketoglutarate

C. Succinyl-CoA

D. Malate

Correct Answer: C. Succinyl-CoA

■ *Listen to Audio 1*

Explanation: Succinyl-CoA and glycine are precursors for δ-aminolevulinic acid (ALA), the first step in heme synthesis. In the heme synthesis, succinyl-CoA combines with glycine to form δ-aminolevulinic acid (ALA), the first step. Succinyl-CoA is replenished via anaplerotic reactions involving amino acids like Valine, Isoleucine, and Methionine , which are converted to Propionyl-CoA before forming Succinyl-CoA. Propionyl CoA from odd fatty acid breakdown enters the pathway by converting to Succinyl CoA. Citrate (Option A) is a 6-carbon molecule formed from acetyl-CoA and oxaloacetate. Citrate serves as an intermediate in the TCA cycle and plays a key role in fatty acid and cholesterol synthesis by transporting acetyl-CoA from the mitochondria to the cytoplasm. α-Ketoglutarate (Option B) is a 5-carbon molecule involved in amino acid metabolism and serves as an intermediate in the Krebs Cycle. It undergoes a Transamination Reaction to form Glutamate, which is a Reversible reaction.(α-Ketoglutarate ↔ Glutamate) Glutamate is derived from α-ketoglutarate and can be synthesized from Histidine, Proline, Arginine, and Glutamine via transamination and deamination reactions. Malate (Option D): Malate is an intermediate in the Krebs Cycle, formed from fumarate and converted into oxaloacetate. Malate can be converted to pyruvate by Malic Enzyme, generating NADPH , which is crucial for fatty acid synthesis. It is crucial as pyruvate can yield both Acetyl CoA (Pyruvate dehydrogenase) and Oxaloacetate (Pyruvate carboxylase), enabling their entry into the Citric cycle.

References:

Vasudevan's Textbook of Biochemistry, 6th Edition, Page 252

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: Which of the following steps in the Citric acid cycle involves oxidative phosphorylation and decarboxylation? Isocitrate → α-Ketoglutarate Succinate → Fumarate Malate → Oxaloacetate α-Ketoglutarate → Succinyl-CoA (Q845006)

A. 1 and 4 are correct

B. 2 and 3 are correct

C. 1, 3, and 4 are correct

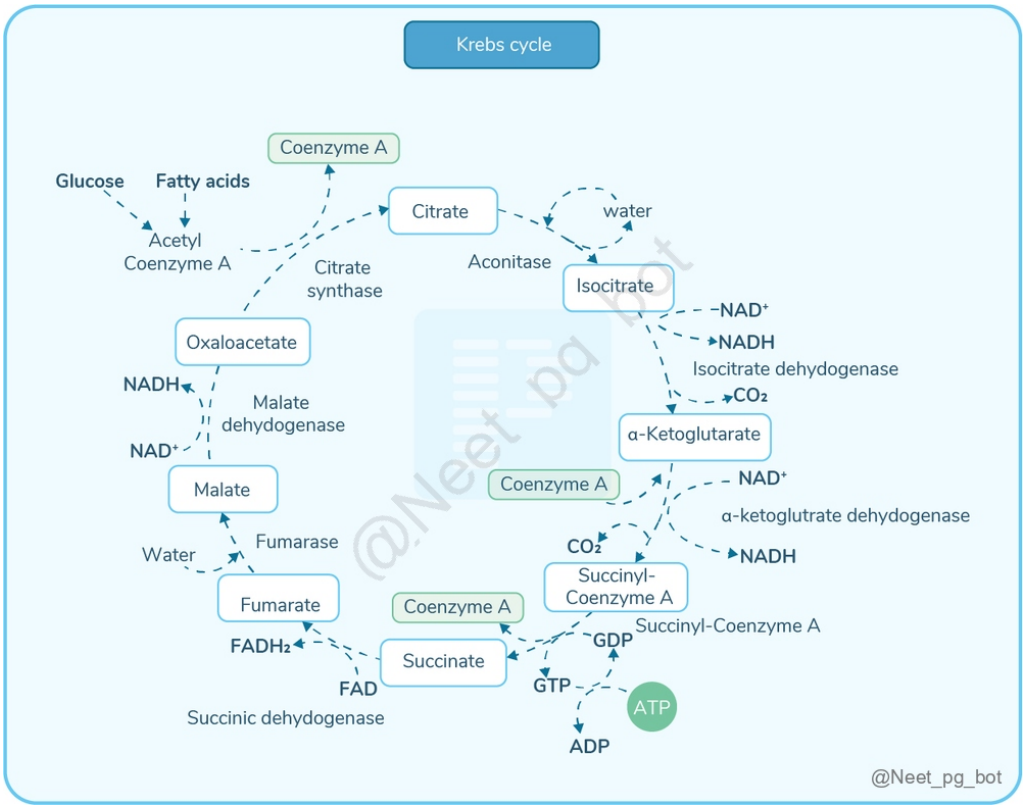
D. All are correct

Correct Answer: A. 1 and 4 are correct

■ *Listen to Audio 1*

Explanation: The enzymes involved in both reactions are Isocitrate Dehydrogenase (Step 1) and α-Ketoglutarate Dehydrogenase (Step 4). Both involve oxidation and removal of CO■ and produce NADH, which is later used in oxidative phosphorylation. Steps 3 and 4 are catalysed by Succinate dehydrogenase, and Malate Dehydrogenase catalyzes the oxidation of malate to oxaloacetate, producing NADH, which enters oxidative phosphorylation.

Krebs cycle: Reactions in Krebs cycle: Reaction Type Enzyme Reaction Product Formed Oxidative Decarboxylation Isocitrate Dehydrogenase Isocitrate → α-Ketoglutarate CO■ + NADH α-Ketoglutarate Dehydrogenase α-Ketoglutarate → Succinyl-CoA CO■ + NADH Oxidation (Production of NADH/FADH■) Isocitrate Dehydrogenase Isocitrate → α-Ketoglutarate NADH α-Ketoglutarate Dehydrogenase α-Ketoglutarate → Succinyl-CoA NADH Succinate Dehydrogenase Succinate → Fumarate FADH■ Malate Dehydrogenase Malate → Oxaloacetate NADH Substrate-Level Phosphorylation Succinyl-CoA Synthetase Succinyl-CoA → Succinate GTP (or ATP) Hydration Fumarase Fumarate → Malate H■O Addition Oxidative Decarboxylation removes CO■ and generates NADH . Oxidation reactions contribute to the Electron Transport Chain by generating NADH/FADH■ . Substrate-level phosphorylation occurs at the Succinyl-CoA to Succinate step. Hydration reaction occurs when Fumarase converts Fumarate to Malate .



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 158,159

↑ *Back to this question in Part A*

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Q18: Which of the following is not a cofactor for the enzyme catalysing α-Ketoglutarate → Succinyl-CoA? (Q844991)

A. Fe2+

B. Thiamine

C. Lipoic Acid

D. Pantothenic acid

Correct Answer: A. Fe2+

■ *Listen to Audio 1*

Explanation: The enzyme responsible for the reaction α-Ketoglutarate → Succinyl-CoA is α-Ketoglutarate Dehydrogenase, in which Fe2+ is not a cofactor. Iron (Fe²■) is a cofactor for Aconitase, which catalyzes the isomerization of citrate to isocitrate. α-Ketoglutarate Dehydrogenase: It is a Multienzyme complex involving 3 subunits and requires 5 Cofactors: Thiamine pyrophosphate (Vit B1) (Option B ruled out) Riboflavin (Vit B2 / FAD) Niacin (Vit B3 / NAD) Lipoic Acid (Option C ruled out) Pantothenic Acid / CoA / Vit B5 (Option D ruled out) It is involved in oxidative decarboxylation in the Krebs cycle but does not participate in oxidative phosphorylation. It catalyses the oxidative decarboxylation of α-ketoglutarate to form succinyl-CoA. α-Ketoglutarate + CoA + NAD■ → Succinyl-CoA + CO■ + NADH (Irreversible) It is Inhibited by Arsenite (Non-Competitive Inhibition). Similar Enzyme Complexes: Enzyme Complex Pathway Reaction Similarity Pyruvate dehydrogenase (PDH) Link reaction Pyruvate → Acetyl-CoA Same 3 subunits + same 5 cofactors α-Ketoglutarate dehydrogenase TCA cycle α-KG → Succinyl-CoA Same mechanism Branched-chain α-ketoacid dehydrogenase BCAA metabolism Leucine, isoleucine, valine metabolism Same structure & cofactors

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 159

↑ *Back to this question in Part A*

@Neet_pg_bot

• [02_Carbohydrate Chemistry] 008 Previous Year Questions

« Back to Folder Directory

[02_Carbohydrate Chemistry] 008 Previous Year Questions — Quick Jump

Q1 (p. 574)	*Q2 (p. 576)*	*Q3 (p. 577)*	*Q4 (p. 578)*	*Q5 (p. 578)*	*Q6 (p. 579)*
Q7 (p. 581)	*Q8 (p. 582)*	*Q9 (p. 584)*	*Q10 (p. 585)*	*Q11 (p. 586)*	*Q12 (p. 587)*
Q13 (p. 588)	*Q14 (p. 589)*	*Q15 (p. 590)*	*Q16 (p. 590)*	*Q17 (p. 591)*	*Q18 (p. 592)*
Q19 (p. 594)	*Q20 (p. 595)*	*Q21 (p. 596)*	*Q22 (p. 596)*	*Q23 (p. 597)*	*Q24 (p. 598)*
Q25 (p. 599)	*Q26 (p. 600)*	*Q27 (p. 600)*	*Q28 (p. 601)*	*Q29 (p. 602)*	*Q30 (p. 604)*
Q31 (p. 604)	*Q32 (p. 606)*	*Q33 (p. 607)*	*Q34 (p. 608)*	*Q35 (p. 609)*	*Q36 (p. 609)*
Q37 (p. 610)	*Q38 (p. 611)*	*Q39 (p. 611)*	*Q40 (p. 612)*	*Q41 (p. 613)*	*Q42 (p. 614)*
Q43 (p. 614)	*Q44 (p. 615)*	*Q45 (p. 616)*	*Q46 (p. 617)*	*Q47 (p. 618)*	*Q48 (p. 618)*
Q49 (p. 619)	*Q50 (p. 620)*	*Q51 (p. 621)*	*Q52 (p. 621)*	*Q53 (p. 622)*	*Q54 (p. 623)*
Q55 (p. 623)	*Q56 (p. 624)*	*Q57 (p. 626)*	*Q58 (p. 626)*	*Q59 (p. 627)*	*Q60 (p. 628)*
Q61 (p. 628)	*Q62 (p. 629)*	*Q63 (p. 630)*	*Q64 (p. 631)*	*Q65 (p. 631)*	*Q66 (p. 632)*
Q67 (p. 633)	*Q68 (p. 634)*				

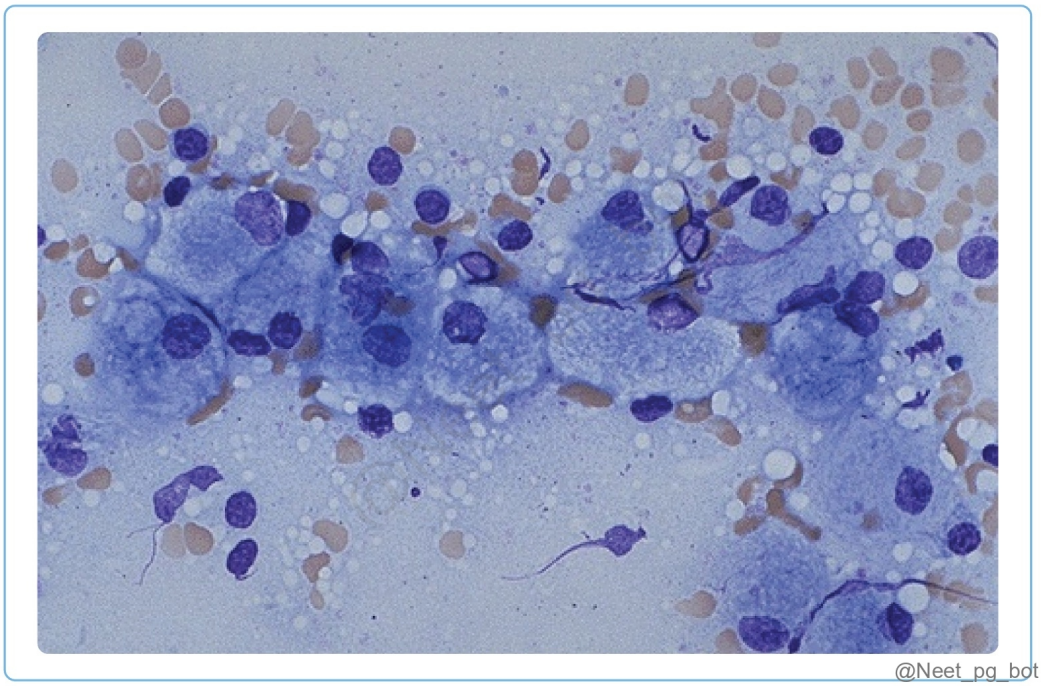
Q1: A child presents with bone pain and hepatosplenomegaly. A trephine biopsy and aspirate shows the following finding. Which of the following is the most likely enzyme deficient in this condition? (Q855278)

A. Hexosaminidase

B. Glucocerebrosidase

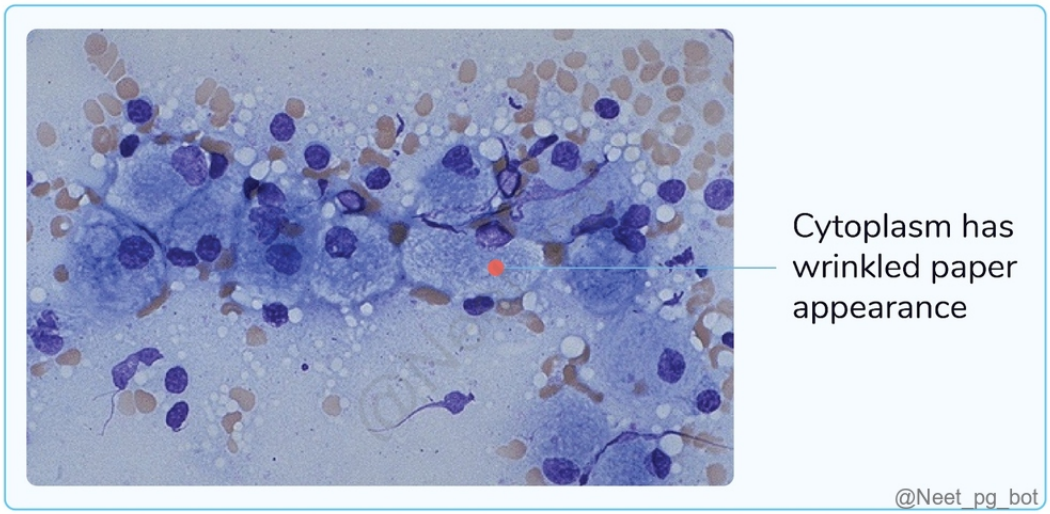
C. Sphingomyelinase

D. Alpha 1,4-glucosidase



Correct Answer: B. Glucocerebrosidase

Explanation: Gaucher's disease is characterized by the accumulation of glucosylceramide in RBCs and platelet membranes due to a deficiency in the enzyme beta-glucosidase (glucocerebrosidase) , leading to anemia, hepatosplenomegaly, and thrombocytopenia. Bone marrow biopsy shows macrophages with a characteristic "wrinkled paper" appearance. Gaucher's disease: Lysosomal storage disease Defective enzyme: Glucocerebrosidase (■-Glucosidase) Defect in metabolism: Accumulation of cerebroside (Glucosylceramide) in RBCs and platelet membranes. (Option B) Pathophysiology: Abnormal RBCs and platelets are engulfed by macrophages leading to accumulation inside macrophages causing wrinkled paper appearance. {{caption_text}} Disease Enzyme deficiency Features Tay-Sachs disease Hexosaminidase A (Option A ruled out) Mental retardation Blindness Muscular weakness Cherry red spots in the macula Fabry disease α-Galactosidase Skin rash Kidney failure (full symptoms only in males X-linked recessive) Krabbe disease β-Galactosidase Mental retardation Near absence of myelin Predominant neurological manifestation and cranial nerves causing deafness and blindness. Gaucher disease β-Glucosidase Enlarged liver and spleen Erosion of long bones Mental retardation in infants Anemia Thrombocytopenia Niemann-Pick disease Sphingomyelinase (Option C ruled out) Enlarged liver and spleen Mental retardation Fatal in early life Cherry red spot in macula Farber disease Ceramidase Hoarseness Dermatitis Skeletal deformation Mental retardation Fatal in early life Alpha 1,4-glucosidase (Option D): Deficiency of alpha 1,4-glucosidase (acid maltase) results in Pompe disease , which is associated with glycogen accumulation, hypotonia, cardiomegaly, and muscle weakness, rather than hepatosplenomegaly and bone pain.



References:

https://www.ncbi.nlm.nih.gov/books/NBK448080/

Harper’s illustrated Biochemistry 32e pg 245,

Vasudevan textbook of Biochemistry 7e pg 194

Image: https://www.tau.ac.il/medicine/tau-only/webpath/hemehtml/heme057.htm

↑ *Back to this question in Part A*

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Q2: Which enzyme deficiency causesVon Gierke's disease? (Q857492)

A. Liver phosphorylase

B. Lysosomal glucosidase

C. Hepatic glycogen synthase

D. Glucose 6-phosphatase

Correct Answer: D. Glucose 6-phosphatase

Explanation: Von Gierke’s disease (Glycogen Storage Disease Type I) is caused by a deficiency of glucose-6-phosphatase, an enzyme responsible for the final step of glycogenolysis and gluconeogenesis. Pathophysiology: Impaired conversion of glucose-6-phosphate to free glucose. Leads to glycogen accumulation in the liver and kidneys, causing hepatomegaly and severe hypoglycemia. Key symptoms: Severe fasting hypoglycemia Hepatomegaly Lactic acidosis Hyperuricemia and hyperlipidemia Liver phosphorylase (Option A): Deficiency causes Hers disease (GSD VI). Lysosomal glucosidase (Option B): Deficiency causes Pompe disease

(GSD II). Hepatic glycogen synthase (Option C): Deficiency causes GSD 0 (reduced glycogen storage). Glycogen storage disease: Type Name Enzyme defect Type 1 Von Gierke's Disease Glucose-6-phophatase Type 2 Pompe's Disease Acid maltase/ alpha glucosidase Type 3 Cori's Disease Debranching enzyme Type 4 Anderson's Disease Branching enzyme Type 5 McArdle's Disease Muscle Phosphorylase Type 6 Hers Disease Liver phosphorylase Type 7 Tarui's Disease Phosphofructokinase- I

References:

Von gierke disease

↑ *Back to this question in Part A*

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Q3: Which enzyme is responsible for the deficiency causing classical galactosemia? (Q856998)

A. Aldose reductase

B. Galactokinase

C. Galactose-1-phosphate uridyl transferase

D. UDP galactose-4-epimerase

Correct Answer: C. Galactose-1-phosphate uridyl transferase

Explanation: Classical galactosemia is a genetic metabolic disorder caused by a deficiency of the enzyme galactose-1-phosphate uridyl transferase (GALT). This enzyme is essential for the proper metabolism of galactose, a sugar found in milk and other dairy products. Role of GALT in Galactose Metabolism: GALT converts galactose-1-phosphate to UDP-galactose, which is required for various biochemical processes, including the synthesis of glycoproteins and glycolipids. A deficiency in GALT leads to the accumulation of galactose-1-phosphate and galactose in tissues, which can cause toxicity and damage to the liver, kidneys, and brain, leading to the symptoms of classical galactosemia. Clinical Features of Classical Galactosemia: Failure to thrive and jaundice in the neonatal period after feeding with milk. Liver damage, cataracts, intellectual disability, and sepsis from E. coli infections (due to impaired immune function). Diagnosis: Elevated galactose-1-phosphate in red blood cells. Genetic testing for mutations in the GALT gene. Treatment: Lactose-free diet (avoiding galactose) to prevent the accumulation of galactose and its metabolites. Aldose reductase (Option A): Involved in the reduction of galactose to galactitol, which is implicated in galactose toxicity but is not the enzyme deficient in classical galactosemia. Galactokinase (Option B): Deficiency causes galactokinase deficiency or non-classical galactosemia, which leads to cataract formation but does not cause the systemic effects seen in classical galactosemia (liver damage, intellectual disability). UDP-galactose-4-epimerase (Option D): Deficiency causes epimerase-deficient galactosemia, a less common form of galactosemia with milder symptoms than classical galactosemia.

References:

Galactosemia

↑ *Back to this question in Part A*

@Neet_pg_bot

Q4: Which one of the following choices does not represent a function of glucagon? (Q856992)

A. Glycolysis

B. Gluconeogenesis

C. Glycogenolysis

D. Ketogenesis

Correct Answer: A. Glycolysis

Explanation: Glucagon is a hormone produced by the alpha cells of the pancreas that primarily increases blood glucose levels, especially during fasting or low glucose states. It achieves this by stimulating processes that increase glucose availability and energy production. Glycolysis is the metabolic pathway that breaks down glucose to produce energy (ATP). Glucagon inhibits glycolysis in the liver to conserve glucose and promote the release of glucose into the bloodstream. Instead of promoting glucose breakdown, glucagon promotes glucose production and release. Gluconeogenesis (Option B): Stimulates the production of glucose from non-carbohydrate sources (amino acids, lactate) in the liver. Glycogenolysis (Option C): Stimulates the breakdown of glycogen into glucose in the liver to release glucose into the bloodstream. Ketogenesis (Option D): Stimulates the production of ketone bodies in the liver from fatty acids during periods of prolonged fasting or carbohydrate restriction.

References:

Glucagon

↑ *Back to this question in Part A*

@Neet_pg_bot

Q5: Glycogen phosphorylase is activated by: (Q855065)

A. Calcium

B. Glucose-6-phosphate

C. Insulin

D. Protein phosphatase

Correct Answer: A. Calcium

Explanation: Glycogen phosphorylase is activated by calcium in tissues, particularly in muscle cells. Calcium and Muscle Contraction: During muscle contraction, calcium ions are released from the sarcoplasmic reticulum in response to a neural signal. Calcium then binds to calmodulin, a calcium-binding protein, forming the calcium-calmodulin complex. Activation of Phosphorylase Kinase: The calcium-calmodulin complex activates phosphorylase kinase, an enzyme that plays a key role in glycogen breakdown. Activation of Glycogen Phosphorylase: The phosphorylation of glycogen phosphorylase activates it, which in turn stimulates the breakdown of glycogen into glucose-1-phosphate, providing energy for muscle contraction. Cofactor of glycogen phosphorylase is pyridoxal phosphate (PLP) Glucose-6-phosphate (Option B): Inhibits glycogen breakdown; favors glycogen synthesis. Insulin (Option C): Promotes dephosphorylation → inactivates glycogen phosphorylase. Protein phosphatase (Option D): Dephosphorylates and inactivates glycogen phosphorylase.

References:

pubmed.ncbi.nlm.nih.gov/11341967/#:~:text=The%20presence%20of%20Mem-CC,-CC%20(0.3%20nM).

↑ *Back to this question in Part A*

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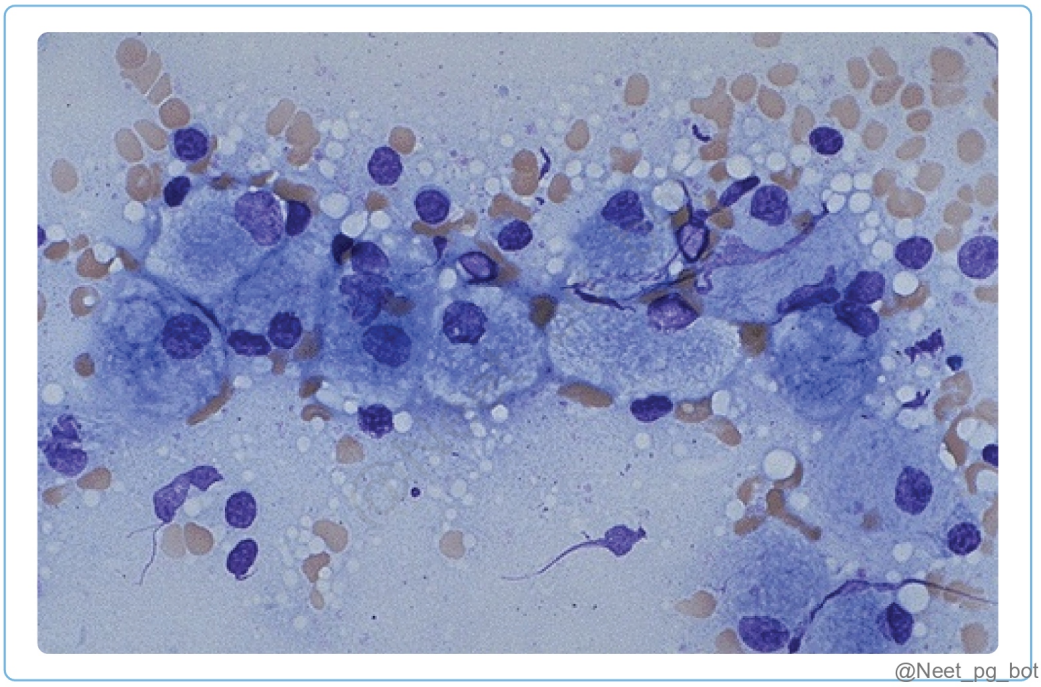
Q6: A baby is being evaluated for delayed developmental milestones. On examination, there is hepatosplenomegaly. Microscopic image of the bone marrow evaluation shown below. What is the diagnosis? (Q855544)

A. Gaucher disease

B. Niemann-pick disease

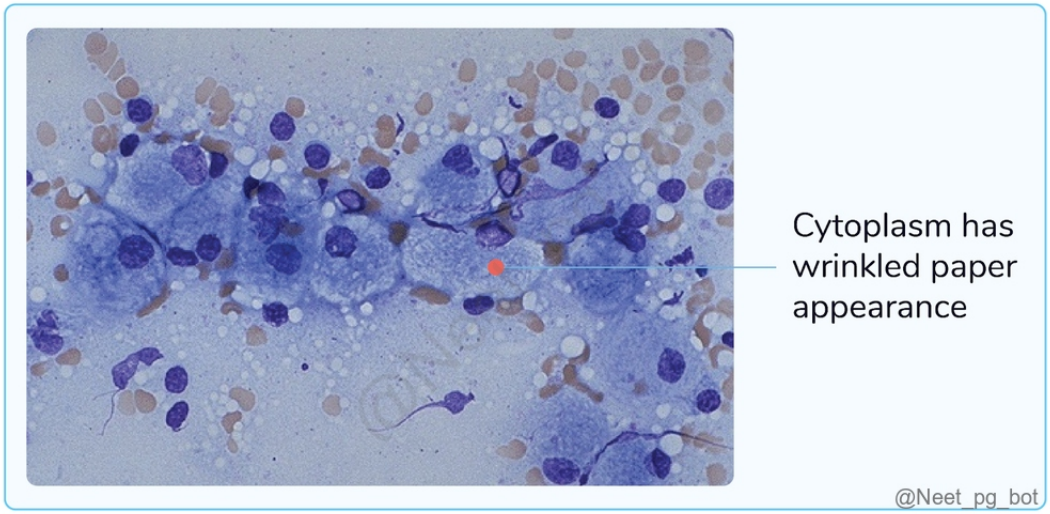
C. Hunter disease

D. Hurler disease



Correct Answer: A. Gaucher disease

Explanation: The given bone marrow image shows Gaucher cells, which have a large macrophage appearance with wrinkled or “crumpled tissue paper” cytoplasm. These cells result from accumulation of glucocerebroside due to β-glucocerebrosidase deficiency. Such cells are pathognomonic for Gaucher disease. Clinically, the child shows hepatosplenomegaly and developmental delay, fitting the disease profile. Bone involvement, anemia, and thrombocytopenia are also common. The combination of clinical features and the characteristic marrow cells strongly supports Gaucher disease. Hence, the diagnosis is Gaucher disease. {{caption_text}} Niemann-Pick disease (Option B): Foamy macrophages with lipid droplets (“soap-bubble” appearance), cherry-red macula. Hunter disease (Option C): Mucopolysaccharidosis with aggressive behavior; no corneal clouding. Hurler disease (Option D): Coarse facies, corneal clouding, but does not show Gaucher cells.



References:

pmc.ncbi.nlm.nih.gov/articles/PMC4017182/

↑ *Back to this question in Part A*

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Q7: A newborn infant is brought to the clinic with jaundice and a white, punctate, and scattered opacity in the central lens. The condition is consistent with Oil drop cataract. Which of the following metabolic disorders is most likely responsible for this presentation? (Q857861)

A. Phenylketonuria (PKU)

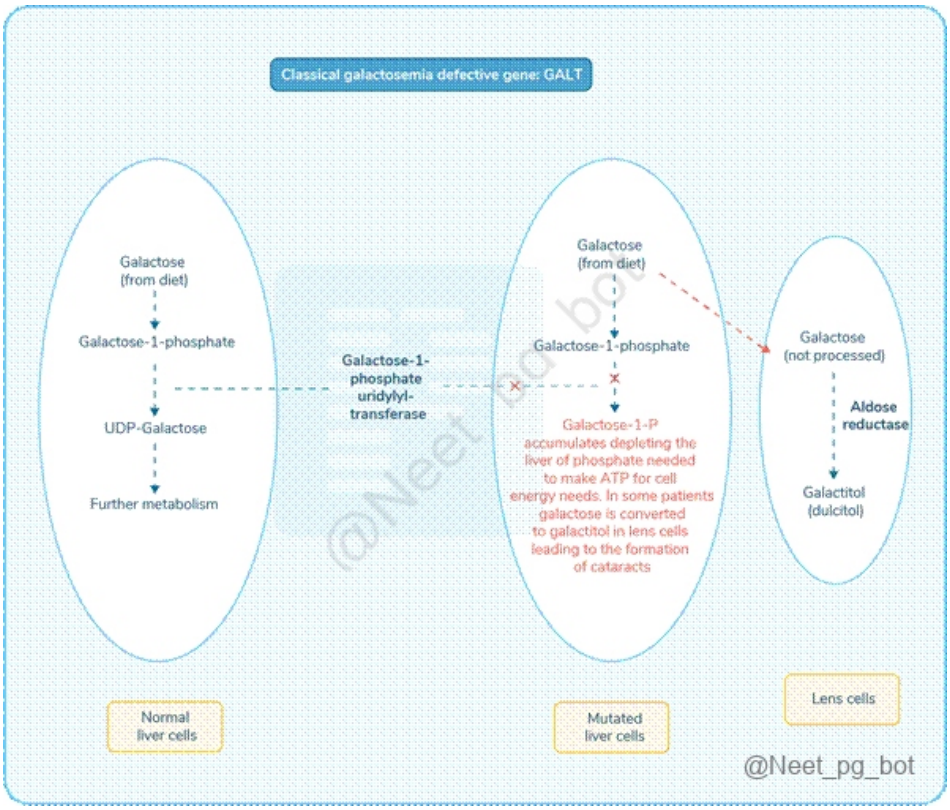
B. Maple syrup urine disease

C. Galactosemia

D. Wilson's disease

Correct Answer: C. Galactosemia

Explanation: The most likely metabolic disorder responsible for the presentation of oil drop cataract in a newborn infant with jaundice and lens opacities is Galactosemia. GALT deficiency, also known as galactosemia, is a genetic disorder that impairs the ability to metabolize galactose properly. Galactose is a sugar found in milk and other dairy products. Galactose-1-phosphate Uridyltransferase (GALT) Deficiency Severely deficient in individuals with Classic Galactosemia in which galactose-1-phosphate and, therefore, galactose accumulate. (Option A) It Is an inborn error of metabolism Accumulation Accumulated galactose is shunted into side pathways like galactitol production. Aldose reductase catalyzes it. Galactose-1 phosphate accumulates in the liver, inhibiting galactokinase and glycogen phosphorylase, leading to hypoglycemia. Bilirubin Reduced bilirubin uptake and conjugation, increasing unconjugated bilirubin in blood Symptoms Hypoglycemia, Hypophosphatemia, Hyperuricemia, Lactic acidosis Hepatomegaly, Jaundice, Severe mental retardation Galactosuria (galactose excreted in urine) Oil drop Cataracts from dulcitol accumulation in the lens, reversible cataract Tubular damage and generalized aminoaciduria Diagnosis Clinical manifestations (e.g., congenital cataract), presence of galactose in urine and elevated blood galactose levels Prenatal diagnosis via amniocentesis Detection of heterozygous parents by elevated galactose levels after a galactose load Complications in untreated cases E. coli sepsis, Fanconi syndrome Feeding problems, Failure to thrive, Hepatocellular damage, Bleeding Treatment Withdrawal of galactose and lactose from the diet. GALT deficiency is a part of the newborn screening panel Phenylketonuria (PKU) (Option A) is a metabolic disorder caused by a deficiency of the enzyme phenylalanine hydroxylase. This leads to the buildup of phenylalanine in the body. It can lead to intellectual disability, developmental delays, and other neurological problems if left untreated. Maple syrup urine disease (Option B) is a metabolic disorder that affects the breakdown of branched-chain amino acids (leucine, isoleucine, and valine). It can cause neurological symptoms and a characteristic sweet odor to the urine. Wilson's disease (Option D) is a disorder of copper metabolism, leading to copper accumulation primarily in the liver and brain. It can cause various neurological and hepatic symptoms.



References:

Lippincott Edition 7 page no:430, Chapter 12. II. FRUCTOSE METABOLISM

Vasudevan Edition 7 page no:139

↑ *Back to this question in Part A*

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Q8: A 2-month-old infant is brought to the pediatrician due to feeding difficulties, vomiting, and failure to thrive. The child's history reveals that the infant was born full-term and had a normal birth weight. On physical examination, the infant appears jaundiced, and the liver is palpable below the costal margin. Oil drop Cataracts are also noted on eye examination. Laboratory tests show elevated levels of galactose in the blood and urine. Based on these clinical findings, which enzyme deficiency is most likely responsible for this presentation? (Q857857)

A. G6PD (Glucose-6-phosphate dehydrogenase)

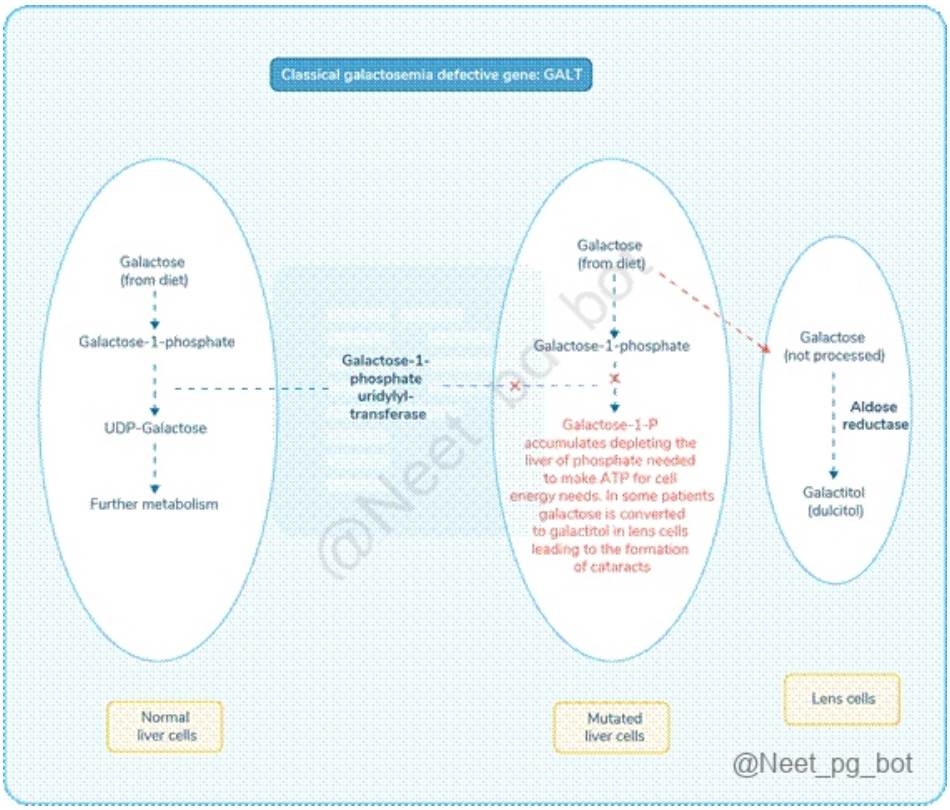
B. GALT (Galactose-1-phosphate uridylyltransferase)

C. ALDH (Aldehyde dehydrogenase)

D. PKU (Phenylalanine hydroxylase)

Correct Answer: B. GALT (Galactose-1-phosphate uridylyltransferase)

Explanation: GALT deficiency, also known as galactosemia, is a genetic disorder that impairs the ability to metabolize galactose properly. Galactose is a sugar found in milk and other dairy products. Galactose-1-phosphate Uridyltransferase (GALT) Deficiency Severely deficient in individuals with Classic Galactosemia in which galactose-1-phosphate and, therefore, galactose accumulate. (Option A) It Is an inborn error of metabolism Accumulation Accumulated galactose is shunted into side pathways like galactitol production. Aldose reductase catalyzes it. Galactose-1 phosphate accumulates in the liver, inhibiting galactokinase and glycogen phosphorylase, leading to hypoglycemia. Bilirubin Reduced bilirubin uptake and conjugation, increasing unconjugated bilirubin in blood Symptoms Hypoglycemia, Hypophosphatemia, Hyperuricemia, Lactic acidosis Hepatomegaly, Jaundice, Severe mental retardation Galactosuria (galactose excreted in urine) Oil drop Cataracts from dulcitol accumulation in the lens Tubular damage and generalized aminoaciduria Diagnosis Clinical manifestations (e.g., congenital cataract), presence of galactose in urine and elevated blood galactose levels Prenatal diagnosis via amniocentesis Detection of heterozygous parents by elevated galactose levels after a galactose load Complications in untreated cases E. coli sepsis, Fanconi syndrome Feeding problems, Failure to thrive, Hepatocellular damage, Bleeding Treatment Withdrawal of galactose and lactose from the diet. GALT deficiency is a part of the newborn screening panel G6PD (Glucose-6-phosphate dehydrogenase) deficiency (Option A) is a genetic disorder that leads to a deficiency of the enzyme glucose-6-phosphate dehydrogenase, which is important for protecting red blood cells from oxidative damage. ALDH (Aldehyde dehydrogenase) deficiency (Option C) is associated with a condition called alcohol-induced flushing, where individuals who carry certain genetic variants of the aldehyde dehydrogenase enzyme experience facial flushing and other symptoms when they consume alcohol. PKU (Phenylalanine hydroxylase) deficiency (Option D) is a genetic disorder characterized by the deficiency of the enzyme phenylalanine hydroxylase, which is responsible for converting phenylalanine to tyrosine.



References:

Lippincott Edition 7 page no:430, Chapter 12. II. FRUCTOSE METABOLISM

Vasudevan Edition 7 page no:139

↑ *Back to this question in Part A*

@Neet_pg_bot

Q9: A child presents with developmental delay, bone pain, and inability to walk. On fundoscopy, a cherry-red spot is observed. There is no organomegaly. What is the most likely diagnosis? (Q855287)

A. Tay-Sachs disease

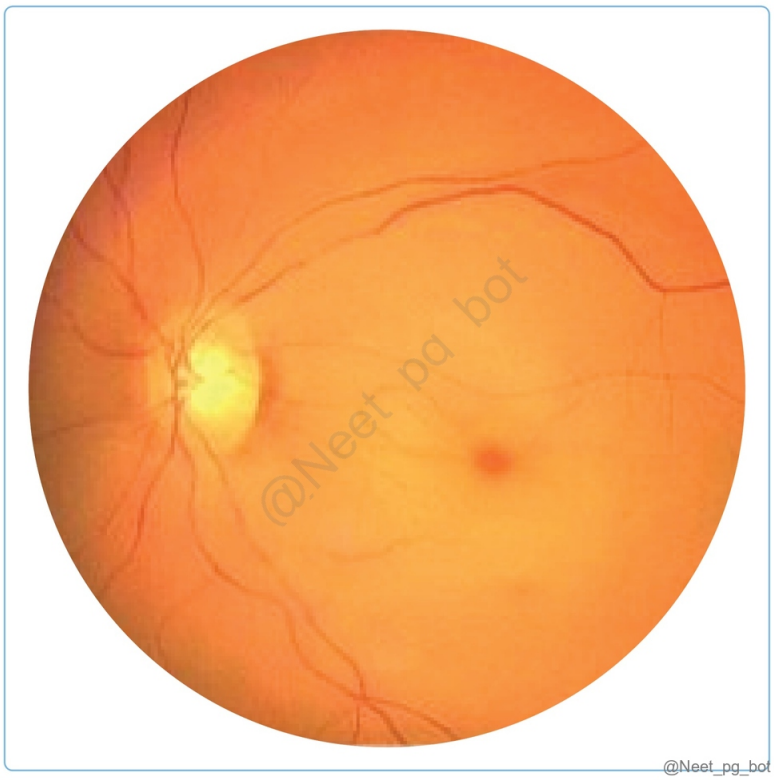
B. Niemann-Pick disease

C. Hurler syndrome

D. Gaucher disease

Correct Answer: A. Tay-Sachs disease

Explanation: Tay–Sachs disease is a GM■ gangliosidosis caused by hexosaminidase A deficiency , leading to accumulation of GM■ gangliosides in neurons. Key features include: Neurodegeneration , developmental delay , and hypotonia , Cherry-red spot on macula (due to retinal ganglion cell storage material surrounding normal fovea) No hepatosplenomegaly , distinguishing it from Niemann–Pick disease. {{caption_text}} Niemann–Pick disease (Option B): Also shows a cherry-red spot, but has massive hepatosplenomegaly due to sphingomyelinase deficiency. Hurler syndrome (Option C): A mucopolysaccharidosis presenting with coarse facial features, corneal clouding, and hepatosplenomegaly, not a cherry-red spot. Gaucher disease (Option D): Due to β-glucocerebrosidase deficiency, causing hepatosplenomegaly and bone pain, but no cherry-red spot. Disease Enzyme Deficiency Tay-Sachs disease Hexosaminidase A, S Fabry disease α-Galactosidase Metachromatic leukodystrophy Arylsulfatase A Krabbe disease β-Galactosidase Gaucher disease β-Glucosidase Niemann-Pick disease types A, B Sphingomyelinase Farber disease Ceramidase



References:

Harper’s illustrated Biochemistry 32e pg 245

https://www.ncbi.nlm.nih.gov/books/NBK564432/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q10: What are the main substrate and metabolic fuel used by RBCs during fasting? (Q855902)

A. Ketone bodies

B. Glucose

C. Free fatty acids

D. Amino acids

Correct Answer: B. Glucose

Explanation: RBCs cannot use fatty acid or ketone bodies as fuel during fasting as they do not have mitochondria where beta-oxidation of fatty acid occurs. Since ketone bodies and free fatty acids are utilized via beta-oxidation which occurs in mitochondria, RBCs cannot utilize them. RBC uses glucose as a substrate for energy production in the fed, fasting, and starvation state. Fuel in Fed, Fasting, and Starvation: Fed state Fasting Starvation Brain Glucose Glucose Ketone body Heart Fatty Acid (FA) FA Ketone body Liver Glucose FA Amino acid Muscle Glucose FA FA and ketone body Adipose tissue Glucose FA FA RBC Glucose Glucose Glucose Main Fuel for Body Glucose Fat Amino acids Ketone body (Option A) are used as a substrate for energy production during starvation phase by vital organs brain and heart primarily. Muscles use FA and ketone bodies for energy during starvation to avoid using proteins as fuel to prevent muscle loss. Amino acid (Option C) is used by liver as a fuel in starvation state. It is the major source of fuel for the body in a starvation state. Free fatty acid (Option D) is used as a fuel in fasting state by Heart (uses fatty acid in both fed and fasting state) Liver Muscle Adipose tissue (uses fatty acid in both fasting and starvation state)

References:

Vasudevan, 7th Edition, Page 101

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: Which of the following is seen in low insulin:glucagon ratio? (Q855240)

A. Activation of lipoprotein lipase

B. Activation of glycogen synthase

C. Activation of phosphofructokinase

D. Activation of hormone-sensitive lipase

Correct Answer: D. Activation of hormone-sensitive lipase

Explanation: Hormone-sensitive lipase (HSL) is activated in response to low insulin:glucagon ratio. HSL breaks down stored triglycerides in adipose tissue into free fatty acids and glycerol, providing energy during fasting. Insulin:Glucagon Bipolar Axis : Insulin : Anabolic hormone promoting glycogenesis, lipogenesis, and protein

synthesis. Glucagon : Catabolic hormone stimulating glycogenolysis, gluconeogenesis, and lipolysis. IGR : High IGR → Anabolic state, reduced glucose production. Low IGR → Catabolic state, increased glucose production. Determinants of IGR : Influenced by insulin and glucagon secretion patterns, dietary composition, and metabolic status. Variability in systemic and portal concentrations complicates measurement accuracy. Activation of lipoprotein lipase (Option A): Lipoprotein lipase is activated by insulin, not glucagon. It facilitates triglyceride uptake from circulating lipoproteins into adipose tissue for storage, which occurs in the fed state. Activation of glycogen synthase (Option B): Glycogen synthase is activated by insulin, promoting glycogen synthesis in the liver and muscle during the fed state. Activation of phosphofructokinase (Option C): Phosphofructokinase (PFK) is a key enzyme in glycolysis. It is activated by insulin and suppressed during fasting (low insulin:glucagon ratio), as glycolysis is reduced in favor of gluconeogenesis.

References:

The Insulin:Glucagon Ratio and the Choice of Glucose-Lowering Drugs - PMC

Vasudevan Textbook of Biochemistry (7th Edition), Page 101, 102

↑ *Back to this question in Part A*

@Neet_pg_bot

Q12: A 4-year-old child presents with easy fatiguability. The mother also complains that the child has increased hunger between meals, which is relieved after food. Liver examination revealed no glycogen. The enzyme most likely deficient is (Q855224)

A. Glycogen phosphorylase

B. Glycogen synthase

C. Debranching enzyme

D. Glucose 6 phosphatase

Correct Answer: B. Glycogen synthase

Explanation: The child has no hepatic glycogen , meaning the liver cannot store glucose. This points to a defect in glycogen synthesis , specifically glycogen synthase deficiency (GSD type 0) . These children develop fasting hypoglycemia with ketones and become hungry between meals, improving after eating. Glycogen storage disease type 0 (0a and 0b): Enzyme Deficiency: Glycogen synthase Affected Gene: GYS2 and GYS1 Clinical Features: Hypoglycemia, Hyperketonemia, early death Mode of Inheritance: Autosomal recessive Management: Supportive care: Frequent protein-rich meals and avoidance of fasting. Cornstarch may be given nighttime Glycogen Storage Disease Enzyme Deficiency Type 0 Glycogen synthase deficiency Type I (von Gierke's Disease) Glucose-6-phosphatase (Option D ruled out) Type II (Pompe's Disease) Acid α-glucosidase (acid maltase) Type III (Cori's Disease) Debranching enzyme (amylo-1,6-glucosidase) (Option C ruled out) Type IV (Andersen's Disease) Branching enzyme Type V (McArdle's Disease) Muscle phosphorylase (myophosphorylase) (Option A ruled out) Type VI (Hers' Disease) Liver phosphorylase Type VII (Tarui's Disease) Phosphofructokinase (muscle

type)

References:

Harper’s Biochemistry 32e pg: 174

Vasudevan Textbook of Biochemistry (7th Edition), Page 129.

Management glycogen storage disease 0

↑ *Back to this question in Part A*

@Neet_pg_bot

Q13: A young boy presents with muscle cramps and dark-colored urine after strenuous exercise. He reports that his symptoms improve after brief rest. Laboratory tests show normal blood glucose levels, no rise in serum lactate after exercise, and elevated creatine kinase. Which of the following is the most likely diagnosis? (Q855193)

A. McArdle disease

B. Hers disease

C. Cori's disease

D. Andersen disease

Correct Answer: A. McArdle disease

Explanation: McArdle disease (Glycogen Storage Disease Type V) results from muscle glycogen phosphorylase deficiency , leading to an inability to break down glycogen in skeletal muscle . Characteristic features include: Exercise intolerance and cramps , Myoglobinuria (dark urine) after exertion , “Second wind” phenomenon , No rise in blood lactate after exercise , Normal liver function and blood glucose . Hers disease (Type VI) (Option B): Affects liver → hepatomegaly and fasting hypoglycemia. Cori disease (Type III) (Option C): Affects both liver and muscle → hypoglycemia, hepatomegaly, mild myopathy. Andersen disease (Type IV) (Option D): Affects liver → progressive cirrhosis and liver failure, not exercise-related. Glycogen Storage Disease Enzyme Deficiency Type 0 Glycogen synthase deficiency Type I (von Gierke's Disease) Glucose-6-phosphatase Type II (Pompe's Disease) Acid α-glucosidase (acid maltase) Type III (Cori's Disease) Debranching enzyme (amylo-1,6-glucosidase) Type IV (Andersen's Disease) Branching enzyme Type V (McArdle's Disease) Muscle phosphorylase (myophosphorylase) Type VI (Hers' Disease) Liver phosphorylase Type VII (Tarui's Disease) Phosphofructokinase (muscle type)

References:

Harper’s Illustrated Biochemistry (23rd Edition), Page 174.

Vasudevan Textbook of Biochemistry (7th Edition), Page 128,129.

Glycogen storage disease type III - NIH Genetic Testing Registry (GTR) - NCBI

Anderson-Fabry disease management:

Anderson's Disease

↑ *Back to this question in Part A*

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Q14: Glycogen phosphorylase is activated by? (Q854622)

A. Glucose 6 phosphatase

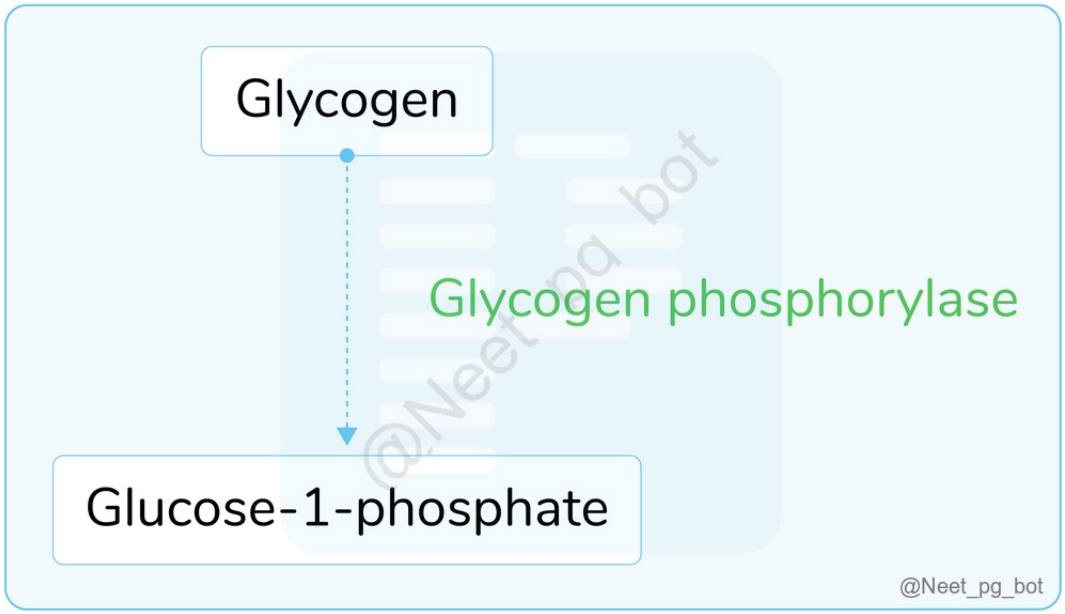
B. ATP

C. Calcium

D. Glucose

Correct Answer: C. Calcium

Explanation: Rate limiting step in glycogenolysis: Glycogen phosphorylase: Phosphorolytic cleavage of the 1→4 linkages of glycogen Different isoenzymes exist in liver, muscle, and brain, encoded by different genes Requires pyridoxal phosphate as coenzyme Activators: cAMP Glucagon - only in liver Epinephrine Calcium 5'AMP Inhibitors: Protein Phosphatase 1 (PP1) Insulin Glucose - only in liver Glucose 6 phosphate ATP Fructose 1 phosphate - only in liver



References:

Harper 32nd edition, page 173

↑ *Back to this question in Part A*

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Q15: Which of the following organs or tissues demonstrate insulin-dependent glucose uptake ? (Q854604)

A. RBC

B. Pancreas

C. Adipose tissue

D. Hepatocytes

Correct Answer: C. Adipose tissue

Explanation: Adipose tissue has GLUT 4 transporter which mediates insulin-stimulated glucose uptake. GLUT proteins are distributed in various tissues according to their specific functions. Glucose Transporters: Transporter Tissue Location Functions GLUT 1 Brain, kidney, colon, placenta, erythrocytes (Option A ruled out) Facilitates glucose uptake GLUT 2 Liver, pancreatic β cells, small intestine, kidney (Option B, D ruled out) Enables rapid glucose uptake or release GLUT 3 Brain, kidney, placenta Assists in glucose uptake GLUT 4 Heart and skeletal muscle, adipose tissue Mediates insulin-stimulated glucose uptake GLUT 5 Small intestine Facilitates fructose absorption SGLT 1 Small intestine and kidney Actively transports glucose against a concentration gradient GLUT 1-5 are facilitative bidirectional transporters, whereas SGLT 1 is a sodium-dependent unidirectional transporter.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 186,187.

↑ *Back to this question in Part A*

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Q16: A newborn infant is brought to the clinic with jaundice and a white, punctate, and scattered opacity in the central lens. The condition is consistent with Oil drop cataract. Which of the following metabolic disorders is most likely responsible for this presentation? (Q820353)

A. Phenylketonuria (PKU)

B. Maple syrup urine disease

C. Galactosemia

D. Wilson's disease

Correct Answer: C. Galactosemia

Explanation: Oil-drop cataract is a classical ocular finding in galactosemia. The disorder is caused by deficiency of galactose-1-phosphate uridyltransferase (GALT). This leads to accumulation of galactitol in the lens. Galactitol causes osmotic damage to lens fibers, producing cataracts. Affected newborns present with jaundice, vomiting, hepatomegaly, and failure to thrive. Early dietary restriction of galactose/lactose can prevent progression. Incorrect Options: Option A - Phenylketonuria (PKU) : Phenylketonuria is a metabolic disorder caused by a deficiency of the enzyme phenylalanine hydroxylase. This leads to the buildup of phenylalanine in the body. However, PKU is not associated with oil drop cataract; instead, it can lead to intellectual disability, developmental delays, and other neurological problems if left untreated. Option B - Maple syrup urine disease: This is a metabolic disorder that affects the breakdown of branched-chain amino acids (leucine, isoleucine, and valine). It can cause neurological symptoms and a characteristic sweet odor to the urine. It is not associated with oil drop cataract. Option D - Wilson's disease: Wilson's disease is a disorder of copper metabolism, leading to copper accumulation primarily in the liver and brain. It can cause various neurological and hepatic symptoms. It is not associated with oil drop cataract.

References:

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↑ *Back to this question in Part A*

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Q17: A patient with a history of strenuous exercise forgot to eat a meal and later consumed alcohol at a party. Which of the following metabolic processes is inhibited by alcohol consumption? (Q860060)

A. Gluconeogenesis

B. Glycogenolysis

C. HMP shunt

D. Ketone body formation

Correct Answer: A. Gluconeogenesis

Explanation: Alcohol consumption inhibits gluconeogenesis. Alcohol metabolism increases the NADH/NAD+ ratio in the liver, which inhibits key enzymes involved in gluconeogenesis, leading to hypoglycemia, especially in a fasting state after strenuous exercise. Alcohol produces a high NADH/NAD+ ratio in the cytoplasm, the high NADH level favours the conversion of pyruvate to lactate, leading to lactic acidosis. Deficiency of pyruvate leads to inadequate formation of oxaloacetate which blocks gluconeogenesis This results in low blood sugar levels. Symptoms of alcohol-induced hypoglycemia include confusion, irritability, and, in severe cases, loss of consciousness. Glycogenolysis (Option B): Glycogenolysis is generally stimulated by alcohol consumption due to the liver's attempt to maintain glucose levels. While alcohol inhibits gluconeogenesis, it does not directly inhibit glycogenolysis. HMP shunt (Option C): The hexose monophosphate (HMP) shunt is primarily involved in the production of NADPH and ribose-5-phosphate. Alcohol does not directly inhibit this pathway. Ketone body formation (Option D): Ketone body formation can actually be promoted in alcohol consumption as the body shifts to using fatty acids for energy when glucose is unavailable.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg.542

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg.140, 478

https://pubmed.ncbi.nlm.nih.gov/12444383/

↑ *Back to this question in Part A*

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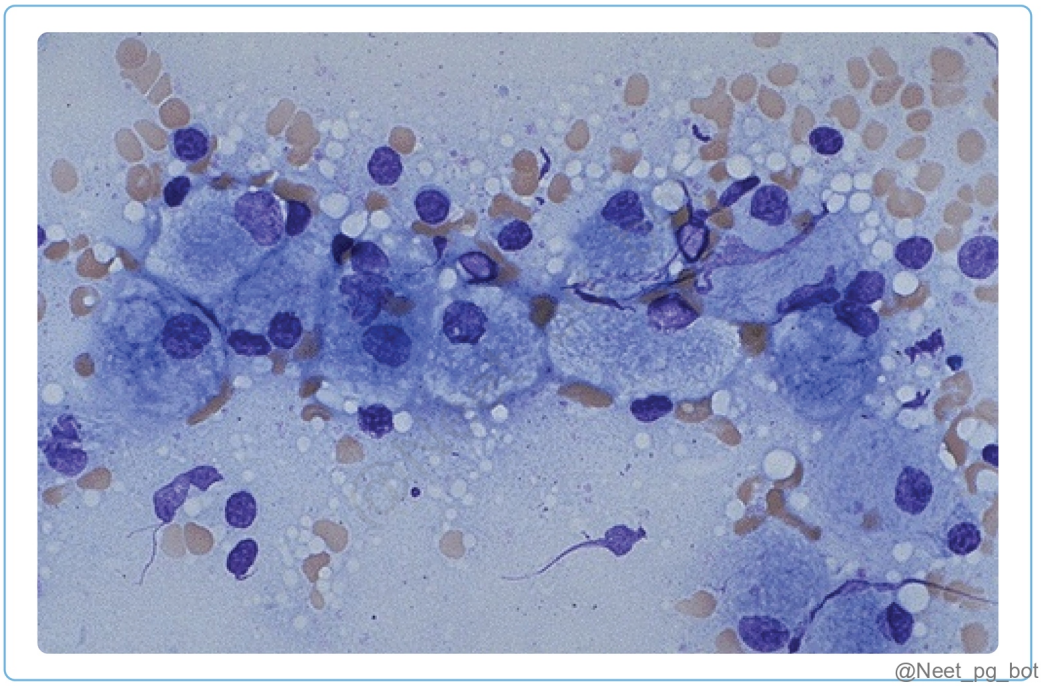
Q18: A 5 year old boy presented with easy fatigability, irritability and inability to concentrate. Labs revealed the following findings a and b (light and oil immersion respectively) on the bone marrow aspiration. Which of the following is the most likely enzyme deficient in this condition? (Q856005)

A. Hexosaminidase

B. Glucocerebrosidase

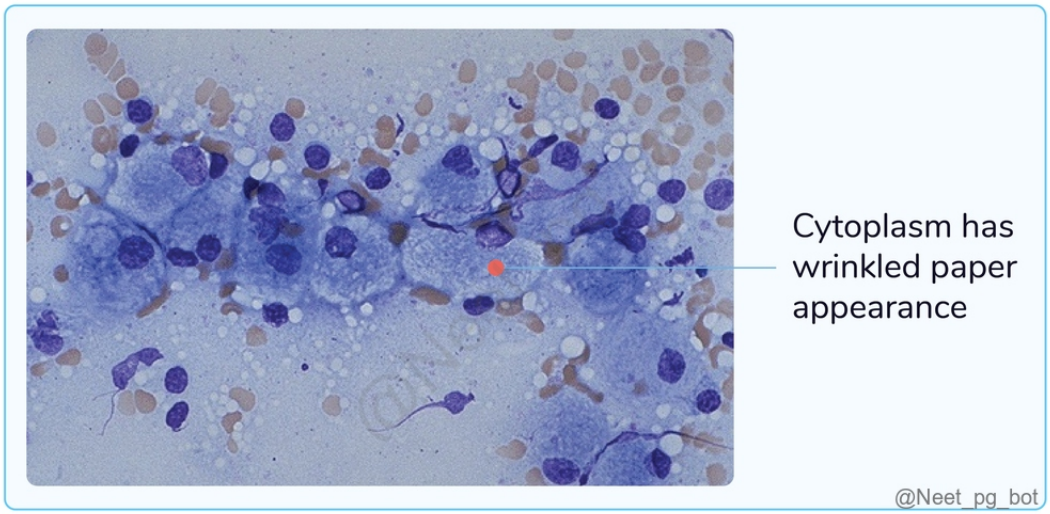
C. Sphingomyelinase

D. N-acetyglucosaminidase



Correct Answer: B. Glucocerebrosidase

Explanation: The Clinical scenario of easy fatiguability, irritability and inability to concentrate along with the microscopic findings of crumpled tissue appearance of macrophages. Gaucher's disease is characterized by the accumulation of glucosylceramide in RBCs and platelet membranes due to a deficiency in the enzyme Glucocerebrosidase , leading to anemia, hepatosplenomegaly, and thrombocytopenia. Bone marrow biopsy shows macrophages with a characteristic "wrinkled paper" appearance. Gaucher's disease: Lysosomal storage disease Defective enzyme: Glucocerebrosidase Defect in metabolism: Accumulation of cerebroside (Glucosylceramide) in RBCs and platelet membranes. (Option B) Pathophysiology: Abnormal RBCs and platelets are engulfed by macrophages leading to accumulation inside macrophages causing wrinkled paper appearance. {{caption_text}} Disease Enzyme deficiency Substance accumulation Features Tay-Sachs disease Hexosaminidase A (Option A ruled out) Ganglioside Mental retardation Blindness Muscular weakness Cherry red spots in the macula Niemann-Pick disease Sphingomyelinase (Option C ruled out) Sphingomyelin Enlarged liver and spleen Mental retardation Fatal in early life Sanfilippo Syndrome N-acetyglucosaminidase (Option D ruled out)



References:

https://www.ncbi.nlm.nih.gov/books/NBK448080/

Harper's illustrated Biochemistry 32e pg 245,

Vasudevan textbook of Biochemistry 7e pg 194

Image: https://www.tau.ac.il/medicine/tau-only/webpath/hemehtml/heme057.htm

↑ *Back to this question in Part A*

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Q19: A A 6-year-old child presents with recurrent abdominal pain. Examination reveals hepatomegaly, short stature and a doll-like facies. The laboratory studies showed anemia, acidosis and a deficiency of glucose-6-phosphatase. Which of the following features is most likely to be seen in this patient? (Q879291)

A. Fasting hypoglycemia

B. Fasting hypoketosis

C. Decreased hepatocellular cytoplasmic accumulation of glycogen

D. Muscle cramps

Correct Answer: A. Fasting hypoglycemia

Explanation: The presentation is classic for Glycogen Storage Disease Type I (Von Gierke disease) due to glucose-6-phosphatase deficiency . Because the liver cannot convert glucose-6-phosphate → glucose , patients are

unable to maintain blood glucose levels during fasting. Hallmark features: Severe fasting hypoglycemia Hepatomegaly (massive accumulation of glycogen + fat) Lactic acidosis Hyperuricemia, hyperlipidemia Doll-like facies, short stature Thus, fasting hypoglycemia is the most characteristic and expected finding. Incorrect Options: Fasting hypoketosis (Option B): Mild hypoketosis can occur, but it is not the hallmark or most likely feature. Increased malonyl-CoA inhibits FA oxidation → ↓ ketone bodies. Decreased hepatocellular cytoplasmic accumulation of glycogen (Option C): Incorrect. Von Gierke shows increased glycogen deposition. Decreased hepatic glycogen is seen in GSD Type 0 (glycogen synthase deficiency) Muscle cramps (Option D): Seen in McArdle disease (GSD Type V) —a defect in muscle glycogen phosphorylase causing exercise intolerance, cramps, and myoglobinuria.

References:

Textbook of Biochemistry - DM Vasudevan, 7th Edition, Page 569 Harper Biochemistry, 32nd Edition, Page 345 Glycogen Storage Disease Type I - StatPearls - NCBI Bookshelf

↑ *Back to this question in Part A*

@Neet_pg_bot

Q20: A 5-year-old girl was washing her doll with shampoo containing rotenone. Her mother noticed her in unconscious state. Which is inhibited by the above chemical? (Q844251)

A. NADH dehydrogenase

B. Succinate dehydrogenase

C. Cytochrome C

D. Cytochrome oxidase

Correct Answer: A. NADH dehydrogenase

Explanation: Rotenone is a potent insecticide and fish poison that rapidly penetrates tissues. It inhibits Complex I (NADH dehydrogenase) of the electron transport chain. By blocking electron transfer from iron–sulfur centers to ubiquinone (CoQ), it halts NADH → CoQ electron flow. This causes marked ATP depletion due to impaired oxidative phosphorylation. More importantly, electron backup at Complex I generates excess ROS, leading to oxidative stress. ROS induce lipid peroxidation, mitochondrial injury, and apoptosis. The combination of energy failure + oxidative damage results in unconsciousness and severe toxicity. Incorrect Options: Options B, C, and D are not inhibited by rotenone.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q21: Which of the following doesn't inhibit Cytochrome? (Q853002)

A. Nitric oxide

B. CO

C. H2S

D. Cyanide

Correct Answer: A. Nitric oxide

Explanation: Electron transport chain Complex IV contains cytochrome a and cytochrome a3 Inhibitors: Carbon monoxide Cyanide Azide Hydrogen sulfide

References:

Vasudevan’s Textbook of Biochemistry for Medical Students, 7th edition, Page 226

↑ *Back to this question in Part A*

@Neet_pg_bot

Q22: Which of the following substances inhibits cytochrome complex IV, a component of the electron transport chain responsible for ATP synthesis? (Q855551)

A. Cyanide

B. Carbon dioxide

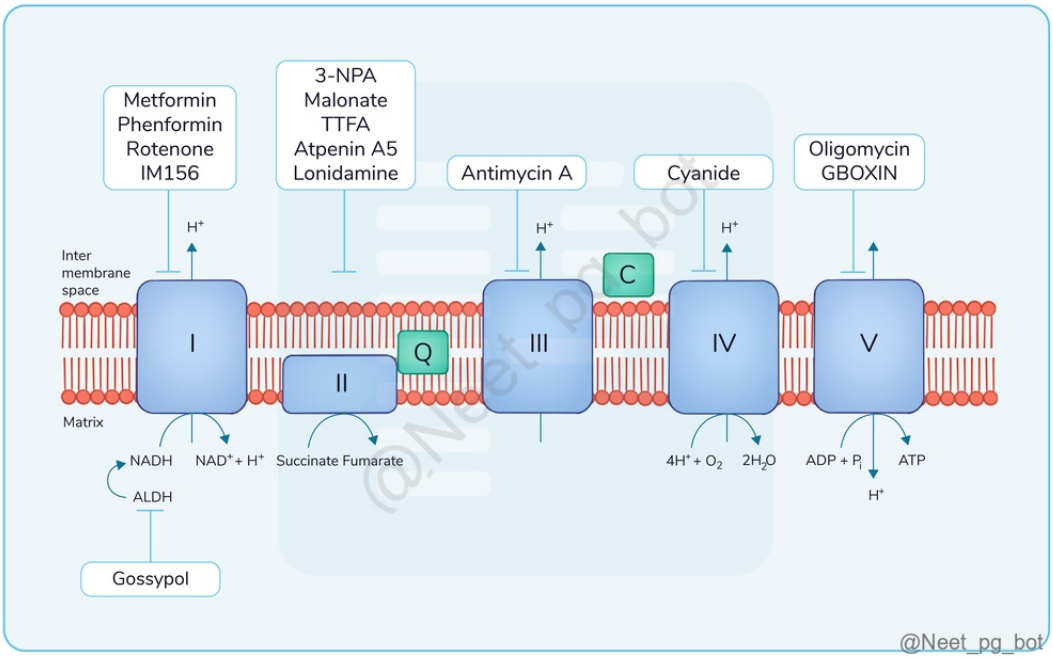
C. Oligomycin

D. Ouabain

Correct Answer: A. Cyanide

Explanation: Cyanide: Powerful inhibitor of cytochrome complex IV is cyanide. It attaches to the enzyme's active site and stops the enzyme from transferring electrons to oxygen. Due to the blockage of the electron transport chain, reducing agents like NADH and FADH2 begin to accumulate. The last enzyme complex in the electron transport chain is cytochrome complex IV, also known as cytochrome c oxidase. When oxygen and protons are combined to form water, it receives electrons from cytochrome c. The ATP synthase uses the proton gradient created by this mechanism to manufacture ATP. inhibitor of Complex IV" data-author="" data-hash="11566"

data-license="" data-source="" data-tags="May2025" src="" /> Carbon dioxide (Option B): Does not inhibit cytochrome complex IV.Bicarbonate ions, which are the predominant type of carbon dioxide carried in the blood, do not directly interact with the electron transport chain. Oligomycin (Option C): Cytochrome complex IV is not inhibited by oligomycin.It is an ATP synthase inhibitor that attaches to the Fo subunit of ATP synthase and stops protons from passing through the enzyme, hence preventing the production of ATP. Ouabain (Option D): Na/K ATPase pump, which keeps the electrochemical gradient across the plasma membrane of cell is inhibited by cardiac glycoside.



References:

ETC inhibitors

↑ *Back to this question in Part A*

@Neet_pg_bot

Q23: Which enzyme deficiency could potentially be the cause of exercise intolerance and cramps in adolescent male patient? (Q855537)

A. Myophosphorylase

B. Hexokinase

C. Glucose-6-phosphatase

D. Hepatic glycogen phosphorylase

Correct Answer: A. Myophosphorylase

Explanation: McArdle disease, or myophosphorylase deficiency, affects glycogen utilization for energy. Type 5 Glycogen storage disorder. Myophosphorylase converts glycogen to glucose-1-phosphate in muscle tissue . Insufficient enzyme activity leads to exercise intolerance due impaired glycogen breakdown. Symptoms include muscle cramps, stiffness, and weakness during exertion, alleviated Hexokinase (Option B): Catalyzes glucose to glucose-6-phosphate conversion in glycolysis.Insufficiency affects glucose uptake, leading to exhaustion and weakness, not exercise intolerance or cramps. Glucose-6-phosphatase (Option C): Converts glucose-6-phosphate to glucose in gluconeogenesis.Deficiency causes hypoglycemia, not exercise intolerance. Hepatic glycogen phosphorylase (Option D): Breaks down liver glycogen. Deficiency affects liver, not causing exercise intolerance or cramps.

References:

McArdle disease

↑ *Back to this question in Part A*

@Neet_pg_bot

Q24: Ketosis is common in type 1 DM due to: - Decreased beta-oxidation of fatty acids Increased mobilization of fat from the adipose cell Decreased acetyl CoA in TCA cycle Increase beta-hydroxy butyryl synthase (Q856515)

A. 2,3 and 4

B. 1,2,3 and 4

C. 4 only

D. 1 and 2 only

Correct Answer: A. 2,3 and 4

Explanation: Ketosis in Type 1 Diabetes Mellitus (T1DM) occurs due to the following mechanisms: Increased mobilization of fat from adipose tissue (statement 2): Lack of insulin leads to unregulated lipolysis, releasing free fatty acids (FFAs) into the bloodstream. Decreased acetyl-CoA utilization in the TCA cycle (statement 3): Due to the depletion of oxaloacetate (used for gluconeogenesis in a hyperglycemic state), acetyl-CoA from beta-oxidation cannot enter the TCA cycle efficiently and is diverted toward ketone body synthesis. Increased activity of beta-hydroxybutyryl-CoA synthase (statement 4): This enzyme catalyzes the synthesis of ketone bodies (e.g., beta-hydroxybutyrate), contributing to ketosis. Decreased beta-oxidation of fatty acids (Statement 1) because beta-oxidation is actually increased in T1DM due to elevated fatty acid mobilization and lack of insulin-mediated inhibition.

References:

DKA

↑ *Back to this question in Part A*

@Neet_pg_bot

Q25: Which enzyme is shared by both glycolysis and gluconeogenesis? (Q856507)

A. Pyruvate kinase

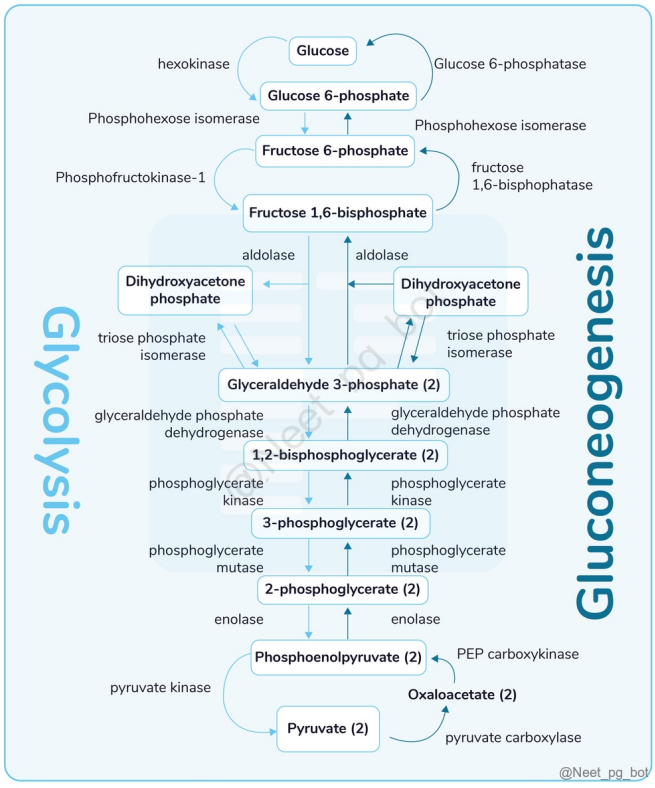
B. Phosphofructokinase

C. Phosphoenolpyruvate carboxy kinase

D. Phosphoglycerate kinase

Correct Answer: D. Phosphoglycerate kinase

Explanation: Phosphoglycerate kinase is involved in a reversible reaction in both glycolysis and gluconeogenesis: Glycolysis : Converts 1,3-bisphosphoglycerate to 3-phosphoglycerate, generating ATP. Gluconeogenesis : Reverses the above reaction, utilizing ATP to convert 3-phosphoglycerate to 1,3-bisphosphoglycerate. The other enzymes listed are specific to one pathway: Pyruvate kinase (Option A): Specific to glycolysis (irreversible). Phosphofructokinase (Option B): Specific to glycolysis (irreversible). Phosphoenolpyruvate carboxykinase (Option C): Specific to gluconeogenesis.



References:

PGK

↑ *Back to this question in Part A*

@Neet_pg_bot

Q26: Glucose is stored in the form of glycogen because (Q856876)

A. It is compact

B. It has multiple reducing ends

C. It can be stored at multiple organs

D. It can provide glucose for about 1 week when needed

Correct Answer: A. It is compact

Explanation: Glycogen is a polysaccharide composed of many glucose molecules linked together through alpha-1,4-glycosidic bonds and branched with alpha-1,6-glycosidic bonds. By storing glucose as glycogen, the body can efficiently pack a large amount of glucose molecules into a compact and readily available form. This compact storage allows for efficient storage of glucose within cells, especially in tissues such as the liver and muscles, where glycogen serves as a reserve energy source. It has multiple reducing ends (Option B): While it is true that glycogen has multiple reducing ends due to its branched structure, this is not the primary reason why glucose is stored as glycogen. It can be stored at multiple organs (Option C): Although glycogen can be stored in multiple organs, such as the liver and muscles, this is not the main reason why glucose is stored as glycogen. It can provide glucose for about 1 week when needed (Option D): The storage capacity of glycogen varies depending on the individual and their glycogen reserves.

References:

Glycogen

↑ *Back to this question in Part A*

@Neet_pg_bot

Q27: Which of the following deficiency is found in congenital lactic acidosis? (Q839919)

A. Transketolase

B. Branched chain ketoacid dehydrogenase

C. Isocitrate dehydrogenase

D. Pyruvate dehydrogenase

Correct Answer: D. Pyruvate dehydrogenase

Explanation: Pyruvate dehydrogenase: Pyruvate dehydrogenase (PDH) is a crucial enzyme that converts pyruvate into acetyl-CoA, linking glycolysis to the citric acid cycle. A deficiency in pyruvate dehydrogenase leads to congenital lactic acidosis. Without functional PDH, pyruvate accumulates and is shunted to lactate production, leading to elevated lactate levels in the blood. Transketolase (Option A): Transketolase is an enzyme of the pentose phosphate pathway, which helps in the production of NADPH and ribose-5-phosphate A deficiency in transketolase is not associated with congenital lactic acidosis. Branched chain Ketoacid dehydrogenase (Option B): This enzyme is involved in the catabolism of branched-chain amino acids (leucine, isoleucine, and valine). A deficiency in branched-chain ketoacid dehydrogenase leads to Maple Syrup Urine Disease (MSUD), not congenital lactic acidosis. MSUD is characterized by the accumulation of branched-chain amino acids and their corresponding keto acids. Isocitrate dehydrogenase (Option C): Isocitrate dehydrogenase is an enzyme in the citric acid cycle that helps in the conversion of isocitrate to α-ketoglutarate, producing NADPH or NADH in the process While important in the citric acid cycle, a deficiency in isocitrate dehydrogenase is not typically associated with congenital lactic acidosis

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

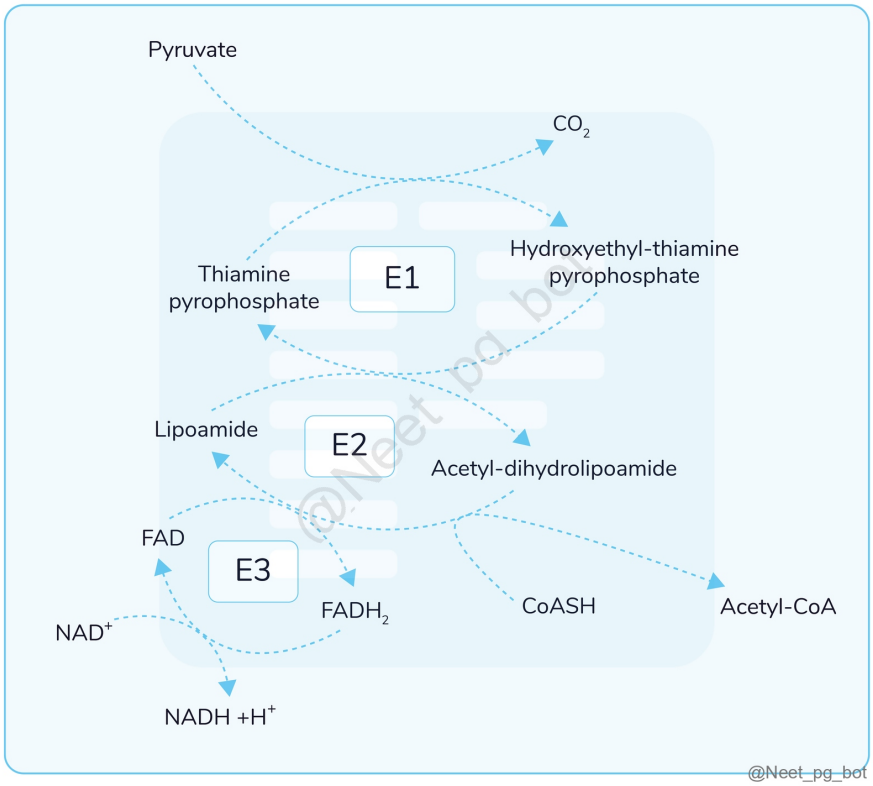
Q28: In the diagram provided, which enzymes are identified as E1 and E2, responsible for the conversion of pyruvate to acetyl CoA? (Q856000)

A. Pyruvate dehydrogenase and dihydrolipoyl transacetylase

B. Pyruvate carboxylase and dihydrolipoyl transacetylase

C. Pyruvate dehydrogenase and dihydrolipoyl dehydrogenase

D. Pyruvate carboxylase and dihydrolipoyl dehydrogenase



Correct Answer: A. Pyruvate dehydrogenase and dihydrolipoyl transacetylase

Explanation: The pyruvate dehydrogenase complex is responsible for the irreversible conversion of pyruvate to acetyl-CoA, a crucial step in aerobic glucose metabolism. A deficiency in this complex leads to the accumulation of pyruvate, which is then shunted towards lactate production, resulting in lactic acidosis. Enzyme Label Full Name Cofactor Function E1 Pyruvate dehydrogenase (decarboxylase) Thiamine pyrophosphate (TPP) Catalyzes oxidative decarboxylation of pyruvate to form hydroxyethyl–TPP. E2 Dihydrolipoyl transacetylase Lipoic acid Transfers the acetyl group from hydroxyethyl–TPP to CoA, forming acetyl-CoA . E3 Dihydrolipoyl dehydrogenase FAD and NAD■ Regenerates oxidized lipoamide by transferring electrons to NAD■ → NADH + H■.

References:

Harper's Illustrated Biochemistry, Ed 32, Pg 168-170.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q29: In this scenario, what is the origin of the blood glucose measured at 7 am the following morning for an individual who had dinner at 8 pm the previous night? (Q855985)

A. Hepatic glycogenolysis

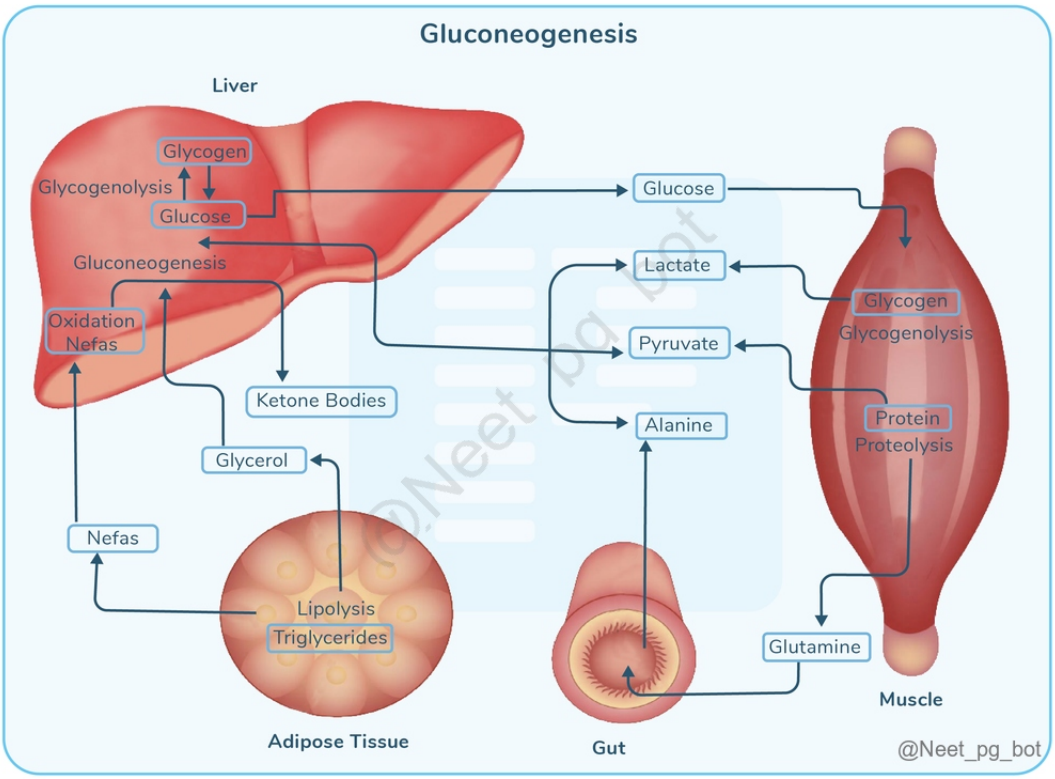
B. Hepatic gluconeogenesis

C. Muscle glycogenolysis

D. Dietary glucose

Correct Answer: A. Hepatic glycogenolysis

Explanation: During fasting: After 4-16 hrs, the primary source of glucose is glycogenolysis After 16-48 hrs, the primary source of glucose is gluconeogenesis (Option B) Gluconeogenesis: It is a process in which glucose is synthesized by non-carbohydrate sources such as amino acids, lactate, glycerol, and propionyl CoA. The major site of gluconeogenesis the liver > kidney. Regulation of gluconeogenesis: Glucagon activates fructose 1,6-bisphosphatase and inhibits PFK-1, favoring gluconeogenesis and inhibiting glycolysis The availability of substrates such as alanine, lactate, and glycerol favors gluconeogenesis Acetyl CoA is an allosteric activator of 1st regulatory step of gluconeogenesis (pyruvate carboxylase) Muscle glycogenolysis (Option C) is used during exercise and muscle activity. Dietary glucose (Option D) is used in the first 4 hrs after meals.



References:

Harper’s Illustrated Biochemistry, Ed 32, Pg 180-189

Lippincott Illustrated Reviews: Biochemistry 7e, pg no: 376-78

↑ *Back to this question in Part A*

@Neet_pg_bot

Q30: You are conducting an experiment on mitochondrial respiration. You add malate/pyruvate and respiration is normal. You add succinate and respiration is normal. When you add another substance in the presence of pyruvate/succinate and malate, respiration is blocked. Which of the following substances is most likely added? (Q855978)

A. Rotenone

B. Antimycin A

C. Oligomycin

D. 2,4-dinitrophenol

Correct Answer: B. Antimycin A

Explanation: In this experiment, respiration proceeds normally when malate/pyruvate (Complex I substrates) and succinate (Complex II substrate) are added. This means both Complex I and II are functioning properly. When respiration stops completely after adding another substance, it indicates the block occurs after Coenzyme Q , where both pathways converge — this points to Complex III inhibition. Antimycin A acts at Complex III (cytochrome bc■ complex) , blocking electron transfer from reduced Coenzyme Q to cytochrome c, which halts the entire electron transport chain and stops oxygen consumption. By contrast: Rotenone (Option A) inhibits Complex I , but respiration would still occur with succinate. Oligomycin (Option C) inhibits ATP synthase (Complex V) , preventing ATP synthesis but not respiration; electrons continue to flow. 2,4-Dinitrophenol (DNP) (Option D) uncouples oxidative phosphorylation, increasing oxygen consumption rather than blocking it.

References:

Harper’s Illustrated Biochemistry, 32nd edition, pg: 126

↑ *Back to this question in Part A*

@Neet_pg_bot

Q31: Which one of the options provided does not serve as a substrate for the synthesis of glucose? (Q856613)

A. Acetyl coenzyme A

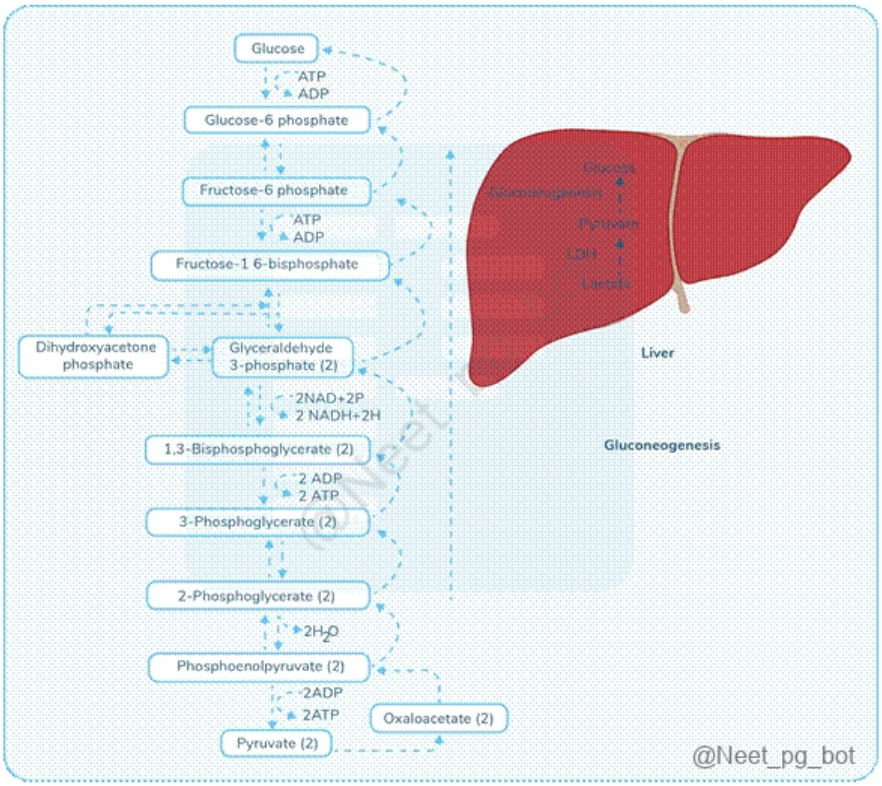
B. Glycerol

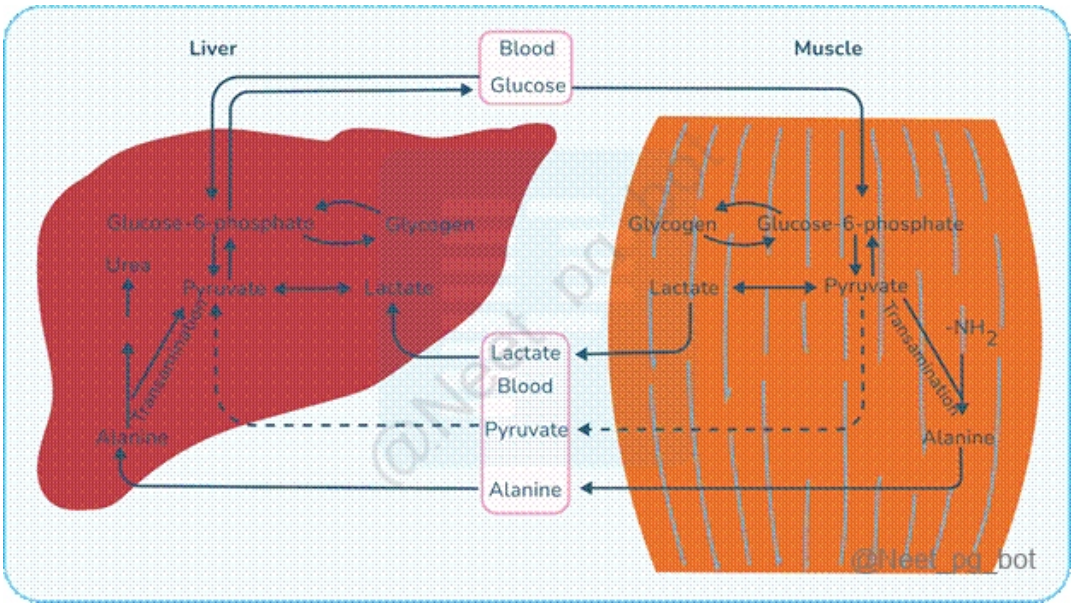
C. Alanine

D. Lactate

Correct Answer: A. Acetyl coenzyme A

Explanation: Acetyl-CoA is NOT a substrate for gluconeogenesis. Although acetyl-CoA is an important metabolic intermediate, it cannot be converted to glucose because the reaction catalysed by pyruvate dehydrogenase is irreversible. Substrates for gluconeogenesis: Lactate (Option D): Produced during anaerobic glycolysis in skeletal muscles and can be converted back to glucose in the liver through the Cori cycle. Glycerol (Option B) is released from the breakdown of triglycerides in adipose tissue and can enter the gluconeogenesis pathway. Alanine (Option C) and Glutamine: Released from the breakdown of proteins, primarily in muscle tissue. Alanine is converted to pyruvate, which enters the gluconeogenesis pathway as part of the glucose-alanine (Cahill) cycle. Propionate : Obtained from the metabolism of odd-chain fatty acids. Cori's cycle/ Glucose Lactate cycle and Cahill cycle/Glucose Alanine cycle





References:

Harper's Biochemistry, 32nd edition, Pg 142, 186, 187

↑ *Back to this question in Part A*

@Neet_pg_bot

Q32: Which of the following poisoning agent does not inhibit cytochrome oxidase? (Q856558)

A. Hydrogen sulfide

B. Carbon monoxide

C. Methane

D. Cyanide

Correct Answer: C. Methane

Explanation: Cytochrome c oxidase is complex 4 in the electron transport chain. It is inhibited by the following: Carbon monoxide Cyanide Hydrogen sulfide Thus, cytochrome oxidase is not inhibited by methane.

References:

Harper's Illustrated Biochemistry, 32nd edition, pg: 126

↑ *Back to this question in Part A*

Q33: A patient with thiamine deficiency develops lactic acidosis. Which of the following enzymes is affected? (Q855957)

A. Pyruvate carboxylase

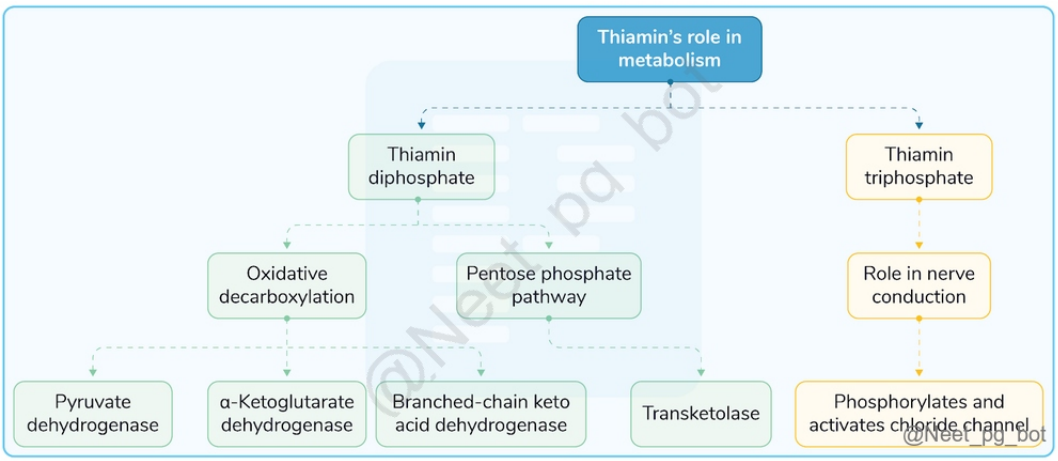
B. Pyruvate dehydrogenase

C. Phosphoenolpyruvate carboxykinase

D. Transketolase

Correct Answer: B. Pyruvate dehydrogenase

Explanation: Thiamine (vitamin B1) is an essential coenzyme involved in several metabolic pathways, including the conversion of pyruvate to acetyl-CoA through the pyruvate dehydrogenase complex (PDC). When deficient, the activity of the pyruvate dehydrogenase complex is impaired. As a result, there is a buildup of pyruvate. This pyruvate is then diverted to other metabolic pathways, like the conversion to lactic acid (lactate), resulting in lactic acidosis. Thiamine deficiency : The Aleurone layer of cereals (food grains) is a rich source of thiamine. Therefore, whole wheat flour and unpolished hand-pound rice have better nutritive value than completely polished refined foods. When the grains are polished, the aleurone layer is usually removed. Biochemical parameters: Thiamine deficiency affects the enzyme transketolase, which is involved in the pentose phosphate pathway. Measuring transketolase activity in red blood cells is a specific test for thiamine deficiency and can confirm the diagnosis. Erythrocyte transketolase activity is reduced; this is the earliest manifestation seen even before clinical disturbances. Conditions Biochemical Role of Thiamine Deficiency Manifestations Dry Beriberi Essential for carbohydrate metabolism and nerve function Peripheral neuritis, sensory disturbances, difficulty walking, and paralysis due to CNS manifestations. Wet Beriberi Thiamine is necessary for the PDH complex and low activity causes Lactic acidosis. Lactic acidosis causes vasodilatation and hence cardiovascular manifestations. Cardiovascular manifestations such as oedema, palpitations, breathlessness, and heart failure. Maple Syrup Urine Disease Cofactor for decarboxylation of branched-chain keto acids Accumulation of branched-chain keto acids leads to convulsions, severe mental retardation, vomiting, acidosis, coma, and death. Lactic Acidosis Cofactor for pyruvate dehydrogenase, converting pyruvate to acetyl-CoA Impaired conversion leads to increased lactate and pyruvate levels, causing lactic acidosis. Pyruvate carboxylase (Option A) is not directly involved in the conversion of pyruvate to acetyl-CoA. Pyruvate carboxylase is responsible for the conversion of pyruvate to oxaloacetate in an anaplerotic reaction that replenishes TCA cycle intermediates. It is not directly affected by thiamine deficiency in the context of lactic acidosis. Phosphoenolpyruvate carboxykinase (Option C) is responsible for the conversion of oxaloacetate to phosphoenolpyruvate (PEP), an important step in gluconeogenesis. It is not directly involved in the conversion of pyruvate to acetyl-CoA and is not typically affected by thiamine deficiency. Transketolase (Option D) is involved in the non-oxidative phase of the pentose phosphate pathway (PPP), which generates ribose-5-phosphate and other sugars. Transketolase requires thiamine pyrophosphate as a cofactor.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 541, 542

↑ *Back to this question in Part A*

@Neet_pg_bot

Q34: Which of the following diagnostic markers is /are incorrectly paired? 1. Beta glucocerebrosidase - Von Gierke disease 2. Lipase - Acute pancreatitis 3. Ceruloplasmin - Hepatolenticular degeneration 4. High LDH1: LDH2 ratio - Myocardial infarction (Q855916)

A. 1 only

B. 1 and 2

C. 1, 2 and 4

D. 2, 3 and 4

Correct Answer: A. 1 only

Explanation: Beta-glucocerebrosidase – Von Gierke disease: Incorrect Von Gierke disease (Glycogen Storage Disease type I) is due to glucose-6-phosphatase deficiency, not beta-glucocerebrosidase. Beta-glucocerebrosidase deficiency causes Gaucher disease, leading to glucocerebroside accumulation in macrophages ("Gaucher cells"). Lipase – Acute pancreatitis : Correct Serum lipase (and amylase) levels are the most specific markers of acute pancreatitis . Ceruloplasmin – Hepatolenticular degeneration: Correct Low ceruloplasmin levels are diagnostic of Wilson's disease (hepatolenticular degeneration) , due to defective copper metabolism. High LDH1:LDH2 ratio – Myocardial infarction: Correct Normally LDH2 > LDH1, but in myocardial infarction , LDH1 > LDH2 ("flipped ratio") due to cardiac tissue damage.

References:

Harper’s Illustrated Biochemistry (23rd Edition), Page 174.

Vasudevan Textbook of Biochemistry (7th Edition), Page 129.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q35: Which among the following absorbs the least water? (Q858469)

A. Mucilage

B. Cellulose

C. Gums

D. Pectin

Correct Answer: B. Cellulose

Explanation: Cellulose absorbs the least water because it is a highly crystalline and rigid polysaccharide. It is composed of long chains of β-glucose units connected by β-1,4-glycosidic bonds, forming strong hydrogen bonds within and between the chains. These extensive hydrogen bonds create a stable, insoluble structure that resists water absorption. Incorrect Options: Mucilage (Option A): Highly hydrophilic and absorbs large amounts of water. Common in plants for water storage and seed germination. Gums (Option C): Hydrophilic polysaccharides that swell and absorb water to form gels.Found in plant exudates. Pectin (Option D): Highly hydrophilic and forms gels by trapping water molecules. Used in food products like jams and jellies for its water-absorbing properties.

References:

Cellulose

↑ *Back to this question in Part A*

@Neet_pg_bot

Q36: Which of the following is deficient in the mucopolysaccharidosis associated with Natowicz disease? (Q852553)

A. Dermatan sulfate

B. Heparan sulfate

C. Keratan sulfate

D. Hyaluronic acid

Correct Answer: D. Hyaluronic acid

Explanation: Natowicz Disease (Mucopolysaccharidosis Type IX - MPS IX) A rare mucopolysaccharidosis (MPS IX) caused by a deficiency of hyaluronidase . Hyaluronidase is needed to degrade the glycosaminoglycan (GAG) hyaluronan (also known as hyaluronic acid). When hyaluronidase is deficient, hyaluronan accumulates in tissues - it is not broken down properly Therefore, the GAG that is “deficient” (i.e. whose breakdown is impaired, leading to accumulation) in Natowicz disease is hyaluronan / hyaluronic acid . Dermatan sulfate (Option A): Incorrect because this is a GAG that accumulates in other types of MPS (e.g. MPS I, VI), not in MPS IX. Heparan sulfate (Option B): Also involved in many other MPS types (e.g. MPS I, II, III), but not in MPS IX. Keratan sulfate (Option C): This GAG is implicated in certain MPS (like MPS IV / Morquio syndrome), but not in MPS IX.

References:

https://pmc.ncbi.nlm.nih.gov/articles/PMC9426054/

https://pmc.ncbi.nlm.nih.gov/articles/PMC5059191/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q37: A 13-year-old boy who accompanied his father to a fish farming region in his hometown accidentally consumes a white odourless chemical. He was under some medication and post-meal he developed toxicity of the drug. Which of the following toxin is most likely to cause this condition? (Q857895)

A. Alkyl guanides

B. BAL

C. Antimycin A

D. Strychnine

Correct Answer: C. Antimycin A

Explanation: Antimycin A Antimycin A is commonly used in fish farming as a piscicide (a chemical toxic to fish) to selectively remove undesirable fish species. It is a colorless to white crystalline compound and is relatively odorless. It is a potent respiratory chain uncoupler. When consumed, it inhibits cellular respiration. It is a mitochondrial toxin that inhibits Complex III of the electron transport chain. This inhibition leads to decreased ATP production and increased oxidative stress. This can amplify the toxicity of some medications- especially those metabolized via oxidative pathways (e.g. anticonvulsants, antibiotics, or chemotherapeutics). Alkyl guanides

(Option A) are a broad class of compounds, some of which can be toxic, but not specifically associated with fish farming or the described scenario. Also, they aren't primarily known for causing drug toxicity by interfering with metabolism in the same direct way as Antimycin A. BAL (British Anti-Lewisite; Dimercaprol) (Option B): This is a chelating agent , not a toxin, and is used as an antidote for heavy metal poisoning. Strychnine (Option D): It's a plant-derived neurotoxin (not used in fish farming). It causes convulsions via spinal inhibition of glycine , not mitochondrial toxicity. It does not primarily cause drug toxicity by interfering with drug metabolism in the way Antimycin A does. Its symptoms would be distinctly neurological, not primarily drug accumulation.

References:

Vasudevan 7th edition , pg no 266

↑ *Back to this question in Part A*

@Neet_pg_bot

Q38: In which of the following locations can cardiolipin be found? (Q830710)

A. Outer mitochondrial membrane

B. Inner mitochondrial membrane

C. Cytosol

D. Mitochondrial matrix

Correct Answer: B. Inner mitochondrial membrane

Explanation: Correct Option B - Inner mitochondrial membrane: This is the correct option. Cardiolipin is a distinctive phospholipid abundant in the inner mitochondrial membrane. It plays a role in the structural integrity of the mitochondria and is involved in several mitochondrial functions, including electron transport chain activity. Incorrect Options: Option A - Outer mitochondrial membrane: The outer mitochondrial membrane primarily consists of phospholipids like phosphatidylcholine and phosphatidylethanolamine. Cardiolipin is not a major component of the outer mitochondrial membrane. Option C - Cytosol: Cardiolipin is not typically found in the cytosol. It is mainly confined to the mitochondrial membrane. Option D - Mitochondrial matrix: Cardiolipin is primarily located in the inner mitochondrial membrane and is not a major component of the mitochondrial matrix.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q39: A 13-year-old boy who accompanied his father to a fish farming region in his hometown accidentally consumes a white odourless chemical. He was under some medication and post-meal he developed toxicity of the drug. Which of the the following

toxin is most likely to cause this condition? (Q820415)

A. Alkyl guanides

B. BAL

C. Antimycin A

D. Strychnine

Correct Answer: C. Antimycin A

Explanation: Antimycin A Antimycin A is commonly used in fish farming as a piscicide (a chemical toxic to fish) to selectively remove undesirable fish species. It is a colorless to white crystalline compound and is relatively odorless. It is a potent respiratory chain uncoupler. When consumed, it inhibits cellular respiration. It is a mitochondrial toxin that inhibits Complex III of the electron transport chain. This inhibition leads to decreased ATP production and increased oxidative stress. This can amplify the toxicity of some medications- especially those metabolized via oxidative pathways (e.g. anticonvulsants, antibiotics, or chemotherapeutics). Alkyl guanides (Option A) are a broad class of compounds, some of which can be toxic, but not specifically associated with fish farming or the described scenario. Also, they aren't primarily known for causing drug toxicity by interfering with metabolism in the same direct way as Antimycin A. BAL (British Anti-Lewisite; Dimercaprol) (Option B): This is a chelating agent , not a toxin, and is used as an antidote for heavy metal poisoning. Strychnine (Option D): It's a plant-derived neurotoxin (not used in fish farming). It causes convulsions via spinal inhibition of glycine , not mitochondrial toxicity. It does not primarily cause drug toxicity by interfering with drug metabolism in the way Antimycin A does. Its symptoms would be distinctly neurological, not primarily drug accumulation.

References:

.https://pmc.ncbi.nlm.nih.gov/articles/PMC6441541/ https://pmc.ncbi.nlm.nih.gov/articles/PMC1217497/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q40: Which of the following minerals is best known for insulin secretion stabilisation? (Q820373)

A. Cadmium

B. Copper

C. Iron

D. Zinc

Correct Answer: D. Zinc

Explanation: Correct Option D - Zinc: Zinc is an essential trace mineral that plays a crucial role in numerous biological processes, including insulin secretion and stabilization. It is involved in the synthesis, storage, and release of insulin from pancreatic beta cells. Zinc also helps in maintaining the structure and stability of insulin molecules. Therefore, of the options provided, zinc is the best-known mineral for insulin secretion stabilization. Incorrect Options: Option A - Cadmium: Cadmium is a toxic heavy metal that can have detrimental effects on health, including disrupting various physiological processes. However, it is not known for its role in insulin secretion stabilization. Option B - Copper: Copper is an essential trace mineral that plays a role in various bodily functions, including the formation of red blood cells and maintaining connective tissues. While it is involved in several enzymatic reactions, it is not primarily known for its role in insulin secretion stabilization. Option C - Iron: Iron is vital for oxygen transport in the blood and is a key component of hemoglobin. It is not primarily associated with insulin secretion stabilization.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q41: Cholera is known to cause diarrhea through its interaction with specific receptors in the human body. Which of the following is known to make it easier for cholera toxin to enter the body through the intestinal mucosa? (Q820337)

A. GLUT 1

B. GM2 ganglioside

C. Cephalin

D. GM1 ganglioside

Correct Answer: D. GM1 ganglioside

Explanation: Correct Option D - GM1 ganglioside: Cholera toxin is produced by the bacterium Vibrio cholerae and is responsible for causing the characteristic symptoms of cholera, including severe diarrhea. Cholera toxin enters the body through the intestinal mucosa and interacts with specific receptors on the surface of intestinal cells. The receptor that facilitates the entry of cholera toxin into the body is the GM1 ganglioside receptor. GM1 ganglioside is a glycosphingolipid, a type of molecule found in the lipid bilayer of cell membranes. Cholera toxin binds to GM1 ganglioside on the surface of intestinal cells, which triggers the toxin to be internalized into the cell through endocytosis. Once inside the cell, the toxin can exert its effects, leading to the disruption of ion transport and fluid balance, ultimately causing severe diarrhea. Option A - GLUT1 (Glucose Transporter 1): GLUT1 is a protein responsible for transporting glucose across cell membranes. It is not directly involved in the interaction between cholera toxin and intestinal cells. Option B - GM2 ganglioside: While GM2 ganglioside is a type of glycosphingolipid, it is not the receptor specifically associated with cholera toxin binding and entry into intestinal cells. Option C - Cephalin: Cephalin is a type of phospholipid and is not known to be directly involved in the interaction between cholera toxin and intestinal cells.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q42: What is the primary mechanism behind tissue damage in long-standing diabetes? (Q852558)

A. Insulin resistance

B. Sorbitol accumulation

C. Hyperglycemia

D. Decreased insulin secretion

Correct Answer: C. Hyperglycemia

Explanation: Hyperglycaemia is the primary mechanism behind tissue damage in long-standing diabetes . Chronic high blood glucose levels lead to various pathophysiological changes , including: Advanced Glycation End Products (AGEs): Hyperglycaemia causes non-enzymatic glycation of proteins and lipids, leading to oxidative stress, inflammation, and vascular damage . Polyol Pathway Activation: Excess glucose is converted into sorbitol , which accumulates in tissues (e.g., retina, nerves, kidneys), leading to osmotic stress and oxidative damage . Protein Kinase C (PKC) Activation: Promotes vascular dysfunction, inflammation, and increased permeability , contributing to complications like diabetic retinopathy and nephropathy . Hexosamine Pathway Dysregulation: Alters gene expression, promoting vascular and renal damage . Insulin resistance (Option A) is incorrect because it plays a role in type 2 diabetes , but the primary cause of tissue damage is chronic hyperglycaemia , not insulin resistance itself. Sorbitol accumulation (Option B) is incorrect because while the polyol pathway contributes to complications like diabetic neuropathy and cataracts , it is not the sole mechanism behind widespread tissue damage. Decreased insulin secretion (Option D) is incorrect because it leads to hyperglycaemia , but it is the high glucose levels that drive long-term tissue damage, not insulin deficiency directly.

References:

https://pmc.ncbi.nlm.nih.gov/articles/PMC6679814/

https://pmc.ncbi.nlm.nih.gov/articles/PMC2996922/

https://pubmed.ncbi.nlm.nih.gov/19489690/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q43: A patient who hails from Mediterranean country and travels to Africa, they contact malaria. After receiving treatment with primaquine, the patient subsequently develops haemolytic anaemia. Which pathway deficiency in the enzyme could potentially be responsible for this condition? (Q855524)

A. Glycolysis

B. Gluconeogenesis

C. Hexose monophosphate (HMP) pathway

D. Luebering-Rapoport pathway

Correct Answer: C. Hexose monophosphate (HMP) pathway

Explanation: The pathway deficiency responsible is the Hexose Monophosphate Pathway (HMP pathway) and enzyme deficiency is G6PD. Hexose Monophosphate Pathway (HMP): The HMP pathway is essential for generating NADPH, which maintains reduced glutathione (GSH) in red blood cells (RBCs). Reduced glutathione protects RBCs from oxidative damage caused by reactive oxygen species (ROS). The enzyme Glucose-6-Phosphate Dehydrogenase (G6PD) is the first and rate-limiting enzyme in the HMP pathway. Primaquine , generates oxidative stress by increasing ROS in RBCs. In G6PD-deficient individuals, the HMP pathway fails to produce adequate NADPH, resulting in depletion of reduced glutathione (GSH). Oxidative damage to hemoglobin (Heinz bodies) and the RBC membrane and subsequent haemolysis. Glycolysis (Option A): Produces ATP but does not generate NADPH required for oxidative stress protection. Gluconeogenesis (Option B): Involves glucose synthesis, not relevant to RBC metabolism. Luebering–Rapoport pathway (Option D): Regulates 2,3-BPG levels in RBCs to modulate oxygen affinity, not oxidative defense.

References:

HMP pathway

↑ *Back to this question in Part A*

@Neet_pg_bot

Q44: Complex IV is inhibited by --? (Q859423)

A. Rotenone

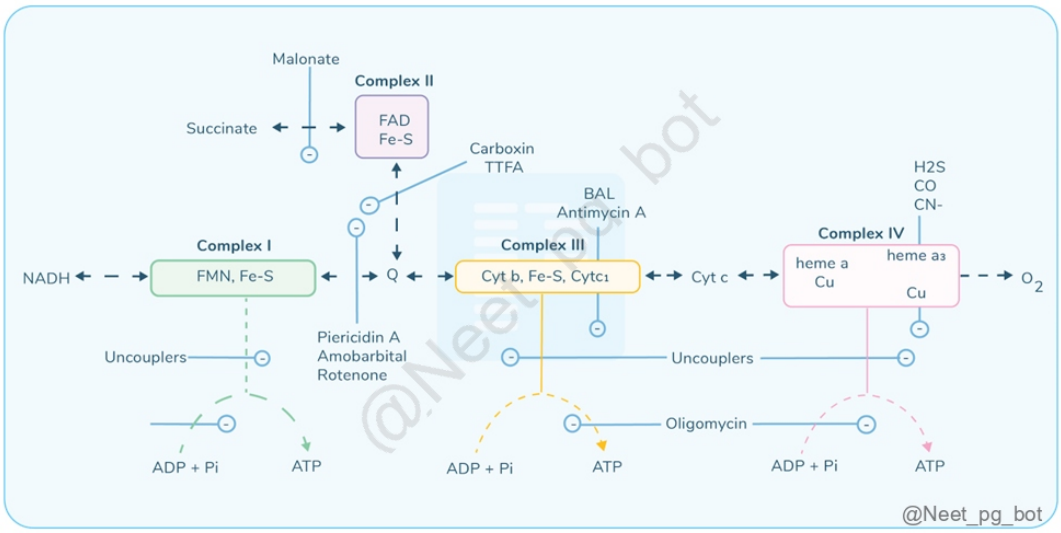
B. Cyanide

C. Carboxin

D. Antimycin A

Correct Answer: B. Cyanide

Explanation: Complex IV, also known as cytochrome c oxidase, is the final enzyme in the electron transport chain, where it catalyzes the transfer of electrons to oxygen, resulting in water formation. Cyanide inhibits Complex IV by binding to the iron in the heme group of cytochrome c oxidase, thereby preventing the transfer of electrons to oxygen. Inhibitors of the Electron Transport Chain (ETC): Inhibitor Target Site Mechanism Rotenone (Option A) Amobarbital Piericidin A Complex I (NADH dehydrogenase) Blocks electron transfer from Complex I to ubiquinone■ TTFA (Thenoyltrifluoroacetone) Carboxin (Option C) Complex II (Succinate dehydrogenase) Blocks electron transfer from Complex II to ubiquinone■ Malonate Complex II Malonate is a competitive inhibitor of Complex II. Antimycin A (Option D) BAL Complex III Cytochrome bc1 complex Blocks electron transfer from Complex III to Cytochrome C. Cyanide (CN–) (Option B) Carbon monoxide (CO) Hydrogen Sulfide (H2S) Complex IV (Cytochrome c Oxidase) Prevents electron transfer from Complex IV to oxygen■ Oligomycin ATP Synthase (Complex V) Blocks the proton channel of ATP synthase, preventing ATP synthesis■ Atractyloside Adenine Nucleotide Translocase Blocks ADP/ATP exchange across the mitochondrial membrane■ {{caption_text}}



References:

Harper’s Illustrated Biochemistry, 32nd edition, pg: 127, 128.

Inhibitors of Oxidative phosphorylation

↑ *Back to this question in Part A*

@Neet_pg_bot

Q45: Which is the enzyme activated by a decrease in insulin: glucagon ratio? (Q854882)

A. Glucokinase

B. Hexokinase

C. Phosphoructokinase

D. Glucose 6-phosphatase

Correct Answer: D. Glucose 6-phosphatase

Explanation: Glucose 6-phosphatase activated by decrease in insulin: glucagon ratio. A decrease in the insulin-to-glucagon ratio shifts the body from an anabolic state (storing energy) to a catabolic state (releasing energy). This leads to activation of enzymes involved in pathways like gluconeogenesis and glycogenolysis, while suppressing enzymes in glycolysis and glycogen synthesis. Glucose 6-phosphatase: Catalyzes the hydrolysis of glucose-6-phosphate to free glucose, the final step in gluconeogenesis and glycogenolysis. Regulation : Activated when the insulin-to-glucagon ratio decreases to release glucose into the bloodstream for energy. It is crucial for maintaining blood glucose levels during fasting or catabolic states. Incorrect Options: Glucokinase (Option A): The gluckinase activity decreases with a decrease in the insulin: glucagon ratio. Hexokinase (Option B): The hexokinase activity decreases with a decrease in the insulin: glucagon ratio. Phosphofructokinase (Option C): A decrease in the insulin: glucagon ratio decreases its activity. Phosphoructokinase activity is regulated by various factors, including ATP, ADP, and citrate concentration.

References:

G6P

↑ *Back to this question in Part A*

@Neet_pg_bot

Q46: Which of the following enzymes are involved in gluconeogenesis? Pyruvate carboxylase Phosphoenol pyruvate kinase Hexokinase Pyruvate kinase Glucose 6 phosphatase (Q856927)

A. 1, 2, and 5 only

B. 2 and 3 only

C. 1 and 5 only

D. 1, 2, and 3 only

Correct Answer: A. 1, 2, and 5 only

Explanation: Gluconeogenesis is the pathway that synthesizes glucose from non-carbohydrate precursors (e.g., lactate, alanine, glycerol). It bypasses the three irreversible steps of glycolysis using alternate enzymes: Glycolytic Step (Irreversible) Gluconeogenic Bypass Enzyme Location Pyruvate → PEP (Pyruvate kinase step) Pyruvate carboxylase and PEP carboxykinase (PEPCK) Mitochondria & Cytosol Fructose-6-phosphate → F-1,6-bisphosphate (PFK-1 step) Fructose-1,6-bisphosphatase Cytosol Glucose → Glucose-6-phosphate

(Hexokinase step) Glucose-6-phosphatase Endoplasmic reticulum (liver, kidney) Hence, the enzymes pyruvate carboxylase , PEP carboxykinase , and glucose-6-phosphatase are directly involved

References:

Gluconeogenesis

↑ *Back to this question in Part A*

@Neet_pg_bot

Q47: Which of the following glycosaminoglycans (GAGs) are responsible for corneal transparency? (Q855981)

A. Keratan sulfate

B. Chondroitin sulfate

C. Heparin sulfate

D. Hyaluronic acid

Correct Answer: A. Keratan sulfate

Explanation: Keratan sulphate is involved in the transparency of cornea Locations of Various Mucopolysaccharides Mucopolysaccharide Primary Locations Functions Hyaluronic Acid (Option D) Connective tissues, tendons, synovial fluid, vitreous humor Serves as a lubricant in joint cavities and contributes to the structure of connective tissues Heparin (Option C) Liver, lungs, spleen, monocytes Acts as an anticoagulant by activating antithrombin III, which inactivates thrombin and other clotting factors Chondroitin Sulfate (Option B) Cartilage, bone, tendons, cornea, skin Provides structural support to connective tissues and contributes to their resilience and elasticity Keratan Sulfate (Option A) Cornea, cartilage Dermatan Sulfate Skin, blood vessels, heart valves Contributes to the structure of the skin, blood vessels, and heart valves

References:

Harper’s Biochemistry Edition 32; Page 610.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q48: Which of the following options is correct with respect to a fed state? 1. Insulin secretion is more 2. Glucagon secretion is less 3. Insulin secretions is less 4. Glucagon secretion is more (Q857486)

A. Only 1

B. Only 3

C. Option 1 and 2 are correct

D. Option 3 and 4 are correct

Correct Answer: C. Option 1 and 2 are correct

Explanation: Insulin secretion is more and glucagon secretion is less in fed state. In the fed state , or postprandial, elevated glucose levels trigger the release of insulin from the pancreatic B cells. As insulin levels rise, there is an increase in glucose uptake, oxidation, and storage in peripheral tissues as well as increases in other anabolic pathways. In the fed state, pancreatic & cells secrete insulin in response to an increase in blood glucose, amíno acids, and fatty acids. Glucagon is secreted from pancreatic a cells, and glucagon secretion is higher in the fasted state and during exercise. Insulin is secreted more in the fed state than in the fasted state. There are two metabolic states- Anabolic and catabolic state. Fed or anabolic state occurs immediately after a meal when the energy of nutrients (carbohydrate, protein, or fat) is transferred to high energy compounds for immediate use or for storage. Peripheral tissues (predominantly skeletal muscle) buffer ingested glucose by storing it as glycogen. Fasted or catabolic state occurs later when the level of available nutrients decreases in the blood, and the stored reserves (initially glycogen) are mobilized to perform work or to generate heat. During prolonged fasting, fat oxidation and ketone bodies are used to meet whole body energy requirements. The secretion of insulin and glucagon hormones is controlled reciprocally by blood glucose levels. High insulin to low glucagon ratio- Fed state/Anabolic. Low insulin to high glucagon ratio- Starved state/Catabolic.

References:

Fed state

↑ *Back to this question in Part A*

@Neet_pg_bot

Q49: A 10-year-old boy rapidly develops hypoglycemia after moderate physical activity. Laboratory evaluation shows elevated ketone bodies, lactic acid, and triglycerides. On examination, hepatomegaly and renomegaly are noted. Which of the following enzyme deficiencies is most likely present in the liver of this child? (Q856947)

A. Glucose-6-phosphatase

B. Phosphoglucomutase

C. Phosphofructokinase

D. Defect in glycogen branching enzyme

Correct Answer: A. Glucose-6-phosphatase

Explanation: This presentation is classic for Von Gierke's disease (Glycogen Storage Disease type I) , caused by deficiency of glucose-6-phosphatase in the liver, kidney, and intestinal mucosa. The enzyme catalyzes the final step of glycogenolysis and gluconeogenesis , converting glucose-6-phosphate → free glucose . Its deficiency leads to severe fasting hypoglycemia , lactic acidosis , hypertriglyceridemia , hyperuricemia , and hepatomegaly/renomegaly due to glycogen accumulation. Incorrect options: Phosphoglucomutase deficiency (Option B): Impairs glycogen metabolism but causes milder symptoms without severe hypoglycemia. Phosphofructokinase deficiency (Option C): Affects muscle glycolysis, leading to exercise intolerance, not hepatic enlargement. Glycogen branching enzyme defect (Option D): Causes Andersen's disease , characterized by cirrhosis, not hypoglycemia with lactic acidosis.

References:

Von gierke disease

↑ *Back to this question in Part A*

@Neet_pg_bot

Q50: Complex IV is inhibited by? (Q793381)

A. Rotenone

B. Cyanide

C. Carboxin

D. Antimycin A

Correct Answer: B. Cyanide

Explanation: Correct Option B: Cyanide compound poisons electron transport chain in the mitochondria within the cells. It stops the body from deriving energy (ATP) from oxygen. Specifically, cyanide attaches with the a3 prosthetic group in the complex IV of cytochrome C oxidase (CCOx) and causes death of the cell rapidly. The generation of ATP is shutdown in the aerobic respiration that is recognized as cyanide toxic actions in the eukaryotes primarily. Incorrect Options: Option A: Rotenone inhibits complex I (NADH CoQ1 reductase) of the respiratory chain and inhibits reduction of ubiquinone. Option C: Like rotenone, carboxin prevents with ubiquinone reduction at the binding site. Option D: Antimycin is one of the strong electron transport chains (ETC) inhibitors. It prevents electron transfer from cytochrome b to cytochrome c. Hence, Flow of electrons inhibits through complex III of the electron transport chain.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q51: What is the composition of lactulose? (Q860047)

A. Glucose + Galactose

B. Glucose + Fructose

C. Galactose + Fructose

D. Fructose + Ribulose

Correct Answer: C. Galactose + Fructose

Explanation: Lactulose is a synthetic disaccharide composed of galactose and fructose. It is commonly used as a laxative and to treat hepatic encephalopathy . Lactulose = Galactose + Fructose (linked by a β-1,4 glycosidic bond). Lactose = Galactose + Glucose (natural milk sugar). Lactulose is not absorbed in the small intestine; it reaches the colon where it is fermented by bacteria to lactic acid and acetic acid, increasing osmotic pressure — hence used as a laxative and in hepatic encephalopathy to reduce ammonia absorption Disaccharide Sugars present Linkage Maltose (Basic thumb rule) 2 glucose residues - glucose + glucose alpha(1,4) Isomaltose (Six atoms) 2 glucose residues - glucose + glucose a(1,6) Trehalose 2 glucose residues - glucose + glucose a(1,1) Sucrose Glucose + fructose (Option B ruled out) a(1,2) Lactose Galactose + glucose (Option A ruled out) beta(1,4) Fructose + Ribulose (Option D): This composition does not correspond to any common disaccharide or sugar.

References:

https://pmc.ncbi.nlm.nih.gov/articles/PMC9519995/

https://www.sciencedirect.com/science/article/abs/pii/B9780128036990000050

Harper’s Illustrated Biochemistry, 32nd Edition, Pg 153

Lippincott illustrated reviews: Biochemistry, 7th edition, pg: 787,788.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q52: A patient’s X-ray shows cardiomegaly, and they present with a "floppy baby" syndrome, hypotonia, macroglossia, and hepatomegaly. What is the most likely diagnosis? (Q844238)

A. Pompe disease

B. Ebstein anomaly

C. Down syndrome

D. Cori's Disease

Correct Answer: A. Pompe disease

Explanation: Correct Option A - Pompe disease: Pompe disease (Glycogen storage disease type II) is caused by a deficiency of lysosomal acid α-1,4-glucosidase (acid maltase) , leading to accumulation of glycogen within lysosomes in cardiac and skeletal muscle. This results in: Cardiomegaly (hypertrophic cardiomyopathy) Severe hypotonia (“floppy baby” syndrome) Macroglossia Hepatomegaly Early death from heart failure or respiratory failure in infancy Incorrect Options: B) Ebstein anomaly: Congenital heart defect involving tricuspid valve displacement; presents with cyanosis and murmur, not hypotonia or macroglossia. C) Down syndrome : May cause hypotonia and macroglossia but not cardiomegaly or glycogen accumulation. D) Cori’s disease : (Glycogen storage disease type III) involves debranching enzyme deficiency, causing hepatomegaly and mild hypoglycemia, but no cardiomyopathy or severe hypotonia.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q53: Match the following with the most appropriate feature. 1. Von Gierke's a. Branched structure in liver biopsy 2. Pompe's b. Glycogen deposition in lysosomes 3. McArdle's c. Hypoglycemia with lactic acidosis 4. Cori's d. Exercise intolerance (Q839906)

A. 1c, 2a,3d,4b

B. 1c, 2b,3d,4a

C. 1a, 2c,3d,4b

D. 1d, 2c,3b,4d

Correct Answer: B. 1c, 2b,3d,4a

Explanation: Von Gierke's Disease: Characterized by hypoglycemia, and lactic acidosis due to the deficiency of glucose-6-phosphatase. Pompe's Disease: Characterized by glycogen deposition in lysosomes due to acid alpha-glucosidase deficiency. McArdle's Disease: Causes exercise intolerance due to the lack of muscle glycogen phosphorylase. Cori's Disease: Characterized by the accumulation of branched glycogen due to deficiency of debranching enzyme. Type Name Enzyme defect 1 Von Gierke's Disease Glucose-6-phosphatase 2 Pompe's Disease Acid maltase/ alpha glucosidase 3 Cori's Disease Debranching enzyme 4 Anderson's Disease Branching enzyme 5 McArdle's Disease Muscle Phosphorylase 6 Hers Disease Hepatic phosphorylase 7 Tarui's Disease

Phosphofructokinase-1

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q54: Which receptor plays a crucial role in glucose uptake by skeletal muscle? (Q860412)

A. GLUT 1

B. GLUT 2

C. GLUT 3

D. GLUT 4

Correct Answer: D. GLUT 4

Explanation: GLUT 4 is the insulin-dependent glucose transporter predominantly found in heart, skeletal muscle and adipose tissue . It translocates to the cell membrane in response to insulin, facilitating glucose uptake during periods of increased energy demand. GLUT proteins are distributed in various tissues according to their specific functions. Transporter Tissue Location Functions GLUT 1 (Option A ruled out) Brain, kidney, colon, placenta, erythrocytes Facilitates glucose uptake GLUT 2 (Option B ruled out) Liver, pancreatic β cells, small intestine, kidney Enables rapid glucose uptake or release GLUT 3 (Option C ruled out) Brain, kidney, placenta Assists in glucose uptake GLUT 4 (Option D) Heart and skeletal muscle, adipose tissue Mediates insulin-stimulated glucose uptake GLUT 5 Small intestine Facilitates fructose absorption SGLT 1 Small intestine and kidney Actively transports glucose against a concentration gradient GLUT 1-5 are facilitative bidirectional transporters, whereas SGLT 1 is a sodium-dependent unidirectional transporter.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg. 186,187.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q55: What is the rate-limiting enzyme of cholesterol synthesis? (Q860403)

A. HMG-CoA reductase

B. 7-alpha hydroxylase

C. Carnitine acyltransferase 1

D. Carnitine acyltransferase 2

Correct Answer: A. HMG-CoA reductase

Explanation: The principal regulatory step in the cholesterol synthesis pathway: HMG-CoA is reduced to mevalonate It is catalyzed by HMG-CoA reductase Regulation of HMG-CoA reductase: Regulation Method Mechanism Sufficient Cholesterol Suppresses transcription of HMG CoA reductase gene Low Dietary Cholesterol Increases transcription of HMG CoA reductase gene AMP-activated Kinase Phosphorylates HMG CoA reductase inactivating it Protein Phosphatase 1 Dephosphorylates HMG CoA reductase activating it Insulin and Thyroxine Increase activity of HMG CoA reductase Cortisol and Glucagon Decrease activity of HMG CoA reductase Statins (e.g., Lovastatin) Competitive inhibition of HMG CoA reductase 7-alpha hydroxylase (Option B): This enzyme is the rate-limiting step in bile acid synthesis, not cholesterol synthesis. Carnitine acyltransferase 1 (Option C): This enzyme is the rate-limiting enzyme in fatty acid oxidation, facilitating the transport of long-chain fatty acids into the mitochondria. Carnitine acyltransferase 2 (Option D): This enzyme is involved in fatty acid oxidation but is not the rate-limiting step. It helps regenerate free carnitine in the mitochondrial matrix.

References:

Harper 32nd edition, page 260

Vasudevan 7th edition, pages 171,173

↑ *Back to this question in Part A*

@Neet_pg_bot

Q56: Which of the following is the rate-limiting enzyme of the hexose monophosphate (HMP) pathway? (Q860387)

A. Glucose-6-phosphatase

B. Glucose-6-phosphate dehydrogenase

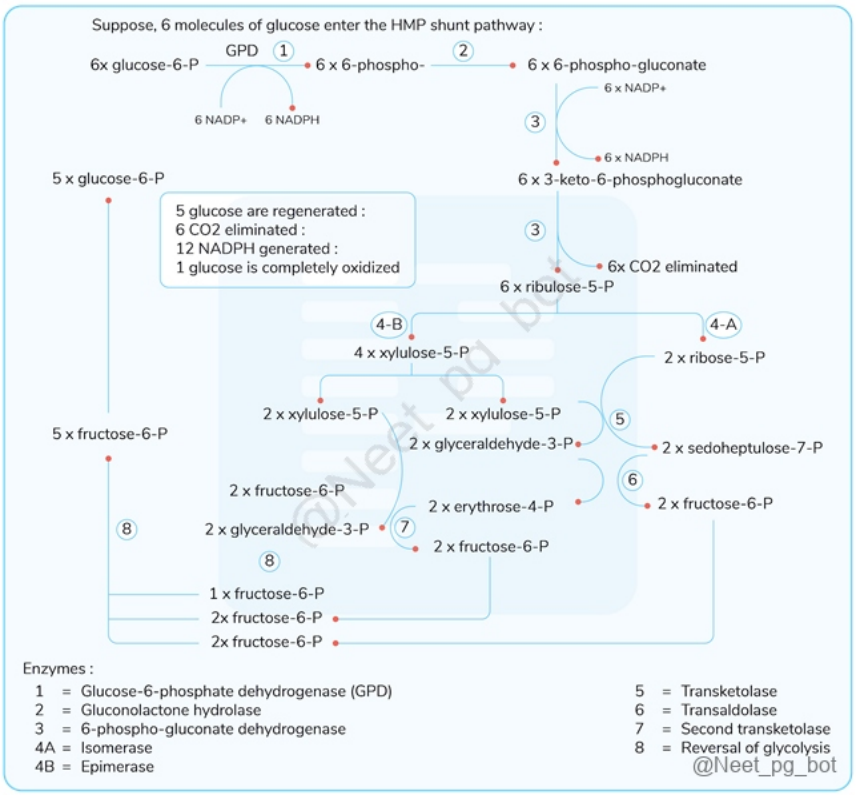
C. Phosphofructokinase-1

D. Glycogen phosphorylase

Correct Answer: B. Glucose-6-phosphate dehydrogenase

Explanation: Glucose-6-phosphate dehydrogenase is the rate-limiting step of the HMP pathway. It catalyzes the oxidation of glucose-6-phosphate to 6-phosphoglucono-δ-lactone, generating NADPH, which is critical for reductive biosynthesis and maintaining cellular redox balance. The pentose phosphate pathway (PPP) is essential

in erythrocytes because it generates NADPH. NADPH is crucial for maintaining glutathione in its reduced form, which is necessary for the activity of glutathione peroxidase. This enzyme removes harmful hydrogen peroxide (H■O■), protecting red blood cells from oxidative damage and preventing hemolysis. Pentose Phosphate Pathway (PPP) The Pentose Phosphate Pathway (PPP), also known as the hexose monophosphate shunt, is an alternative metabolic pathway for glucose that doesn't generate ATP. Location: Cytosol It serves two primary functions: NADPH Production The PPP is a major source of NADPH, vital for biosynthesis (fatty acids and steroids). NADPH Production: RBCs rely on glycolysis for ATP due to the lack of mitochondria and depend solely on the PPP for NADPH, as they lack mitochondrial dehydrogenases. Glutathione Maintenance: NADPH keeps glutathione reduced, enabling glutathione peroxidase to neutralize hydrogen peroxide and prevent oxidative damage. Impact of NADPH Deficiency : Low NADPH reduces glutathione, impairs glutathione peroxidase, increases oxidative damage , and leads to hemolysis . Ribose-5-Phosphate (R-5-P) Synthesis Ribose-5-phosphate is essential for nucleotide synthesis needed for RNA and DNA. Homeostasis between NADPH and R-5-P production. When NADPH demand exceeds R-5-P's need, the non-oxidative phase converts R-5-P to glycolytic intermediates (fructose-6-phosphate and glyceraldehyde-3-phosphate). If the demand for R-5-P is higher than for NADPH, the non-oxidative phase generates R-5-P from fructose-6-phosphate and glyceraldehyde-3-phosphate, bypassing the oxidative steps. Differences from Glycolysis Uses NADP+ instead of NAD+ and generates CO2, not ATP. Glucose-6-phosphatase (Option A): This enzyme catalyzes the conversion of glucose-6-phosphate to glucose during gluconeogenesis and glycogenolysis. It is not involved in the HMP pathway. Phosphofructokinase-1 (Option C): This enzyme is the rate-limiting enzyme of glycolysis , not the HMP pathway. Glycogen phosphorylase (Option D): This enzyme catalyzes the breakdown of glycogen to glucose-1-phosphate in glycogenolysis and is not related to the HMP pathway.



References:

Harper’s biochemistry 32ed Pg 191;194-196

↑ *Back to this question in Part A*

@Neet_pg_bot

Q57: A diabetic patient is advised to exercise. Which of the following glucose transporters is insulin-sensitive? (Q844249)

A. GLUT4

B. GLUT1

C. GLUT3

D. GLUT2

Correct Answer: A. GLUT4

Explanation: Correct Option A - GLUT4: This glucose transporter is insulin-sensitive and is primarily found in adipose tissue and muscle. During exercise, insulin facilitates glucose uptake via GLUT4, helping regulate blood glucose levels. Incorrect Options: Options B, C, and D are Insulin-independent transporters.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q58: A neonate presents with poor feeding and vomiting, cataract, and acidosis with a positive Benedict's test on urine and negative urinary glucose. Which enzyme is likely defective? (Q844276)

A. Galactose-1-phosphate uridyl transferase

B. Aldolase B

C. Fructokinase

D. Glucose-6-phosphatase

Correct Answer: A. Galactose-1-phosphate uridyl transferase

Explanation: Correct Option A - Galactose-1-phosphate uridyl transferase: This presentation is classic for galactosemia due to deficiency of galactose-1-phosphate uridyl transferase (GALT) — the most common and severe form. Pathophysiology: In normal metabolism: Galactose → Galactose-1-phosphate → Glucose-1-phosphate (via galactokinase and GALT) In GALT deficiency , galactose-1-phosphate accumulates in

the liver, kidneys, and brain. Excess galactose is reduced to galactitol (dulcitol) in the lens → cataracts . Accumulation causes hepatic dysfunction , renal tubular damage , and metabolic acidosis . Urine Findings: Benedict's test positive (detects reducing sugars like galactose, fructose, lactose — not just glucose) Urine glucose test negative (since glucose is absent) Incorrect Options: B) Aldolase B: Defective in hereditary fructose intolerance — presents after fructose ingestion (e.g., fruit, sucrose) with hypoglycemia , vomiting , and hepatomegaly , not cataracts. C) Fructokinase: Causes essential fructosuria , a benign condition with positive Benedict's test but no symptoms . D) Glucose-6-phosphatase: Defective in Von Gierke's disease (Glycogen storage disease type I) — presents with hypoglycemia , hepatomegaly , and lactic acidosis , not with cataracts or positive Benedict's test.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q59: A patient with high triglycerides esterified with long-chain fatty acids presents with fatigue, and muscle biopsy shows fat vacuoles. What is the most likely diagnosis? (Q844255)

A. Carnitine deficiency

B. Fatty Acid Synthase defect

C. Lipoprotein Lipase (LPL) deficiency

D. Medium-chain acyl-CoA dehydrogenase (MCAD) deficiency

Correct Answer: A. Carnitine deficiency

Explanation: Correct Option A - Carnitine deficiency: Carnitine deficiency impairs the transport of long-chain fatty acids into the mitochondrial matrix for β-oxidation . As a result: Fatty acids accumulate in muscle cytoplasm (fat vacuoles), ATP production decreases → fatigue, weakness , and exercise intolerance , Hypoketotic hypoglycemia may occur during fasting due to impaired fatty acid oxidation. The condition can be primary (defect in carnitine transporter) or secondary (from liver disease, malnutrition, or valproate use). Incorrect Options: B) Fatty Acid Synthase defect: Affects fatty acid synthesis, not oxidation; would not cause fat accumulation or myopathy. C) Lipoprotein Lipase (LPL) deficiency: Causes hypertriglyceridemia and pancreatitis, not muscle lipid vacuolation. D) Medium-chain acyl-CoA dehydrogenase (MCAD) deficiency: Causes hypoketotic hypoglycemia but does not affect long-chain fatty acid transport ; fat vacuoles are less prominent.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q60: A breastfed infant presents with lethargy, hepatomegaly, and cataracts. Which of the following enzyme deficiencies is most likely responsible for this presentation? (Q866651)

A. Galactokinase

B. Galactose-1-phosphate uridyltransferase (GALPUT)

C. Aldolase B

D. Fructokinase

Correct Answer: B. Galactose-1-phosphate uridyltransferase (GALPUT)

Explanation: The infant’s lethargy, hepatomegaly, and cataracts after breastfeeding indicate classic galactosemia caused by Galactose-1-phosphate uridyltransferase deficiency, leading to toxic buildup of galactose-1-phosphate and galactitol, resulting in liver damage, cataracts, and neurological issues. Classic Galactosemia Galactose-1-phosphate Uridyltransferase (GALT) Deficiency (Option B) Severely deficient in individuals with Classic Galactosemia in which galactose-1-phosphate and, therefore, galactose accumulate. It Is an inborn error of metabolism Accumulation Accumulated galactose is shunted into side pathways like galactitol production. Aldose reductase catalyzes it. Galactose-1 phosphate accumulates in the liver, inhibiting galactokinase and glycogen phosphorylase, leading to hypoglycemia. Bilirubin Reduced bilirubin uptake and conjugation, increasing unconjugated bilirubin in blood Symptoms Hypoglycemia, Hypophosphatemia, Hyperuricemia, Lactic acidosis Hepatomegaly, Jaundice, Severe mental retardation Galactosuria (galactose excreted in urine) Cataracts from dulcitol accumulation in the lens Tubular damage and generalized aminoaciduria Diagnosis Clinical manifestations (e.g., congenital cataract), presence of galactose in urine and elevated blood galactose levels Prenatal diagnosis via amniocentesis Detection of heterozygous parents by elevated galactose levels after a galactose load Complications in untreated cases E. coli sepsis, Fanconi syndrome Feeding problems, Failure to thrive, Hepatocellular damage, Bleeding Treatment Withdrawal of galactose and lactose from the diet. GALT deficiency is a part of the newborn screening panel Incorrect Options: Galactokinase (Option A) causes isolated cataracts but milder disease without severe liver involvement. Aldolase B (Option C) causes hereditary fructose intolerance, presenting with hypoglycemia and liver problems after fructose ingestion. Fructokinase (Option D) causes essential/fructosuria, a benign condition without serious symptoms.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q61: A patient with a history of strenuous exercise skipped a meal and later consumed alcohol at a party. Alcohol inhibits which of the following biochemical processes,

potentially leading to hypoglycemia? (Q852549)

A. Gluconeogenesis

B. Glycogenolysis

C. Hexose Monophosphate (HMP) Shunt

D. Ketone Body Formation

Correct Answer: A. Gluconeogenesis

Explanation: Alcohol metabolism increases NADH/NAD+ ratio: Ethanol is metabolized in the liver by alcohol dehydrogenase and acetaldehyde dehydrogenase , leading to excessive NADH production. Inhibition of Gluconeogenesis: High NADH shifts metabolic balance , preventing conversion of lactate to pyruvate and malate to oxaloacetate , both of which are essential for gluconeogenesis. Hypoglycemia Risk: After exercise and fasting, glycogen stores are depleted. The body relies on gluconeogenesis for glucose , but alcohol blocks this pathway , leading to hypoglycemia after drinking. Alcohol-Induced Hypoglycemia Feature Effect of Alcohol Key Mechanism ↑ NADH inhibits gluconeogenesis Primary Effect ↓ Glucose production → Hypoglycemia Clinical Features Weakness, dizziness, confusion, seizures Risk Factors Fasting, exercise, malnutrition, chronic alcoholism Treatment IV dextrose, thiamine in alcoholics Glycogenolysis (Option B): Incorrect because alcohol does not directly inhibit glycogen breakdown. However, after prolonged fasting, glycogen stores are already depleted. Hexose Monophosphate (HMP) Shunt (Option C): Incorrect because this pathway primarily generates NADPH for anabolic processes and does not significantly contribute to blood glucose levels. Ketone Body Formation (Option D): Incorrect because alcohol metabolism can actually promote ketogenesis, especially in chronic alcoholism, rather than inhibiting it.

References:

Harper’s illustrated Biochemistry 32nd edition, pages 142, 162,163,171, 186, 187

↑ *Back to this question in Part A*

@Neet_pg_bot

Q62: Which of the following is excreted in the urine of patients with pentosuria? (Q852461)

A. L-Xylulose

B. D-Ribose

C. D-Ribulose

D. D-Lactose

Correct Answer: A. L-Xylulose

Explanation: L-Xylulose is excreted in the urine of patients with essential pentosuria , a rare benign metabolic disorder caused by a deficiency of L-xylulose reductase . This leads to the accumulation and urinary excretion of L-xylulose , which can be mistaken for glucose in urine tests, but patients remain asymptomatic with no metabolic consequences. Essential Pentosuria Feature Essential Pentosuria Cause Deficiency of L-xylulose reductase Metabolic Effect Increased excretion of L-xylulose in urine Clinical Symptoms None (asymptomatic) Diagnosis Urine sugar analysis + Chromatography Treatment No treatment needed D-Ribose (Option B) is incorrect because it is a naturally occurring sugar involved in nucleotide metabolism and is not significantly excreted in pentosuria. D-Ribulose (Option C) is incorrect because it is an intermediate in the pentose phosphate pathway but is not associated with pentosuria. D-Lactose (Option D) is incorrect because lactose is a disaccharide and is not involved in pentose metabolism or pentosuria.

References:

Harper’s Biochemistry: Pg 194

Vasudevan’s Textbook of Biochemistry for Medical Students, 7th Edition, Page 131, 316, 317.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q63: During starvation, which of the following hormones is primarily produced to maintain blood glucose levels? (Q852554)

A. Glucagon

B. Insulin

C. Amylase

D. Somatostatin

Correct Answer: A. Glucagon

Explanation: Role of Glucagon in Starvation: During starvation, glucagon is the primary hormone that maintains blood glucose levels. It promotes gluconeogenesis (glucose synthesis) and glycogenolysis (breakdown of glycogen) in the liver. Increases lipolysis in adipose tissue, providing free fatty acids for energy. Phases of Starvation: Early fasting (first few hours): Blood glucose is maintained by glycogenolysis. Prolonged fasting (after 24 hours): Gluconeogenesis becomes the primary glucose source, with glucagon playing a key role. Later starvation (after 3-5 days): Ketogenesis increases as an alternative fuel. Hormonal Changes in Starvation Hormone Effect in Starvation Glucagon Increases to stimulate gluconeogenesis and glycogenolysis. Insulin Decreases to reduce glucose uptake by tissues. Cortisol Increases to promote protein catabolism for gluconeogenesis. Epinephrine Increases to enhance glycogen breakdown and lipolysis. Growth Hormone (GH) Increases to reduce glucose utilization and promote lipolysis. Insulin (Option B): Incorrect because insulin decreases during starvation to limit glucose uptake by tissues. Amylase (Option C): Incorrect because amylase is a digestive enzyme, not a

hormone involved in blood glucose regulation. Somatostatin (Option D): Incorrect because somatostatin inhibits both insulin and glucagon but does not directly regulate blood glucose during starvation.

References:

Nelson Textbook of Paediatrics Edition 21, Page 778-781

↑ *Back to this question in Part A*

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Q64: When blood glucose concentration is suddenly increased to 2–3 times the normal level and maintained at that level, what will be the typical pattern of blood glucose change observed over time? (Q866337)

A. Rapid rise followed by sustained high glucose levels

B. Continuous increase in insulin secretion without plateau

C. Rise in glucose followed by a fall below baseline

D. Initial rise and then stabilization slightly above baseline

Correct Answer: C. Rise in glucose followed by a fall below baseline

Explanation: When plasma glucose suddenly increases and stays elevated, insulin secretion occurs in two phases : Phase 1: A rapid burst of preformed insulin causes a quick fall in glucose. Phase 2: A slower, sustained insulin release maintains glucose control. As a result, glucose levels initially rise , then overshoot downward , producing a temporary fall below baseline before normalizing — known as reactive hypoglycemia .

References:

Guyton and Hall Textbook of Medical Physiology, 15th Ed., Ch. 79 — “Insulin, Glucagon, and Diabetes Mellitus.” Harrison’s Principles of Internal Medicine, 22nd Ed., Ch. 417 — “Glucose Homeostasis and Insulin Secretion.”

↑ *Back to this question in Part A*

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Q65: After a high-carbohydrate meal, weight gain is explained by all except? (Q865420)

A. Malonyl CoA

B. Tricarboxylate transporter

C. Acetyl-CoA carboxylase

D. Mitochondrial carnitine

Correct Answer: D. Mitochondrial carnitine

Explanation: After a high-carbohydrate meal, excess glucose is converted into fatty acids and stored as fat, contributing to weight gain. This process involves: Malonyl CoA: An intermediate in fatty acid synthesis that inhibits fatty acid oxidation. Tricarboxylate transporter: Transports citrate from mitochondria to cytosol, providing acetyl-CoA for fatty acid synthesis. Acetyl-CoA carboxylase: The key enzyme that converts acetyl-CoA to malonyl-CoA, initiating fatty acid synthesis. Mitochondrial carnitine (Option D) transports fatty acids into mitochondria for β-oxidation (fat breakdown). After a high-carb meal, fat oxidation is inhibited, so mitochondrial carnitine doesn't contribute to weight gain.

References:

Mitochondrial Fatty Acid Oxidation in Obesity - PMC

↑ *Back to this question in Part A*

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Q66: Cancer cells take up excess glucose because? (Q865417)

A. Lactate is produced even in the presence of Oxygen

B. High NADH/NAD ratio

C. High GLUT2

D. Absence of Oxygen

Correct Answer: A. Lactate is produced even in the presence of Oxygen

Explanation: Cancer cells demonstrate the Warburg Effect by favoring the conversion of glucose to lactate even in the presence of adequate oxygen, a process known as aerobic glycolysis. The phenomenon in cancer cells where, even in the presence of oxygen, glucose is primarily converted to lactate through aerobic glycolysis. (Option A) Significance: This allows faster ATP production and provides intermediates for neoplastic cell growth and multiplication. It increases NADPH production, protecting neoplastic cells from oxidative stress. High NADH/NAD ratio (Option B): Although the NADH/NAD■ balance affects cellular metabolism, it is not the primary reason cancer cells take up excess glucose. The Warburg effect drives increased glucose uptake regardless of NADH levels. High GLUT2 (Option C): GLUT2 is a glucose transporter mainly found in liver, pancreatic beta cells, and kidney, not commonly involved in cancer cell glucose uptake. Cancer cells typically overexpress GLUT1 or other transporters to increase glucose intake. Absence of Oxygen (Option D): While low oxygen

(hypoxia) can stimulate glucose uptake, many cancer cells continue to produce lactate and consume large amounts of glucose even when oxygen is plentiful, which is the hallmark of aerobic glycolysis, not simply hypoxia-induced metabolism.

References:

Vasudevan's Textbook of Biochemistry, 7th Edition, Page 114.

Warburg Effect

↑ *Back to this question in Part A*

@Neet_pg_bot

Q67: A child presents with fatigue and hepatomegaly. Liver enzymes (ALT, AST) are elevated. Ketosis was significant. Liver biopsy shows excess glycogen accumulation. After feeding, blood glucose levels rise, but there is no rise in glucose after overnight fasting. Which of the following enzyme deficiencies is most likely responsible for this presentation? (Q865410)

A. Glucose-6-phosphatase

B. Liver phosphorylase

C. Muscle phosphorylase

D. Phosphofructokinase

Correct Answer: A. Glucose-6-phosphatase

Explanation: The child's presentation—fatigue, hepatomegaly, elevated liver enzymes, marked ketosis, glycogen accumulation in the liver, fasting hypoglycemia with normal postprandial glucose—strongly points toward a Glucose-6-phosphatase deficiency Von Gierke's Disease (Glycogen Storage Disease Type I) Cause Deficiency of glucose-6-phosphatase enzyme Pathophysiology Inability to convert glucose-6-phosphate to glucose; leads to accumulation of glycogen, lactate, and triglycerides Symptoms Severe hypoglycemia Hepatomegaly and Renomegaly Growth retardation Lactic acidosis Hyperuricemia and Hyperlipidemia Diagnosis Biochemical tests (hypoglycemia, lactic acidosis, hyperuricemia, hyperlipidemia) Genetic testing Management Frequent feedings with slow-release carbohydrates Blood glucose monitoring Medications for associated conditions Liver transplantation (in severe cases) Liver phosphorylase (GSD VI – Hers disease) (Option B) causes mild hypoglycemia and hepatomegaly, but symptoms are less severe. Gluconeogenesis is intact, so fasting hypoglycemia and ketosis are not as pronounced as in glucose-6-phosphatase deficiency. Muscle phosphorylase (GSD V – McArdle disease) (Option C) affects only skeletal muscle. Presents with exercise intolerance and muscle cramps, not fasting hypoglycemia or hepatomegaly. Phosphofructokinase (GSD VII – Tarui disease) (Option D) involves muscle and red blood cells. Causes muscle pain and hemolysis, but does not affect the liver or cause fasting hypoglycemia. Learning Objectives: Glycogen Storage Disease Enzyme Deficiency Type 0 Glycogen synthase deficiency Type I (von Gierke's Disease) Glucose-6-phosphatase Type II (Pompe's Disease)

Acid α-glucosidase (acid maltase) Type III (Cori's Disease) Debranching enzyme (amylo-1,6-glucosidase) Type IV (Andersen's Disease) Branching enzyme Type V (McArdle's Disease) Muscle phosphorylase (myophosphorylase) Type VI (Hers' Disease) Liver phosphorylase Type VII (Tarui's Disease) Phosphofructokinase (muscle type)

References:

Harper’s Illustrated Biochemistry, 23rd Edition, Page 174.

Vasudevan Textbook of Biochemistry, 7th Edition, Page 128,129.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q68: In a fasting state, glucagon promotes the production of glucose by which of the following pathways? (Q793392)

A. Glycolysis and gluconeogenesis

B. Gluconeogenesis and glycogenolysis

C. Glycogenesis and gluconeogenesis

D. Only gluconeogenesis

Correct Answer: B. Gluconeogenesis and glycogenolysis

Explanation: Correct Option B - Gluconeogenesis and glycogenolysis: Gluconeogenesis is a metabolic process in the body in which glucose is produced from a non-carbohydrate substrate of carbon. Glycogenolysis is biochemical process in which glycogen is broken down into glucose and glucose-1-phosphate. During the fasting state, glucose is produced from glycogen in the liver which is called glycogenolysis and also via gluconeogenesis from glycerol and oxaloacetate. Incorrect Options: Option A - Glycolysis and gluconeogenesis: Glycolysis is a chain of reactions that produce energy from glucose by breaking into two molecules of three carbon molecule compounds called pyruvates. Hence, this is incorrect. Option C - Glycogenesis and gluconeogenesis: Glycogenesis is formation of glycogen from primary carbohydrate that is stored in the muscle, liver cells of animals from the glucose molecule. Hence, this is incorrect. Option D - Only gluconeogenesis: Gluconeogenesis and glycogenolysis are both pathways used in the production of glucose during the fast.

↑ *Back to this question in Part A*

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• [03_Lipid Chemistry] 009 Lipid Chemistry

« Back to Folder Directory

[03_Lipid Chemistry] 009 Lipid Chemistry — Quick Jump

Q1 (p. 635) *Q2 (p. 636)* *Q3 (p. 637)* *Q4 (p. 638)* *Q5 (p. 639)* *Q6 (p. 640)*
Q7 (p. 641) *Q8 (p. 642)* *Q9 (p. 643)* *Q10 (p. 644)* *Q11 (p. 645)* *Q12 (p. 646)*
Q13 (p. 647) *Q14 (p. 649)* *Q15 (p. 649)* *Q16 (p. 650)* *Q17 (p. 651)* *Q18 (p. 652)*
Q19 (p. 654)

Q1: In a patient with a history of recurring attacks of pancreatitis, eruptive xanthomas, increased plasma triglyceride levels (2,000 mg/dL), and milky plasma. Which of the following is the most likely deficiency? (Q882115)

A. Lipoprotein Lipase

B. LDL receptors

C. HMG-CoA reductase

D. ABCA1 receptor

Correct Answer: A. Lipoprotein Lipase

Explanation: The patient's presentation, including recurring attacks of pancreatitis, eruptive xanthomas, markedly increased plasma triglyceride levels (2,000 mg/dL), and milky plasma, is suggestive of Familial Hyperchylomicronemia, specifically Type I Hyperlipoproteinemia. Familial Hyperchylomicronemia, or Type I Hyperlipoproteinemia, is often caused by a deficiency in Lipoprotein Lipase (LPL) or its cofactor, apolipoprotein C-II (apo C-II). LPL is crucial for the hydrolysis of triglycerides in chylomicrons and very low-density lipoproteins (VLDLs), allowing the release of free fatty acids. Presentation: Elevated triglyceride levels can lead to the formation of chylomicrons, causing pancreatitis as they accumulate in the pancreas. These are lipid deposits in the skin resulting from the deposition of chylomicrons in the dermis. Markedly elevated triglycerides, as observed in the patient, are characteristic of familial hyperchylomicronemia. The milky appearance of the plasma is due to the presence of excessive chylomicrons. Treatment: Reduction of dietary fat. Fibrates and omega-3 fatty acid supplements to reduce triglyceride levels. LDL receptor (Option B): LDL receptor defect leads to accumulation of LDL, not TGs HMG CoA reductase (Option C): It is a rate-limiting enzyme of cholesterol biosynthesis. Complete deficiency of HMG CoA reductase is lethal and Incompatible with life ABCA1 receptor (Option D): ABCA1 defect leads to Tangier’s disease with decreased HDL

References:

Harper's Illustrated Biochemistry, 31st Edition, Page 258

↑ *Back to this question in Part A*

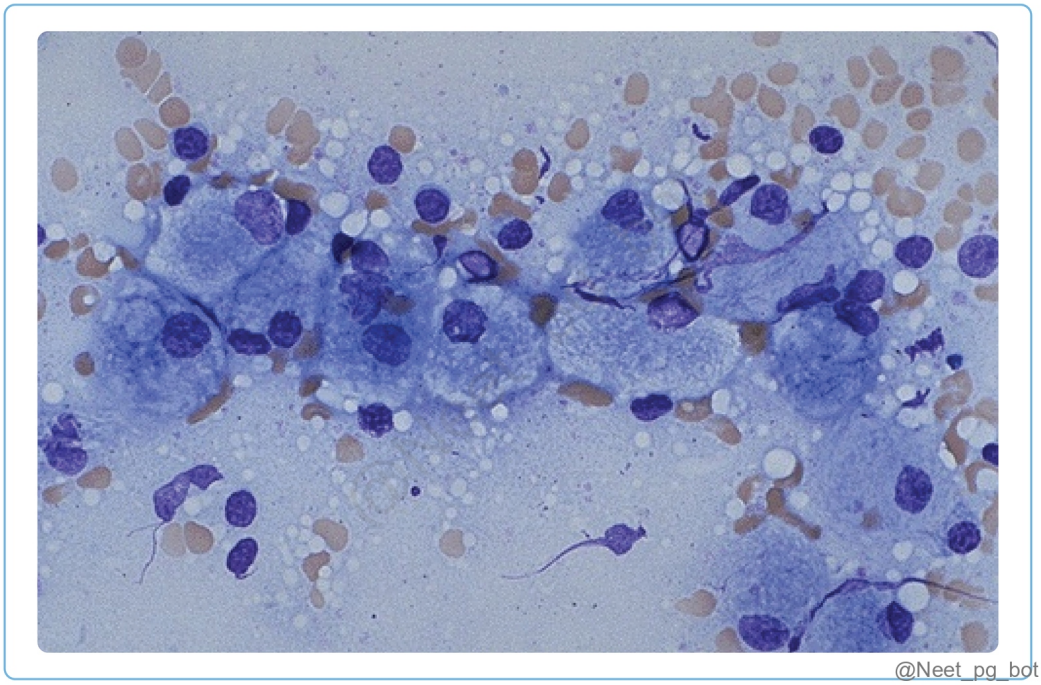
Q2: A patient presents with bone pain, fatigue and hepatosplenomegaly. Blood tests reveal anemia and thrombocytopenia. Bone marrow biopsy shows macrophages with a characteristic "wrinkled paper" appearance. Which substrate accumulates in Gaucher's disease due to the deficient enzyme activity? (Q843064)

A. Sphingomyelin

B. Glucosylceramide

C. Ganglioside

D. Ceramide

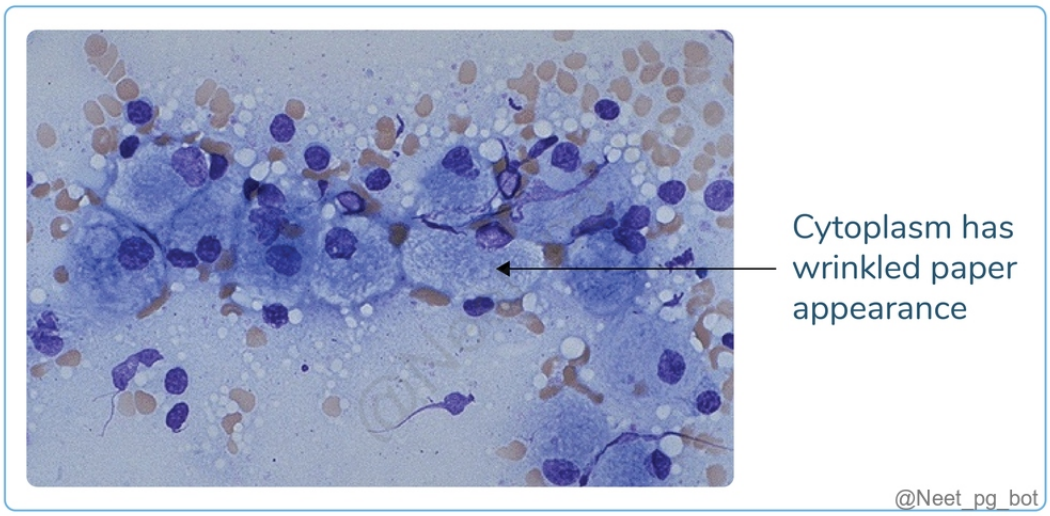


Correct Answer: B. Glucosylceramide

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Explanation: Gaucher's disease → deficiency of β-glucosidase (glucocerebrosidase) → accumulation of glucosylceramide . Features: hepatosplenomegaly, anemia, bone pain, "wrinkled paper" macrophages. Takeaway: Gaucher's = β-glucosidase deficiency → glucosylceramide accumulation . Substrate Disease Where It Accumulates Enzyme Deficiency Key Clinical Features Sphingomyelin (Option A) Niemann-Pick disease Sphingomyelinase Neurodegeneration, hepatosplenomegaly, cherry-red spot Glucosylceramide (Option B) Gaucher disease β-glucocerebrosidase Bone pain, anemia, HSM, wrinkled paper macrophages Ganglioside (GM2) (Option C) Tay-Sachs disease Hexosaminidase A Neurodegeneration, cherry-red spot , no HSM Ceramide (Option

D) Farber disease Ceramidase Painful joints, subcutaneous nodules, hoarseness



References:

www.ncbi.nlm.nih.gov/books/NBK448080/

Harperis's Illustrated Biochemistry,32nd Edition, Page 245

Vasudevan, 7th Edition, Page 194

↑ *Back to this question in Part A*

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Q3: In a patient with a history of recurring attacks of pancreatitis, eruptive xanthomas, increased plasma triglyceride levels (2,000 mg/dL), and milky plasma. Which of the following is the most likely deficiency? (Q882162)

A. Lipoprotein Lipase

B. LDL receptors

C. HMG-CoA reductase

D. ABCA1 receptor

Correct Answer: A. Lipoprotein Lipase

Explanation: The patient's presentation, including recurring attacks of pancreatitis, eruptive xanthomas, markedly increased plasma triglyceride levels (2,000 mg/dL), and milky plasma, is suggestive of Familial Hyperchylomicronemia, specifically Type I Hyperlipoproteinemia. Familial Hyperchylomicronemia, or Type I

Hyperlipoproteinemia, is often caused by a deficiency in Lipoprotein Lipase (LPL) or its cofactor, apolipoprotein C-II (apo C-II). LPL is crucial for the hydrolysis of triglycerides in chylomicrons and very low-density lipoproteins (VLDLs), allowing the release of free fatty acids. Presentation: Elevated triglyceride levels can lead to the formation of chylomicrons, causing pancreatitis as they accumulate in the pancreas. These are lipid deposits in the skin resulting from the deposition of chylomicrons in the dermis. Markedly elevated triglycerides, as observed in the patient, are characteristic of familial hyperchylomicronemia. The milky appearance of the plasma is due to the presence of excessive chylomicrons. Treatment: Reduction of dietary fat. Fibrates and omega-3 fatty acid supplements to reduce triglyceride levels. LDL receptor (Option B): LDL receptor defect leads to accumulation of LDL, not TGs HMG CoA reductase (Option C): It is a rate-limiting enzyme of cholesterol biosynthesis. Complete deficiency of HMG CoA reductase is lethal and Incompatible with life ABCA1 receptor (Option D): ABCA1 defect leads to Tangier's disease with decreased HDL

References:

Harper's Illustrated Biochemistry, 31st Edition, Page 258

↑ *Back to this question in Part A*

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Q4: Which enzyme in snake venom hydrolyzes only sn-2 acyl bond of phospholipids? (Q843438)

A. Phospholipase A2

B. Phospholipase B

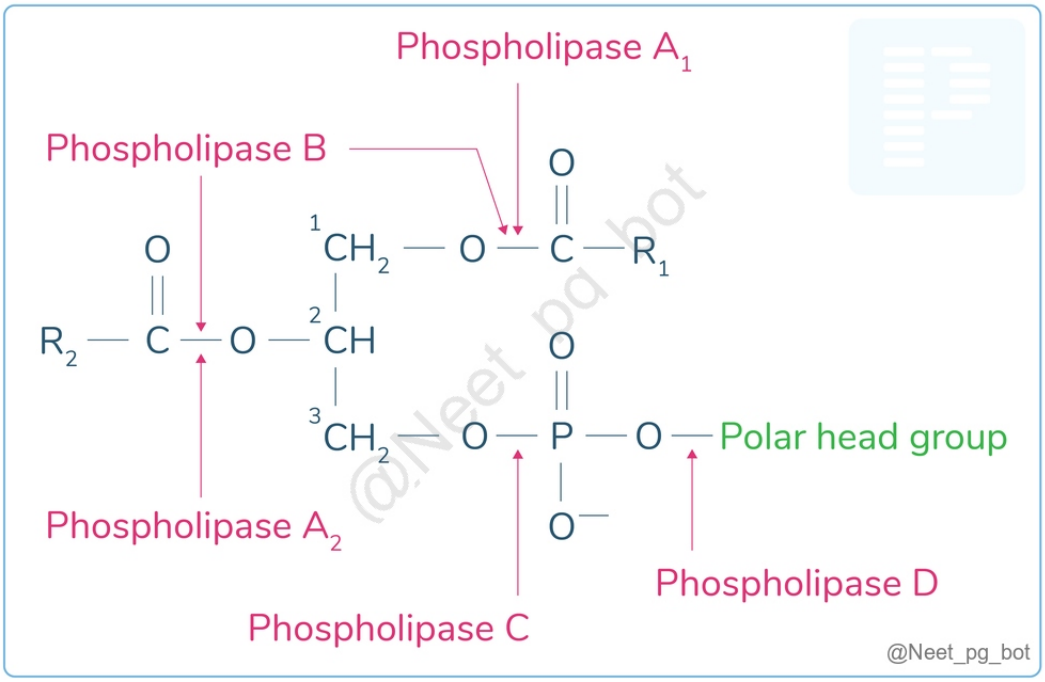
C. Phospholipase C

D. Phospholipase D

Correct Answer: A. Phospholipase A2

■ *Listen to Audio 1*

Explanation: Phospholipase A2 acts on the 2nd carbon atom causing hydrolysis of the ester bond in phospholipids leading to cell membrane disruption and inflammation. Phospholipase A2: (Option A) Acts at the 2nd carbon atom. Hydrolysis of the ester bond in phospholipids Release of arachidonic acid. Causes cell membrane disruption. Phospholipase B (Option B) has both PLA1 and PLA2 activities. Hence, it can hydrolyze both the sn-1 and sn-2 acyl bonds of phospholipids. Phospholipase C (Option C) hydrolyzes the phosphodiester bond of phospholipids, particularly phosphatidylinositol. Phospholipase D (Option D) hydrolyzes the phosphodiester bond at the phosphate group.



References:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3873700/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q5: Which of the following fatty acids is needed for humans from diet as humans cannot create double bonds beyond the 9th carbon from the carboxyl end of a fatty acid chain? (Q843434)

A. Alpha-linolenic acid

B. Palmitic acid

C. Oleic acid

D. Stearic acid

Correct Answer: A. Alpha-linolenic acid

■ *Listen to Audio 1*

Explanation: Alpha-linolenic acid is an essential fatty acid. It cannot be synthesized by the human body and must be obtained through the diet Essential fatty acids Non-essential fatty acids Cannot be synthesized by the human body and must be obtained through the diet. Humans cannot create double bonds beyond the 9th carbon atom from

the carboxyl end of a fatty acid chain. Linoleic Acid (LA): An omega-6 fatty acid. Alpha-linolenic acid (ALA) : An omega-3 fatty acid. (Option A) Can be synthesized by the human body from other dietary fats Palmitic acid (Option B ruled out) Oleic acid (Option C ruled out) Stearic acid (Option D ruled out) Arachidonic acid Eicosapentaenoic acid (EPA) Docosahexaenoic acid (DHA) Gamma-linolenic acid (GLA)

References:

Harper Biochemistry, 32nd Edition, Page. 232, 233

↑ *Back to this question in Part A*

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Q6: Which of the following lipids is characterized by its amphipathic nature, allowing it to form micelles in aqueous solutions? (Q843070)

A. Triacylglycerols

B. Cholesteryl esters

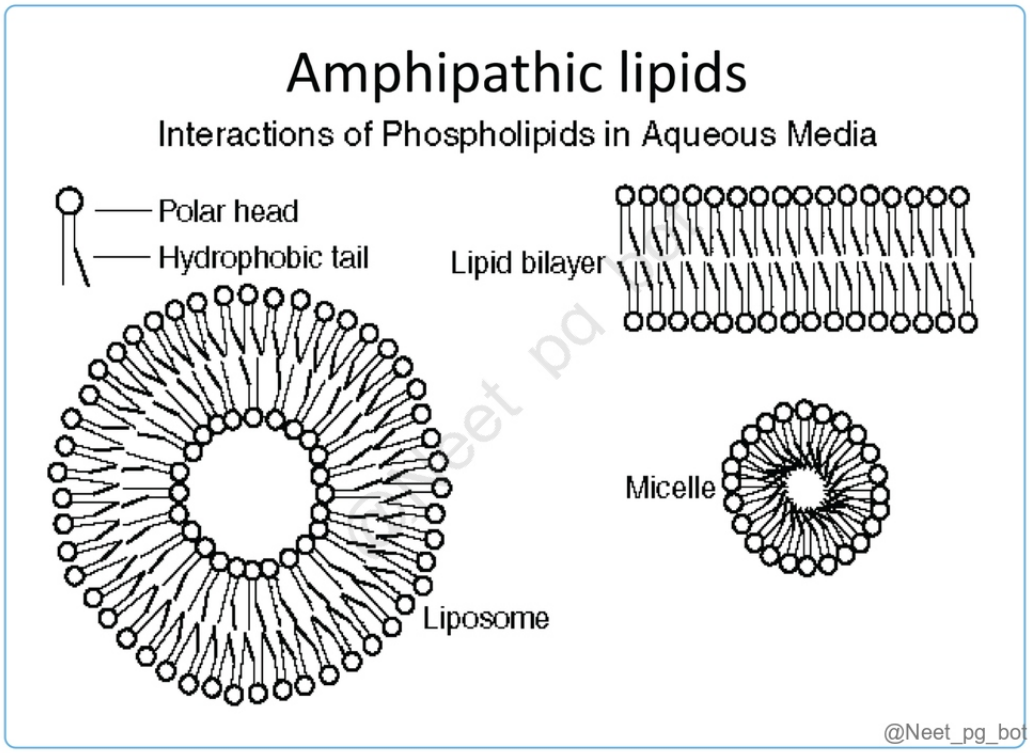
C. Phospholipids

D. Nonpolar lipids

Correct Answer: C. Phospholipids

■ *Listen to Audio 1*

Explanation: Phospholipids are amphipathic lipids with both hydrophobic and hydrophilic properties. They have a polar head group (hydrophilic) and nonpolar tails (hydrophobic), enabling them to form micelles when in aqueous solutions. This property is crucial for various biological functions, including fat digestion and membrane formation. Lipids Categorized by Solubility: 1. Nonpolar Lipids: (Option D ruled out) Characteristics: Hydrophobic, insoluble in water, dominated by nonpolar hydrocarbon groups. Examples: Triacylglycerols (Triglycerides): Esters of glycerol with three fatty acid chains; primary fat storage in adipose tissue. (Option A ruled out) Cholesteryl Esters: Cholesterol esterified to a fatty acid; used for cholesterol storage and transport. (Option B ruled out) 2. Amphipathic Lipids: Characteristics: Both hydrophobic and hydrophilic; contain a polar head group and a nonpolar tail. Functions and Structures: Membranes: Bilayer structure with hydrophobic tails inward and hydrophilic heads outward Micelles: Spherical structures with hydrophobic tails in the center and hydrophilic heads on the surface; aid in fat digestion. Liposomes: Spherical vesicles with lipid bilayers enclosing an aqueous compartment Emulsions: Larger particles of nonpolar lipids dispersed in water, stabilized by amphipathic lipids; prevent coalescence. Examples: Phospholipids (Option C) Sphingolipids Cholesterol



References:

Harper 32nd edition page 205, 206, 209, 215, 264, 248, 259.

↑ *Back to this question in Part A*

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Q7: Which of the following pairs correctly associates a disease with its corresponding enzyme deficiency? (Q843068)

A. Tay-Sachs disease - α-Galactosidase

B. Gaucher disease - β-Glucosidase

C. Farber disease - Sphingomyelinase

D. Fabry disease - Ceramidase

Correct Answer: B. Gaucher disease - β-Glucosidase

■ *Listen to Audio 1*

Explanation: Gaucher disease is caused by a deficiency of β-Glucosidase, leading to the accumulation of glucosylceramide. Disease Enzyme Deficiency Tay-Sachs disease Hexosaminidase A Fabry disease

α-Galactosidase Metachromatic leukodystrophy Arylsulfatase A Krabbe disease β-Galactocerebrosidase Gaucher disease β-Glucosidase Niemann-Pick disease types A, B Sphingomyelinase Farber disease Ceramidase

References:

Harper 32nd edition page 245

↑ *Back to this question in Part A*

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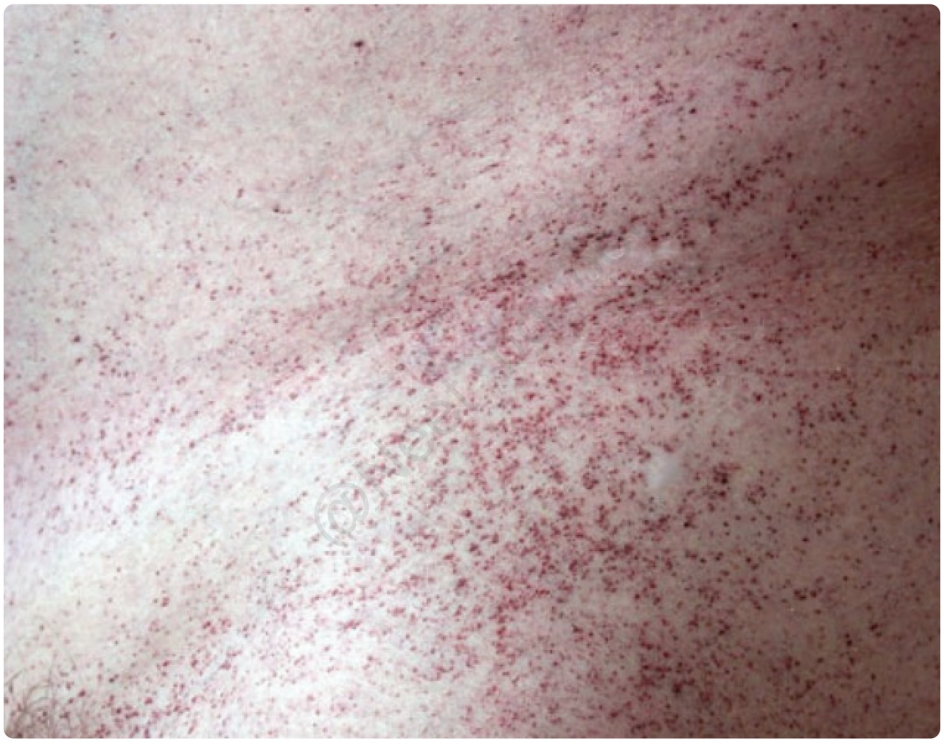
Q8: A 9-year-old boy presents with episodic pain in his extremities, skin finding as shown in the image, and progressive renal dysfunction. A biopsy of the skin reveals the accumulation of ceramide trihexoside. An α-galactosidase deficiency is suspected. What is the diagnosis? (Q843067)

A. Tay-Sachs disease

B. Fabry disease

C. Krabbe disease

D. Gaucher disease



Correct Answer: B. Fabry disease

■ *Listen to Audio 1*

Explanation: Fabry disease is due to a deficiency of α-galactosidase , leading to the accumulation of ceramide trihexoside , resulting in symptoms such as episodic pain, angiokeratomas (as shown in the image), and renal dysfunction. Classical Clinical Features: Acroparesthesia: Episodic burning pain in hands & feet (early symptom) Angiokeratomas: Dark red-purple skin lesions (trunk, groin) Renal failure: Progressive → proteinuria → ESRD Hypohidrosis: ↓ Sweating Heat intolerance: Due to autonomic dysfunction Diagnostic Findings: Urine microscopy: Maltese cross bodies (fat globules) Eye exam: Cornea verticillata Enzyme assay: ↓ α-galactosidase activity Genetic testing: Confirms diagnosis Disease Enzyme Deficiency Lipid Accumulating Clinical Symptoms Tay-Sachs disease (Option A ruled out) Hexosaminidase A, S Cer—Glc—Gal(NeuAc) GalNAc GM2 Ganglioside Mental retardation, blindness, and muscular weakness Cherry red spot Fabry disease (Option B) α-Galactosidase Cer—Glc—Gal—Gal Globotriaosylceramide Skin rash (Angiokeratoma) , kidney failure (symptoms manifest fully only in males; X-linked recessive) Metachromatic leukodystrophy Arylsulfatase A Cer—Gal—OSO3 3-Sulfogalactosylceramide Mental retardation and psychological disturbances in adults; demyelination Krabbe disease (Option C ruled out) β-Galactosidase Cer—Gal Galactosylceramide Mental retardation; myelin is almost absent Gaucher disease (Option D ruled out) β-Glucosidase Cer—Glc Glucosylceramide Enlarged liver and spleen, erosion of long bones, mental retardation in infants Niemann-Pick disease types A, B Sphingomyelinase Cer—P—choline Sphingomyelin Enlarged liver and spleen, mental retardation; fatal in early life Cherry red spot Farber disease Ceramidase Acyl—Sphingosine Ceramide Hoarseness, dermatitis, skeletal deformation, mental retardation; fatal in early life Abbreviations: Cer, ceramide; Gal, galactose; Glc, glucose; NeuAc, N-acetylneuraminic acid; site of deficient enzyme reaction.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 245

↑ *Back to this question in Part A*

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Q9: A 6-month-old infant presents with a hoarse cry, painful and swollen joints, and skin nodules. The child also shows signs of developmental delay and mental retardation. Histological analysis reveals the accumulation of a certain substance in the tissues, which confirms the diagnosis of Farber disease. Which enzyme deficiency is responsible for this condition? (Q843066)

A. Hexosaminidase A

B. Arylsulfatase A

C. Ceramidase

D. Sphingomyelinase

Correct Answer: C. Ceramidase

■ *Listen to Audio 1*

Explanation: Farber disease is caused by a deficiency of ceramidase, leading to the accumulation of ceramide and presenting with a hoarse cry, painful and swollen joints, skin nodules, and mental retardation. Disease Enzyme Deficiency Clinical Symptoms Tay-Sachs disease Hexosaminidase A, S (Option A ruled out) Mental retardation, blindness, muscular weakness Fabry disease α-Galactosidase Skin rash, kidney failure (symptoms manifest fully only in males; X-linked recessive) Metachromatic leukodystrophy Arylsulfatase A (Option B ruled out) Mental retardation and psychological disturbances in adults; demyelination Krabbe disease β-Galactosidase Mental retardation; myelin almost absent Gaucher disease β-Glucosidase Enlarged liver and spleen, erosion of long bones, mental retardation in infants Niemann-Pick disease types A, B Sphingomyelinase (Option D ruled out) Enlarged liver and spleen, mental retardation; fatal in early life Farber disease Ceramidase (Option C) Hoarseness, dermatitis, skeletal deformation, mental retardation; fatal in early life Abbreviations: Cer, ceramide; Gal, galactose; Glc, glucose; NeuAc, N-acetylneuraminic acid; , site of deficient enzyme reaction.

References:

Harper 32nd edition page 245

↑ *Back to this question in Part A*

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Q10: A 6-month-old infant presents with hepatosplenomegaly, progressive neurodegeneration, and a history of frequent respiratory infections. A biopsy shows lipid-laden macrophages and a genetic test confirms the diagnosis. Which of the following enzyme deficiencies is most likely responsible for this condition? (Q843065)

A. Hexosaminidase A

B. Arylsulfatase A

C. Sphingomyelinase

D. β-Glucosidase

Correct Answer: C. Sphingomyelinase

■ *Listen to Audio 1*

Explanation: Sphingomyelinase deficiency is responsible for Niemann-Pick disease types A and B, which present with hepatosplenomegaly, and neurodegeneration, biopsy shows lipid-laden macrophages (foam cells) and are often fatal in early life. Disease Enzyme Deficiency Clinical Symptoms Tay-Sachs disease Hexosaminidase A, S (Option A ruled out) Mental retardation, blindness, muscular weakness Fabry disease α-Galactosidase Skin rash, kidney failure (symptoms manifest fully only in males; X-linked recessive) Metachromatic leukodystrophy Arylsulfatase A (Option B ruled out) Mental retardation and psychological disturbances in adults; demyelination

Krabbe disease Galactocerebrosidase Mental retardation; myelin almost absent Gaucher disease β-Glucosidase (Option D ruled out) Enlarged liver and spleen, erosion of long bones, mental retardation in infants Niemann-Pick disease types A, B Sphingomyelinase (Option C) Enlarged liver and spleen, mental retardation; fatal in early life Farber disease Ceramidase Hoarseness, dermatitis, skeletal deformation, mental retardation; fatal in early life Abbreviations: Cer, ceramide; Gal, galactose; Glc, glucose; NeuAc, N-acetylneuraminic acid; , site of deficient enzyme reaction.

References:

Harper 32nd edition page 245

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: Which of the following correctly matches the type of lipid with its description? (Q843051)

A. Fats - Liquid at room temperature, often containing polyunsaturated fatty acids.

B. Phospholipids - Contain glycerol and are the main lipid constituents of cell membranes.

C. Glycolipids - Esters of fatty acids that never contain sphingosine.

D. Sphingophospholipids - Cerebrosides is a subtype

Correct Answer: B. Phospholipids - Contain glycerol and are the main lipid constituents of cell membranes.

■ *Listen to Audio 1*

Explanation: Classification of lipids 1.Simple Lipids Esters of fatty acids with various alcohols. Further classified into fats, oils & waxes Fats Esters of fatty acids with glycerol; solid at room temperature. (Option A ruled out) Triacylglycerol with saturated fatty acids. Oils Fats in liquid state at room temperature. Triacylglycerols with polyunsaturated fatty acids. Waxes Esters of fatty acids with higher molecular weight monohydric alcohols. 2.Complex Lipids Esters of fatty acids with alcohol and other groups. Further classified based on additional groups present as Phospholipids, glycolipids and other complex lipids Phospholipids: Contain phosphoric acid; Main lipid constituents of membranes. (Option B) Glycerophospholipids Alcohol is glycerol. Phosphatidylcholine, phosphatidylethanolamine. Sphingophospholipids Alcohol is sphingosine Sphingomyelin, found in myelin sheaths. Glycolipids: (glycosphingolipids) Contain fatty acid, sphingosine, and carbohydrate; Involved in cell adhesion and recognition (Option C ruled out) Cerebrosides (Option D ruled out) Galactosylceramide, a key component of myelin. Globosides Cell adhesion and Pathogen identification Gangliosides Highly concentrated in nerve tissues and function in cell-cell recognition, communication, and as receptors for hormones and bacterial toxins. 3.Precursor/Derived Lipids Formed from hydrolysis of simple and complex lipids; Also act as precursors for lipid formation. Fatty acids, cholesterol, fatty aldehydes, ketone bodies, hydrocarbons, lipid-soluble vitamins, hormones.

References:

Harper’s biochemistry 32nd edition page 206,209,212

↑ *Back to this question in Part A*

@Neet_pg_bot

Q12: A 4-month-old infant presents developmental delay. MRI shows demyelination in the central nervous system. Which of the following is the most likely diagnosis, and what enzyme deficiency causes this condition? (Q843063)

A. Niemann-Pick disease, Sphingomyelinase

B. Krabbe disease, β-Galactosidase

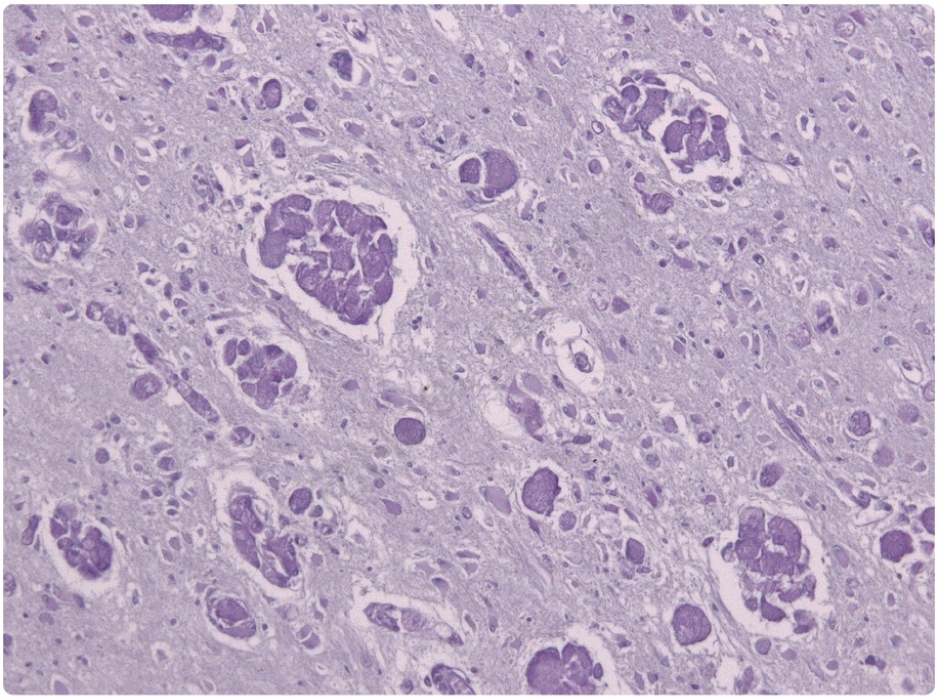
C. Farber disease, Ceramidase

D. Gaucher disease, β-Glucosidase

Correct Answer: B. Krabbe disease, β-Galactosidase

■ *Listen to Audio 1*

Explanation: An infant with developmental delay and associated MRI findings of demyelination points towards the diagnosis of Krabbe's disease, also known as globoid cell leukodystrophy, an autosomal recessive fatal disorder of infancy. Krabbe disease occurs due to defects in β-Galactosidase (or galactocerebrosidase) encoded by GALC gene, leading to white matter accumulation of galactosylceramide, which is normally found almost exclusively in the myelin sheath. Both peripheral and central myelin are affected, resulting in spasticity and cognitive impairment coupled with deceptively normal or even absent deep tendon reflexes. The infantile form of Krabbe disease is rapidly progressive, and patients present in early infancy with irritability, seizures, and hypertonia. Optic atrophy is evident in the first years of life, and cognitive development is severely impaired. As the disease progresses, optic atrophy and severe developmental delay become apparent; affected children exhibit opisthotonos and typically die before 3 years of age. Histopathology demonstrates typical globoid bodies: On imaging of brain, thalamic hyperdensities are also seen. Disease Enzyme deficiency Features Tay-Sachs disease Hexosaminidase A, Mental retardation Blindness Muscular weakness Cherry red spots in macula Fabry disease α-Galactosidase Skin rash(angiokeratoma) Kidney failure (full symptoms only in males - X-linked recessive) Krabbe disease (Option B) β-Galactosidase Mental retardation Near absence of myelin Predominant neurological manifestation and cranial nerves causing deafness and blindness. Gaucher disease (Option D ruled out) β-Glucosidase Enlarged liver and spleen Erosion of long bones Mental retardation in infants Anemia Thrombocytopenia Niemann-Pick disease (Option A ruled out) Sphingomyelinase Enlarged liver and spleen Mental retardation Fatal in early life Cherry red spot in macula Farber disease (Option C ruled out) Ceramidase Hoarseness Dermatitis Skeletal deformation Mental retardation Fatal in early life



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 245. Vasudevan's Textbook of Biochemistry, 7th Edition, Page 194.

↑ *Back to this question in Part A*

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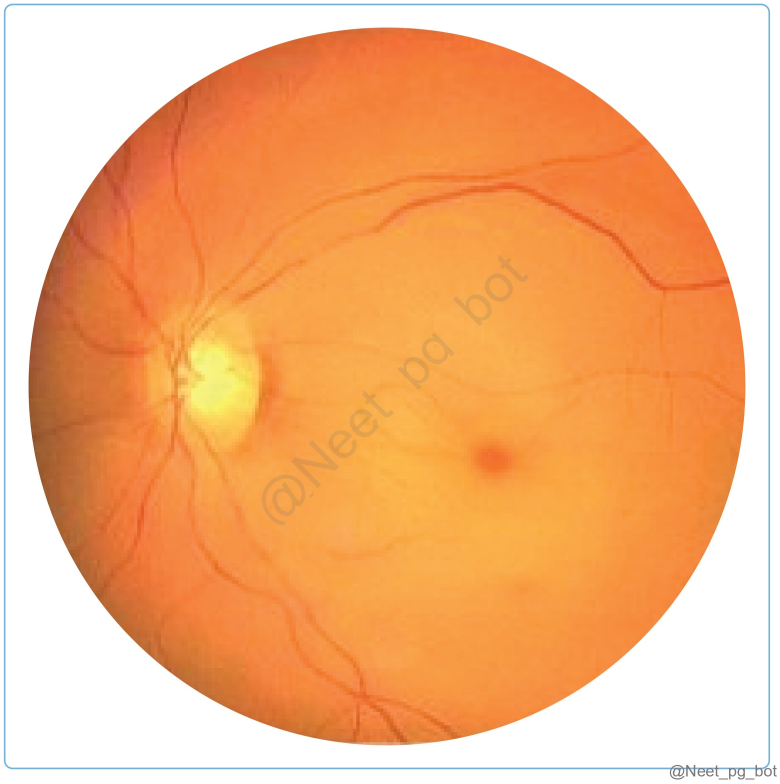
Q13: A 6-month-old infant has developmental delay, muscle weakness, and on eye examination, a cherry-red spot on the macula is noted. What is the most likely diagnosis, and which enzyme deficiency is responsible? (Q843062)

A. Niemann-Pick disease, Sphingomyelinase

B. Gaucher disease, Glucocerebrosidase

C. Tay-Sachs disease, Hexosaminidase A

D. Sandhoff disease, Beta-galactosidase



Correct Answer: C. Tay-Sachs disease, Hexosaminidase A

■ *Listen to Audio 1*

Explanation: In this scenario, the clinical features of developmental delay and muscular weakness, and fundoscopic image show cherry red spot in the macula are consistent with the diagnosis of Tay-Sachs disease. It is an inborn error of metabolism due to the failure of degradation of gangliosides, which is caused by a deficiency in the enzyme hexosaminidase A. Lipid Storage Diseases (Sphingolipidoses): They are a group of lysosomal storage diseases. Disease Enzyme deficiency Features Tay-Sachs disease (Option C) Hexosaminidase A Mental retardation Blindness Muscular weakness Cherry red spots in the macula Fabry disease α-Galactosidase Skin rash Kidney failure (full symptoms only in males X-linked recessive) Krabbe disease β-Galactosidase Mental retardation Near absence of myelin Predominant neurological manifestation and cranial nerves causing deafness and blindness. Gaucher disease (Option B ruled out) β-Glucosidase Enlarged liver and spleen Erosion of long bones Mental retardation in infants Anemia Thrombocytopenia Niemann-Pick disease (Option A ruled out) Sphingomyelinase Enlarged liver and spleen Mental retardation Fatal in early life Cherry red spot in macula Farber disease Ceramidase Hoarseness Dermatitis Skeletal deformation Mental retardation Fatal in early life Sandhoff disease (Option D): Lysosomal storage disease. Deficient enzyme: Hexosaminidase B Accumulation of GM2 ganglioside and goboside Cherry red spot is also seen in: Central retinal artery occlusion (CRAO) Tay-Sach's disease Sandhoff's disease Mucopolysaccharidoses Gaucher's disease

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 245.

https://www.ncbi.nlm.nih.gov/books/NBK564432/

↑ *Back to this question in Part A*

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Q14: Which enzyme deficiency is responsible for GM1 gangliosidosis? (Q843060)

A. Beta-glucosidase

B. Beta-galactosidase

C. Alpha-galactosidase

D. Hexosaminidase

Correct Answer: B. Beta-galactosidase

■ *Listen to Audio 1*

Explanation: Beta-galactosidase removes galactose in GM 1 ganglioside and converts to GM 2 ganglioside . In the absence of the enzyme, it causes GM 1 gangliosidosis. Disease Enzyme deficiency Features Tay-Sachs disease (GM2 Gangliosidoses) Hexosaminidase A (Option D ruled out) Mental retardation Blindness Muscular weakness Cherry red spots in macula Fabry disease α-Galactosidase (Option C ruled out) Angiokeratomas (reddish purple spots) Kidney failure (full symptoms only in males X-linked recessive) GM1 gangliosidosis β-Galactosidase (Option B) Mental retardation Organomegaly Skeletal deformities Gaucher disease β-Glucosidase (Option A ruled out) Enlarged liver and spleen Erosion of long bones Mental retardation in infants Anemia Thrombocytopenia Niemann-Pick disease Sphingomyelinase Enlarged liver and spleen Mental retardation Fatal in early life Note: GM1 ganglioside is the primary surface receptor on intestinal epithelial cells for cholera toxin, triggering the severe diarrhea (rice-water diarrhea) characteristic of the disease. The toxin's B-subunits bind specifically to GM1, facilitating endocytosis and intracellular transport to the endoplasmic reticulum, where it activates adenylate cyclase and causes massive water/electrolyte loss.

References:

Harper 32nd edition page 245

↑ *Back to this question in Part A*

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Q15: Which component is present in gangliosides but not in cerebrosides or globosides? (Q843059)

A. Monosaccharides

B. Ceramide

C. Fatty acid

D. N-acetyl Neuraminic Acid

Correct Answer: D. N-acetyl Neuraminic Acid

■ *Listen to Audio 1*

Explanation: Gangliosides contain oligosaccharides as part of their structure, whereas cerebrosides contain only a single monosaccharide attached to ceramide. Ganglioside is a glycolipid, which is made of ceramide, oligosaccharide with NANA (N-acetyl Neuraminic Acid). Glycolipids: Also known as glycosphingolipids as alcohol in glycolipids are sphingosine. Glycosphingolipids are a combination of (Option B ruled out) ceramide with one or more sugar residues. Examples: cerebroside, globoside, and ganglioside. Glycolipids Composition Cerebroside Ceramide + monosaccharide (glucose or galactose) (Option A ruled out) Globoside Ceramide + oligosaccharide (glucose and galactose) Ganglioside Ceramide + oligosaccharide + NANA (N-acetyl Neuraminic Acid) (Option D) Ceramide: Ceramide is synthesised in the endoplasmic reticulum from the amino acid serine. Ceramide = Fatty acid (Option C ruled out) + Sphingosine

References:

Harper 32nd edition page 243, 244

↑ *Back to this question in Part A*

@Neet_pg_bot

Q16: Which of the following are the correct components of sphingomyelin? (Q843057)

A. Sphingosine + Fatty acid + Choline

B. Ceramide + Phosphate + Choline

C. Sphingosine + Phosphate + Choline

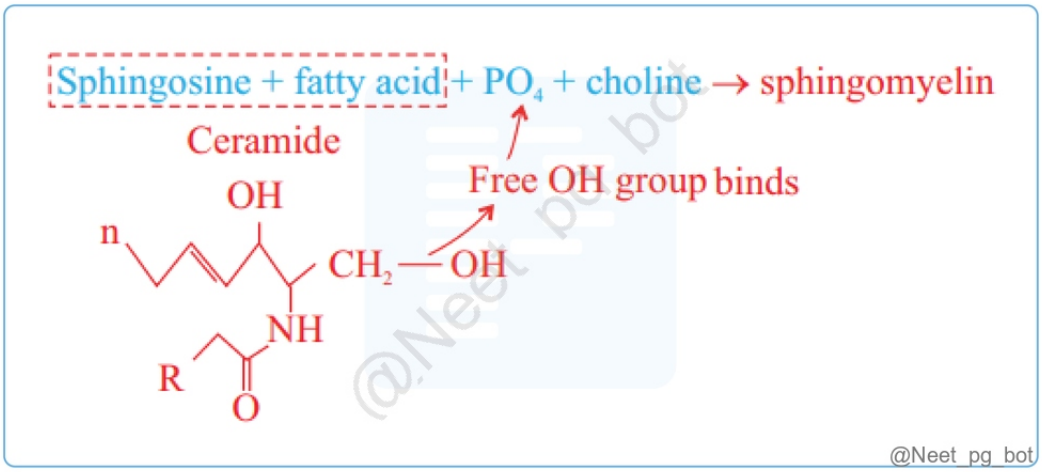
D. Ceramide + Phosphate + Fatty acids

Correct Answer: B. Ceramide + Phosphate + Choline

■ *Listen to Audio 1*

Explanation: Sphingosine and fatty acids combine to form Ceramide . The phosphate group attaches to the free OH group in ceramide to create its derivative. Choline, when added to this derivative, forms Sphingomyelin Sphingomyelin is a type of lipid classified as a phospholipid and is an important component of cell membranes. Abundant in the myelin sheath that surrounds nerve fibers. Found in the outer leaflet of the cell membrane lipid bilayer and is a key component of lipid rafts. Plays an important role in cell signaling and apoptosis. Synthesis occurs primarily in the Golgi apparatus, Incorrect Options Sphingosine + Fatty acid + Choline (Option A): This combination forms ceramide and choline but lacks the phosphate group , which is essential for sphingomyelin. Hence, incorrect. Sphingosine + Phosphate + Choline (Option C): Missing the fatty acid component that binds to

sphingosine to form ceramide. So, it is incomplete. Incorrect. Ceramide + Phosphate + Fatty acids (Option D): Includes two fatty acids , which is incorrect. Only one fatty acid is present in ceramide; the head group should be choline , not another fatty acid.



References:

Harper 32nd edition page 210-211

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: A 30-year-old woman presents with a history of four consecutive miscarriages over the past three years. After a thorough evaluation, a diagnosis is made . Which of the following phospholipids is specifically antigenic in this disease? (Q843056)

A. Phosphatidylserine

B. Phosphatidylinositol

C. Cardiolipin

D. Phosphatidylethanolamine

Correct Answer: C. Cardiolipin

■ *Listen to Audio 1*

Explanation: The condition described is Antiphospholipid Syndrome (APS), a disorder characterized by recurrent miscarriages, thrombosis, and the presence of antiphospholipid antibodies. Cardiolipin is a phospholipid that becomes antigenic in Antiphospholipid Syndrome, leading to the production of anti-cardiolipin antibodies. Other phospholipids (Phosphatidylserine, Phosphatidylinositol, Phosphatidylethanolamine) are not antigenic

Phospholipid Location Function Synthesis Significance Cardiolipin (Option C) Inner mitochondrial membrane optimal function of numerous enzymes involved in mitochondrial energy metabolism. Stabilizes the respiratory chain complexes and supercomplexes Unique dimeric structure consisting of four fatty acid chains Synthesized in the mitochondria from phosphatidylglycerol and cytidine diphosphate-diacylglycerol (CDP-DAG) Antigenic Phosphatidylserine (Option A) is involved in cell signaling and apoptosis but is not antigenic. Phosphatidylinositol (Option B) is involved in cell signaling pathways and membrane dynamics but is not antigenic. Phosphatidylethanolamine (Option D) is a common phospholipid in cell membranes but is not antigenic.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 240, 241. Lippincott Illustrated Reviews, 7th Edition, 592-599.

↑ *Back to this question in Part A*

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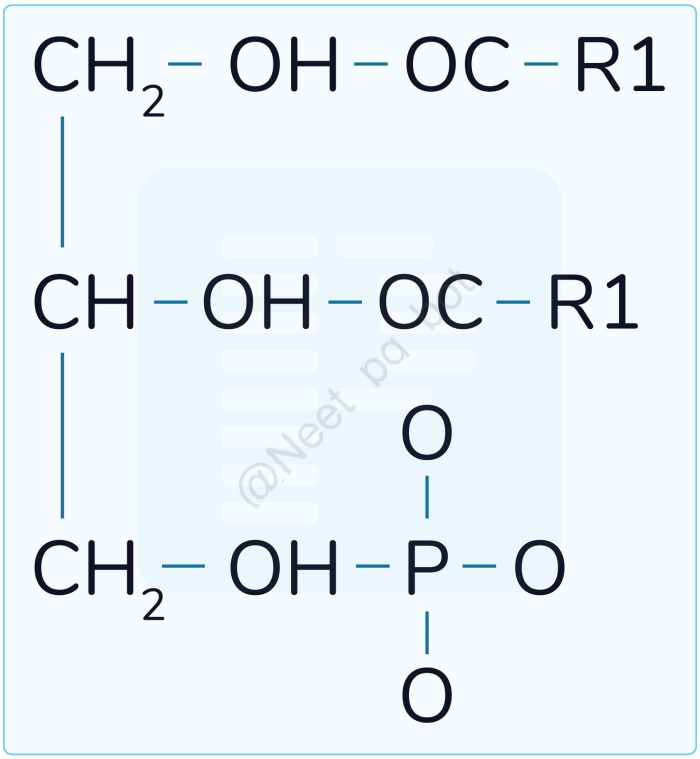
Q18: What compound is formed when choline is added to the following structure? (Q843054)

A. Lecithin

B. Cephalin

C. Cardiolipin

D. Inositol

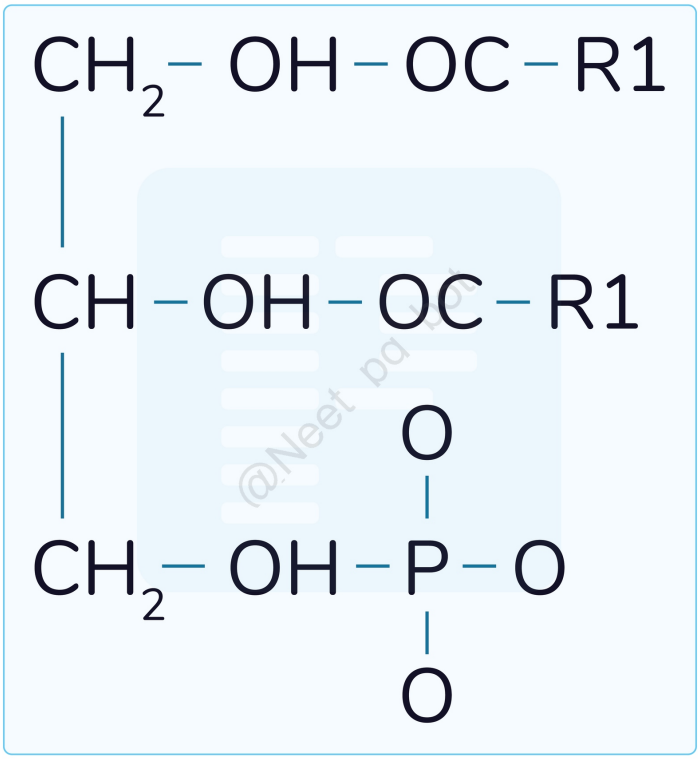


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Correct Answer: A. Lecithin

■ *Listen to Audio 1*

Explanation: This structure is called phosphatidate - phosphatide when combined with choline results in the formation of Phosphatidylcholine also known as Lecithin . Formation of Phosphotidate: When the first two OH groups of a glycerol molecule are esterified with fatty acids. And the third OH group bonds with a phosphate group This combination forms phosphatidate , which is a precursor to all glycerophospholipids. Lecithin: In phosphatidylcholine, if the first two fatty acids are palmitic acid, the compound is known as Dipalmitoyl phosphatidyl choline . This substance is a key component of pulmonary surfactant. It is synthesized by type 2 pneumocytes in the lungs. In the later stages of the third trimester, it is produced by fully developed fetal lungs. Premature neonates often lack this surfactant due to the underdevelopment of their lungs. A lecithin:sphingomyelin (L:S) ratio less than 2:1 poses a risk of developing respiratory distress syndrome in neonates. Phospholipids: Phospholipid Composition Lecithin (Option A) Free phosphate + choline Phosphatidylserine Free phosphate + Serine Phosphatidylinositol Free phosphate + Inositol (Option D ruled out) Cephalin (Option B ruled out) Free phosphate + Ethanolamine Cardiolipin (Option C ruled out) Combination of Free phosphate group of 2 phosphatidate



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References:

Harper's Illustrated Biochemistry, 32nd Edition. Pages 209-211.

↑ *Back to this question in Part A*

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Q19: A premature baby presents with infant respiratory distress syndrome. The neonatologist says the neonate is yet to produce a type of phospholipid adequately. All of the following belong to the same class except? (Q843052)

A. Cephalin

B. Cardiolipin

C. Lecithin

D. Cerebrosides

Correct Answer: D. Cerebrosides

■ *Listen to Audio 1*

Explanation: The neonate in respiratory distress syndrome fails to produce Lecithin (phosphatidylcholine), a phospholipid crucial for surfactant production. Cephalin, Cardiolipin, and Lecithin are all phospholipids. Cerebroside is a glycolipid and not a phospholipid. Classification of Phospholipids(esters containing phosphate groups with fatty acid and alcohol.) Glycerophospholipids Alcohol: Glycerol Phosphatidylcholine (Lecithin) (Option C ruled out) Most abundant in cell membranes, major source of choline. Phosphatidylethanolamine (Cephalin) (Option A ruled out) Found in membranes; differs from phosphatidylcholine by choline substitution. Phosphatidylserine Similar to phosphatidylethanolamine; found in cell membranes. Phosphatidylinositol Contains myoinositol. Cardiolipin (Option B ruled out) Found in mitochondria; synthesized from phosphatidylglycerol. Lysophospholipids Contains one acyl radical; e.g., lysophosphatidylcholine (lysolecithin). Plasmalogens 10–30% of phospholipids in brain/heart; ether link instead of ester. Sphingophospholipids Alcohol: Sphingosine Sphingomyelins Found in myelin sheaths and cell membrane lipid rafts.

References:

Harper’s biochemistry 32nd edition page 209- 210

↑ *Back to this question in Part A*

@Neet_pg_bot

• [03_Lipid Chemistry] 010 Fatty Acid Synthesis And Oxidation - I

« Back to Folder Directory

[03_Lipid Chemistry] 010 Fatty Acid Synthesis And Oxidation - I — Quick Jump

Q1 (p. 656)	*Q2 (p. 657)*	*Q3 (p. 658)*	*Q4 (p. 659)*	*Q5 (p. 660)*	*Q6 (p. 661)*
Q7 (p. 662)	*Q8 (p. 663)*	*Q9 (p. 664)*	*Q10 (p. 665)*	*Q11 (p. 666)*	*Q12 (p. 667)*
Q13 (p. 668)	*Q14 (p. 668)*	*Q15 (p. 669)*	*Q16 (p. 670)*	*Q17 (p. 670)*	*Q18 (p. 672)*
Q19 (p. 672)	*Q20 (p. 673)*	*Q21 (p. 674)*	*Q22 (p. 675)*	*Q23 (p. 675)*	*Q24 (p. 676)*

Q1: Which of the following is an end product of beta-oxidation of odd-chain fatty acids? (Q845005)

A. Propionyl-CoA

B. Malonyl CoA

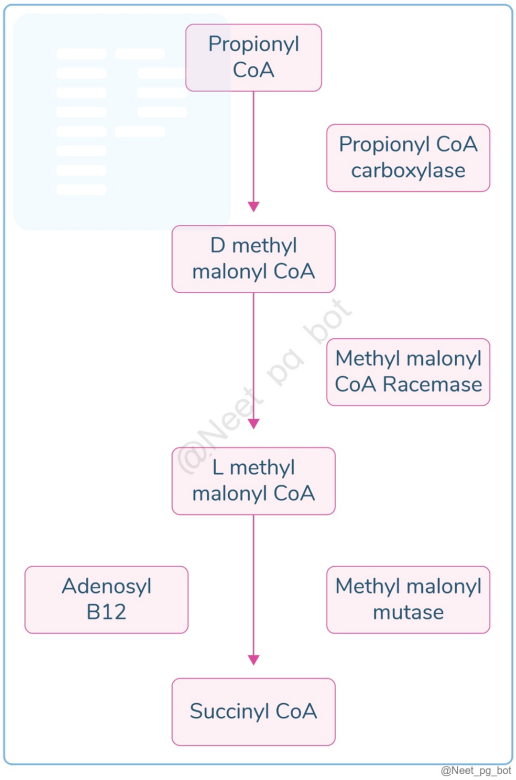
C. Succinyl CoA

D. Pyruvate

Correct Answer: A. Propionyl-CoA

■ *Listen to Audio 1*

Explanation: Propionyl-CoA is the actual end product of beta-oxidation of odd-chain fatty acids. The last cycle of beta-oxidation of odd-chain fatty acids produces propionyl-CoA, which is then converted to succinyl-CoA. β-Oxidation of Odd-Chain Fatty Acids: Proceeds like even-chain oxidation, yielding acetyl-CoA units until a final propionyl-CoA remains. Fate of Propionyl-CoA: 1. Carboxylation: → D-methylmalonyl-CoA (biotin-dependent). 2. Racemization: → L-methylmalonyl-CoA. 3. Rearrangement: → Succinyl-CoA (vitamin B■■–dependent). 4. TCA entry → Oxaloacetate → Gluconeogenesis.



References:

Vasudevan Biochemistry, 7th Edition, Pages 154, 155.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: Which of the following organs can utilize ketone bodies for energy, and which enzyme is involved in their metabolism? (Q845004)

A. Liver- Thiophorase

B. Heart muscle- Acetyl-CoA carboxylase

C. Brain- Thiophorase

D. Liver- Carboxylase

Correct Answer: C. Brain- Thiophorase

■ *Listen to Audio 1*

Explanation: The brain can use ketone bodies as an energy source, and Thiophorase is the enzyme that converts acetoacetate to acetoacetyl CoA , which is utilized by the brain . KETOSIS Ketone Bodies Produced in the liver Utilized by extrahepatic tissues due to the absence of Thiophorase enzyme in the liver Tissues that use Ketone Bodies Almost all tissues except: Liver Red blood cells (RBCs) Tissues that prefer ketone bodies: Heart muscle Renal cortex Placenta Tissues that use ketone bodies only in the absence of glucose: Skeletal muscle Brain Additional Tissues Using Ketone Bodies Intestinal mucosal cells Adipocytes Enzyme Involved Thiophorase : Converts acetoacetate to acetoacetyl CoA Fatty Acids as Primary Energy Source During Starvation Skeletal muscles Heart Liver Energy Production Pathway Acetoacetyl CoA enters the beta-oxidation pathway to produce energy Liver- Thiophorase (Option A): The liver produces ketone bodies but does not utilize them due to the absence of the enzyme Thiophorase. Thiophorase is involved in ketone body metabolism but is not present in the liver. Heart muscle- Acetyl-CoA carboxylase (Option B): While the heart muscle can utilize ketone bodies, the enzyme mentioned, acetyl-CoA carboxylase, is not involved in their metabolism. Instead, the correct enzyme is Thiophorase. Liver- Carboxylase (Option D): The liver does not utilize ketone bodies due to the lack of Thiophorase, and Carboxylase is not involved in ketone body metabolism.

References:

Vasudevan's Textbook of Biochemistry, 7th Edition, Page 164.

Harper's Illustrated Biochemistry, 32nd Edition, Page 221.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q3: Which of the following statements accurately describes the role of glucagon in ketogenesis during starvation? (Q845003)

A. Glucagon decreases lipolysis and reduces ketogenesis.

B. Glucagon inhibits gluconeogenesis and promotes ketogenesis.

C. Glucagon increases lipolysis and stimulates ketogenesis.

D. Glucagon decreases fatty acid entry into mitochondria and inhibits ketogenesis.

Correct Answer: C. Glucagon increases lipolysis and stimulates ketogenesis.

■ *Listen to Audio 1*

Explanation: Glucagon increases lipolysis, releasing more fatty acids that the liver converts into ketone bodies, thereby stimulating ketogenesis. Causes of Ketosis: Diabetes Mellitus: Insulin deficiency → ↑Lipolysis, ↑FA oxidation → ↑Acetyl-CoA, ↓TCA activity → Ketone overproduction. Starvation: Gluconeogenesis depletes oxaloacetate → excess Acetyl-CoA → ketogenesis. High glucagon promotes ketone use by brain (≈75% energy). Other causes: Prolonged vomiting or hyperemesis. Regulation: Glucagon ↑: ↓Malonyl-CoA, ↑Lipolysis, ↑Ketogenesis. Insulin ↑: ↑Malonyl-CoA, ↓Lipolysis, ↓Ketogenesis. Control points: Lipolysis, FA mitochondrial entry (CAT-I), and Acetyl-CoA oxidation. Glucagon decreases lipolysis and reduces ketogenesis. (Option A):

Glucagon, in fact, increases lipolysis, which provides more fatty acids for ketogenesis. Glucagon inhibits gluconeogenesis and promotes ketogenesis. (Option B): Glucagon activates gluconeogenesis and also promotes ketogenesis, but it does not inhibit gluconeogenesis. Glucagon decreases fatty acid entry into mitochondria and inhibits ketogenesis. (Option D): Glucagon does not decrease fatty acid entry into mitochondria; rather, it promotes ketogenesis by increasing lipolysis and the availability of fatty acids.

References:

Harper Biochemistry, 32nd Edition, Page 223, 224, 225

Vasudevan Biochemistry, 7th Edition, Page 165

↑ *Back to this question in Part A*

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Q4: Which one of the following is the major regulator of carnitine acyltransferase I (CAT-I) activity? (Q844996)

A. Insulin

B. Glucagon

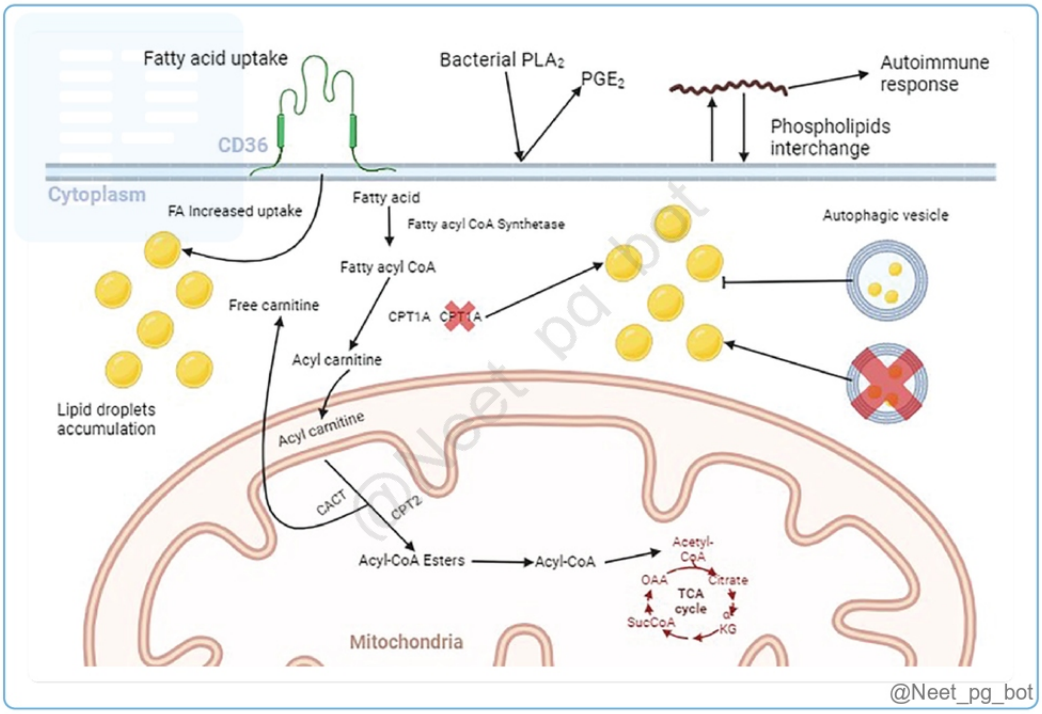
C. Malonyl CoA

D. ATP

Correct Answer: C. Malonyl CoA

■ *Listen to Audio 1*

Explanation: Malonyl CoA is the major regulator of CAT-I activity. Malonyl CoA inhibits CAT-I, thus preventing the entry of long-chain acyl groups into the mitochondrial matrix The fate of fatty acids in the cell: Fatty Acid Transport & Regulation: Entry: FAs bind membrane receptors → activated by fatty acyl-CoA synthetase . Transport: CPT1A: Converts acyl-CoA → acylcarnitine (outer membrane). CACT: Exchanges acylcarnitine ↔ carnitine (inner membrane). CPT2: Reforms acyl-CoA in matrix for β-oxidation. Regulation (CAT-I): Insulin and glucagon do not act directly on CAT-I but indirectly regulate it through changes in malonyl-CoA levels .(Options A & B) Low energy (↑glucagon): ↓Malonyl-CoA → ↑β-oxidation High energy (↑insulin): ↑Malonyl-CoA → ↓β-oxidation → FA stored as TAG ATP (Option D) does not directly regulate CAT-I activity.



References:

Vasudevan Textbook of Biochemistry, 7th Edition, Page 165

↑ *Back to this question in Part A*

@Neet_pg_bot

Q5: Which one of the following does not occur during β-oxidation of fatty acids? (Q844998)

A. Dehydrogenation of fatty acyl-CoA to trans-enoyl-CoA

B. Formation of acetyl-CoA from β-keto fatty acyl-CoA

C. Conversion of trans-enoyl-CoA to saturated fatty acyl-CoA

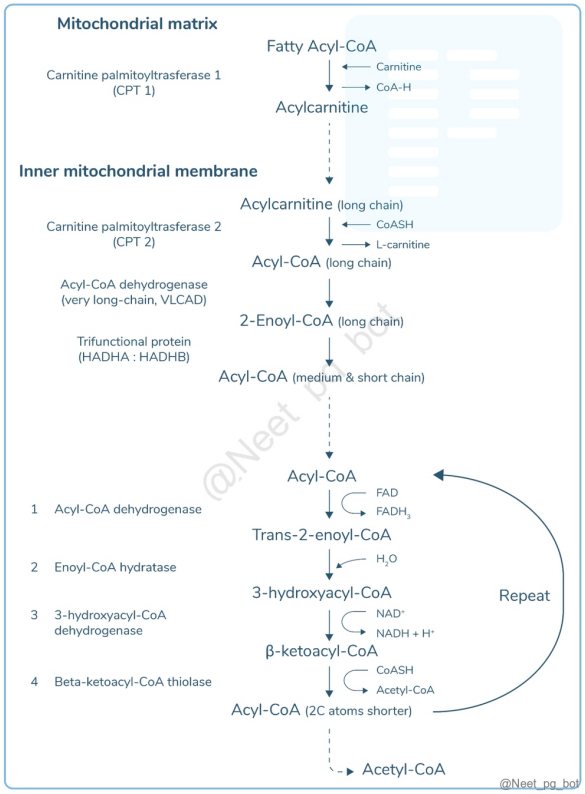
D. Hydration of trans-enoyl-CoA to β-hydroxy fatty acyl-CoA

Correct Answer: C. Conversion of trans-enoyl-CoA to saturated fatty acyl-CoA

■ *Listen to Audio 1*

Explanation: Trans-enoyl-CoA is hydrated to form β-hydroxy fatty acyl-CoA. No step converts trans-enoyl-CoA directly to a saturated fatty acyl-CoA. Steps of beta-oxidation: Step 1: FAD-Linked Dehydrogenation Catalyzed by Acyl-CoA dehydrogenase Fatty acyl-CoA → trans-enoyl-CoA (FADH■ produced). (Option A ruled out) FADH■

generates 1.5 ATP in the electron transport chain. Step 2: Hydration Catalyzed by enoyl-CoA hydratase. Converts trans-enoyl-CoA to β-hydroxy fatty acyl-CoA (only L isomer formed). (Option D ruled out) Step 3 :NAD■-Dependent Dehydrogenation Catalyzed by β-hydroxy acyl-CoA dehydrogenase β-Hydroxy fatty acyl-CoA → β-keto fatty acyl-CoA (NADH produced). NADH generates 2.5 ATP in the electron transport chain Cleavage Catalyzed by Acyl CoA Acetyltransferase (Thiolase) β-Keto fatty acyl-CoA undergoes thiolytic cleavage. Produces one acetyl-CoA and a shorter fatty acyl-CoA for further cycles. (Option B ruled out)



References:

Vasudevan Textbook of Biochemistry, 7th Edition, Page 165

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: What is the Total and Net ATP used in the complete oxidation of Arachidonic acid? (Q844999)

A. 136, 134

B. 134, 136

C. 120, 122

D. 120, 118

Correct Answer: A. 136, 134

■ *Listen to Audio 1*

Explanation: The oxidation of arachidonic acid occurs in the mitochondria via thiolysis when C2 units of it degrade once at a time to generate FADH2, NADH, and Acetyl-CoA. N = number of carbons in the fatty acid, here, N= 20 (Arachidonic acid is a 20C chain fatty acid) N/2 = of acetyl CoA produced N/2 – 1 = rounds need to fully metabolize the fatty acid, FADH2, NADH produced. Number of Acetyl-CoA produced = 20/2 = 10 Number of NADH and FADH2 produced = 20/2 —1 = 9 each. The 10 Acetyl-CoA then moves in the citrate cycle to produce 3 NADH, 1 FADH2, and 1 GTP per Acetyl-CoA. So, the number of NADH produced = 30, The number of FADH2 produced = 10 The number of GTP = 10. The ATP conversions: 1 GTP= 1 ATP 1 NADH = 2.5 ATP 1 FADH2= 1.5 ATP. The number of ATPs produced before the Acetyl-CoA moves in the citrate cycle= 9 FADH2 + 9 NADH = 9(1.5) + 9(2.5) = 36 ATPs The number of ATPs produced when the Acetyl-CoA moves in the citrate cycle. 30 NADH + 10 FADH2 + 1 GTP = 30(2.5) + 10(1.5) + 10(1) = 100 ATPs Total ATPs produced= 100 +36 = 136 2 equivalents of ATP (ATP■ AMP +PPi) are used to activate fatty acids to make acyl-CoA; therefore the net yield is 2 fewer ATP molecules. The true yield is 134 ATP (136 – 2 ATP). Net ATPs produced=134

References:

Harper Biochemistry, 32nd Edition, Page 220

Vasudevan Textbook of Biochemistry, 7th Edition, Page 153

↑ *Back to this question in Part A*

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Q7: Which of the following is NOT an inhibitor of Carnitine Acyl Transferase-1 (CAT-1)? (Q845000)

A. Sulfonylureas

B. Malonyl-CoA

C. AMPK

D. Insulin

Correct Answer: C. AMPK

■ *Listen to Audio 1*

Explanation: AMPK activation leads to increased fatty acid oxidation by promoting mitochondrial biogenesis and improving fatty acid metabolism. It is a metabolic regulator that often enhances Carnitine Acyl Transferase-1 (CAT-1)/ Carnitine Palmitoyl Transferase-1 CPT-1 activity rather than inhibiting it. CARNITINE ACYL TRANSFERASE-1 (CPT-1) STIMULATORS INHIBITORS Starvation FFA (Free Fatty Acids) AMPK

(Adenosine Monophosphate-Activated Protein Kinase) Glucagon Well fed state Malonyl-CoA (Option B ruled out) Sulfonylureas (Option A ruled out) Insulin (Option D ruled out) Etomoxir Oxfenicine Perhexiline PPARα antagonist

References:

Harper Biochemistry, 32nd Edition, Page 223, 224

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2646053/

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https://onlinelibrary.wiley.com/doi/full/10.1111/j.1467-789X.2009.00642.x

↑ *Back to this question in Part A*

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Q8: A 3-y/o child is brought to the emergency department with symptoms of severe fatigue, vomiting, and episodes of hypoglycemia. Blood tests reveal low glucose levels without elevated ketone bodies, as indicated by a negative Rothera and Benedict tests. Further examination indicates a fatty liver. The child's mother gives h/o acute fatty liver during her pregnancy with the child. Which of the following deficiencies is most likely responsible for these symptoms? (Q845001)

A. CPT-I Deficiency

B. Long-Chain 3-Hydroxyacyl-CoA Dehydrogenase Deficiency

C. Jamaican Vomiting Sickness

D. Carnitine Deficiency

Correct Answer: B. Long-Chain 3-Hydroxyacyl-CoA Dehydrogenase Deficiency

■ *Listen to Audio 1*

Explanation: Long-Chain 3-Hydroxyacyl-CoA Dehydrogenase Deficiency The scenario describes a child with severe fatigue, vomiting, nonketotic hypoglycemia, and fatty liver, alongside a maternal history of acute fatty liver of pregnancy (AFLP). These features are characteristic of Long-Chain 3-Hydroxyacyl-CoA Dehydrogenase Deficiency. This condition affects fatty acid oxidation and can manifest in both the child and mother, particularly with AFLP during pregnancy. Other deficiencies listed do not specifically present with this combination of symptoms and maternal history. FATTY ACID OXIDATION DEFECTS CONGENITAL DEFECTS ACQUIRED DEFECTS 1. CPT-I Deficiency: Site : Liver Pathophysiology : Leading to decreased fatty acid oxidation and ketogenesis, which can result in hypoglycemia (Option A ruled out) 2. CPT-II Deficiency: Site: Primarily affecting skeletal muscle and potentially the liver in severe cases 3. Defects in β-oxidation Enzymes: 2 types: Long and Short-Chain 3-Hydroxyacyl-CoA Dehydrogenase Deficiency: Can cause nonketotic

hypoglycemia, coma, and fatty liver. May also contribute to acute fatty liver during pregnancy 3-Ketoacyl-CoA Thiolase and HMG-CoA Lyase Deficiency: These deficiencies not only disrupt fatty acid oxidation but also affect the breakdown of leucine, a ketogenic amino acid. 1. Carnitine Deficiency: Common in preterm babies Cause : Inadequate biosynthesis or renal leakage. Hemolysis Pathophysiology : Impaired fatty acid oxidation→hypoglycemia & Lipid accumulation→muscular weakness (Option D ruled out) 2. Jamaican Vomiting Sickness: Cause : Consuming unripe ackee fruit containing the toxin hypoglycin, Pathophysiology : This condition inhibits medium- and short-chain acyl-CoA dehydrogenase, leading to hypoglycemia (Option C ruled out) CLINICAL FEATURES Hypoglycemia Nonketotic Hypoglycemia Lipid Accumulation Dicarboxylic Aciduria :Excretion of C6–C10 ω-dicarboxylic acids with nonketotic hypoglycemia due to MCAD deficiency . Muscular Weakness due to the buildup of lipids in muscles Coma Acute Fatty Liver of Pregnancy: Caused by LCHAD deficiency , influenced by hormonal changes. Neurological Symptoms: From defects in fatty acid and leucine oxidation.

References:

Harper Biochemistry, 32nd Edition, Page 224, 225

↑ *Back to this question in Part A*

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Q9: Which enzyme requires vitamin B12 for the isomerization of L-methylmalonyl-CoA to succinyl-CoA during odd-chain fatty acid oxidation? (Q845002)

A. Propionyl-CoA carboxylase

B. Methyl malonyl-CoA mutase

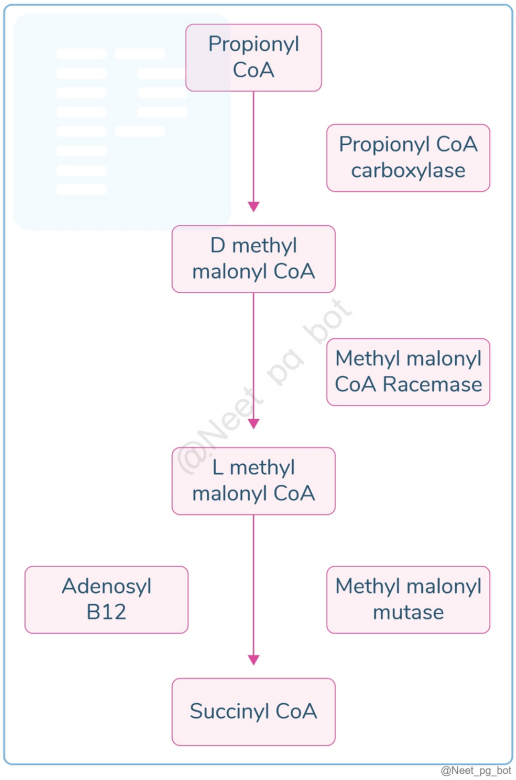
C. Methyl malonyl-CoA racemase

D. Biotin

Correct Answer: B. Methyl malonyl-CoA mutase

■ *Listen to Audio 1*

Explanation: Methylmalonyl-CoA mutase is the enzyme responsible for converting L-methylmalonyl-CoA to succinyl-CoA, a crucial step in the oxidation of odd-chain fatty acids. This reaction requires vitamin B12 as a cofactor. In vitamin B12 deficiency, the activity of methyl malonyl-CoA mutase is greatly reduced, leading to the accumulation of methyl malonyl-CoA and the subsequent excretion of methylmalonic acid in the urine (methylmalonic aciduria) and neurological side effects leading to mental retardation. Propionyl-CoA carboxylase (Option A) is another enzyme in the pathway that carboxylates propionyl-CoA to D-methylmalonyl-CoA and requires biotin, not vitamin B12. Methylmalonyl-CoA racemase (Option C) converts D-methylmalonyl-CoA to L-methylmalonyl-CoA, but this reaction also does not require vitamin B12. Biotin (Option D) is a cofactor for other carboxylation reactions but is not directly involved in the isomerization of succinyl-CoA.



References:

Harper Biochemistry, 32nd Edition, Page 181, 182, 224, 225

Vasudevan Biochemistry, 7th Edition, Page 155

↑ *Back to this question in Part A*

@Neet_pg_bot

Q10: During activation of long-chain fatty acids, ATP is hydrolyzed directly to AMP and pyrophosphate (PPi) by which enzyme? (Q885194)

A. Acyl-CoA dehydrogenase

B. Acyl-CoA synthetase (thiokinase)

C. Carnitine palmitoyltransferase I

D. Inorganic pyrophosphatase

Correct Answer: B. Acyl-CoA synthetase (thiokinase)

Explanation: Acyl-CoA synthetase (thiokinase) catalyses the activation of free fatty acids to fatty acyl-CoA , a high-energy intermediate required for further metabolism. Unlike most kinase reactions, this enzyme uses ATP and cleaves it directly to AMP and pyrophosphate (PPi) , not ADP. The released PPi is rapidly hydrolysed to 2Pi by inorganic pyrophosphatase, making the reaction effectively irreversible . Because ATP is converted to AMP + PPi (and PPi → 2Pi) , the energetic cost of fatty acid activation is equivalent to two high-energy phosphate bonds . The energy from ATP is conserved in the high-energy thioester bond between the fatty acid and Coenzyme A . Multiple isoforms of acyl-CoA synthetase exist, and the long-chain enzymes typically act on C12-C20 fatty acids . Acyl-CoA dehydrogenase (Option A): This enzyme catalyses the first step of β-oxidation (introducing a double bond and producing FADH■). It does not activate fatty acids and does not cleave ATP to AMP + PPi . Carnitine palmitoyltransferase I (Option C): CPT I is involved in the transport of long-chain fatty acids into mitochondria by transferring the acyl group to carnitine. It does not use ATP and does not perform fatty acid activation . Inorganic pyrophosphatase (Option D): This enzyme hydrolyses PPi to 2Pi , helping drive reactions forward, but it does not activate fatty acids and is not responsible for ATP → AMP + PPi cleavage .

References:

https://pmc.ncbi.nlm.nih.gov/articles/PMC2846691/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: A newborn presents with characteristic facial features (mongoloid facies, hypertelorism), hypotonia, seizures, and hepatomegaly. Accumulation of very-long-chain fatty acids (VLCFA) is found. The most likely diagnosis is: (Q885207)

A. Refsum disease

B. MCAD deficiency

C. Zellweger Syndrome

D. Adrenoleukodystrophy

Correct Answer: C. Zellweger Syndrome

Explanation: Zellweger syndrome (cerebrohepatorenal syndrome) is a peroxisomal biogenesis disorder caused by mutations in PEX genes , leading to the absence of functional peroxisomes . As a result, there is an accumulation of very-long-chain fatty acids (VLCFA) , bile acid intermediates , and phytanic acid in tissues. The hallmark metabolic defect is failure of peroxisomal β-oxidation of VLCFA , causing toxic buildup, especially in the brain and liver . Clinically, infants present with severe hypotonia (floppy infant) , neonatal seizures , dysmorphic facial features (e.g., high forehead, flattened nasal bridge, hypertelorism), and hepatomegaly with jaundice . Neuropathologically, there are neuronal migration defects , contributing to profound developmental impairment. Skeletal abnormalities such as chondrodysplasia punctata (stippled epiphyses) may be seen. The prognosis is poor , and the condition is usually fatal within the first year of life , with management being supportive only . Refsum disease (Option A): A defect of α-oxidation of phytanic acid, presenting later in life with retinitis pigmentosa,

neuropathy, and ataxia , not a lethal neonatal syndrome with VLCFA accumulation. MCAD deficiency (Option B): A mitochondrial β-oxidation defect causing hypoketotic hypoglycemia and metabolic crises, but no dysmorphic facies or peroxisomal dysfunction . Adrenoleukodystrophy (Option D): An X-linked peroxisomal disorder with VLCFA accumulation, but typically presents in childhood with demyelination and adrenal insufficiency , not with severe neonatal dysmorphism and hypotonia.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 225, 236, 587

↑ *Back to this question in Part A*

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Q12: During fatty acid oxidation, acyl-carnitine is formed as an intermediate in which process? (Q885195)

A. Activation of fatty acids

B. Transport across the inner mitochondrial membrane

C. Beta-oxidation spiral

D. Ketone body synthesis

Correct Answer: B. Transport across the inner mitochondrial membrane

Explanation: Acyl-carnitine is a transport intermediate in the carnitine shuttle that allows long-chain fatty acids to enter the mitochondrial matrix for oxidation. Fatty acids are first activated to fatty acyl-CoA on the outer mitochondrial membrane, but this molecule cannot cross the inner mitochondrial membrane directly. The enzyme Carnitine Palmitoyltransferase I (CPT-I) , located on the outer mitochondrial membrane , transfers the acyl group from CoA to carnitine, forming acyl-carnitine . This acyl-carnitine is transported across the inner mitochondrial membrane by the carnitine-acylcarnitine translocase . Inside the matrix, Carnitine Palmitoyltransferase II (CPT-II) converts acyl-carnitine back to fatty acyl-CoA , which then enters β-oxidation . This transport step is inhibited by malonyl-CoA , ensuring that fatty acid oxidation is suppressed during active fatty acid synthesis (lipogenesis) . Activation of fatty acids (Option A): Activation converts free fatty acids to fatty acyl-CoA using acyl-CoA synthetase. Acyl-carnitine is not formed during this step. Beta-oxidation spiral (Option C): β-oxidation occurs in the mitochondrial matrix and uses fatty acyl-CoA , not acyl-carnitine, as the substrate. Ketone body synthesis (Option D): Ketogenesis uses acetyl-CoA derived from fatty acid oxidation in the liver mitochondria and does not involve acyl-carnitine as an intermediate.

References:

https://pmc.ncbi.nlm.nih.gov/articles/PMC11641656/

↑ *Back to this question in Part A*

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Q13: Which enzyme catalyses the initial energy-requiring "activation" step necessary for fatty acid oxidation to occur? (Q885201)

A. Acyl-CoA dehydrogenase

B. Enoyl-CoA hydratase

C. Thiolase

D. Fatty Acyl-CoA synthetase (Thiokinase)

Correct Answer: D. Fatty Acyl-CoA synthetase (Thiokinase)

Explanation: Fatty Acyl-CoA synthetase (Thiokinase) Explantion: Fatty acyl-CoA synthetase (thiokinase) catalyses the activation of free fatty acids to fatty acyl-CoA , which is a prerequisite for fatty acids to enter metabolic pathways such as β-oxidation . For long-chain fatty acids, this enzyme is located on the outer mitochondrial membrane (and also exists in the ER and peroxisomes in other contexts). The reaction uses ATP and converts it to AMP + pyrophosphate (PPi) , making the energetic cost equivalent to two high-energy phosphate bonds . The PPi is rapidly hydrolysed by inorganic pyrophosphatase, which makes the reaction effectively irreversible . The energy from ATP is conserved in the high-energy thioester bond of fatty acyl-CoA. Multiple isoforms of this enzyme exist, specific for short-, medium-, long-, and very-long-chain fatty acids . The activated acyl-CoA can then be used for fatty acid oxidation or lipid synthesis . Acyl-CoA dehydrogenase (Option A): This enzyme catalyses the first step of the β-oxidation spiral inside mitochondria and does not activate fatty acids. Enoyl-CoA hydratase (Option B): This enzyme catalyses the hydration step of β-oxidation and acts only after the fatty acid has already been activated. Thiolase (Option C): Thiolase catalyses the final cleavage step of β-oxidation , producing acetyl-CoA, and is not involved in the activation of fatty acids.

References:

DM Vasudevan, Textbook of Biochemistry, 7th Edition, Page 149

↑ *Back to this question in Part A*

@Neet_pg_bot

Q14: In each cycle of beta-oxidation, which of the following is not produced? (Q885202)

A. 1 FADH■

B. 1 NADH

C. 1 Acetyl-CoA

D. 1 GTP

Correct Answer: D. 1 GTP

Explanation: Each cycle of β-oxidation produces 1 FADH■ , 1 NADH , and 1 acetyl-CoA . The first step (acyl-CoA dehydrogenase) reduces FAD to FADH■ , and the third step (β-hydroxyacyl-CoA dehydrogenase) reduces NAD■ to NADH . The final thiolysis step releases one acetyl-CoA and a fatty acyl-CoA shortened by two carbons , allowing the cycle to repeat. GTP is not produced in β-oxidation. GTP is generated by substrate-level phosphorylation in the TCA cycle , specifically at the succinyl-CoA synthetase step, not during fatty acid β-oxidation. The reduced cofactors FADH■ and NADH produced in β-oxidation enter the electron transport chain , yielding approximately 1.5 ATP per FADH■ and 2.5 ATP per NADH . 1 FADH■ (Option A): Produced in the first oxidation step of β-oxidation by acyl-CoA dehydrogenase. 1 NADH (Option B): Produced in the third step of β-oxidation by β-hydroxyacyl-CoA dehydrogenase. 1 Acetyl-CoA (Option C): Released in the thiolysis step of each cycle as two carbons are cleaved from the fatty acyl chain.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 219, 220

↑ *Back to this question in Part A*

@Neet_pg_bot

Q15: A straight-chain fatty acid with 2N carbon atoms undergoes beta-oxidation. The net ATP yield can be calculated by which formula? (Q885204)

A. 10N - 2

B. 12N - 4

C. 14N - 6

D. 17N - 8

Correct Answer: C. 14N - 6

Explanation: For a saturated straight-chain fatty acid with 2N carbons , the number of β-oxidation cycles is (N - 1) , and the number of acetyl-CoA molecules formed is N . Each cycle produces 1 FADH■ and 1 NADH , giving: (N - 1) FADH■ × 1.5 ATP = 1.5(N - 1) (N - 1) NADH × 2.5 ATP = 2.5(N - 1) Each acetyl-CoA entering the TCA cycle yields 10 ATP , so: N acetyl-CoA × 10 ATP = 10N Total ATP produced = 10N + 1.5(N - 1) + 2.5(N - 1) = 14N - 4 Activation of the fatty acid to acyl-CoA costs 2 ATP equivalents , so the net yield is: 14N - 6 ATP For example, palmitic acid (C16, N = 8) yields: 14 × 8 - 6 = 112 - 6 = 106 ATP .

References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 220, 224

↑ *Back to this question in Part A*

@Neet_pg_bot

Q16: A child presents with hypoketotic hypoglycemia and dicarboxylic aciduria (C6-C10 dicarboxylic acids in urine). The most likely diagnosis is: (Q885205)

A. Refsum disease

B. Zellweger syndrome

C. Medium-chain acyl-CoA dehydrogenase (MCAD) deficiency

D. CPT-I deficiency

Correct Answer: C. Medium-chain acyl-CoA dehydrogenase (MCAD) deficiency

Explanation: MCAD deficiency impairs β-oxidation of medium-chain fatty acids , leading to reduced production of acetyl-CoA . As a result, patients develop hypoketotic hypoglycemia due to failure of ketogenesis and impaired support of gluconeogenesis . When β-oxidation is blocked, fatty acids are diverted to ω-oxidation in the microsomes , producing dicarboxylic acids (C6-C10) that appear in the urine (dicarboxylic aciduria). Clinically, affected children are often well until fasting or infection triggers a metabolic crisis, presenting with vomiting, lethargy, seizures, coma, or sudden death . MCAD deficiency is a known cause of sudden infant death syndrome (SIDS) . Diagnosis is supported by elevated octanoylcarnitine (C8) on newborn screening or acylcarnitine profile. Management focuses on avoidance of fasting , a high-carbohydrate diet , and measures such as cornstarch supplementation to maintain glucose levels. Refsum disease (Option A): Caused by a defect in α-oxidation of phytanic acid, leading to retinitis pigmentosa, neuropathy, and ataxia , not hypoketotic hypoglycemia with dicarboxylic aciduria. Zellweger syndrome (Option B): A peroxisomal biogenesis disorder causing severe hypotonia, seizures, and dysmorphic features in neonates, not an isolated defect of fatty acid β-oxidation. CPT-I deficiency (Option D): Causes hypoketotic hypoglycemia due to impaired transport of long-chain fatty acids into mitochondria, but it does not classically produce the characteristic medium-chain dicarboxylic aciduria seen in MCAD deficiency.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 225

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: Which of the following enzymes is correctly paired with its function in metabolic processes? (Q844988)

A. Acetyl CoA Carboxylase (ACC) converts acetyl-CoA to citrate, initiating fatty acid synthesis.

B. Carnitine Palmitoyltransferase I (CPT-I) regulates entry of all fatty acids into mitochondria for beta-oxidation.

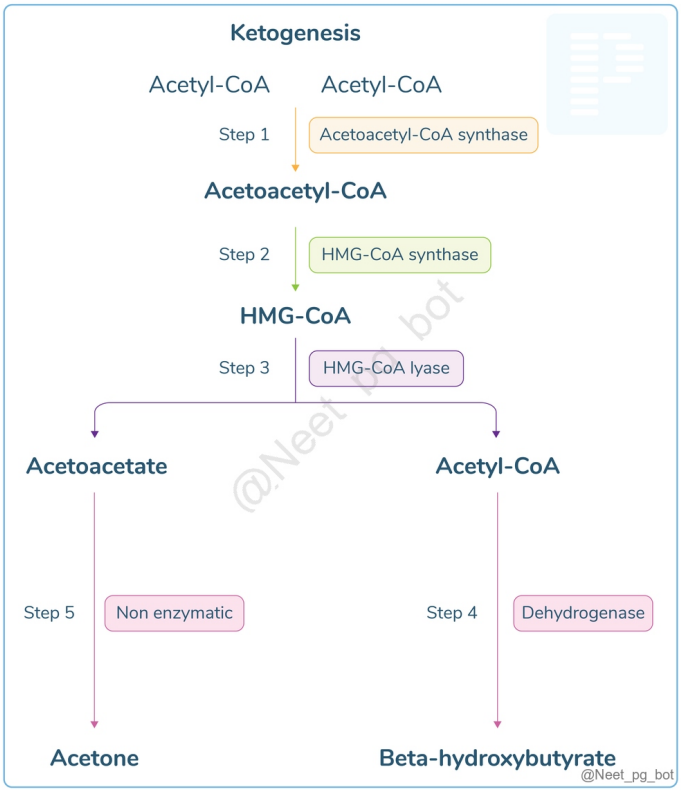
C. HMG-CoA Synthase catalyzes ketone body production from acetyl-CoA.

D. Acetyl CoA Carboxylase (ACC) converts acetyl-CoA to malonyl-CoA, the rate-limiting step in fatty acid Oxidation

Correct Answer: C. HMG-CoA Synthase catalyzes ketone body production from acetyl-CoA.

Listen to Audio 1

Explanation: HMG-CoA Synthase catalyzes a key step in ketogenesis, producing ketone bodies from acetyl-CoA when glucose is limited. Rate Limiting Enzymes: Acetyl CoA Carboxylase (ACC) : Converts acetyl-CoA to malonyl-CoA, the first committed step in fatty acid synthesis. (Options A & D ruled out) Regulated by citrate (activator) and palmitoyl-CoA (inhibitor). Insulin activates ACC, while glucagon and epinephrine inhibit it. Carnitine acyltransferase I (CAT-I) : Regulates the entry of long-chain fatty acids (typically 14-20 carbon atoms) into mitochondria for beta-oxidation. (Option B ruled out) Inhibited by malonyl-CoA to prevent simultaneous synthesis and breakdown of fatty acids. Activated when glucagon levels rise and malonyl-CoA decreases. HMG-CoA Synthase : Catalyzes a key step in ketogenesis, producing ketone bodies from acetyl-CoA. Involved in a key regulatory step in ketogenesis.



References:

Vasudevan's Textbook of Biochemistry for Medical Students, 7th Edition, Pages 154, 157, 160, 164, 165

↑ *Back to this question in Part A*

@Neet_pg_bot

Q18: Which of the following statements about alpha-oxidation is true? (Q885186)

A. It is a major energy-yielding process

B. It occurs primarily in mitochondria

C. It is not an energy-yielding process

D. It produces FADH■ and NADH

Correct Answer: C. It is not an energy-yielding process

Explanation: Alpha-oxidation is not coupled to ATP synthesis and is therefore not an energy-yielding pathway (Option A) . It is a specialised pathway for the metabolism of branched-chain fatty acids , especially phytanic acid , which cannot undergo beta-oxidation directly due to a methyl group at the beta-carbon. This process occurs in the peroxisomes (Option B) and involves the removal of one carbon atom from the carboxyl end of the fatty acid. The key enzyme is phytanoyl-CoA hydroxylase (PhyH) , which hydroxylates the alpha-carbon and requires vitamin C, Fe^{2}■, and alpha-ketoglutarate as cofactors. The primary role of alpha-oxidation is not to generate ATP, NADH, or FADH■ (Option D) , but to convert phytanic acid into pristanic acid , which can then undergo beta-oxidation for energy production. Clinically, a deficiency of alpha-oxidation leads to Refsum disease , characterised by the accumulation of phytanic acid , causing neurological deficits, retinitis pigmentosa, and ataxia .

References:

DM Vasudevan, Textbook of Biochemistry, 7th Edition, Page 155

↑ *Back to this question in Part A*

@Neet_pg_bot

Q19: Which of the following enzymes is the most important target for the regulation of fatty acid synthesis by citrate? (Q875985)

A. Hydratase

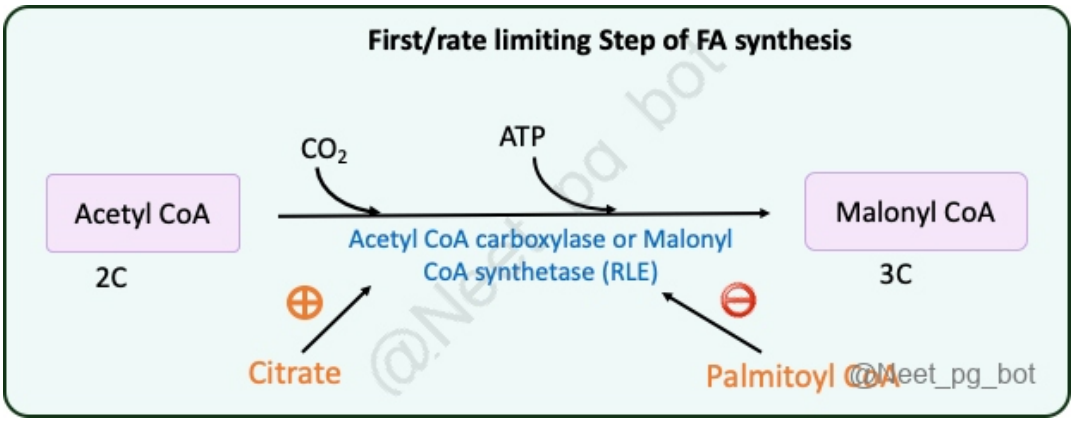
B. Acetyl-CoA carboxylase

C. Enoyl reductase

D. Acetyl transacetylase

Correct Answer: B. Acetyl-CoA carboxylase

Explanation: Correct option B: Acetyl-CoA carboxylase It is the rate-limiting enzyme of fatty acid synthesis. It is activated by citrate. It is Inhibited by Palmitoyl CoA. It Converts Acetyl-CoA to Malonyl-CoA, It needs biotin ATP and CO2. As ATP is used, the name of the enzyme is also Malonyl CoA Synthetase. Incorrect options Option A: Hydratase Hydratase enzyme is used for the addition or removal of water from any any substrate. Option C: Enoyl reductase It is a part of FA synthase and used for the synthesis of fatty acids. Option D: Acetyl transacetylase This enzyme transfers the acetyl group from acetyl-CoA to beta-galactosides, glucosides, and lactosides.



References:

Harper 30th /e pg. 237

↑ *Back to this question in Part A*

@Neet_pg_bot

Q20: Which of the following fatty acid metabolism primarily takes place in the peroxisome? (Q844995)

A. Very long chain FA

B. Medium Chain FA

C. Short chain FA

D. Long chain FA

Correct Answer: A. Very long chain FA

■ *Listen to Audio 1*

Explanation: Very long-chain fatty acids undergo oxidation in peroxisomes Fatty acids oxidation Mitochondria Short-chain fatty acids (Option C ruled out) Medium-chain fatty acids (Option B ruled out) Long-chain fatty acids (Option D ruled out) Peroxisomes Very long-chain fatty acids (>22C) Functions of peroxisomes Breakdown of Very-Long-Chain Fatty Acids (VLCFAs) Energy Production by reducing the length of fatty acid chains, making them suitable for further oxidation in mitochondria. Cholesterol Metabolism : Contribute to the shortening of the side chain of cholesterol in bile acid formation. Involved in the synthesis of ether glycerolipids , which are essential components of cellular membranes. Participate in the synthesis of cholesterol and dolichol , which are important for various cellular functions.

References:

Harper Biochemistry, 32nd Edition, Page 220

Lippincott Textbook of Biochemistry, 7th Edition, Page 556,560,563,566

↑ *Back to this question in Part A*

@Neet_pg_bot

Q21: Which of the following inhibits Acetyl CoA Carboxylase? (Q844994)

A. Glucagon

B. NADH

C. ATP

D. Insulin

Correct Answer: A. Glucagon

■ *Listen to Audio 1*

Explanation: Acetyl-CoA carboxylase is inhibited by Glucagon . Glucagon inhibits acetyl-CoA carboxylase by keeping the enzyme in the inactive phosphorylated state, which in turn reduces Malonyl-CoA and makes CPT-I (carnitine palmitoyltransferase-I) active, and causes beta-oxidation of fatty acids in mitochondria. Glucagon is secreted when the energy charge is low and AMP levels are high. Acetyl CoA Carboxylase Stimulated by High energy level states Inhibited by Low energy level states. NADH (Option B ruled out) FADH2 ATP (Option C ruled out) Citrate Insulin: enhances the uptake of glucose by adipocytes and increases the activity of pyruvate dehydrogenase, acetyl CoA carboxylase, and glycerol phosphate acyl transferase (Option D ruled out) ADP NAD FAD Glucagon(catabolic hormone) (Option A) Acyl CoA Epinephrine The nutritional state of the organism is the main factor regulating the rate of lipogenesis Diets : Carbohydrate-rich diet: Increases lipogenesis Fat-rich diet: Decreases lipogenesis eg. Keto diet Other situations where lipogenesis is decreased: Restricted caloric intake Diabetes mellitus

References:

Vasudevan Textbook of Biochemistry, 7th Edition, Page 159-160

Harper's Illustrated Biochemistry, 32nd Edition, Page 230

↑ *Back to this question in Part A*

@Neet_pg_bot

Q22: Which of the following correctly describes the products and by-products of the palmitic acid synthesis reaction? (Q844993)

A. 1 Palmitate + 7 CO■ + 6 H■O + 8 CoA–SH + 14 NADPH

B. 1 Palmitate + 7 CO■ + 6 H■O + 8 CoA–SH + 14 NADP+

C. 1 Palmitate + 7 CO■ + 8 H■O + 8 CoA–SH + 14 NADP■

D. 1 Palmitate + 7 CO■ + 6 H■O + 8 CoA–SH + 14 NADP■ + 14 H■

Correct Answer: B. 1 Palmitate + 7 CO■ + 6 H■O + 8 CoA–SH + 14 NADP+

■ *Listen to Audio 1*

Explanation: 1 Palmitate + 7 CO■ + 6 H■O + 8 CoA–SH + 14 NADP+ accurately describes the products of palmitic acid synthesis, including 1 palmitate, 7 CO■, 6 H■O, 8 CoA–SH, and 14 NADP+. Reaction Equation: CH 3 CO–S–CoA + 7HOOCCHCO–S–CoA + 14NADPH + 14H + → CH 3 (CH 2) 14 COOH + 7CO 2 + 6H 2 O + 8CoA–SH + 14 NADP+ Synthesis of Palmitate (16-carbon fatty acid): Reactants: 1 molecule of acetyl-CoA (provides carbons 15 and 16) 7 molecules of malonyl-CoA (provides remaining carbons) 14 molecules of NADPH 14 hydrogen protons (H■) Products: 1 molecule of palmitate 7 molecules of carbon dioxide (CO■) 6 molecules of water (H■O) 8 molecules of coenzyme A (CoA–SH) 14 molecules of NADP■ 1 palmitate + 7 CO■ + 6 H■O + 8 CoA–SH + 14 NADPH (Option A) NADPH is used during the synthesis, not produced. 1 palmitate + 7 CO■ + 8 H■O + 8 CoA–SH + 14 NADP■ (Option C) In palmitic acid synthesis, 6 molecules of H20 are produced, not 8. 1 palmitate + 7 CO■ + 6 H■O + 8 CoA–SH + 14 NADP■ + 14 H■ (Option D) This option shows extra protons (14 H■), which are not produced in the reaction, rather they are used up along with 14 NADPH to give 14 NADP+.

References:

Harper's Illustrated Biochemistry, Edition 32, Page 228

↑ *Back to this question in Part A*

@Neet_pg_bot

Q23: Which of the following statements about the fatty acid synthesis mechanism is NOT TRUE? (Q844992)

A. Acetyl-CoA is converted to malonyl-CoA by acetyl-CoA carboxylase.

B. The FAS complex is a homodimer with two identical subunits and seven enzymes

C. NADPH is primarily generated by the citric acid cycle.

D. Palmitate is the main product, which can be converted into shorter fatty acids.

Correct Answer: C. NADPH is primarily generated by the citric acid cycle.

■ *Listen to Audio 1*

Explanation: NADPH is primarily generated through the pentose phosphate pathway, not the citric acid cycle. Fatty Acid Synthesis: Rate-limiting step: Acetyl-CoA → Malonyl-CoA (via acetyl-CoA carboxylase). (Option A ruled out) FAS complex: Fatty Acid Synthase (FAS) complex is a homodimer , and each subunit contains seven enzyme activities plus an acyl carrier protein (ACP) domain .. (Option B ruled out) Process: Priming: Acetyl-CoA and malonyl-CoA bind to ACPs. Condensation → Reduction → Dehydration → Reduction: Chain extends by 2C per cycle using NADPH . Elongation Cycle: Repeats six times to form 16-carbon palmitate bound to ACP. (Option D ruled out) Thioesterase: Releases free palmitate. Mammary Thioesterase: Produces shorter (C8–C12) fatty acids for milk lipids. NADPH sources: PPP, malic enzyme, isocitrate dehydrogenase.

References:

Harper’s Illustrated Biochemistry, Edition 32, Page 227, 228

↑ *Back to this question in Part A*

@Neet_pg_bot

Q24: Which of the following statements about the Fatty acid synthase (FAS) complex is true? (Q844989)

A. The FAS complex synthesizes fatty acids exclusively in the mitochondria.

B. The primary product of the FAS complex is palmitic acid, a 16-carbon saturated fatty acid.

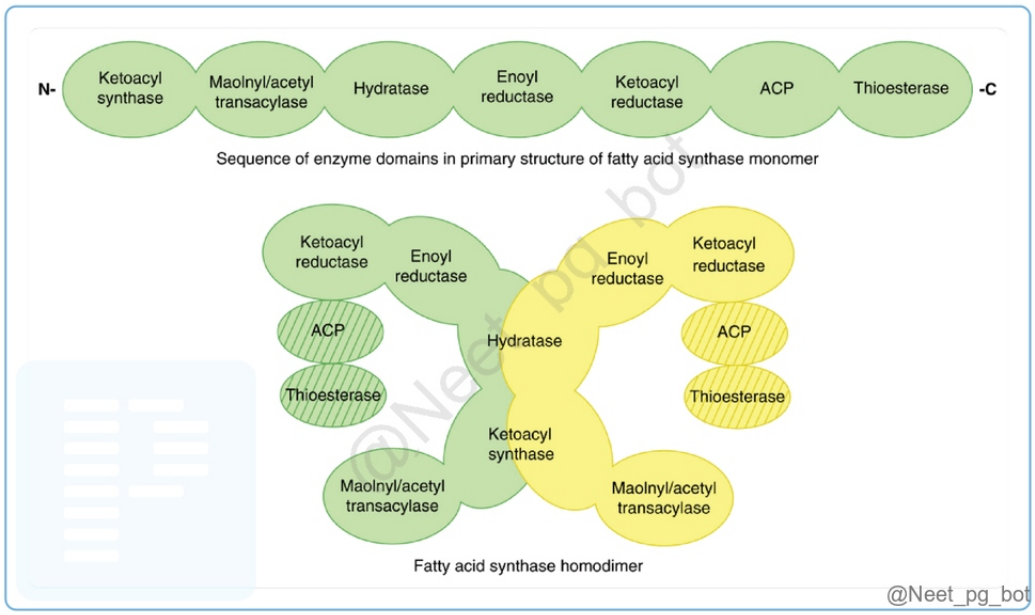
C. The FAS complex utilizes the same enzymes and intermediates as those found in β-oxidation.

D. Each subunit of the FAS complex consists of a single enzyme responsible for fatty acid synthesis.

Correct Answer: B. The primary product of the FAS complex is palmitic acid, a 16-carbon saturated fatty acid.

■ *Listen to Audio 1*

Explanation: The primary product of the FAS complex is palmitic acid, which is a 16-carbon saturated fatty acid synthesized during fatty acid synthesis. Fatty Acid Synthase (FAS) Complex Overview: Location : Cytoplasm (Option A ruled out) Active in liver, adipose tissue, lactating mammary gland, kidney, and brain. Actions : De novo synthesis of fatty acids, primarily palmitic acid (16-carbon saturated fatty acid). Not the reverse of β-oxidation; it involves different intermediates, enzymes, coenzymes, and regulatory mechanisms. (Option C ruled out) Structure : Multi-enzyme complex organized as a homodimer (two identical subunits). Each subunit contains three domains, six enzymes, and an acyl carrier protein. (Option D ruled out) Enhances efficiency by facilitating interactions between intermediates and active sites. Subunits : Domain 1 (Condensing): Acetyl transacylase, Malonyl transacylase, β-Ketoacyl synthase Domain 2 (Reduction): ACP, β-Ketoacyl reductase, Dehydratase, Enoyl reductase Domain 3 (Releasing): Thioesterase



References:

Vasudevan’s Textbook of Biochemistry for Medical Students, Edition 7, Page 156, 157, 158

Harper’s Illustrated Biochemistry, Edition 32, Page 227

↑ *Back to this question in Part A*

@Neet_pg_bot

• [03_Lipid Chemistry] 011 Cholesterol Metabolism Lipoproteins - I

« Back to Folder Directory

[03_Lipid Chemistry] 011 Cholesterol Metabolism Lipoproteins - I — Quick Jump

Q1 (p. 678)	*Q2 (p. 679)*	*Q3 (p. 679)*	*Q4 (p. 680)*	*Q5 (p. 681)*	*Q6 (p. 682)*
Q7 (p. 684)	*Q8 (p. 685)*	*Q9 (p. 686)*	*Q10 (p. 687)*	*Q11 (p. 688)*	*Q12 (p. 689)*
Q13 (p. 690)	*Q14 (p. 692)*	*Q15 (p. 693)*	*Q16 (p. 694)*	*Q17 (p. 694)*	*Q18 (p. 695)*
Q19 (p. 696)	*Q20 (p. 697)*				

Q1: Which of the following best describes Tangier disease? (Q845019)

A. Low VLDL levels

B. Low HDL levels

C. Low LDL levels

D. Low LDL receptor activity

Correct Answer: B. Low HDL levels

■ *Listen to Audio 1*

Explanation: Tangier disease It is marked by extremely low levels of high-density lipoprotein (HDL) cholesterol due to mutations in the ABCA1 gene on chromosome 9q31 , which impairs cholesterol transport. Defects in ABCA1 are characterized by two clinical hallmarks; enlarged lipid-laden tonsils and low serum HDL. Also known as Familial alpha-lipoprotein deficiency/Hypo alpha lipoproteinemia. This mutation causes a decreased level of Apo A1,where HDL is not properly formed. Leads to the accumulation of cholesterol in various tissues such as the tonsils, liver, spleen, and corneas. Clinical features : Include enlarged orange-colored tonsils, peripheral neuropathy, hepatosplenomegaly and early-onset cardiovascular disease. Low VLDL levels (Option A) are not characteristic of Tangier disease. VLDL primarily carries triglycerides, and its levels are not significantly affected in Tangier disease. Low LDL levels (Option C) are not specifically affected in Tangier disease. LDL primarily carries cholesterol to tissues, and its levels can vary independently of HDL levels. Low LDL receptor activity (Option D): This describes familial hypercholesterolemia, not Tangier disease. Familial hypercholesterolemia is characterized by high LDL cholesterol levels due to defective LDL receptors on cell surfaces.

References:

Lippincott Biochemistry, 7th Edition, Page 678

↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: A 12-year-old boy presents with developmental delays, visual impairment, and recurrent episodes of ataxia. Laboratory tests reveal low serum levels of triglycerides, cholesterol, and fat-soluble vitamins. A blood smear shows the presence of acanthocytes. Which of the following findings is most likely present in this patient? (Q844854)

A. Elevated levels of chylomicrons in the bloodstream.

B. Absence of apolipoprotein B-containing lipoproteins.

C. Normal absorption of dietary fats.

D. High serum levels of low-density lipoprotein (LDL).

Correct Answer: B. Absence of apolipoprotein B-containing lipoproteins.

■ *Listen to Audio 1*

Explanation: Abetalipoproteinemia is a rare genetic disorder characterized by the inability to synthesize apolipoprotein B (Apo B), which is essential for the formation of certain lipoproteins. Pathophysiology : Caused by mutations in the microsomal triglyceride transfer protein (MTP) gene. Leads to impaired assembly and secretion of lipoproteins, particularly chylomicrons and very low-density lipoprotein (VLDL). (Option A and D ruled out) Results in low serum levels of triglycerides, cholesterol, and fat-soluble vitamins (A, D, E, K). Clinical Features : Fat malabsorption leads to steatorrhea (fatty stools). (Option C ruled out) Deficiencies in fat-soluble vitamins can cause: Retinitis pigmentosa (leading to blindness). Neurological deficits (e.g., ataxia, developmental delays). Presence of acanthocytes (spiny red blood cells) on blood smear. Management : Dietary management to provide essential fatty acids and fat-soluble vitamins. Supplementation with medium-chain triglycerides (MCTs) may be beneficial. Lomitapide (AEGR-733/BMS-201038) is an FDA-approved MTP inhibitor used for homozygous familial hypercholesterolemia to lower lipids, whereas in Abetalipoproteinemia patients need to increase them.

References:

Vasudevan Biochemistry, 7th Edition, Page 341, 342

↑ *Back to this question in Part A*

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Q3: A researcher is studying the mechanisms of lipoprotein lipase (LPL) activity in post-heparinized blood samples. They conduct a mixing study to differentiate between a deficiency of apolipoprotein C-II (Apo C-II) and LPL deficiency. Which of the

following outcomes from the mixing study would most accurately indicate an Apo C-II deficiency? (Q843472)

A. The addition of pooled normal plasma results in no change in LPL activity.

B. LPL activity normalizes after mixing with normal plasma.

C. A significant increase in triglyceride levels is observed after mixing.

D. LPL activity decreases following the addition of normal plasma.

Correct Answer: B. LPL activity normalizes after mixing with normal plasma.

■ *Listen to Audio 1*

Explanation: If the LPL activity normalises after mixing, it is an Apo C II defect. Post-heparinized blood samples are used because heparin in circulation competes with heparan sulfate on vessel walls, causing enzymes to detach from the vessel wall and attach to circulating heparin. If LPL activity is low in post-heparinized individuals, it is either an Apo C II defect or an LPL defect. Mixing study to differentiate Apo C defect from LPL defect: In the patients post heparinized plasma, add an equal amount of pooled normal plasma which acts as a source of apo C II. If LPL activity normalizes it is an apo C II defect and it will respond to FFP administration. If LPL activity does not normalize it is an LPL defect (Option A ruled out). An increase in triglyceride levels is not indicative of Apo C-II deficiency and does not relate directly to the expected outcomes of the mixing study. (Option C ruled out) Decrease in LPL activity after mixing would suggest an error in the experiment or another underlying issue, not an Apo C-II deficiency. (Option Druled out)

References:

Vasudevan Biochemistry, 7th Edition, Page 176

↑ *Back to this question in Part A*

@Neet_pg_bot

Q4: Match the following contributing factors with their corresponding effects on fatty liver: A) Choline Deficiency 1) Increases NADH, hindering fatty acid oxidation and promoting triacylglycerol accumulation. B) Orotic Acid Interference 2) Impairs VLDL secretion, resulting in triacylglycerol accumulation and a fatty liver. C) Vitamin E and Selenium 3) Involved in pyrimidine synthesis. Disrupts VLDL glycosylation, hindering release. D) Ethanol Consumption 4) Protect against liver damage from lipid peroxidation (Q843471)

A. A-2, B-3, C-4, D-1

B. A-3, B-4, C-2, D-1

C. A-4, B-1, C-3, D-2

D. A-1, B-2, C-4, D-3

Correct Answer: A. A-2, B-3, C-4, D-1

■ *Listen to Audio 1*

Explanation: Fatty Liver-Contributing Factors Choline Deficiency Choline is crucial for lipid metabolism. Deficiency impairs VLDL secretion , resulting in triacylglycerol accumulation and a fatty liver. Orotic Acid Interference Involved in pyrimidine synthesis Disrupts VLDL glycosylation , hindering release. Nutritional Deficiencies Deficiencies in essential fatty acids, vitamins (linoleic acid, pyridoxine, pantothenic acid) , and protein contribute to fatty liver. Ethanol Consumption Ethanol metabolism increases NADH, hindering fatty acid oxidation and promoting triacylglycerol accumulation. Vitamin E and Selenium Protect against liver damage from lipid peroxidation , especially in choline deficiency-induced fatty liver. Note: Resmetirom is a new FDA-approved oral, liver-directed, thyroid hormone receptor beta–selective agonist in development for the treatment of NASH with liver fibrosis.

References:

Harper Biochemistry, 32nd Edition, Page 254, 255 Vasudevan Biochemistry, 7th Edition, Page 162, 163

↑ *Back to this question in Part A*

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Q5: Which among the following is a true statement on electrophoretic mobility of lipoproteins seen during serum electrophoresis? (Q843470)

A. HDL shows the least mobility due to high lipid content.

B. Lipoproteins with higher protein content move slower towards the anode.

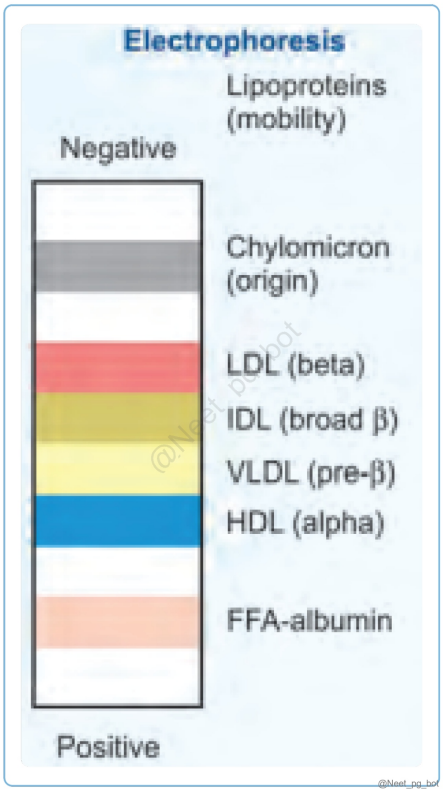
C. Lipoproteins with higher protein content migrate faster towards the anode

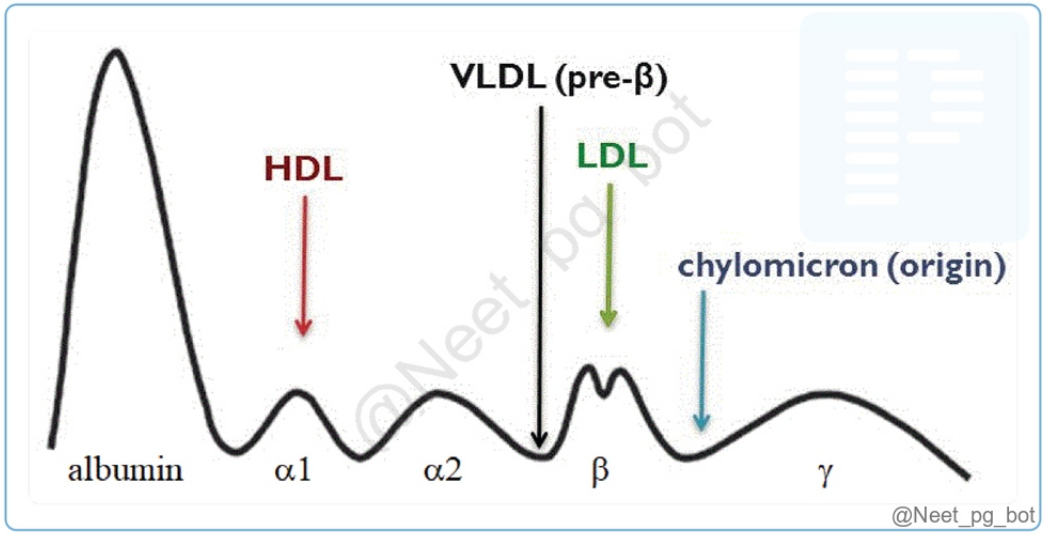
D. Lipoproteins identified by dye color after staining with lipid dyes, not electrophoretic mobility.

Correct Answer: C. Lipoproteins with higher protein content migrate faster towards the anode

■ *Listen to Audio 1*

Explanation: The serum is applied on cellulose acetate, electric current is applied for 2 hours, the strip is dried and stained with lipid dyes, such as Oil Red O. Higher protein content will move faster towards the anode and those with less proteins have minimum mobility. HDL shows the highest mobility, followed by VLDL, IDL, and LDL. (Option A ruled out) Lipoproteins with higher protein content move faster towards the anode. (Option B ruled out) Staining helps visualize lipoproteins , their electrophoretic mobility is crucial for identifying and classifying them. (Option D ruled out)





References:

Vasudevan Biochemistry, 7th Edition, Page 174, 175 https://pubmed.ncbi.nlm.nih.gov/10828082/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: A 45-year-old male presents with symptoms of peripheral neuropathy, enlarged tonsils, and orange-colored tonsils. His lipid profile shows very low levels of high-density lipoprotein (HDL) cholesterol. Further investigation reveals that the patient has a rare genetic condition leading to severe HDL deficiency. Given these findings, which of the following gene mutations is most likely responsible for this condition? (Q843026)

A. ABCA1 gene mutation

B. LDLR gene mutation

C. APOE gene mutation

D. PCSK9 gene mutation

Correct Answer: A. ABCA1 gene mutation

Listen to Audio 1

Explanation: The patient presents findings most likely points towards the diagnosis of Tangier disease. Tangier disease is a rare, autosomal recessive disorder caused by mutations in the ABCA1 (ATP-binding cassette, sub-family A, member 1) gene . Pathophysiology: The ABCA1 protein is crucial for the efflux of cholesterol and phospholipids to apolipoprotein A-I (ApoA-I), forming HDL particles. Mutations in the ABCA1 gene impair this process, leading to a shortage of HDL and cholesterol accumulation in tissues such as the tonsils, liver, spleen, and peripheral nerves. Clinical Features: Greyish-Orange Tonsils: (Hallmark feature) Tonsils, become enlarged and acquire a greyish-orange hue due to the accumulation of cholesterol esters. Hepatosplenomegaly : Due to cholesterol and phospholipid accumulation. Peripheral Neuropathy: Multiple nerves can be individually affected due to cholesterol deposition in peripheral nerves, leading to peripheral neuropathy. LDLR gene mutations (Option B) are associated with familial hypercholesterolemia, which primarily affects low-density lipoprotein (LDL) cholesterol levels, not HDL levels. APOE gene mutation (Option C) is associated with various lipid disorders and Alzheimer's disease. While APOE influences lipid metabolism, it does not specifically cause the severe HDL deficiency. PCSK9 gene mutations (Option D) are linked to familial hypercholesterolemia, affecting LDL cholesterol levels rather than HDL. These mutations result in increased LDL and cholesterol levels and are not associated with HDL deficiency.



References:

Vasudevan Textbook of Biochemistry, 7th Edition, Page 342.

↑ *Back to this question in Part A*

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Q7: A 45-year-old woman presents with a history of palmar xanthoma. She reports a family history of similar symptoms. Laboratory tests reveal elevated cholesterol and triglyceride levels, with a cloudy appearance of her plasma.What is the most likely diagnosis for this patient? (Q843191)

A. Type I (Familial chylomicronemia syndrome)

B. Type IIa (Familial hypercholesterolemia)

C. Type III (Familial dysbetalipoproteinemia)

D. Type IV (Familial hypertriglyceridemia)

Correct Answer: C. Type III (Familial dysbetalipoproteinemia)

■ *Listen to Audio 1*

Explanation: TYPE III (Familial Dysbetalipoproteinemia) Lipoprotein Fraction Elevated Broad β-VLDL, Chylomicrons Cholesterol Level Increased Triglyceride Level Increased Appearance of Plasma Cloudy Metabolic Defect Abnormal Apo E Apo CII increased Features Palmar xanthomas Increased incidence of vascular disease Management Weight reduction Restrict intake of fat and cholesterol Administer polyunsaturated fatty acids (PUFA) and drugs Type I (Familial chylomicronemia syndrome) (Option A): Characterized by lipoprotein lipase deficiency, causing severe hypertriglyceridemia, eruptive xanthomas, and a creamy layer over clear plasma. Type IIa (Familial hypercholesterolemia) (Option B): Results from an LDL receptor defect , leading to high LDL levels, atherosclerosis, and tuberous xanthomas. Type IV (Familial hypertriglyceridemia) (Option D) : Associated with elevated VLDL levels, glucose intolerance, and a cloudy or milky appearance of plasma, but not typically with palmar xanthomas.



References:

Harper Biochemistry, 32nd Edition, Page 268 Vasudevan Biochemistry, 7th Edition, Page 343

↑ *Back to this question in Part A*

@Neet_pg_bot

Q8: Which of the following statements accurately distinguishes Type IIa from Type IIb Hyperlipoproteinemias? (Q843190)

A. Plasma after 24 hrs- Type IIa: clear; Type IIb: slightly cloudy.

B. Clinical Features- Type IIa: eruptive xanthoma ; Type IIb: corneal arcus.

C. Type IIa: VLDL overproduction; Type IIb: LDL and VLDL overproduction.

D. Management - Type IIa: restrict fat, supplement MCT; Type IIb: weight reduction, drug therapy.

Correct Answer: A. Plasma after 24 hrs- Type IIa: clear; Type IIb: slightly cloudy.

■ *Listen to Audio 1*

Explanation: Feature Type IIa (Familial Hypercholesterolemia) Type IIb (Combined Hyperlipidemia) Defect Defective LDL receptors or Apo B-100 ligand region mutation Increased Apo B, Apo C-II Lipoprotein Fraction Elevated (Option C) LDL LDL and VLDL Main Lipid Abnormality Hypercholesterolemia Combined hyperlipidemia (elevated cholesterol and triglycerides) Plasma after 24 ho urs (Option A ruled out) Clear Cloudy Features (Option B ruled out) Atherosclerosis Coronary artery disease (early onset) Tendon xanthomas Corneal arcus. Management (Option D ruled out) Statins PUFA Lifestyle modifications (diet, exercise) Statins PUFA Lifestyle modifications (diet, exercise)

References:

Harper Biochemistry, 32nd Edition, Page 268 Vasudevan biochemistry, 7th Edition, Page 343

↑ *Back to this question in Part A*

@Neet_pg_bot

Q9: A 5-year-old boy presents with recurrent episodes of severe abdominal pain and hepatomegaly. Physical examination reveals eruptive xanthomas on his trunk and limbs. Blood tests show a creamy layer over clear plasma after overnight refrigeration. What metabolic defect is most likely present in this patient? (Q843189)

A. LDL receptor defect

B. Apo B-100 mutation

C. Lipoprotein lipase deficiency

D. Apo E abnormality

Correct Answer: C. Lipoprotein lipase deficiency

■ *Listen to Audio 1*

Explanation: Lipoprotein lipase deficiency : This defect causes familial chylomicronemia syndrome (Type I), leading to hypertriglyceridemia, eruptive xanthomas, hepatomegaly, and a creamy layer over clear plasma. Hyperlipoproteinemia Metabolic defect Lipid profile (Increase in:) Inheritance Type I Lipoprotein lipase

deficiency Defect in Apo-C2 Chylomicrons, TG, cholesterol Autosomal Recessive Type II A LDL receptor defect; Apo B-100 mutation (Option A and B) Cholesterol Autosomal Dominant Type II B Increased Apo B and Apo CII Cholesterol, LDL, TG Autosomal Dominant Type III Abnormal Apo E; increased Apo CII(Option D) Chylomicrons, VLDL Autosomal Recessive Type IV Overproduction of VLDL; increased Apo CII VLDL, TG Autosomal Dominant Type V Secondary to other conditions VLDL, TG Autosomal Dominant

References:

Harper Biochemistry, 32nd Edition, Page 268 Vasudevan Biochemistry, 7th Edition, Page 343

↑ *Back to this question in Part A*

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Q10: Match the apolipoproteins with their respective functions in lipid metabolism: Apolipoprotein Function 1) Apolipoprotein A-I a. Enhances lipoprotein lipase activity, facilitating triglyceride hydrolysis. 2) Apolipoprotein B-100 b. Involved in the transport of dietary lipids from the intestine to other tissues. 3) Apolipoprotein C-II c. Helps in reverse cholesterol transport, removing excess cholesterol from tissue back to liver. 4) Apolipoprotein E d. Essential for binding to LDL receptors on various tissues. (Q843188)

A. 1-c, 2-d, 3-a, 4-b

B. 1-b, 2-c, 3-d, 4-a

C. 1-c, 2-d, 3-d, 4-a

D. 1-d, 2-a, 3-c, 4-b

Correct Answer: A. 1-c, 2-d, 3-a, 4-b

■ *Listen to Audio 1*

Explanation: Apolipoprotein Associated Lipoproteins Function Apolipoprotein A-I HDL (High-Density Lipoprotein) Major protein component of HDL facilitates reverse cholesterol transport, removing excess cholesterol from tissues and transporting it back to the liver. Apolipoprotein B-100 LDL (Low-Density Lipoprotein) Main apolipoprotein of LDL; essential for binding to LDL receptors on various tissues , facilitating cholesterol uptake. Apolipoprotein B-48 Chylomicrons Main apolipoprotein of chylomicrons; involved in the transport of dietary lipids from the intestine to other tissues. Apolipoprotein C-II Chylomicrons, VLDL (Very-Low-Density Lipoprotein) Acts as a cofactor for lipoprotein lipase, enhancing triglyceride hydrolysis and aiding in lipoprotein metabolism. Apolipoprotein C-III VLDL, HDL Inhibits lipoprotein lipase activity, affecting triglyceride metabolism and lipoprotein clearance. Apolipoprotein E Chylomicrons, VLDL, HDL Facilitates the uptake of triglyceride-rich lipoproteins by cells through interactions with specific receptors; involved in lipid transport and metabolism.

References:

Harper’s Illustrated Biochemistry, 23rd Edition, Page 249-251.

Vasudevan Textbook of Biochemistry, 7th Edition, Page 174, 175.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: Following are the substrates of cholesterol synthesis. Arrange them in order of appearance in the cholesterol synthesis pathway. Mevalonate Squalene Lanosterol Zymosterol Desmosterol (Q843016)

A. 1,2,3,4,5

B. 5,4,3,2,1

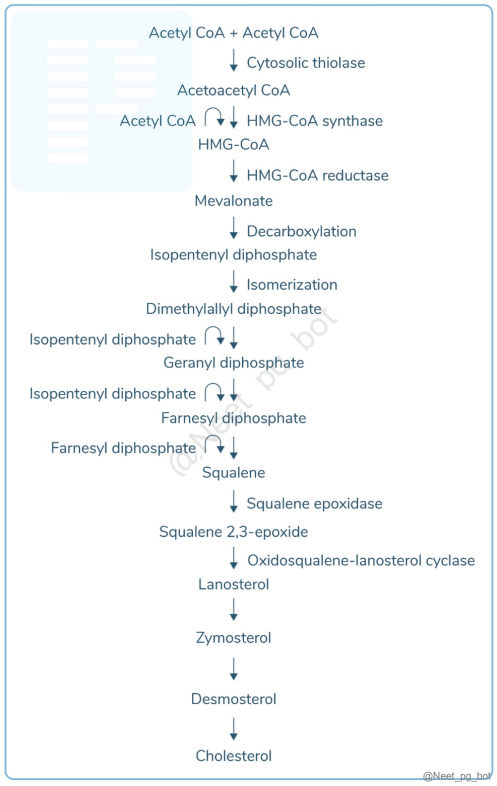
C. 4,3,1,2,5

D. 1,3,5,2,4

Correct Answer: A. 1,2,3,4,5

■ *Listen to Audio 1*

Explanation: The substrates of cholesterol synthesis appear in the following order: Mevalonate Isopentenyl pyrophosphate (IPP) Farnesyl pyrophosphate (FPP) Squalene Lanosterol Zymosterol Desmosterol Cholesterol So, the correct order of the listed substrates in the cholesterol synthesis pathway is: Mevalonate → Squalene → Lanosterol → Zymosterol → Desmosterol. Cholesterol synthesis pathway:



References:

Harper's Biochemistry, 32nd Edition, Page 260

↑ *Back to this question in Part A*

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Q12: A 45-year-old male presents with a history of progressive kidney dysfunction and recurrent episodes of hemolytic anemia. On examination, the patient is found to have corneal opacification on slit-lamp examination. His lipid profile shows high levels of cholesterol and lipoprotein X. What is the most likely diagnosis? (Q843025)

A. Tangier disease

B. Familial hypercholesterolemia

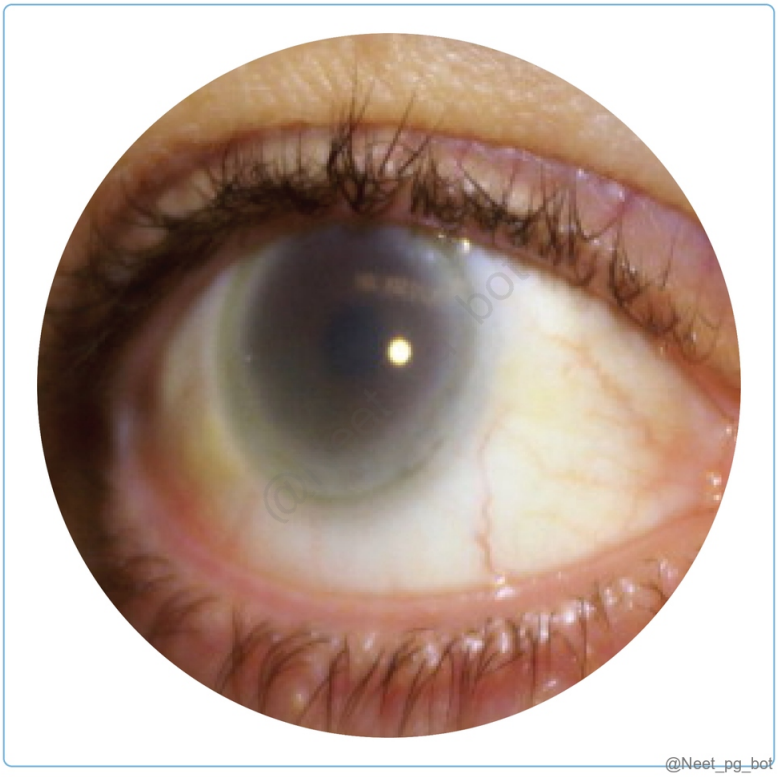
C. LCAT deficiency

D. Niemann-Pick disease

Correct Answer: C. LCAT deficiency

■ *Listen to Audio 1*

Explanation: The most likely diagnosis for the given clinical scenario is LCAT deficiency (Lecithin-Cholesterol Acyltransferase deficiency). LCAT deficiency exists in two primary forms: Partial LCAT Deficiency (Fish eye disease) Complete LCAT Deficiency (Norum's disease) LCAT Enzyme Activity Partial loss, mainly affecting corneal lipid metabolism Severe impairment or absence, leading to systemic issues Corneal Appearance Fish-eye appearance due to cholesterol ester accumulation Fisheye disease: similar corneal opacification Systemic Involvement Less severe, primarily ocular Severe: hemolytic anemia, CKD, early myocardial infarction, progressive End-stage renal disease Tangier disease (Option A) is associated with very low HDL levels and characteristic features such as orange tonsils and peripheral neuropathy. Familial hypercholesterolemia (Option B) typically presents with high LDL levels, xanthomas, and early cardiovascular disease, but not hemolytic anemia or corneal opacification. Niemann-Pick disease (Option D) is a lysosomal storage disorder involving neurological symptoms and organomegaly.



References:

Harper's Illustrated Biochemistry, 23rd Edition, Page 268.

Vasudevan Textbook of Biochemistry, 7th Edition, Page 344

↑ *Back to this question in Part A*

@Neet_pg_bot

Q13: Which of the following best describes the primary function of Very Low-Density Lipoprotein (VLDL) in the body? (Q843024)

A. Transport of dietary cholesterol to the liver

B. Transport of triglycerides from the liver to peripheral tissues

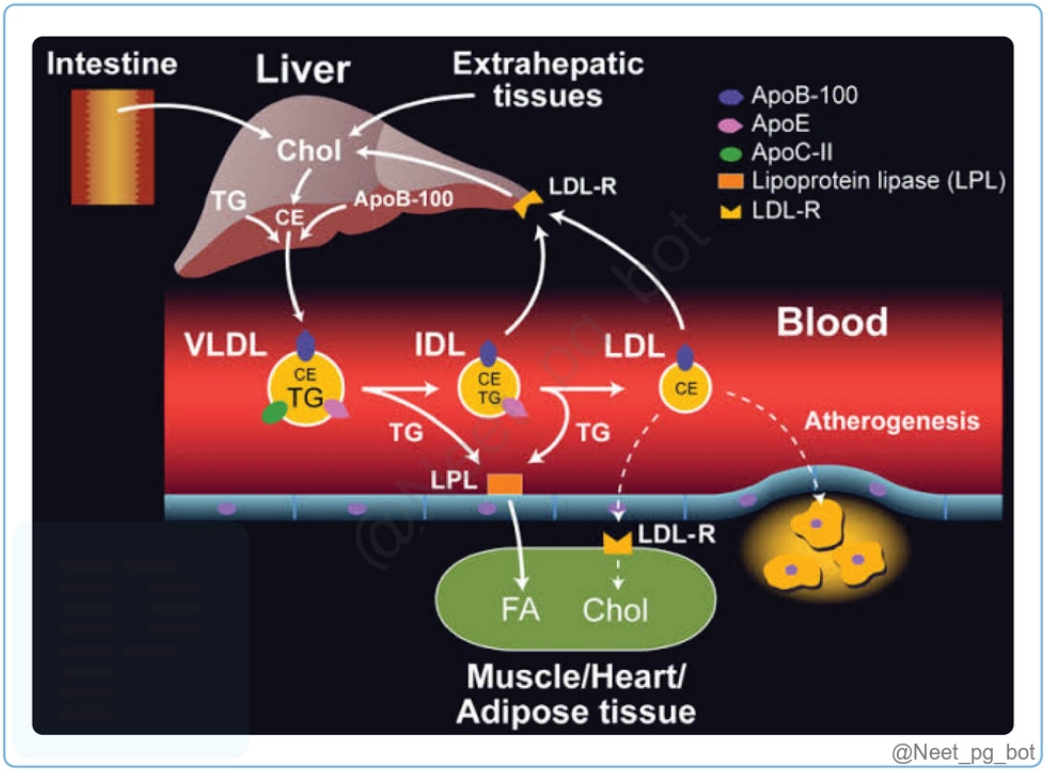
C. Removal of cholesterol from peripheral tissues to the liver

D. Storage of excess cholesterol in adipose tissue

Correct Answer: B. Transport of triglycerides from the liver to peripheral tissues

■ *Listen to Audio 1*

Explanation: VLDL is synthesized in the liver and its main role is to transport triglycerides to peripheral tissues , such as muscle and adipose tissue, where they can be used for energy or stored. VLDL is rich in triglycerides and also contains cholesterol and apolipoproteins. As VLDL releases triglycerides to cells, it becomes intermediate density lipoprotein(IDL) and eventually low density lipoprotein(LDL). This conversion of VLDL to IDL and then to LDL is referred to as lipoprotein cascade pathway Transport of dietary cholesterol to the liver (Option A) - This function is primarily carried out by chylomicrons, not VLDL. Chylomicrons are responsible for transporting dietary lipids, including cholesterol, from the intestines to the liver. Removal of cholesterol from peripheral tissues to the liver (Option C) - This function is primarily carried out by high-density lipoprotein (HDL), not VLDL. HDL is involved in reverse cholesterol transport, carrying cholesterol away from the tissues and back to the liver for excretion. Storage of excess cholesterol in adipose tissue (Option D) - This is not a function of VLDL. Excess cholesterol can be stored in various tissues, but VLDL primarily transports triglycerides, while LDL and other mechanisms are involved in cholesterol storage and transport.



References:

Harper's Biochemistry, 32nd Edition, Page 250 & 251

↑ *Back to this question in Part A*

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Q14: The lipid profile of 55 Y/O female is given below: Triglycerides 150 mg/dL Total cholesterol 350 mg/dL HDL 90 mg/dL Calculate LDL level of this patient. (Q843023)

A. 250 mg/dL

B. 230 mg/dL

C. 200 mg/dL

D. 270 mg/dL

Correct Answer: B. 230 mg/dL

■ *Listen to Audio 1*

Explanation: LDL of the patient can be calculated using Friedwald's equation: Total cholesterol = HDL+LDL+TG/5 mg/dL Substituting the values, 350 = 90+LDL+150/5 LDL = 350-90-30 = 230 mg/dL Friedwald's equation is accurate when triglycerides levels are below 400 mg/dL.

References:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6960042/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q15: Which enzyme facilitates the conversion of discoidal HDL into spheroidal HDL in HDL metabolism? (Q843022)

A. Lipoprotein lipase (LPL)

B. Lecithin-cholesterol acyltransferase (LCAT)

C. Hepatic lipase

D. Cholesteryl ester transfer protein (CETP)

Correct Answer: B. Lecithin-cholesterol acyltransferase (LCAT)

■ *Listen to Audio 1*

Explanation: Lecithin-cholesterol acyltransferase is the enzyme responsible for the conversion of discoidal HDL (aka Nascent HDL- amphipathic) into spheroidal HDL (aka Mature HDL- hydrophobic). It catalyzes the transfer of an acyl group from lecithin (phosphatidylcholine) to cholesterol, forming cholesteryl esters. HDL Metabolism HDL Formation: Formed in the liver and intestines as nascent, discoidal particles rich in apolipoprotein A-I (apoA-I). Cholesterol Efflux: ABCA1 Transporter , transfers free cholesterol and phospholipids to nascent HDL. ABCG1 and SR-BI Transporters facilitate cholesterol efflux to HDL. Maturation of HDL: Lecithin-cholesterol acyltransferase (LCAT) converts discoidal HDL into spherical HDL by forming cholesterol esters that move to the core of HDL particles. HDL Remodeling: Phospholipid transfer protein (PLTP) and cholesteryl ester transfer protein (CETP) modify HDL size and composition by transferring lipids between lipoproteins. Selective Uptake and HDL Recycling: Scavenger Receptor Class B Type I (SR-BI) facilitates selective uptake of cholesterol esters from HDL into hepatocytes. CETP transfers cholesteryl esters from HDL to VLDL and LDL for liver uptake. Excretion: The liver converts cholesterol into bile acids or secretes it into bile for excretion via intestines. Lipoprotein lipase (LPL) (Option A) is an enzyme that hydrolyzes triglycerides in lipoproteins into free fatty acids and glycerol. Hepatic lipase (Option C) primarily hydrolyzes triglycerides and phospholipids in intermediate-density lipoproteins (IDL) and low-density lipoproteins (LDL). Cholesteryl ester transfer protein (Option D) (CETP), facilitates the transfer of cholesteryl esters and triglycerides between lipoproteins.

References:

Harper's Illustrated Biochemistry, 23rd Edition, Page 253

Textbook of Biochemistry for Medical Students,Vasudevan, 7th Edition, Page 179, 180.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q16: What is Apo B48 primarily associated with? (Q843021)

A. High-density lipoproteins (HDL)

B. Chylomicrons

C. Very low-density lipoproteins (VLDL)

D. Low-density lipoproteins (LDL)

Correct Answer: B. Chylomicrons

■ *Listen to Audio 1*

Explanation: Apolipoprotein B-48 is an essential component of chylomicrons. Chylomicrons are lipoproteins that transport dietary lipids from the intestines to other locations in the body. Apo B48 is synthesized in the intestines and is integral to the structure and function of chylomicrons, ensuring proper lipid absorption and transport. Lipoprotein Type Major Apolipoproteins associated Chylomicrons Apo B48, ApoA-I, ApoA-II, ApoC-II, ApoE VLDL ApoB-100, ApoC-II, ApoE LDL ApoB-100 HDL ApoA-I, ApoA-II, ApoC-II, ApoE

References:

Harper’s Illustrated Biochemistry, 23rd Edition, Page 248, 249.

Textbook of Biochemistry for Medical Students,Vasudevan, 7th Edition, Page 173.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: Match the lipoprotein with its function. Lipoprotein Function 1.Chylomicron a.Transports triglycerides from the liver to muscle and adipose tissue 2.Very low-density lipoprotein (VLDL) b.Transports cholesterol from peripheral tissue to the liver 3. Low-density lipoprotein (LDL) c.Transports cholesterol from the liver to peripheral tissues 4. High-density lipoprotein (HDL) d.Transports triglycerides from the gut to muscle and adipose tissue (Q843020)

A. 1-d, 2-a, 3-c, 4-b

B. 1-a, 2-b, 3-c, 4-d

C. 1-a, 2-b, 3-d, 4-c

D. 1-c, 2-d, 3-b, 4-a

Correct Answer: A. 1-d, 2-a, 3-c, 4-b

■ *Listen to Audio 1*

Explanation: Since lipids are insoluble in water, they are transported in blood as lipoproteins Lipoprotein Function Chylomicron Transports triglycerides from the gut to liver, muscle and adipose tissue Very low-density lipoprotein (VLDL) Transports triglycerides from the liver to muscle and adipose tissue Intermediate-density lipoprotein (IDL) VLDL remnant Low-density lipoprotein (LDL) Transports cholesterol from the liver to peripheral tissues High-density lipoprotein (HDL) Transports cholesterol from peripheral tissue to the liver

References:

Harper's Biochemistry, 32nd Edition, Page 251

Textbook of Biochemistry for Medical Students,Vasudevan, 7th Edition, Page 173-175

↑ *Back to this question in Part A*

@Neet_pg_bot

Q18: Which of the following is true regarding bile acid synthesis regulation? 7α-hydroxylation is the step which is suppressed Cholic acid suppresses the synthesis Farnesoid X receptor is activated Transcription of the CYP7A1 gene is suppressed (Q843019)

A. 1,2,3

B. 1,3,4

C. 2,3

D. 2,3,4

Correct Answer: B. 1,3,4

■ *Listen to Audio 1*

Explanation: Chenodeoxycholic acid (not cholic acid) is the most potent bile acid inhibitor. It activates FXR , which suppresses CYP7A1 transcription , reducing 7α-hydroxylase activity (rate-limiting step). This leads to ↓ bile acid synthesis. Mechanisms of Regulation: 1. Feedback Inhibition (Most Important) Bile acids (especially chenodeoxycholic acid) inhibit CYP7A1 Prevents excess bile acid formation Negative feedback loop: ↑ Bile acids

→ ↓ CYP7A1 activity → ↓ bile acid synthesis 2. Nuclear Receptor Regulation Bile acids activate FXR (Farnesoid X receptor) FXR → ↑ SHP (small heterodimer partner) SHP → inhibits CYP7A1 gene expression 3. Enterohepatic Circulation ~95% of bile acids are reabsorbed in terminal ileum Returned to liver via portal circulation High return → suppresses synthesis Loss (e.g., diarrhea, ileal disease) → increases synthesis 4. Hormonal Regulation Hormone Effect Insulin ↑ Stimulates synthesis Glucagon ↓ Inhibits synthesis Thyroid hormone ↑ Increases CYP7A1 activity 5. Cholesterol Availability ↑ Cholesterol → ↑ bile acid synthesis Helps maintain cholesterol homeostasis 6. Drug Influence Bile acid sequestrants (e.g., cholestyramine) : Prevent reabsorption ↑ bile acid synthesis from cholesterol ↓ plasma cholesterol

References:

Harper's Biochemistry, 32nd Edition, Page 266

↑ *Back to this question in Part A*

@Neet_pg_bot

Q19: Which of the following statements is true regarding the fate of cholesterol? Chenodeoxycholic acid and cholic acid are the primary bile acids Glycine-taurine conjugation happens in liver peroxisomes Intestinal bacteria produce secondary bile acids (Q843018)

A. 1, 2

B. 1, 2, 3

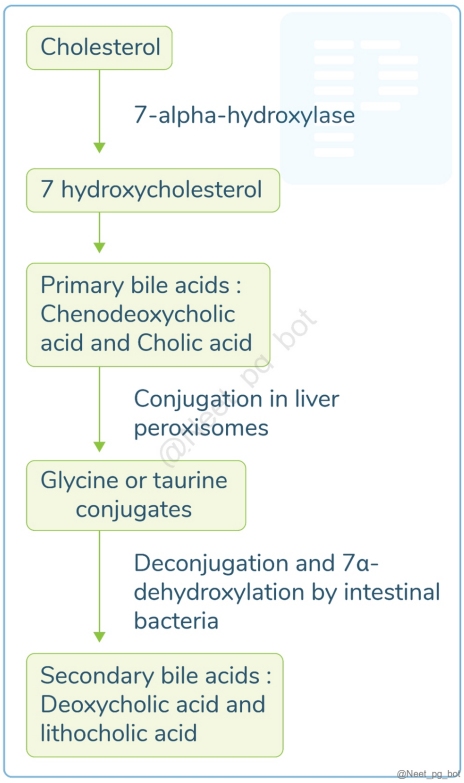
C. 2, 3

D. Only 1

Correct Answer: B. 1, 2, 3

■ *Listen to Audio 1*

Explanation: Chenodeoxycholic acid and cholic acid are the primary bile acids. Deoxycholic acid and lithocholic acid are secondary bile acids. Primary bile acids are conjugated in liver peroxisomes. They are converted into glycine or taurine conjugates. Intestinal bacteria deconjugate and de-hydroxylase the glycine-taurine conjugates to form secondary bile acids. Fate of cholesterol: Note: The anions of bile acids and their glycine or taurine conjugates are referred to as bile salts. In the alkaline environment of bile, they are expected to exist in this form.



References:

Harper's Biochemistry, 32nd Edition, Page 265

↑ *Back to this question in Part A*

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Q20: Which of the following enzymes catalyzes the principal regulatory step of cholesterol synthesis? (Q843017)

A. HMG CoA synthase

B. HMG CoA reductase

C. Cytosolic thiolase

D. Squalene epoxidase

Correct Answer: B. HMG CoA reductase

■ *Listen to Audio 1*

Explanation: The principal regulatory step in the cholesterol synthesis pathway: HMG-CoA is reduced to mevalonate, catalyzed by HMG-CoA reductase Site- cytoplasm and smooth endoplasmic reticulum Regulation of HMG-CoA reductase: Category Regulation Method Mechanism Allosteric Modification Statins (e.g., Lovastatin) Competitive inhibition of HMG CoA reductase Covalent Modification AMP-activated Kinase Phosphorylates HMG CoA reductase, inactivating it Protein Phosphatase 1 Dephosphorylates HMG CoA reductase, activating it Induction or Repression Sufficient Cholesterol Suppresses transcription of the HMG CoA reductase gene Low Dietary Cholesterol Increases transcription of the HMG CoA reductase gene Insulin and Thyroxine Increase the activity of HMG CoA reductase (induction) Cortisol and Glucagon Decrease the activity of HMG CoA reductase (repression) HMG CoA synthase (Option A) catalyzes the formation of HMG-CoA from acetyl-CoA and acetoacetyl-CoA, which is an upstream step in the cholesterol synthesis pathway but not the rate-limiting step. - HMG CoA Synthase is also a part of ketone body synthesis pathway (mitochondrial) - Cholesterol pathway enzyme is cytoplasmic Cytosolic thiolase (Option C) is involved in an upstream step of cholesterol synthesis by converting two molecules of acetyl-CoA into acetoacetyl-CoA, not directly involved in the regulatory step. Squalene epoxidase (Option D) catalyzes the conversion of squalene to squalene-2,3-epoxide, a downstream step in the cholesterol synthesis pathway, but it is not a regulatory step.

References:

Harper's Biochemistry, 32nd Edition, Page 260

Textbook of Biochemistry for Medical Students,Vasudevan, 7th Edition, Page 171, 173

↑ *Back to this question in Part A*

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• [03_Lipid Chemistry] 012 Previous Year Questions

« Back to Folder Directory

[03_Lipid Chemistry] 012 Previous Year Questions — Quick Jump

Q1 (p. 699)	*Q2 (p. 699)*	*Q3 (p. 700)*	*Q4 (p. 701)*	*Q5 (p. 701)*	*Q6 (p. 702)*
Q7 (p. 703)	*Q8 (p. 704)*	*Q9 (p. 704)*	*Q10 (p. 705)*	*Q11 (p. 706)*	*Q12 (p. 707)*
Q13 (p. 708)	*Q14 (p. 710)*	*Q15 (p. 711)*	*Q16 (p. 712)*	*Q17 (p. 712)*	*Q18 (p. 714)*
Q19 (p. 715)	*Q20 (p. 715)*	*Q21 (p. 716)*	*Q22 (p. 717)*	*Q23 (p. 718)*	*Q24 (p. 719)*
Q25 (p. 720)	*Q26 (p. 720)*	*Q27 (p. 721)*	*Q28 (p. 722)*	*Q29 (p. 723)*	*Q30 (p. 723)*
Q31 (p. 724)	*Q32 (p. 725)*	*Q33 (p. 726)*	*Q34 (p. 727)*	*Q35 (p. 728)*	*Q36 (p. 728)*
Q37 (p. 729)	*Q38 (p. 730)*	*Q39 (p. 730)*	*Q40 (p. 731)*	*Q41 (p. 732)*	*Q42 (p. 732)*
Q43 (p. 733)	*Q44 (p. 735)*	*Q45 (p. 736)*	*Q46 (p. 736)*		

Q1: Which of the cycles listed below is absent in muscle tissue? (Q856932)

A. Glycogenolysis

B. Ketogenesis

C. Glycogenesis

D. Lipolysis

Correct Answer: B. Ketogenesis

Explanation: Ketogenesis is absent in muscle tissue because: It occurs exclusively in the liver (mitochondria of hepatocytes). The liver has the necessary enzyme HMG-CoA synthase, which is absent in muscle. Purpose of Ketogenesis: Converts acetyl-CoA into ketone bodies (acetoacetate, β-hydroxybutyrate). Ketone bodies are then released into the blood for use by peripheral tissues, including muscle, during fasting or low glucose states. Glycogenolysis (Option A): Muscle breaks down glycogen for its own energy needs during exercise. Glycogenesis (Option C): Muscle stores glycogen for later energy use. Lipolysis (Option D): Muscle can break down triglycerides to obtain fatty acids for energy.

References:

Ketogenesis

↑ *Back to this question in Part A*

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Q2: Which of the following enzymes has the highest catalytic efficiency? (Q856482)

A. Km of an enzyme = 10 micromole and Kcat = 20 per sec

B. Km of an enzyme = 2000 nanomole and Kcat = 50 per sec

C. Km of an enzyme = 2 micromole and Kcat = 200 per sec

D. Km of an enzyme = 4 micromole and Kcat = 200 per sec

Correct Answer: C. Km of an enzyme = 2 micromole and Kcat = 200 per sec

Explanation: Km of an enzyme = 2 micromole and Kcat = 200 per sec Catalytic efficiency, also known as specificity constant, is a measure of how effectively an enzyme converts substrate to product. It is calculated as the ratio of the catalytic rate constant (Kcat) to the Michaelis constant (Km). Km of an enzyme = 2 micromole and Kcat = 200 per sec : Catalytic efficiency = Kcat / Km = 200 / 2 = 100 per micromole per sec. Catalytic efficiency refers to the measure of how effectively an enzyme catalyzes a specific reaction. It is a measure of the enzyme's ability to convert substrate molecules into products within a given time frame. Catalytic efficiency is commonly expressed using the Michaelis-Menten equation, which relates the rate of the enzymatic reaction to the concentration of the substrate. The two important parameters used to evaluate catalytic efficiency are the turnover number (kcat) and the Michaelis constant (Km).

References:

Catalytic efficiency

↑ *Back to this question in Part A*

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Q3: A 6-month-old infant is brought to the emergency department with lethargy, vomiting, and poor feeding. The episode occurred after prolonged fasting. Laboratory results reveal hypoglycemia, low ketone levels (hypoketosis), and mild hepatomegaly. These findings suggest a defect in fat metabolism. Which of the following is the most likely underlying disorder? (Q866670)

A. Her’s disease

B. Medium-chain acyl-CoA dehydrogenase (MCAD) deficiency

C. Hereditary fructose intolerance

D. Glycogen storage disease type I (Von Gierke disease)

Correct Answer: B. Medium-chain acyl-CoA dehydrogenase (MCAD) deficiency

Explanation: This presentation is classic for medium-chain acyl-CoA dehydrogenase (MCAD) deficiency, a fatty acid β-oxidation defect. Prolonged fasting - Body normally switches to fatty acid oxidation for energy and ketone production. MCAD deficiency - Inability to break down medium-chain fatty acids → impaired ketogenesis and gluconeogenesis. Clinical Features: Hypoglycemia (due to no gluconeogenesis from fat-derived acetyl-CoA) Hypoketosis (unbale to synthesize ketone bodies from fatty acids) Mild hepatomegaly (fat accumulation in liver) Typical age: Symptoms often appear between 3–24 months, especially after illness or prolonged fasting. Incorrect Options: Her's disease (GSD type VI) (Option A): Liver phosphorylase deficiency → fasting hypoglycemia + hepatomegaly, but ketones are normal because fat oxidation is intact. Hereditary fructose intolerance (Option C): Aldolase B deficiency → symptoms after fructose ingestion, not prolonged fasting; causes hypoglycemia, vomiting, jaundice. GSD type I (Von Gierke disease) (Option D): Severe fasting hypoglycemia, lactic acidosis, hyperuricemia, hyperlipidemia, but normal or high ketones .

↑ *Back to this question in Part A*

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Q4: A patient presents with orange-colored tonsils. Laboratory investigations reveal triglyceride level of 140 mg/dL and HDL cholesterol of 5 mg/dL. What is the most likely diagnosis? (Q866332)

A. Familial hypercholesterolemia

B. Tangier disease

C. Type I hyperlipoproteinemia

D. Abetalipoproteinemia

Correct Answer: B. Tangier disease

Explanation: The presence of orange-colored tonsils, extremely low HDL cholesterol (5 mg/dL), and normal triglyceride levels (140 mg/dL) are hallmark features of Tangier Disease , a rare inherited disorder of lipid metabolism. Incorrect Options: Familial hypercholesterolemia (Option A) is characterized by high LDL cholesterol, not low HDL, and typically presents with tendon xanthomas, not orange-colored tonsils. Type I hyperlipoproteinemia (Familial chylomicronemia) (Option C) involves markedly elevated triglyceride levels, often exceeding 1000 mg/dL, and is not associated with low HDL or orange tonsils. Abetalipoproteinemia (Option D) is marked by low or absent ApoB-containing lipoproteins such as VLDL, LDL, and chylomicrons, and presents with fat malabsorption, acanthocytosis, and neurological symptoms, but not orange-colored tonsils.

↑ *Back to this question in Part A*

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Q5: A patient presents with elevated total cholesterol, subcutaneous xanthomas, and a positive family history of similar findings. Triglyceride levels are normal (<140 mg/dL). What is the most likely type of familial dyslipidemia? (Q866333)

A. Type I

B. Type IIa

C. Type IIb

D. Type II

Correct Answer: B. Type IIa

Explanation: The combination of elevated total cholesterol, normal triglyceride levels (<140 mg/dL), subcutaneous xanthomas, and a positive family history is characteristic of Type IIa Familial Dyslipidemia, also known as Familial Hypercholesterolemia . Feature Type IIa (Familial Hypercholesterolemia) (Option B) Type IIb (Combined Hyperlipidemia) (Option C ruled out) Defect Defective LDL receptors or Apo B-100 ligand region mutation Increased Apo B, Apo C-II Lipoprotein Fraction Elevated LDL LDL and VLDL Main Lipid Abnormality Hypercholesterolemia Combined hyperlipidemia (elevated cholesterol and triglycerides) Plasma after 24 hours Clear Cloudy Features Atherosclerosis Coronary artery disease (early onset) Tendon xanthomas Corneal arcus. Management Statins PUFA Lifestyle modifications (diet, exercise) Statins PUFA Lifestyle modifications (diet, exercise) Incorrect Options: Type I (Option A) dyslipidemia is characterized by elevated chylomicrons, leading to severely high triglyceride levels, not elevated cholesterol. It typically presents with pancreatitis, rather than xanthomas or isolated high cholesterol. Type II (Option D) is a non-specific term that does not differentiate between Type IIa and IIb, and therefore is not adequate for precise diagnosis.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: A patient presents with xanthomas on the Achilles tendon. Which of the following is the most likely diagnosis? (Q844257)

A. Familial hypercholesterolemia

B. Tangier disease

C. Familial hyperchylomicronemia

D. Type III hyperlipoproteinemia

Correct Answer: A. Familial hypercholesterolemia

Explanation: Correct Option A - Familial hypercholesterolemia: Familial hypercholesterolemia (Type IIa hyperlipoproteinemia) is caused by a defect in the LDL receptor or ApoB-100 , leading to markedly elevated LDL cholesterol levels . Characteristic clinical findings include: Tendon xanthomas , especially on the Achilles tendon and extensor tendons of the hands , Xanthelasma , arcus cornealis , Premature atherosclerosis , myocardial infarction, and coronary artery disease. Tangier disease (Option B): Due to ABCA1 transporter defect , causes low HDL , orange tonsils , and peripheral neuropathy — not tendon xanthomas. Familial hyperchylomicronemia (Type I) (Option C): Caused by LPL or ApoC-II deficiency , presents with eruptive xanthomas and pancreatitis , not tendon xanthomas. Type III hyperlipoproteinemia (Option D): Caused by ApoE mutation , presents with palmar xanthomas and tuberoeruptive xanthomas , not Achilles tendon involvement.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q7: A patient presents to the emergency room with severe abdominal pain, and serum triglyceride levels are 1500 mg/dL. What is the most likely underlying defect? (Q844261)

A. Apo B-48 defect

B. Apo B-100 defect

C. Apo C-II defect

D. LDL receptor defect

Correct Answer: C. Apo C-II defect

Explanation: Correct Option C - Apo C-II defect: Apo C-II is a cofactor for lipoprotein lipase (LPL) , the enzyme responsible for hydrolyzing triglycerides in chylomicrons and VLDL into free fatty acids. A deficiency of Apo C-II (or LPL itself) results in familial hyperchylomicronemia (Type I hyperlipoproteinemia) , characterized by: Massive hypertriglyceridemia (>1000 mg/dL) Recurrent episodes of acute pancreatitis (due to elevated chylomicrons) Eruptive xanthomas Lipemia retinalis (milky retinal vessels) Despite very high triglycerides, atherosclerosis risk is low , as LDL and cholesterol are usually normal. Incorrect Options: A) Apo B-48 defect: Leads to impaired chylomicron formation → abetalipoproteinemia , presenting with fat malabsorption , not hypertriglyceridemia. B) Apo B-100 defect: Causes defective LDL formation → Type IIb hyperlipoproteinemia , with elevated LDL, not chylomicrons. D) LDL receptor defect: Causes familial hypercholesterolemia (Type IIa) with elevated LDL and tendon xanthomas , not pancreatitis.

References:

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↑ *Back to this question in Part A*

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Q8: A patient with multiple tendon xanthomas is found to have a serum cholesterol level of 398 mg/dL and an LDL level of 220 mg/dL. What is the most likely defect? (Q844263)

A. Lipoprotein lipase deficiency

B. LDL receptor defect

C. Defective Apo B-100

D. PCSK9 gain-of-function mutation

Correct Answer: B. LDL receptor defect

Explanation: The most likely defect in a patient with multiple tendon xanthomas, elevated serum cholesterol levels, and LDL levels is an LDL receptor defect. LDL levels in the blood remain high because the liver and other cells cannot efficiently remove LDL from the bloodstream. This leads to elevated cholesterol levels and the formation of xanthomas, resulting from the accumulation of cholesterol in tissues. LDL receptor defect One allele affected – LDL receptor defective individual (LDL receptor activity <25% of normal) Gene dose effect: Milder Both alleles affected – LDL receptor-negative individual (LDL receptor activity <8% of normal) Gene dose effect: Severe Clinical features: Eruptive xanthoma, recurrent pancreatitis, and Lipemia retinalis. LPL (Lipoprotein lipase) activity is low. Lipoprotein lipase deficiency (Option A): Causes ↑ chylomicrons and ↑ triglycerides, not predominantly LDL . Defective Apo B-100 (Option C): Also causes increased LDL but tendon xanthomas and severe elevation are more typical with receptor defects. PCSK9 gain-of-function (Option D): Can cause high LDL but is much rarer than LDL receptor defects.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q9: Which gene defect causes familial hypercholesterolemia? (Q839947)

A. Apo E

B. Apo C II

C. LDL receptor

D. Apo B48

Correct Answer: C. LDL receptor

Explanation: Correct Option C - LDL receptor: Familial hypercholesterolemia is most commonly caused by mutations in the gene coding for the LDL receptor. This receptor is responsible for the uptake of LDL cholesterol from the bloodstream into the liver. Defects in this receptor lead to high levels of LDL cholesterol in the blood. Incorrect Options: Option A - Apo E: Mutations in the Apo E gene are associated with different lipid disorders, such as familial dysbetalipoproteinemia, but not typically familial hypercholesterolemia. Option B - Apo C II: Apo C II is a cofactor for lipoprotein lipase, and defects in this gene are linked to hypertriglyceridemia, not hypercholesterolemia. Option D - Apo B48: Apo B48 is a form of apolipoprotein B that is involved in chylomicron formation and is not related to familial hypercholesterolemia. The defect in familial hypercholesterolemia involves Apo B100, not Apo B48.

↑ *Back to this question in Part A*

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Q10: Hormone-sensitive lipase is activated by all of the following EXCEPT? (Q857872)

A. Glucagon

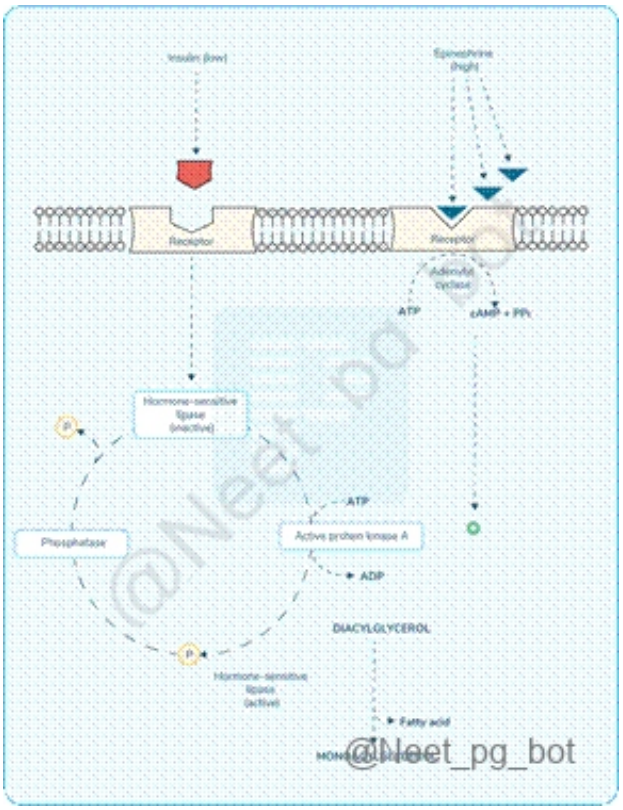
B. Epinephrine

C. ACTH

D. Insulin

Correct Answer: D. Insulin

Explanation: Insulin inhibits the activation of hormone-sensitive lipase Hormone-sensitive lipase is present in the adipose tissues which breaks down fats stored in the adipose tissue to FA + glycerol during fasting and starvation. Since it breaks fats, it is considered a catabolic enzyme. It is activated by the catabolic hormone like glucagon and inhibited by the anabolic hormone insulin. Peripheral lipolysis: It is a process in which triacylglycerols are hydrolyzed into free fatty acids (FFA) and glycerol. This process is mediated by hormone-sensitive lipase (HSL). This process occurs in adipose tissue The lipase acts on triacylglycerols and converts them into diacylglycerol , the preferred substrate for HSL. The glycerol released from the adipose tissue is transported to the liver and participates in glycolysis or gluconeogenesis. Free fatty acids bind to albumin and are transported to muscles, where they are activated into their CoA derivatives and oxidized in mitochondria for ATP. Regulation: HSL is activated when phosphorylated with Protein kinase A, which in turn is dependent on cAMP produced in the adipocyte when catecholamines activate adenylyl cyclase. Insulin dephosphorylates HSL and inactivates it.(Option D) The counter-regulatory hormones of insulin, such as glucagon (Option A), epinephrine (Option B), and cortisol, stimulate the activity of HSL. ACTH (Adrenocorticotropic Hormone) (Option C) While cortisol plays a role in metabolism, it does not directly activate hormone-sensitive lipase. Instead, cortisol can indirectly influence lipid metabolism by affecting other regulatory pathways.



References:

Lippincott Illustrated Reviews: Biochemistry 7e, pg no: 553-56

↑ *Back to this question in Part A*

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Q11: A patient presented with reduced levels of high-density lipoprotein and an ABCA1 mutation. On examination, tonsils appeared as shown in the image. What is the diagnosis? (Q855549)

A. Tangier's disease

B. Abetalipoproteinemia

C. Hyperlipoproteinemia

D. Wolman's disease



Correct Answer: A. Tangier's disease

Explanation: Tangier disease is a rare, autosomal recessive disorder caused by mutations in the ABCA1 (ATP-binding cassette, sub-family A, member 1) gene. Pathophysiology: The ABCA1 protein is crucial for the efflux of cholesterol and phospholipids to apolipoprotein A-I (ApoA-I), forming HDL particles. Mutations in the ABCA1 gene impair this process, leading to a shortage of HDL and cholesterol accumulation in tissues such as the tonsils, liver, spleen, and peripheral nerves. Clinical Features: Greyish-Orange Tonsils: (Hallmark feature) Tonsils, become enlarged and acquire greyish-orange hue due to the accumulation of cholesterol esters Hepatosplenomegaly: Due to cholesterol and phospholipid accumulation. Peripheral Neuropathy: Multiple nerves can be individually affected due to cholesterol deposition in peripheral nerves, leading to peripheral neuropathy.

References:

Reference:Vasudevan Textbook of Biochemistry, 7th Edition, Page 342.

↑ *Back to this question in Part A*

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Q12: What is the primary source of reducing equivalents for liver fatty acid synthesis? (Q856924)

A. Hexose monophosphate shunt pathway

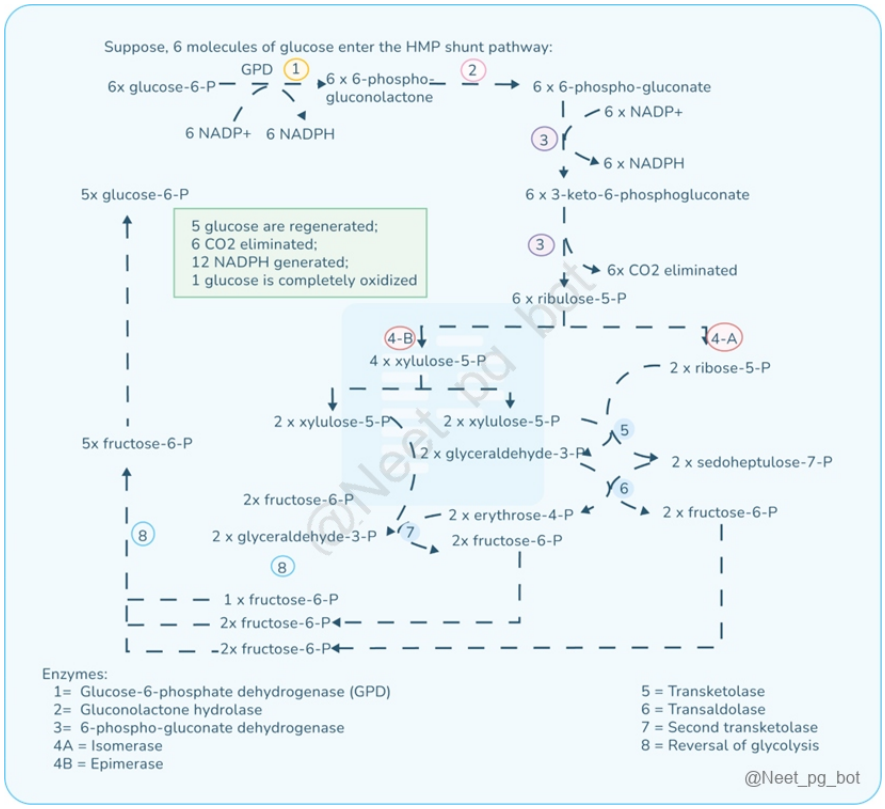
B. Glycolysis

C. Tricarboxylic acid cycle

D. Uronic acid pathway

Correct Answer: A. Hexose monophosphate shunt pathway

Explanation: The primary source of reducing equivalents (NADPH) for fatty acid synthesis in the liver is the Hexose Monophosphate Shunt (HMP), also called the Pentose Phosphate Pathway (PPP). Fatty acid synthesis involves the conversion of acetyl-CoA into palmitate. This process requires reducing equivalents (NADPH) for the reduction reactions catalyzed by fatty acid synthase. NADPH provides the electrons needed for the reduction of carbonyl groups during fatty acid elongation. HMP Shunt Provides NADPH: Occurs in the cytosol. Glucose-6-phosphate enters the pathway. Key enzyme: Glucose-6-phosphate dehydrogenase (G6PD). Produces 2 NADPH per glucose molecule oxidized. Ribose-5-phosphate (for nucleotide synthesis) is another product.



References:

HMP pathway

↑ *Back to this question in Part A*

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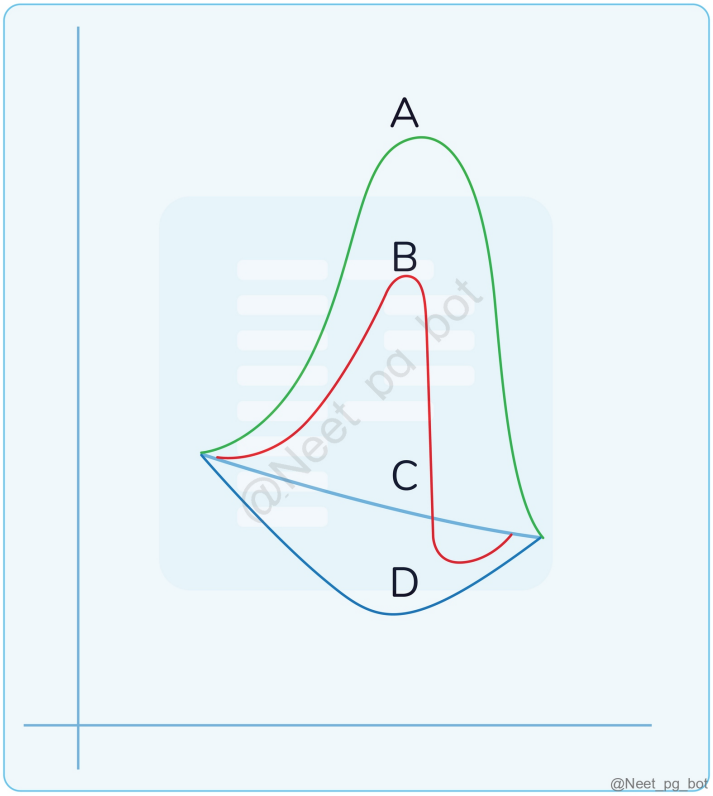
Q13: The Gibbs free energy curve of an enzyme-catalysed reaction is given by: (Q856930)

A. Curve B

B. Curve A

C. Curve D

D. Curve C



Correct Answer: A. Curve B

Explanation: The enzyme-catalyzed reaction corresponds to Curve B , which shows a lower activation energy than the uncatalyzed reaction (Curve A) , while both have the same ΔG (C → D) . Curve A – Represents the uncatalyzed reaction , which has a higher activation energy (Ea) . Curve B – Represents the enzyme-catalyzed reaction , which has a lower activation energy , hence proceeds faster. C → D – Represent the reactants and products , respectively. Enzymes lower the activation energy barrier by stabilizing the transition state, without altering the overall Gibbs free energy (ΔG) or the reaction equilibrium.

References:

Gibbs free energy

↑ *Back to this question in Part A*

Q14: What is the primary source of cholesterol synthesis? (Q856980)

A. Malonyl-CoA

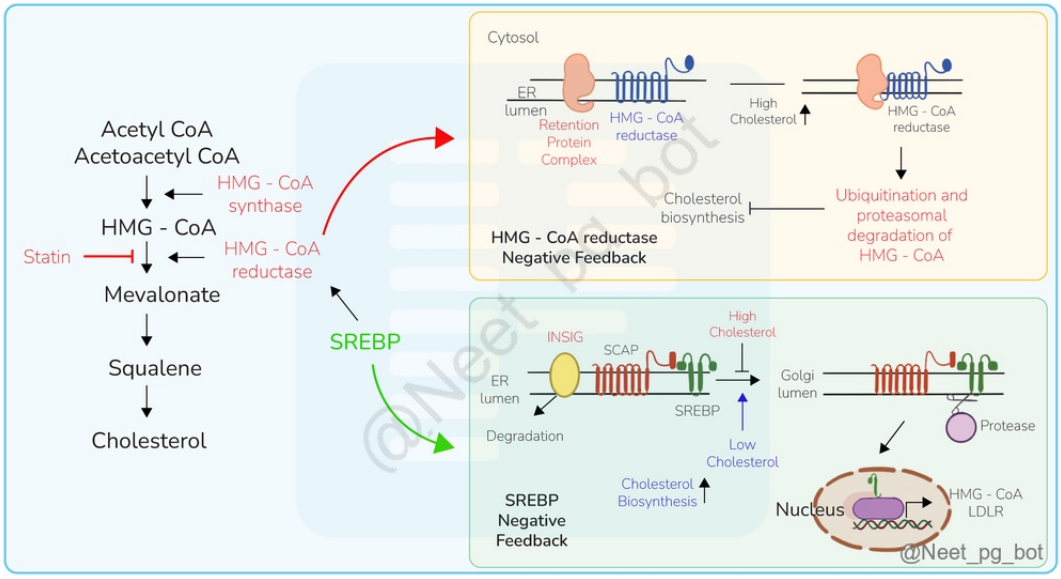
B. Pyruvate

C. Oxaloacetate

D. Acetyl-CoA

Correct Answer: D. Acetyl-CoA

Explanation: Cholesterol is synthesized from acetyl-CoA in the liver through a multi-step process called the mevalonate pathway. Acetyl-CoA → HMG-CoA (via HMG-CoA synthase) HMG-CoA → Mevalonate (via HMG-CoA reductase, the rate-limiting step) Mevalonate is further converted into isoprenoid units, which eventually lead to cholesterol formation. Malonyl CoA (Option A): Involved in fatty acid synthesis. Pyruvate (Option B): Amino acid, not involved in cholesterol synthesis. Oxaloacetate (Option C): Part of the TCA cycle, not cholesterol synthesis.



References:

Cholestrol synthesis

↑ *Back to this question in Part A*

Q15: Enzyme deficient in Gaucher disease : (Q857482)

A. Betagalacto cerebrosidase

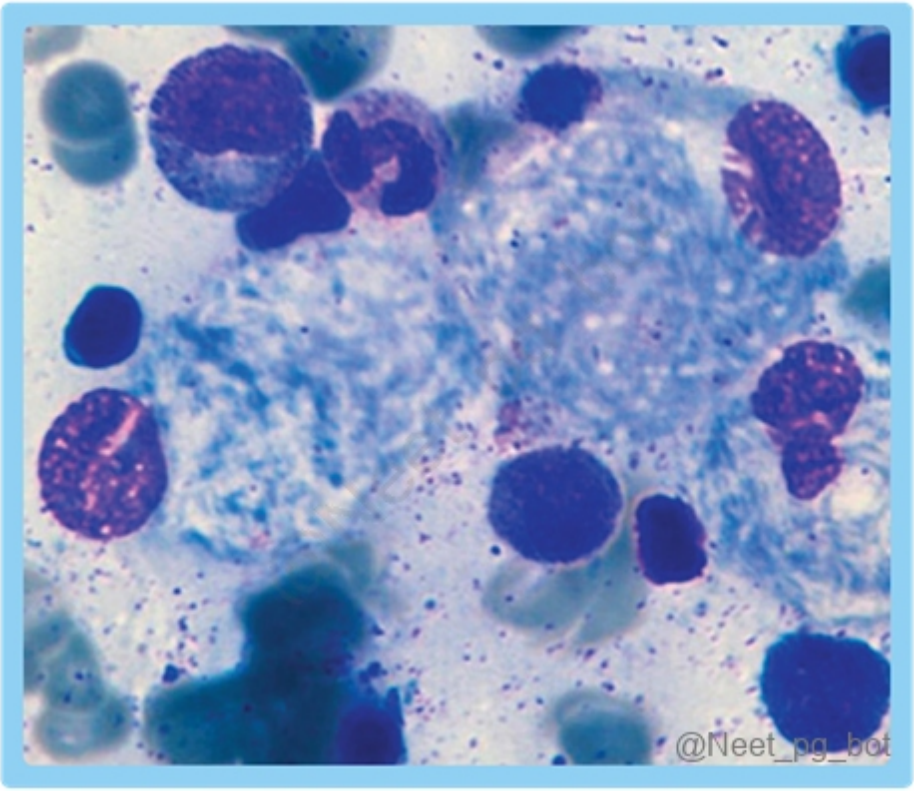
B. Glucose-6-phosphate

C. Betaglucocerebrosidase

D. Alpha glucocerebrosidase

Correct Answer: C. Betaglucocerebrosidase

Explanation: Gaucher disease , caused by a defect in beta- glucosidase (glucocerebrosidase), leads to the accumulation of glucosylceramide (glucocerebroside) within macrophages. Lysosomal storage disorder of autosomal recessive inheritance. These macrophages, engorged with glucosylceramide, are known as Gaucher cells. The accumulation affects the bone marrow and spleen, leading to anaemia and thrombocytopenia. Gaucher cells have characteristic appearance of crumbled tissue paper look due to accumulation of glucosylceramide in the cytoplasm of macrophages, forming fibril like structures. {{caption_text}}



References:

Gaucher disease

↑ *Back to this question in Part A*

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Q16: A 5-year-old girl is brought to the clinic by her mother due to the abnormalities in the genital area. On examination, there is the presence of pubic and axillary hair, indicating early androgen effects. Which of the following enzyme deficiencies is the least likely cause of her symptoms? (Q857557)

A. 17α-hydroxylase deficiency

B. 21-hydroxylase deficiency

C. 11β-hydroxylase deficiency

D. 3β-hydroxy steroid dehydrogenase deficiency

Correct Answer: A. 17α-hydroxylase deficiency

Explanation: 17α-hydroxylase deficiency results in low blood levels of estrogens, androgens, and cortisol. This deficiency causes a compensatory increase in adrenocorticotropic hormone (ACTH), which stimulates the production of 11-deoxycorticosterone and corticosterone. Elevated levels of these mineralocorticoids lead to volume expansion, hypertension, and hypokalemia. Clinically, females present with delayed puberty and primary amenorrhea, often with hypertension. In females, there may be hyperandrogenism or signs of precocious puberty . Treatment involves glucocorticoid replacement (like hydrocortisone) to suppress excess ACTH and normalize hormone levels. In addition, sex steroid hormone therapy (estrogen and progesterone) is used to induce secondary sexual characteristics and regulate menstrual cycles. Variants of congenital adrenal hyperplasia (CAH): Ambiguous genitalia in female Under virilization in Males With salt wasting & Hyperkalemia 21-hydroxylase deficiency (Option B ruled out) 3β-hydroxysteroid dehydrogenase deficiency (Option D ruled out) With Hypertension 11β-hydroxylase deficiency (Option C ruled out) 17α-hydroxylase deficiency (Option A)

References:

CAH

↑ *Back to this question in Part A*

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Q17: Insulin inhibits which of the following lipase enzymes? (Q855896)

A. Lipoprotein lipase

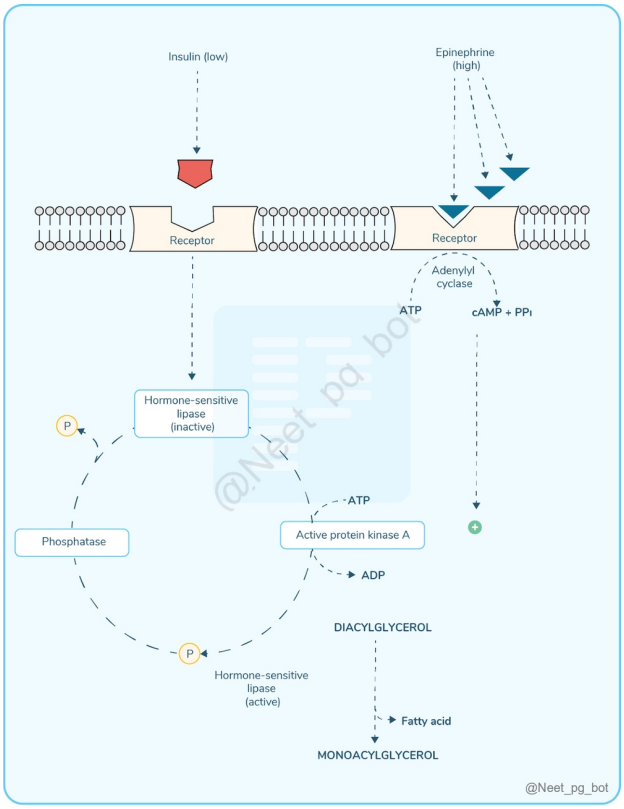
B. Pancreatic lipase

C. Gastric lipase

D. Hormone sensitive lipase

Correct Answer: D. Hormone sensitive lipase

Explanation: Hormone-sensitive lipase is present in the adipose tissues which breaks down fats stored in the adipose tissue to FA + glycerol during fasting and starvation. Since it breaks fats, it is considered a catabolic enzyme. It is activated by the catabolic hormone like glucagon and inhibited by the anabolic hormone insulin. Peripheral lipolysis: It is a process in which triacylglycerols are hydrolyzed into free fatty acids (FFA) and glycerol. This process is mediated by hormone-sensitive lipase (HSL). This process occurs in adipose tissue The lipase acts on triacylglycerols and converts them into diacylglycerol , the preferred substrate for HSL. The glycerol released from the adipose tissue is transported to the liver and participates in glycolysis or gluconeogenesis. Free fatty acids bind to albumin and are transported to muscles, where they are activated into their CoA derivatives and oxidized in mitochondria for ATP. Regulation: HSL is activated when phosphorylated with Protein kinase A, which in turn is dependent on cAMP produced in the adipocyte when catecholamines activate adenylyl cyclase. Insulin dephosphorylates HSL and inactivates it. The counter-regulatory hormones of insulin, such as glucagon, epinephrine, and cortisol, stimulate the activity of HSL. Lipoprotein lipase (Option A) is present in the endothelium of blood vessels. It breaks down fats in the blood to FA+ glycerol, which gets absorbed into adipose tissue and gets stored as triglycerides. It is considered an anabolic enzyme. It is activated by insulin and inhibited by glucagon. Pancreatic lipase (Option B) is a primary lipase enzyme that breaks down triglyceride substrates to monoglyceride + free fatty acid for absorption and storage. It is activated by insulin. Gastric lipase (Option C) is present in gastric juice where fats are broken down to triglycerides for absorption and storage. It is activated by the anabolic hormone insulin.



References:

Lippincott Illustrated Reviews: Biochemistry 7th Edition, Page no: 553-56

↑ *Back to this question in Part A*

@Neet_pg_bot

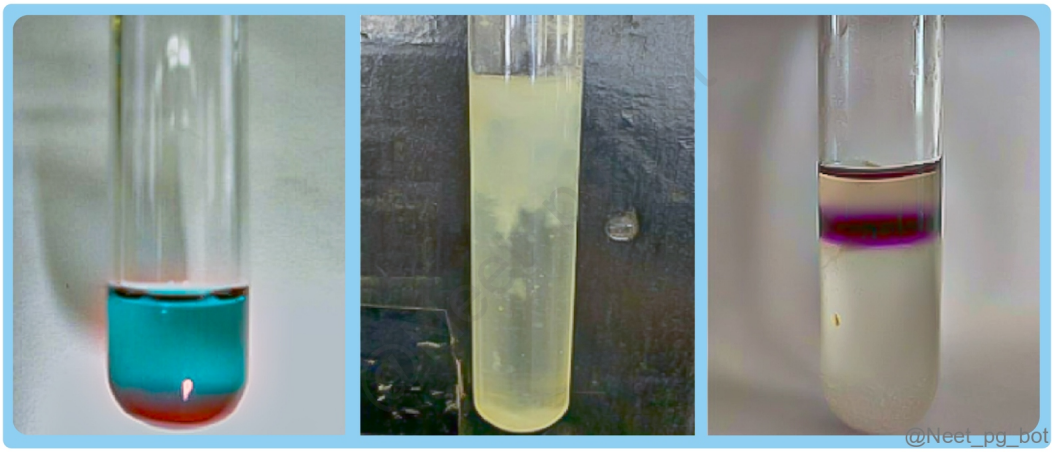
Q18: Which of the following tests will be positive in the urine of a patient who has undergone prolonged starvation? (Q855964)

A. 1 and 3

B. Only 3

C. 1, 2 and 3

D. 1 and 2



Correct Answer: B. Only 3

Explanation: Rothera's test is done for the detection of ketone bodies which are produced as a result of starvation/fasting . The purple color ring at the junction of 2 liquids is a positive test due to acetoacetate and acetone Benedict test (Image 1) : Used for the detection of reducing sugars in a complex Simple carbohydrates containing a free ketone or aldehyde functional group can be identified with this test. The test is based on Benedict’s reagent (also known as Benedict’s solution) When exposed to reducing sugars, the reactions undergone by Benedict’s reagent result in the formation of a brick-red precipitate, which indicates a positive Benedict test. Heat coagulation test (Image 2) is done for the detection of proteins in a sample

References:

Vasudevan Biochemistry, 7th Edition, Page 166, 365

↑ *Back to this question in Part A*

@Neet_pg_bot

Q19: The enzyme that is not used in cholesterol synthesis is: (Q856879)

A. Thiolase

B. HMG CoA reductase

C. HMG CoA synthase

D. HMG CoA lyase

Correct Answer: D. HMG CoA lyase

Explanation: HMG CoA lyase: Cholesterol synthesis involves several key enzymes, but HMG CoA lyase is not one of them. HMG CoA lyase: This enzyme is involved in ketogenesis , where it converts HMG CoA to acetoacetate (a ketone body), and it is not involved in the cholesterol biosynthesis pathway. Enzymes involved in cholesterol synthesis: Thiolase : This enzyme catalyzes the first step in cholesterol synthesis, where two acetyl-CoA molecules are combined to form acetoacetyl-CoA. HMG CoA synthase: This enzyme catalyzes the next step, combining acetoacetyl-CoA with another acetyl-CoA to form HMG CoA (3-hydroxy-3-methylglutaryl-CoA). HMG CoA reductase : This is the rate-limiting enzyme in cholesterol biosynthesis. It converts HMG CoA to mevalonate, a crucial step in the pathway.

References:

Cholesterol synthesis

↑ *Back to this question in Part A*

@Neet_pg_bot

Q20: The process of apolipoprotein B48 synthesis involves which of the following? (Q854877)

A. RNA alternate splicing

B. RNA editing

C. DNA editing

D. RNA interference

Correct Answer: B. RNA editing

Explanation: Apolipoprotein B (apoB) is crucial in lipid metabolism, transporting cholesterol and triglycerides in the bloodstream. It has two isoforms: apoB100 synthesized in the liver, part of VLDL, and apoB48 synthesized in the intestine, part of chylomicrons. ApoB48 synthesis involves "A-to-I RNA editing," where a specific cytosine is converted to uridine, creating a premature stop codon and producing apoB48. ApoB48 is found in chylomicrons, mediates the transport of dietary triglycerides from the intestine to peripheral tissues. Incorrect Options: RNA alternate splicing (Option A): While alternate splicing generates different mRNA isoforms, it's not directly related to apoB48 synthesis. DNA editing (Option C): ApoB48 synthesis involves RNA editing, not DNA editing. RNA interference (Option D): RNAi regulates gene expression but does not directly impact apoB48 synthesis.

References:

RNA editing

↑ *Back to this question in Part A*

@Neet_pg_bot

Q21: Which of the options aids in the transportation of fatty acids through the inner mitochondria membrane? (Q855530)

A. Acyl carrier protein

B. Carnitine

C. Lecithin-Cholesterol acyltransferase

D. Albumin

Correct Answer: B. Carnitine

Explanation: The carnitine shuttle system transports long-chain fatty acids into the mitochondrial matrix for β-oxidation. It involves: Carnitine palmitoyltransferase I (CPT-I): Converts fatty acyl-CoA to fatty acyl-carnitine. Translocase : Moves fatty acyl-carnitine across the inner mitochondrial membrane. Carnitine palmitoyltransferase II (CPT-II): Converts fatty acyl-carnitine back to fatty acyl-CoA inside the matrix. Movement of fatty acids across inner mitochondrial membrane where they are oxidised to produce energy depends on carnitine. Acyl carrier protein (Option A): Involved in movement of fatty acids within the cytosol, but not in the movement of fatty acids across mitochondrial membrane. Lecithin-Cholesterol acyltransferase (Option C): Involved in metabolism of phospholipids and cholesterol. Albumin (Option D): Functions as carrier and aids in the transportation of numerous chemicals in the blood, including fatty acids, but not directly.

References:

Carnitine

↑ *Back to this question in Part A*

@Neet_pg_bot

Q22: What is the enzyme defect if a child with anemia, thrombocytopenia, bony pain and hepatosplenomegaly shows a "crumpled tissue paper" appearance on light microscopy of bone marrow aspirate? (Q855548)

A. Beta- glucosidase

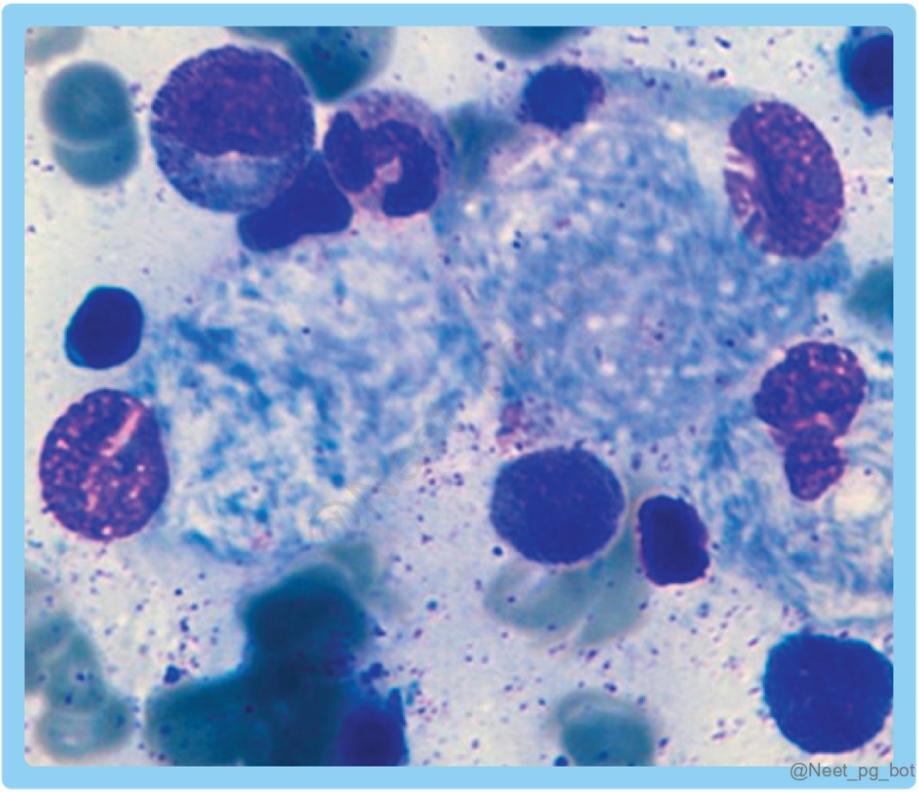
B. Sphingomyelinase

C. Hexosaminidase

D. Glucose6-phosphatase

Correct Answer: A. Beta- glucosidase

Explanation: Gaucher disease , caused by a defect in beta- glucosidase (glucocerebrosidase), leads to the accumulation of glucosylceramide (glucocerebroside) within macrophages. Lysosomal storage disorder of autosomal recessive inheritance. These macrophages, engorged with glucosylceramide, are known as Gaucher cells. The accumulation affects the bone marrow and spleen, leading to anaemia and thrombocytopenia. Gaucher cells have characteristic appearance of crumbled tissue paper look due to accumulation of glucosylceramide in the cytoplasm of macrophages, forming fibril like structures. {{caption_text}} Sphingomyelinase (Option B): Niemann-Pick disease results from a deficiency in sphingomyelinase.Its bone marrow findings involve foamy histiocytes with lipid-filled cytoplasm. Hexosaminidase (Option C): Tay-Sachs disease, characterized by hexosaminidase deficiency, doesn't typically cause anemia, thrombocytopenia, or bone pain.Its bone marrow findings may show an onion-skin appearance. Glucose 6-phosphatase (Option D): Von Gierke disease, due to glucose-phosphatase deficiency, may lead to hepatosplenomegaly and bone pain but not anemia or thrombocytopenia.



References:

Gaucher disease

↑ *Back to this question in Part A*

@Neet_pg_bot

Q23: Which of the following statements about β-oxidation of fatty acids is incorrect? (Q879278)

A. CPT-1 and CPT-2 are in the mitochondrial membranes

B. ATP is directly produced during β-oxidation

C. CoA is released in the cytosol during acyl-carnitine formation

D. Carnitine is released into the matrix

Correct Answer: B. ATP is directly produced during β-oxidation

Explanation: β-oxidation occurs in the mitochondrial matrix , where fatty acids are broken down to produce acetyl-CoA, NADH, and FADH■ . However, ATP is not directly produced during the β-oxidation steps

themselves. The ATP is generated later via oxidative phosphorylation , using the NADH and FADH■ produced during β-oxidation. (Option B) Carnitine Shuttle (facilitates transport): CPT-1 (outer membrane) converts acyl-CoA to acyl-carnitine, releasing CoA in the cytosol, and CPT-2 (inner membrane) regenerates acyl-CoA in the matrix, releasing free carnitine. (Option A, C, D ruled out)

References:

DM Vasudevan's Textbook of Biochemistry, 7th Edition, Page 151-152.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q24: What is the process of converting unconjugated substances into conjugated forms called? (Q860059)

A. Glycation

B. Glucuronidation

C. Glycosylation

D. Oxidation

Correct Answer: B. Glucuronidation

Explanation: Glucuronidation is a biochemical process in which a glucuronic acid molecule is attached to a substance, making it more water-soluble and easier to excrete. This process is essential for the detoxification and elimination of substances like bilirubin, drugs, and hormones in the liver. Synthetic Reaction Description Examples Glucuronidation Conjugation with glucuronic acid via UDP-glucuronosyl transferases (UGTs). Chloramphenicol, aspirin, paracetamol, morphine, bilirubin, steroid hormones, thyroxine. Acetylation Conjugation with acetyl CoA, mediated by N-acetyl transferases. Isoniazid, sulfonamides, hydralazine, dapsone, clonazepam. Methylation Methylation of amines and phenols by methyl transferases using methionine or cysteine as donors. Adrenaline, histamine, methyldopa, captopril, mercaptopurine. Sulfation Conjugation of phenols and steroids with sulfate by sulfotransferases (SULTs). Chloramphenicol, methyldopa, adrenal steroids. Glycine Conjugation Conjugation with glycine for drugs with carboxylic acid groups. Salicylates, nicotinic acid. Glutathione Conjugation Conjugation of reactive intermediates with glutathione via glutathione-S-transferases. Paracetamol (reactive intermediates), quinones, epoxides. Ribonucleoside Synthesis Conjugation for activation of purine and pyrimidine antimetabolites. Cancer chemotherapeutic agents. Glycation (Option A): Glycation is the non-enzymatic attachment of sugars to proteins or lipids, typically seen in conditions like diabetes. It does not involve the conjugation of substances in the way glucuronidation does. Glycosylation (Option C): Glycosylation involves the enzymatic attachment of a sugar molecule (often glucose or mannose) to proteins or lipids, typically in the endoplasmic reticulum and Golgi apparatus. It is distinct from glucuronidation and is not primarily involved in detoxification. Oxidation (Option D): Oxidation refers to the chemical reaction where a molecule loses electrons, often resulting in the addition of oxygen or the removal of hydrogen. While oxidation can modify molecules, it does not specifically refer to the conjugation process like glucuronidation.

References:

KDT- Essentials of Medical Pharmacology, 8th Ed, Pg: 31

↑ *Back to this question in Part A*

@Neet_pg_bot

Q25: A man is trapped in a tunnel for 5 consecutive days without access to food but survives. What is the primary source of energy for his brain during this period? (Q866318)

A. Gluconeogenesis

B. Glycogenolysis

C. Ketosis

D. Lipolysis

Correct Answer: C. Ketosis

Explanation: When a person is deprived of food for 3–5 days, the brain adapts by using ketone bodies—primarily β-hydroxybutyrate and acetoacetate—produced through Ketosis as its main energy source, thereby conserving muscle protein from being broken down for gluconeogenesis. KETOSIS Causes Diabetes Mellitus: Untreated diabetes mellitus → Insulin deficiency → Increases lipolysis and fatty acid oxidation → Raises acetyl CoA levels → Overproduction of ketone bodies (relies on fat metabolism for energy). Starvation: Reduced dietary glucose shifts oxaloacetate to gluconeogenesis. Increased lipolysis converts excess acetyl CoA to ketone bodies. High glucagon levels promote ketogenesis. Limited glucose boosts free fatty acid mobilization, with the liver converting them to ketones, regulated by carnitine palmitoyltransferase-I (CPT-I). Hyperemesis in early pregnancy can also cause ketosis. Ketone Bodies Produced in the liver Utilized by extrahepatic tissues due to the absence of Thiophorase enzyme in the liver Tissues that use Ketone Bodies Almost all tissues except: Liver Red blood cells (RBCs) Tissues that prefer ketone bodies: Heart muscle Renal cortex Placenta Tissues that use ketone bodies only in the absence of glucose: Skeletal muscle Brain Additional Tissues Using Ketone Bodies Intestinal mucosal cells Adipocytes Enzyme Involved Thiophorase : Converts acetoacetate to acetoacetyl CoA Fatty Acids as Primary Energy Source During Starvation Skeletal muscles Heart Liver Energy Production Pathway Acetoacetyl CoA enters the beta-oxidation pathway to produce energy Gluconeogenesis (Option A) is active early in fasting, but not the main source for brain energy after 5 days. Glycogenolysis (Option B): Liver glycogen is depleted within ~24 hours of fasting. Lipolysis (Option D): Breaks down fat into free fatty acids and glycerol, but fatty acids can't cross the blood-brain barrier — only ketones can.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q26: A diabetic patient presents with high VLDL due to the combination of which of the following? (Q853004)

A. High HSL, low LPL

B. High LPL, low HSL

C. High HSL, High LPL

D. Low LPL, Low HSL

Correct Answer: A. High HSL, low LPL

Explanation: In uncontrolled diabetes, insulin deficiency increases hormone-sensitive lipase (HSL) activity → excess free fatty acid release from adipose tissue. At the same time, lipoprotein lipase (LPL) activity decreases because LPL is insulin-dependent. This combination leads to increased hepatic VLDL production and reduced clearance , causing high VLDL levels. HSL (Hormone-Sensitive Lipase): This enzyme is involved in the breakdown of stored triglycerides into free fatty acids and glycerol. In a diabetic patient, increased levels of HSL can contribute to higher levels of free fatty acids, which can promote VLDL (Very Low-Density Lipoprotein) synthesis in the liver. LPL (Lipoprotein Lipase): LPL is responsible for the hydrolysis of triglycerides in VLDL and chylomicrons, converting them into free fatty acids and glycerol. A low level of LPL activity would impair the clearance of VLDL from the bloodstream, leading to high VLDL levels. In diabetes, high HSL activity and low LPL activity both contribute to increased levels of VLDL. High HSL promotes the production of triglycerides in the liver, while low LPL reduces the breakdown of these triglycerides in the bloodstream, resulting in elevated VLDL levels. High LPL, low HSL (Option B): LPL would lower VLDL, not raise it. High HSL, high LPL (Option C): increased LPL would compensate by clearing VLDL. Low LPL, low HSL (Option D): Less substrate (FFA) for VLDL production, so VLDL won't rise significantly.

References:

Vasudevan's Textbook of Biochemistry for Medical Students, 7th edition, Pages 323-330

↑ *Back to this question in Part A*

@Neet_pg_bot

Q27: Cholera is known to cause diarrhea through its interaction with specific receptors in the human body. Which of the following is known to make it easier for cholera toxin to enter the body through the intestinal mucosa? (Q857854)

A. GLUT 1

B. GM2 ganglioside

C. Cephalin

D. GM1 ganglioside

Correct Answer: D. GM1 ganglioside

Explanation: Correct Option: D) GM1 ganglioside: Cholera toxin is produced by the bacterium Vibrio cholerae and is responsible for causing the characteristic symptoms of cholera, including severe diarrhea. Cholera toxin enters the body through the intestinal mucosa and interacts with specific receptors on the surface of intestinal cells. The receptor that facilitates the entry of cholera toxin into the body is the GM1 ganglioside receptor. GM1 ganglioside is a glycosphingolipid, a type of molecule found in the lipid bilayer of cell membranes. Cholera toxin binds to GM1 ganglioside on the surface of intestinal cells, which triggers the toxin to be internalized into the cell through endocytosis. Once inside the cell, the toxin can exert its effects, leading to the disruption of ion transport and fluid balance, ultimately causing severe diarrhea. GLUT1 (Glucose Transporter 1) (Option A) is a protein responsible for transporting glucose across cell membranes. GM2 ganglioside (Option B) is a type of glycosphingolipid, it is not the receptor specifically associated with cholera toxin binding and entry into intestinal cells. Cephalin (Option C) is a type of phospholipid and is not known to be directly involved in the interaction between cholera toxin and intestinal cells.

References:

https://pmc.ncbi.nlm.nih.gov/articles/PMC3089049/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q28: In which of the following locations can cardiolipin be found? (Q857900)

A. Outer mitochondrial membrane

B. Inner mitochondrial membrane

C. Cytosol

D. Mitochondrial matrix

Correct Answer: B. Inner mitochondrial membrane

Explanation: Correct Option: B) Inner mitochondrial membrane Cardiolipin is a distinctive phospholipid abundant in the inner mitochondrial membrane. It plays a role in the structural integrity of the mitochondria and is involved in several mitochondrial functions, including electron transport chain activity. Cardiolipin: Location : Inner mitochondrial membrane Function: Optimal function of numerous enzymes involved in mitochondrial energy metabolism. Stabilizes the respiratory chain complexes and supercomplexes Synthesis: Unique dimeric structure consisting of four fatty acid chains Synthesized in the mitochondria from phosphatidylglycerol and cytidine diphosphate-diacylglycerol (CDP-DAG) Outer mitochondrial membrane (Option A) primarily consists of phospholipids like phosphatidylcholine and phosphatidylethanolamine. Cytosol (Option C): Cardiolipin is not found in the cytosol. It is mainly confined to the mitochondrial membrane. Mitochondrial matrix (Option D) Cardiolipin is primarily located in the inner mitochondrial membrane and is not a major component of the

mitochondrial matrix.

References:

Harper 32e 240, 241

Lippincott Illustrated reviews 7e 592-599

↑ *Back to this question in Part A*

@Neet_pg_bot

Q29: Fat is stored in which form in tissue? (Q856950)

A. Fatty acid

B. Cholesterol

C. TGs

D. Cholesterol esters

Correct Answer: C. TGs

Explanation: Fat is primarily stored in tissues, especially adipose tissue, in the form of triglycerides (TGs). Energy-dense storage: TGs are highly energy-rich molecules, storing more energy per gram than carbohydrates or proteins. Structure : Composed of glycerol backbone esterified with three fatty acids. Hydrophobic nature : Stored in a compact, anhydrous form, ideal for long-term energy storage. Fatty acids (Option A): Not stored as free fatty acids due to toxicity and membrane disruption.Instead, fatty acids are esterified into TGs for storage. Cholesterol (Option B): Stored only in small amounts for membrane synthesis and hormone production.Not a primary energy storage molecule. Cholesterol esters (Option D): Mostly stored in the liver and lipoproteins for lipid transport, not as a primary energy reserve in adipose tissue.

References:

Fat storage

↑ *Back to this question in Part A*

@Neet_pg_bot

Q30: Cholesterol is synthesized from? (Q857480)

A. Acetyl coA

B. Phenylalanine

C. Malonyl coA

D. Oxaloacetate

Correct Answer: A. Acetyl coA

Explanation: Cholesterol is synthesized from acetyl-CoA in the liver through a multi-step process called the mevalonate pathway. Acetyl-CoA → HMG-CoA (via HMG-CoA synthase) HMG-CoA → Mevalonate (via HMG-CoA reductase, the rate-limiting step) Mevalonate is further converted into isoprenoid units, which eventually lead to cholesterol formation. Phenylalanine(Option B): Amino acid, not involved in cholesterol synthesis. Malonyl CoA (Option C): Involved in fatty acid synthesis. Oxaloacetate (Option D): Part of the TCA cycle, not cholesterol synthesis.

References:

Cholestrol synthesis

↑ *Back to this question in Part A*

@Neet_pg_bot

Q31: Which of the following substances is not characterized by amphipathicity? (Q856600)

A. Sphingolipids

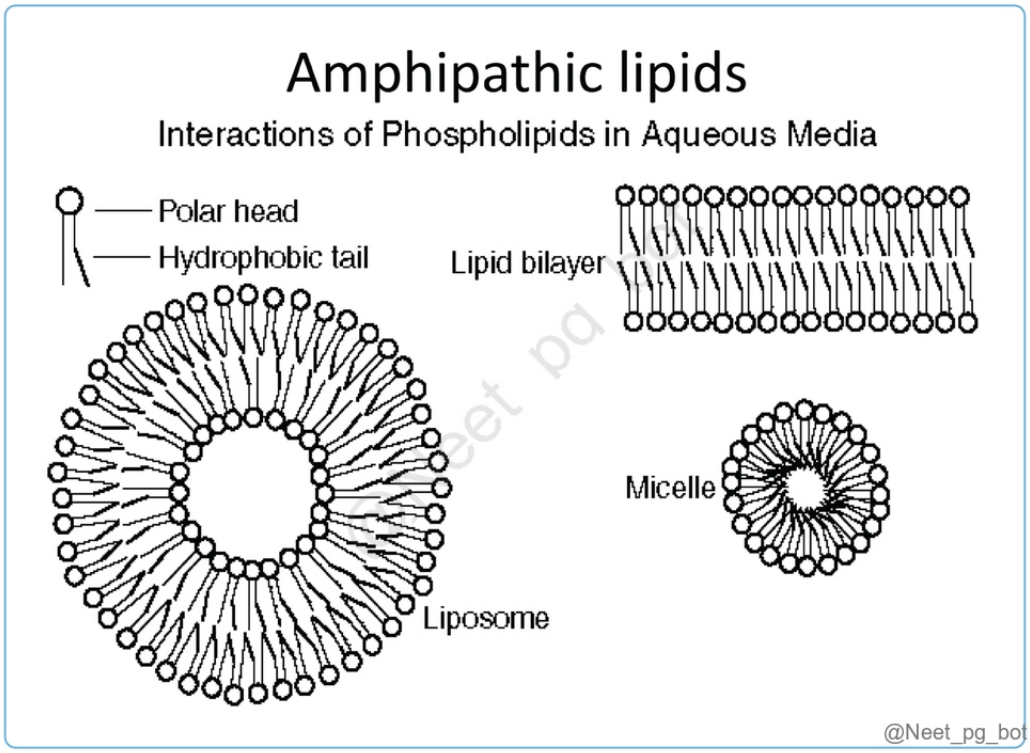
B. Phosphoglycerol

C. Triglycerides

D. Glycolipids

Correct Answer: C. Triglycerides

Explanation: Triglycerides are not Amphipathicitic in nature. Amphipathic Lipids: Characteristics: Both hydrophobic and hydrophilic; contain a polar head group and a nonpolar tail. Functions and Structures: Membranes: Bilayer structure with hydrophobic tails inward and hydrophilic heads outward Micelles: Spherical structures with hydrophobic tails in the center and hydrophilic heads on the surface; aid in fat digestion. Liposomes: Spherical vesicles with lipid bilayers enclosing an aqueous compartment Emulsions: Larger particles of nonpolar lipids dispersed in water, stabilized by amphipathic lipids; prevent coalescence. Examples: Phospholipids Sphingolipids Cholesterol



References:

Harper's illustrated Biochemistry 32e pg 205, 206, 209, 215, 264, 248, 259

↑ *Back to this question in Part A*

@Neet_pg_bot

Q32: What could be the possible diagnosis for a patient who exhibited tendon xanthomas, with his lipid profile indicating serum cholesterol levels of 283 mg/dL and serum LDL levels of 202 mg/dL, and subsequently initiated on lovastatin treatment? (Q856010)

A. Familial hypercholesterolemia

B. Tangier's disease

C. Lipoprotein lipase deficiency

D. Familial hyperchylomicronemia

Correct Answer: A. Familial hypercholesterolemia

Explanation: The Clinical scenario of Tendon xanthomas, raised serum cholesterol and ldl levels indicate Familial Hypercholestrolemia. Feature Type IIa (Familial Hypercholesterolemia) Defect Defective LDL receptors or Apo B-100 ligand region mutation Lipoprotein Fraction Elevated LDL Main Lipid Abnormality Hypercholesterolemia Laboratory findings Raised serum LDL and Cholesterol Features . Atherosclerosis Coronary artery disease (early onset) Tendon xanthomas Management Statins PUFA Lifestyle modifications (diet, exercise) Incorrect Options: Tangier disease (Option B) is associated with very low HDL levels and characteristic features such as orange tonsils and peripheral neuropathy. Lipoprotein lipase deficiency(Option C) Familial Hyperchylomicronemia (Option D) Causes severe hypertriglyceridemia eruptive xanthomas, hepatomegaly, and a creamy layer over clear plasma, not typically elevated LDL.

References:

Lippincot biochemistry 7th Ed-pg 678

Harper biochemistry 32nd edition pg: 268

Vasudevan biochemistry 7th edition pg: 343

↑ *Back to this question in Part A*

@Neet_pg_bot

Q33: What diagnostic tool is used to determine the cause of chest pain at rest for 6 hours and an electrocardiogram indicating myocardial ischemia in a patient? (Q859574)

A. LDH 2

B. LDH 3

C. LDH 1

D. LDH 4

Correct Answer: C. LDH 1

Explanation: LDH-1 is the isoenzyme predominantly found in the heart muscle, making it the most relevant for diagnosing conditions related to the heart, particularly myocardial infarction (heart attack). During a myocardial infarction (MI), heart muscle cells are damaged or destroyed, causing the release of LDH-1 into the bloodstream. LDH-1 increases significantly in the case of heart injury, and its levels can be 5-10 times higher than in normal conditions during a heart attack. LDH in Myocardial Infarction: Total LDH activity is increased during a heart attack (myocardial infarction). H4 isoenzyme increases 5-10 times more than other forms during myocardial infarction. The size of the myocardial infarct correlates with the peak value and area under the LDH curve. Increased total LDH levels are observed in: Hemolytic anemias Hepatocellular damage Muscular dystrophy Carcinomas Leukemias Any condition causing necrosis of body cells. LDH Isoenzymes: LDH enzyme is a tetramer with 4 subunits: either H (heart) or M (muscle) polypeptide chains. There are 5 possible isoenzymes

based on combinations of H and M chains: H4 H3M1 H2M2 M3H1 M4 Isoenzymes are separated using cellulose acetate electrophoresis at pH 8.6. The reaction is identified with NAD+ and nitroblue tetrazolium. Isoenzyme Locations and Patterns: M4 form is predominantly found in skeletal muscles. H4 form is predominantly found in the heart. In normal conditions, LDH-2 (H3M1) concentration is higher than LDH-1 (H4) , but in myocardial infarction, the pattern reverses (known as the "flipped pattern"). Isoenzyme Activity at 60°C for 30 minutes Electrophoretic Mobility at pH 8.6 Tissue of Origin Percentage in Human Serum (Mean) LDH-1 Not destroyed Fastest Heart muscle 30% LDH-2 Not destroyed Faster RBC (Option A) 35% LDH-3 Partially destroyed Fast Brain (Option B) 20% LDH-4 Destroyed Slow Liver (Option D) 10% LDH-5 Destroyed Slowest Skeletal muscle 5%

References:

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg. 304

↑ *Back to this question in Part A*

@Neet_pg_bot

Q34: Which substance is involved in the conjugation process in the liver? (Q860320)

A. Hyaluronic Acid

B. Glucuronic Acid

C. Gluconic Acid

D. Glycolic Acid

Correct Answer: B. Glucuronic Acid

Explanation: Glucuronidation is a biochemical process in which a glucuronic acid molecule is attached to a substance, making it more water-soluble and easier to excrete. This process is essential for the detoxification and elimination of substances like bilirubin, drugs, and hormones in the liver. Synthetic Reaction Description Examples Glucuronidation Conjugation with glucuronic acid via UDP-glucuronosyl transferases (UGTs). Chloramphenicol, aspirin, paracetamol, morphine, bilirubin, steroid hormones, thyroxine. Acetylation Conjugation with acetyl CoA, mediated by N-acetyl transferases. Isoniazid, sulfonamides, hydralazine, dapsone, clonazepam. Methylation Methylation of amines and phenols by methyl transferases using methionine or cysteine as donors. Adrenaline, histamine, methyldopa, captopril, mercaptopurine. Sulfation Conjugation of phenols and steroids with sulfate by sulfotransferases (SULTs). Chloramphenicol, methyldopa, adrenal steroids. Glycine Conjugation Conjugation with glycine for drugs with carboxylic acid groups. Salicylates, nicotinic acid. Glutathione Conjugation Conjugation of reactive intermediates with glutathione via glutathione-S-transferases. Paracetamol (reactive intermediates), quinones, epoxides. Ribonucleoside Synthesis Conjugation for activation of purine and pyrimidine antimetabolites. Cancer chemotherapeutic agents. Hyaluronic Acid (Option A): Hyaluronic acid is a glycosaminoglycan involved in extracellular matrix formation and lubrication of joints. It does not play a role in hepatic conjugation processes. Gluconic Acid (Option C): Gluconic acid is a product of glucose oxidation and is not involved in the conjugation process in the liver. Glycolic Acid (Option D): Glycolic acid is an alpha-hydroxy

acid commonly associated with skincare and is not involved in hepatic conjugation.

References:

KDT- Essentials of Medical Pharmacology, 8th Ed, Pg: 31

↑ *Back to this question in Part A*

@Neet_pg_bot

Q35: A man presents with high VLDL and chylomicrons, with eruptive xanthomas. What is the most likely diagnosis? (Q866645)

A. Familial hyperlipidemia

B. Familial dysbetalipoproteinemia

C. Familial hypertriglyceridemia

D. Familial hypercholesterolemia

Correct Answer: C. Familial hypertriglyceridemia

Explanation: Elevated VLDL and chylomicrons with eruptive xanthomas indicate high triglycerides, characteristic of Familial Hypertriglyceridemia (Type IV Hyperlipoproteinemia) , presenting with increased VLDL, raised triglycerides, and small yellowish skin papules. Inheritance: Autosomal dominant Lipid profile: Elevated VLDL and triglycerides Pathophysiology: Overproduction or decreased clearance of VLDL Clinical features: Eruptive xanthomas (small yellow papules on skin) Pancreatitis risk due to very high triglycerides No significant increase in LDL cholesterol Management: Lifestyle changes, fibrates, and omega-3 fatty acids Incorrect Options: Familial hyperlipidemia (Option A) is a broad term; not specific. Familial dysbetalipoproteinemia (Type III) (Option B) shows elevated IDL and chylomicron remnants, often with tuberoeruptive xanthomas, not mainly VLDL. Familial hypercholesterolemia (Option D) mainly shows elevated LDL cholesterol, leading to tendon xanthomas, not high VLDL/chylomicrons.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q36: The active site of an enzyme is most complementary to? (Q857356)

A. Allosteric activator

B. Substrate

C. Product

D. Transition state

Correct Answer: B. Substrate

Explanation: Properties of Enzyme Nature of Enzymes Enzymes are proteins, heat-sensitive, water-soluble Efficiency Enzymes can accelerate reactions by factors 10^6 to 10^9 or more. Specificity Enzymes act on specific substrates and can distinguish stereoisomers. Cofactors/Coenzymes Some enzymes need non-protein molecules (cofactors or coenzymes) to be active. Substrate Binding Enzymes bind substrates at multiple points, creating chiral products from non-chiral substrates. Transition State Enzymes stabilize the transition state, lowering activation energy. Reversibility Enzymes can catalyze both forward and reverse reactions. Regulation Activity is regulated by pH, temperature, inhibitors, and activators. Active site The region where the substrate binds and undergoes a chemical reaction It is most complimentary to enzyme-substrate

References:

Harper 32nd edition, page 59,60

https://pubmed.ncbi.nlm.nih.gov/35534116/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q37: In which organelle is DNA present? (Q820383)

A. Mitochondria

B. Lysosome

C. Endoplasmic reticulum

D. Cytoplasm

Correct Answer: A. Mitochondria

Explanation: DNA is present in the mitochondria, which are double-membraned organelles found in the cells of most eukaryotic organisms. Mitochondrial DNA (mtDNA) is a small circular molecule separate from the nuclear DNA. It carries genetic information essential for the functioning of mitochondria and plays a crucial role in energy production through oxidative phosphorylation. Lysosome (Option B): Lysosomes are membrane-bound organelles containing enzymes responsible for breaking down various cellular waste materials and substances. They do not contain DNA; their primary function is related to digestion and waste disposal within the cell. Endoplasmic Reticulum (Option C): The endoplasmic reticulum (ER) is an organelle involved in the synthesis, folding, modification, and transport of proteins and lipids. It does not contain DNA. The ER is composed of membranous

structures and is mainly responsible for the manufacturing and transportation of molecules within the cell. Cytoplasm (Option D): The cytoplasm is the gel-like substance found inside the cell, which includes various organelles and is the site of many cellular processes. While the cytoplasm contains various molecules and structures necessary for cell function, DNA itself is not typically present in the cytoplasm. DNA is primarily found within the nucleus of eukaryotic cells, where it houses the genetic information required for cell function and development.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q38: In which organelle is DNA present? (Q857869)

A. Mitochondria

B. Lysosome

C. Endoplasmic reticulum

D. Cytoplasm

Correct Answer: A. Mitochondria

Explanation: DNA is present in the mitochondria, which are double-membraned organelles found in the cells of most eukaryotic organisms. Mitochondrial DNA (mtDNA) is a small circular molecule separate from the nuclear DNA. It carries genetic information essential for the functioning of mitochondria and plays a crucial role in energy production through oxidative phosphorylation. Lysosome (Option B) are membrane-bound organelles containing enzymes responsible for breaking down various cellular waste materials and substances. Their primary function is related to digestion and waste disposal within the cell. Endoplasmic Reticulum (Option C) is an organelle involved in the synthesis, folding, modification, and transport of proteins and lipids. The ER is composed of membranous structures and is mainly responsible for the manufacturing and transportation of molecules within the cell. Cytoplasm (Option D) is the gel-like substance found inside the cell, which includes various organelles and is the site of many cellular processes. The cytoplasm contains various molecules and structures necessary for cell function, DNA is not present in the cytoplasm. DNA is primarily found within the nucleus of eukaryotic cells, where it houses the genetic information required for cell function and development.

References:

Vasudevan Edition 7 page no:10-23.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q39: What is the cause of elevated H+ ions in the intermembrane space of mitochondria? (Q855538)

A. Decreased oxidative phosphorylation

B. Increased ATP production

C. Reduced chemiosmosis

D. Water retention in inner mitochondrial membrane

Correct Answer: A. Decreased oxidative phosphorylation

Explanation: Decreased oxidative phosphorylation Elevated H■ ions in the intermembrane space of mitochondria occur due to active proton pumping during oxidative phosphorylation . In this process: Electrons from NADH and FADH■ pass through the electron transport chain (Complexes I, III, and IV) . The released energy drives H■ ions from the mitochondrial matrix into the intermembrane space , creating a proton gradient (electrochemical potential). This proton motive force is later used by ATP synthase (Complex V) to synthesize ATP as H■ flows back into the matrix via chemiosmosis . Thus, oxidative phosphorylation is the primary cause of H■ accumulation in the intermembrane space. Increased ATP production (Option B): This is a result of proton gradient utilization, not its cause. Reduced chemiosmosis (Option C): Would decrease H■ re-entry, stalling ATP synthesis but not explaining the normal physiological gradient formation. Water retention in inner mitochondrial membrane (Option D): Unrelated to proton concentration or electron transport.

References:

pmc.ncbi.nlm.nih.gov/articles/PMC2712118/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q40: Zellweger syndrome results from abnormality in formation of: (Q855055)

A. Peroxisomes

B. Lysosomes

C. Mitochondria

D. Nucleus

Correct Answer: A. Peroxisomes

Explanation: Zellweger syndrome/Cerebrohepatorenal syndrome results from an abnormality in the formation of peroxisomes , which are cellular organelles responsible for various metabolic functions, including: Defective

Peroxisomal Biogenesis: Mutations in PEX genes disrupt the assembly and function of peroxisomes. Key Consequence: Impaired breakdown of very long-chain fatty acids (VLCFAs), branched-chain fatty acids and other metabolic substrates leads to severe metabolic dysfunction. It is part of the peroxisome biogenesis disorder (PBD) spectrum. Accumulates C26-C38 polyenoic acids in brain tissue and exhibit generalised loss of peroxisomal functions. Management: Docosahexaenoic acid, Lorenzo oil and Cholic acid.

References:

www.ncbi.nlm.nih.gov/books/NBK560676/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q41: Refsum disease is caused due to absence of which of the following enzymes? (Q855061)

A. Absence of alpha oxidation in peroxisomes.

B. Absence of beta oxidation in peroxisomes.

C. Absence of alpha oxidation in lysosomes.

D. Absence of beta oxidation in lysosomes.

Correct Answer: A. Absence of alpha oxidation in peroxisomes.

Explanation: Refsum disease is a rare inherited disorder caused by mutation in PHYH gene , leading to deficiency of the enzyme phytanoyl-CoA hydroxylase , which is involved in the alpha-oxidation of phytanic acid (a fatty acid). Alpha oxidation occurs in peroxisomes and aids in fatty acid breakdown. Triad : Cerebellar Ataxia Retinitis pigmentosa Chronic polyneuropathy

References:

pubmed.ncbi.nlm.nih.gov/32809453/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q42: The subcellular organelle which is associated with the killing of viruses and bacteria is? (Q857485)

A. Peroxisome

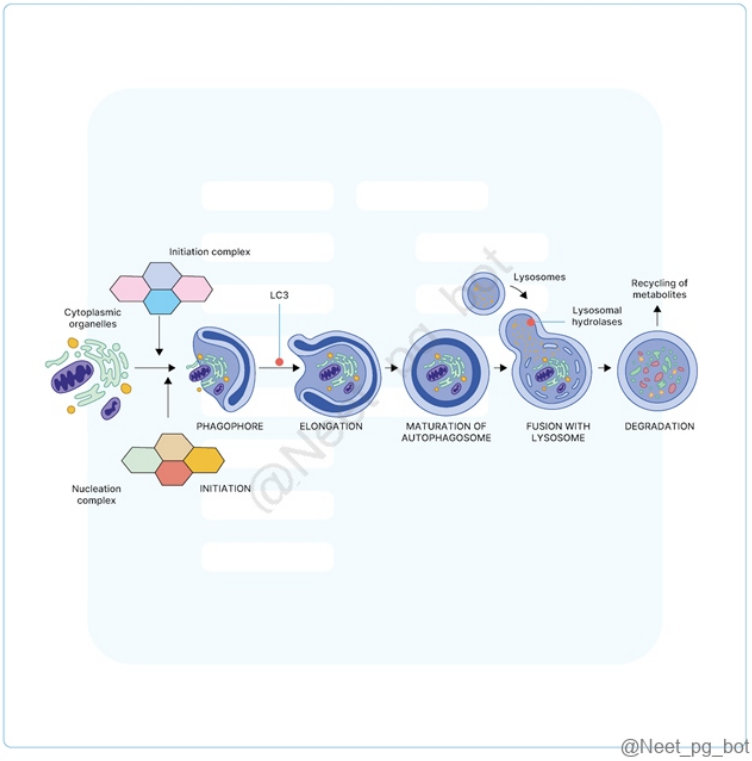
B. Mitochondria

C. Lysosome

D. Golgi apparatus

Correct Answer: C. Lysosome

Explanation: Lysosomes are membrane-bound organelles containing hydrolytic enzymes (like proteases, lipases, and nucleases) that digest and destroy pathogens, including viruses and bacteria, through a process called phagocytosis. After engulfment by phagocytes (macrophages), the pathogen is enclosed in a phagosome, which fuses with a lysosome to form a phagolysosome, where enzymatic degradation occurs. {{caption_text}} Peroxisome (Option A): Involved in fatty acid oxidation and reactive oxygen species detoxification. Mitochondria (Option B): Powerhouse of the cell, involved in ATP production. Golgi apparatus (Option D): Involved in protein modification, packaging, and transport.



References:

Lysosome

↑ *Back to this question in Part A*

@Neet_pg_bot

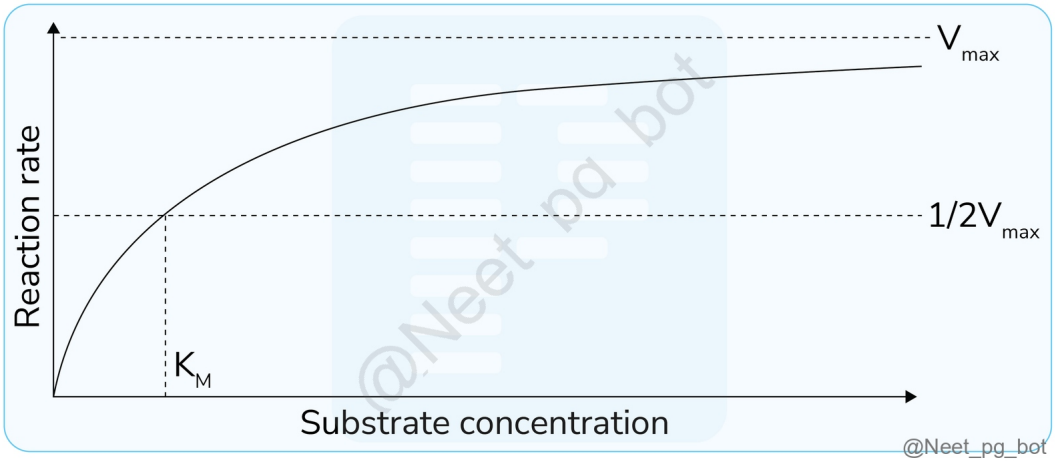
Q43: The enzyme kinetics depicted in the graph is suggestive of (Q855904)

A. Allosteric activation

B. Cooperative binding

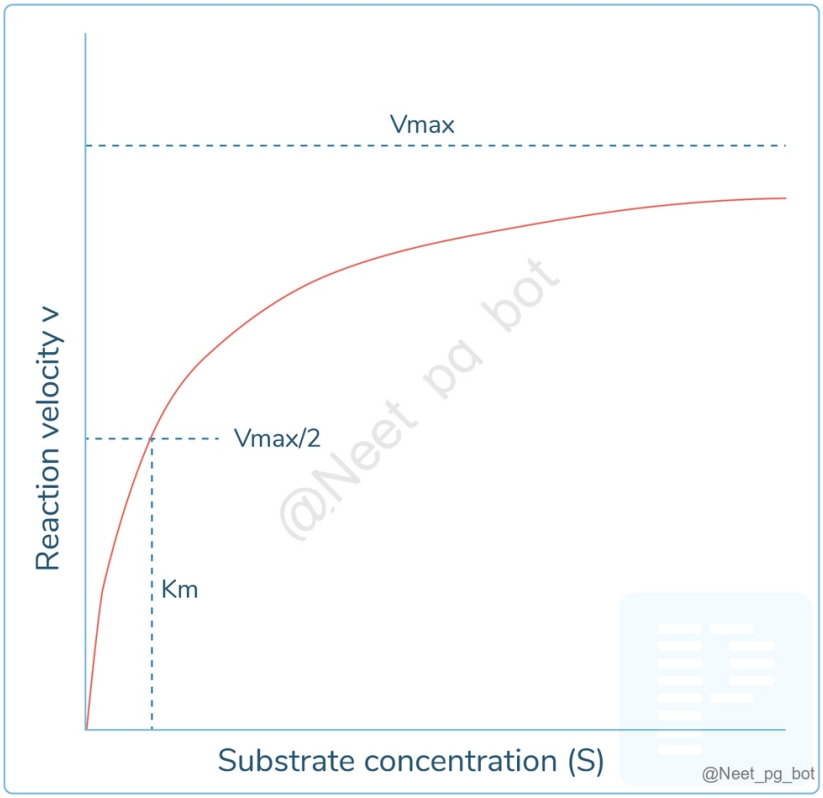
C. Michaelis Menten kinetics

D. Sigmoid curve



Correct Answer: C. Michaelis Menten kinetics

Explanation: The graph depicted is of Michaelis Menten's kinetics. Michaelis Menten's kinetics is intended to explain the broad mechanism and rate of enzyme-catalyzed reactions. The Michaelis-Menten equation is widely used to analyze enzyme kinetics and determine important parameters such as Vmax and Km. Michalis Menten equation: V = Vmax x S / (Km + S) Where, V = Velocity of reaction Vmax = Maximum velocity of the reaction S = Substrate concentration Km = Michaelis constant = Substrate concentration at which the velocity of the reaction is half the maximal velocity (Vmax/2) Allosteric activation (Option A) They are regulatory enzymes /multi-subunit enzymes. The activator shifts the curve to the left while the inhibitor shifts the cure to right Cooperative binding (Option B) One substrate binding to an enzyme increases the affinity for other substrates for enzymes. Sigmoid curve (Option D) In the presence of allosteric modifiers the graph of enzyme–catalyzed reaction obtained is a sigmoid / S-shaped graph.



References:

Harper, 32nd Edition, Page 76-77

↑ *Back to this question in Part A*

@Neet_pg_bot

Q44: Which mechanism is responsible for the inhibition of the acetylcholinesterase enzyme by organophosphorus compounds? (Q856597)

A. Non-competitive inhibition

B. Reversible competitive inhibition

C. Irreversibleinhibition

D. Suicidal inhibition

Correct Answer: C. Irreversibleinhibition

Explanation: Organophosphorus compounds, such as certain pesticides and nerve agents, can irreversibly inhibit the activity of acetylcholinesterase (AChE The inhibition caused by organophosphorus compounds is considered

irreversible because these compounds form strong covalent bonds with the active site of the enzyme . They covalently bind to the serine residue within the active site of AChE, forming a stable complex that cannot be easily reversed or dissociated. Reversible competitive inhibition (Option A) occurs when an inhibitor molecule competes with the substrate for binding to the active site of the enzyme. Non-competitive inhibition (Option B) occurs when an inhibitor binds to a site on the enzyme other than the active site, altering its conformation and reducing its activity. Suicidal inhibition (Option D) is another term used to describe the irreversible nature of inhibition caused by organophosphorus compounds. It refers to the fact that the inhibitor "kills" the enzyme by permanently modifying it, leading to its inactivation.

References:

Textbook of Biochemistry by DM Vasudevan, 7th edition, pg 59-62

↑ *Back to this question in Part A*

@Neet_pg_bot

Q45: A TPN bag consists of 100g glucose, 30g amino acids, and 40g lipids. What is the amount of calories delivered? (Q856864)

A. 840 KCal

B. 880 KCal

C. 640 KCal

D. 680 KCal

Correct Answer: B. 880 KCal

Explanation: To calculate the number of calories delivered in the TPN (Total Parenteral Nutrition) bag, we need to know the caloric value of each macronutrient. The caloric values per gram for each macronutrient are as follows: Glucose: 4 calories per gram Amino acids: 4 calories per gram Lipids (fats): 9 calories per gram Now let's calculate the calories for each component of the TPN bag: Glucose: 100g of glucose * 4 calories/gram = 400 calories. Amino acids: 30g of amino acids * 4 calories/gram = 120 calories. Lipids: 40g of lipids * 9 calories/gram = 360 calories. Now, let's add up the calories from each component: 400 calories (glucose) + 120 calories (amino acids) + 360 calories (lipids) = 880 calories

References:

Calories

↑ *Back to this question in Part A*

@Neet_pg_bot

Q46: In a patient who is tall, obese, known to have hypertension, and has recently been diagnosed with metabolic syndrome during a routine examination, which of the following findings are characteristic findings? (Q793475)

A. Increase in uric acid and insulin

B. Raised blood pressure and normal insulin

C. Increase in triglycerides and total cholesterol

D. Raised blood sugar and low insulin

Correct Answer: A. Increase in uric acid and insulin

Explanation: Metabolic syndrome is defined by central obesity, hypertension, insulin resistance, and dyslipidemia. Insulin resistance causes the pancreas to secrete more insulin → hyperinsulinemia. Thus, insulin levels are high, not normal or low. Hyperinsulinemia contributes to increased sympathetic activity and worsens hypertension. High insulin decreases renal uric acid excretion via proximal tubule reabsorption, raising uric acid levels. Obesity and fructose-rich diets increase purine breakdown, producing more uric acid. Therefore, metabolic syndrome commonly shows high insulin + high uric acid. This combination is a hallmark metabolic abnormality in these patients. Incorrect Options: Option B) Raised blood pressure and normal insulin : BP is high, but insulin is not normal → patients show hyperinsulinemia. Option C) Increase in triglycerides and total cholesterol : ↑Triglycerides is correct, but total cholesterol is not a defining feature, making this option incomplete. Option D) Raised blood sugar and low insulin : Low insulin occurs in late-stage diabetes, not in metabolic syndrome. Early metabolic syndrome shows high insulin, not low insulin.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

• [04_Amino Acids and Proteins Chemistry] 013 Protein Chemistry Hemoglobin Myoglobin Epp Chromatography

« Back to Folder Directory

[04_Amino Acids and Proteins Chemistry] 013 Protein Chemistry Hemoglobin Myoglobin Epp Chromatography — Quick Jump

Q1 (p. 738)	*Q2 (p. 738)*	*Q3 (p. 740)*	*Q4 (p. 741)*	*Q5 (p. 742)*	*Q6 (p. 743)*
Q7 (p. 744)	*Q8 (p. 746)*	*Q9 (p. 747)*	*Q10 (p. 748)*	*Q11 (p. 750)*	*Q12 (p. 751)*
Q13 (p. 752)	*Q14 (p. 753)*	*Q15 (p. 754)*	*Q16 (p. 755)*	*Q17 (p. 757)*	*Q18 (p. 757)*
Q19 (p. 758)	*Q20 (p. 759)*	*Q21 (p. 760)*	*Q22 (p. 764)*	*Q23 (p. 764)*	*Q24 (p. 765)*
Q25 (p. 766)	*Q26 (p. 767)*				

Q1: Which of the following channels is primarily responsible for active magnesium reabsorption in the distal convoluted tubule? (Q876252)

A. TRPM6

B. ENaC

C. NKCC2

D. Aquaporin-2

Correct Answer: A. TRPM6

Explanation: Correct Option A - TRPM6: TRPM6 (Transient Receptor Potential Melastatin 6) is a magnesium-specific channel in the distal convoluted tubule, mediating active Mg^{2}■ reabsorption. NKCC2 is for Na■, K■, 2Cl■ in TAL; ENaC is for Na■ in DCT/collecting duct; Aquaporin-2 is water channel in collecting duct.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: Which of the following statements about selenocysteine and Pyrrolysine is correct? (Q844902)

A. Pyrrolysine is incorporated into proteins at UGA codons with the help of a specific tRNA.

B. The incorporation of selenocysteine requires a special mRNA structure known as the selenocysteine insertion sequence (SECIS).

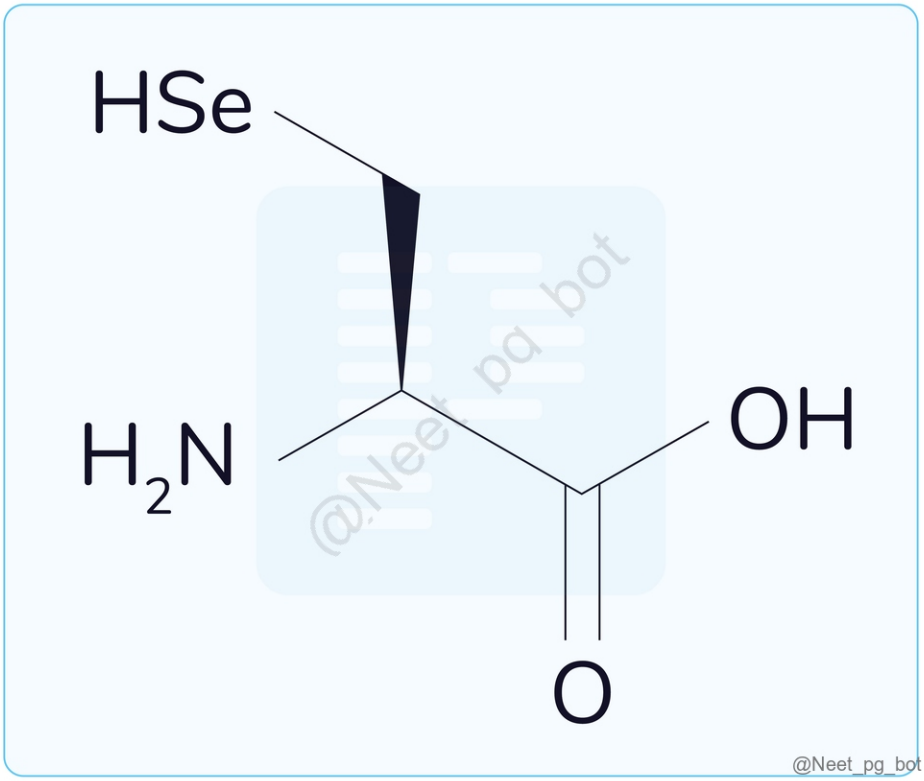
C. Selenocysteine is synthesized from cysteine during protein translation.

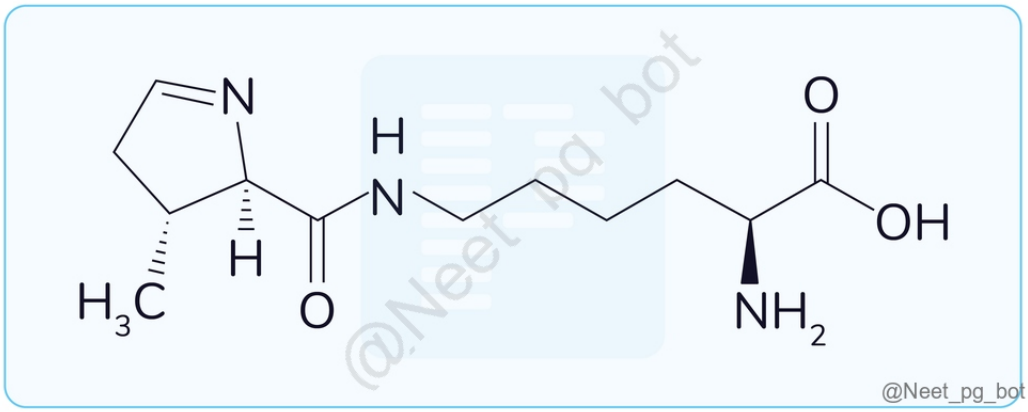
D. Pyrrolysine is only found in eukaryotic organisms and not in prokaryotes.

Correct Answer: B. The incorporation of selenocysteine requires a special mRNA structure known as the selenocysteine insertion sequence (SECIS).

■ *Listen to Audio 1*

Explanation: Selenocysteine : The 21st amino acid , derived from serine. Found in ~25 human selenoproteins (e.g., peroxidases, reductases). Incorporated via tRNASec and the UGA codon with help of the SECIS element . Functions in redox reactions and T■ → T■ conversion by deiodinases. {{caption_text}} Pyrrolysine: The 22nd amino acid , derived from lysine. Found in some methanogenic archaea and bacteria . Incorporated via tRNAPyl using the UAG codon (normally a stop codon). Functions in methane-producing enzymes that metabolize methylamines. {{caption_text}}





References:

Harper's Illustrated Biochemistry, 32nd edition, Page 18

↑ *Back to this question in Part A*

@Neet_pg_bot

Q3: Which of the following statements correctly describes the metabolic fate of specific amino acids? (Q844900)

A. All amino acids can be classified as either glucogenic or ketogenic without exceptions.

B. Aspartate is a purely ketogenic amino acid.

C. Phenylalanine can be converted to both glucose and ketone bodies.

D. Lysine is exclusively glucogenic.

Correct Answer: C. Phenylalanine can be converted to both glucose and ketone bodies.

■ *Listen to Audio 1*

Explanation: Phenylalanine is classified as both glucogenic and ketogenic because its catabolism produces: Fumarate → Enters the TCA cycle , contributing to gluconeogenesis (glucose production). Acetoacetate → A precursor for ketone bodies . This dual fate allows phenylalanine to contribute to both energy sources. Metabolism of Amino Acids: Glucogenic vs. Ketogenic Classification of Amino Acids Amino acids are classified based on their metabolic fate: Type Definition Examples: Purely Glucogenic Amino acids that are converted into pyruvate or TCA cycle intermediates (e.g., oxaloacetate, α-ketoglutarate) and contribute to glucose synthesis via gluconeogenesis . Aspartate, Alanine, Glycine, Serine, Cysteine Purely Ketogenic Amino acids that yield only acetyl-CoA or acetoacetate , leading to the formation of ketone bodies . Leucine, Lysine Both Glucogenic & Ketogenic Amino acids that can be converted into both glucose and ketone bodies , contributing to energy metabolism through multiple pathways. Phenylalanine, Tyrosine, Tryptophan, Isoleucine, Threonine All amino acids can be classified as either glucogenic or ketogenic without exceptions. (Option A):Incorrect because some

amino acids (e.g., ornithine and citrulline) are involved in the urea cycle rather than energy metabolism. Aspartate is a purely ketogenic amino acid. (Option B):Incorrect because aspartate is purely glucogenic , converting into oxaloacetate , which enters the gluconeogenic pathway . Lysine is exclusively glucogenic. (Option D):Incorrect because lysine is purely ketogenic , forming acetoacetate , which is used for ketone body synthesis , and does not contribute to glucose production.

References:

Harper's Illustrated Biochemistry, 32nd edition, Page 142

↑ *Back to this question in Part A*

@Neet_pg_bot

Q4: A patient with anaemia has elevated levels of 2,3-Bisphosphoglycerate (2,3-BPG) in their red blood cells. How does this increased 2,3-BPG level affect the patient's haemoglobin in terms of oxygen delivery? (Q843515)

A. Forms salt bridges with positively charged residues forming a relaxed structure

B. Disrupts salt bridges formed in the taut structure, decreasing oxygen affinity

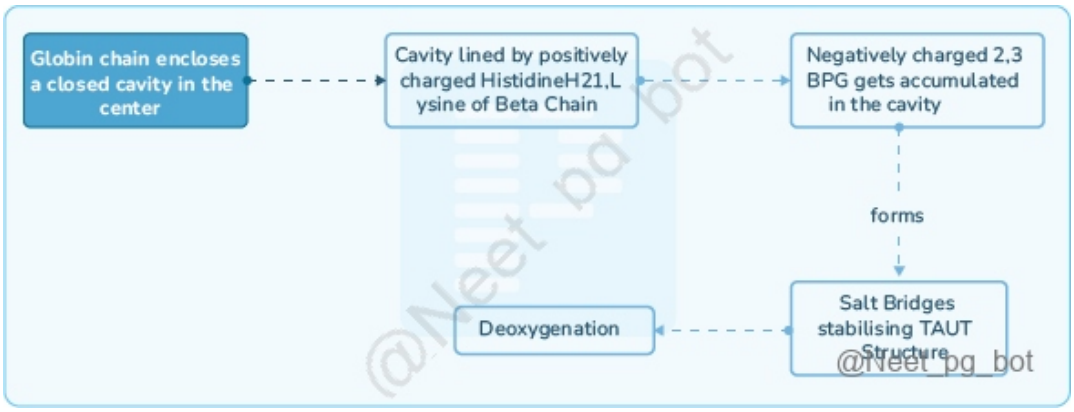
C. Forms salt bridges with positively charged residues stabilising taut structure

D. Binds to the heme directly, decreasing the oxygen affinity

Correct Answer: C. Forms salt bridges with positively charged residues stabilising taut structure

■ *Listen to Audio 1*

Explanation: Increased levels of 2,3-BPG stabilise the taut (T) structure of haemoglobin by forming salt bridges with positively charged residues. This stabilisation lowers haemoglobin's affinity for oxygen, promoting oxygen release to tissues, which can be beneficial in conditions like anaemia. Effect of 2,3 BPG on Hb: Reduces the affinity of Hb for oxygen and helps unload oxygen to tissues. A by-product of glycolysis. (Rapaport Leubering Cycle) Mechanism: 2,3-Bisphosphoglycerate (2,3-BPG) binds with a pocket formed by the β chains in the deoxygenated state of hemoglobin. Forms salt bridges with positively charged residues forming a relaxed structure (Option A): Incorrect because salt bridges stabilize the T form , not the R form. Disrupts salt bridges formed in the taut structure, decreasing oxygen affinity (Option B): Incorrect because 2,3-BPG forms rather than disrupts salt bridges, stabilizing the T form. Binds to the heme directly, decreasing the oxygen affinity (Option D): Incorrect because 2,3-BPG does not bind to heme ; it binds between β-chains in deoxygenated Hb.



References:

Harper's Illustrated Biochemistry, 32nd edition, Page 55

↑ *Back to this question in Part A*

@Neet_pg_bot

Q5: Which of the following techniques is most widely used to estimate a patient's HbA1c level for monitoring long-term glucose control? (Q843526)

A. Cation exchange chromatography

B. Anion exchange chromatography

C. Gel filtration chromatography

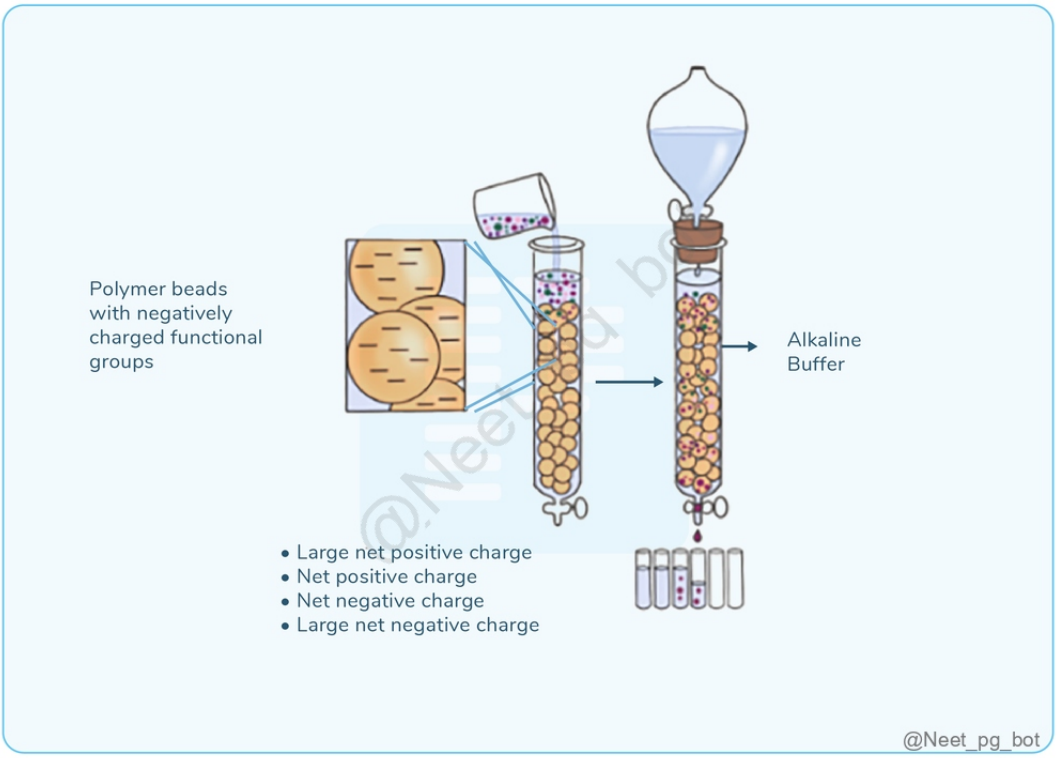
D. Paper chromatography

Correct Answer: A. Cation exchange chromatography

■ *Listen to Audio 1*

Explanation: Chromatography is a separation technique that distinguishes solutes based on their distribution between a stationary and a mobile phase. Solutes with a higher affinity for the mobile phase move faster, while those with a higher affinity for the stationary phase move slower or not at all. Multiple solutes can be separated using this method. Types of Chromatography: Types of Chromatography Description Cation exchange chromatography (Option A) Carboxymethyl Sepharose: A cation exchange resin used in HbA1c estimation. Separates positively charged proteins on an anionic stationary phase; impurities are washed off, and target proteins are eluted for analysis. In this, positively charged proteins bind to the negatively charged stationary phase. Anion exchange chromatography (Option B) Separates negatively charged proteins using a cationic stationary phase (e.g., quaternary ammonium); anions are retained while other components pass through. Gel filtration chromatography (Option C) Also known as size exclusion chromatography, this technique separates proteins based on their size. Smaller proteins get trapped in the pores of the gel matrix, slowing their movement, while larger proteins move quickly through the column. Paper chromatography (Option D) It is a type of planar

chromatography conducted on a flat surface. This method separates amino acids based on their polarity. Nonpolar amino acids move faster, while polar amino acids stay closer to the application point. Cation exchange chromatography



References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 66,67,68

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: Based on which characteristic the technique SDS-PAGE is used to separate proteins? (Q843525)

A. Charge

B. Molecular Weight

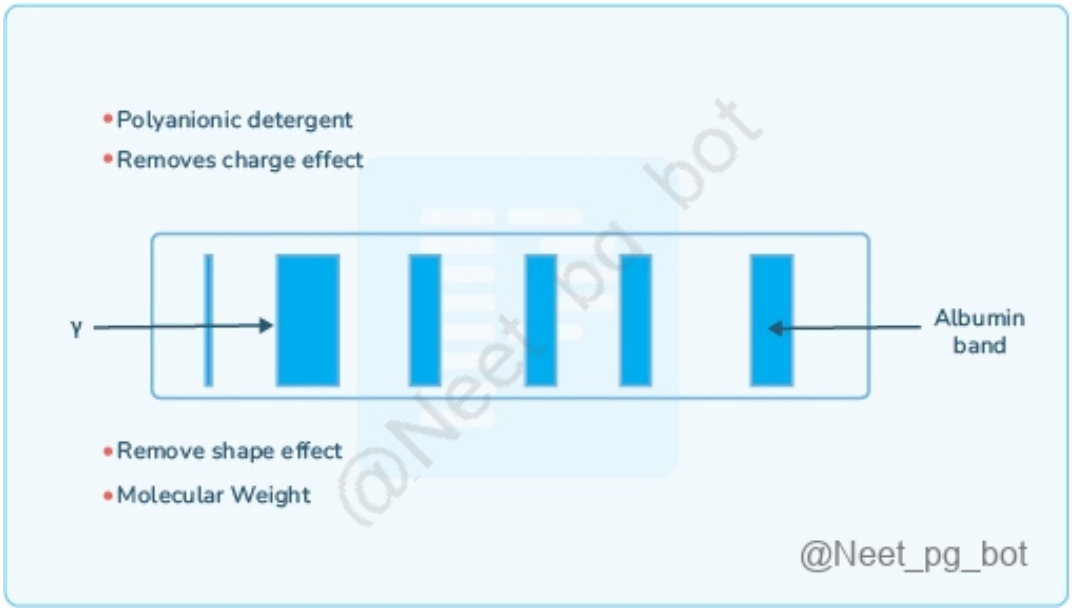
C. Size

D. Shape

Correct Answer: B. Molecular Weight

■ *Listen to Audio 1*

Explanation: The SDS-PAGE technique (Sodium Dodecyl Sulfate Polyacrylamide Gel Electrophoresis) separates proteins based on their molecular weight. In this technique, proteins are denatured and bound by SDS (Sodium Dodecyl Sulfate), which is an anionic detergent. SDS binds to proteins and imparts a uniform negative charge proportional to the protein's mass, effectively masking the protein's original charge and shape. (Option A and D ruled out) The denatured proteins are then subjected to an electric field within a polyacrylamide gel matrix. Since SDS provides a uniform charge-to-mass ratio, proteins move through the gel based on molecular weight , with smaller proteins migrating faster than larger ones. Molecular Weight vs. Size: (Option C ruled out) Molecular weight refers to the mass of a molecule. Size refers to the physical dimensions of a molecule. While larger molecules generally have higher molecular weights, factors like protein shape and conformation can influence their size. SDS coating of the protein removes the influence that shape has on the migration of the protein. SDS-PAGE Separation: SDS-PAGE eliminates the influence of protein shape and charge, ensuring that separation is primarily determined by molecular weight.



References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 62,66

↑ *Back to this question in Part A*

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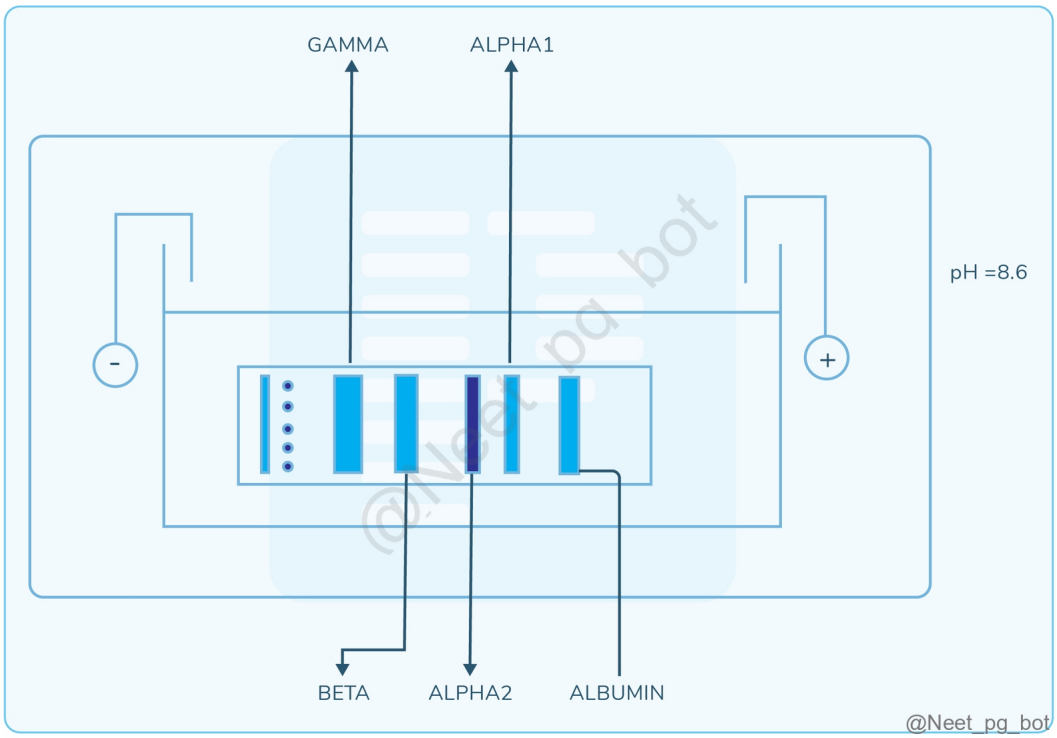
Q7: The electrophoresis pattern in the image shows a prominent alpha-2 band. Which disease is most likely associated with this finding? (Q843524)

A. Nephrotic syndrome

B. Multiple myeloma

C. Decompensated chronic liver disease

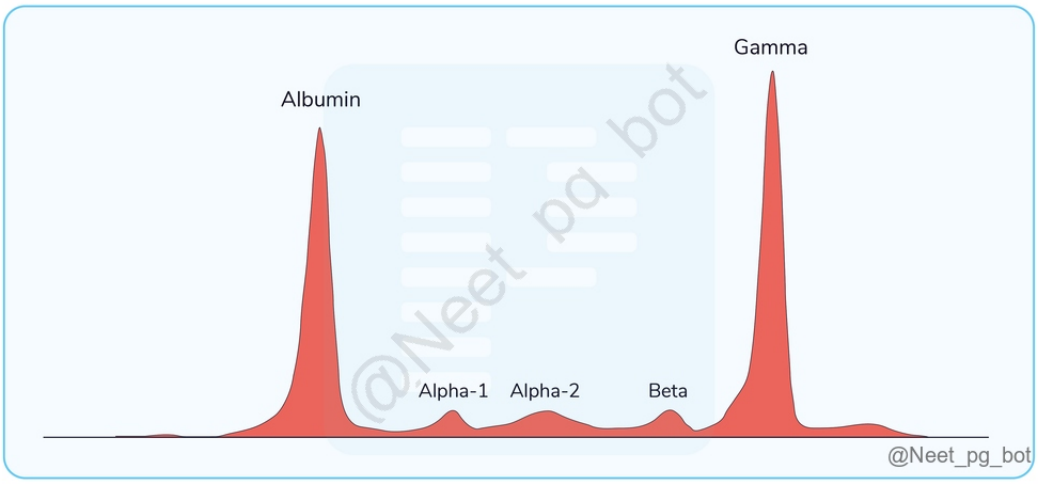
D. Nephritic syndrome

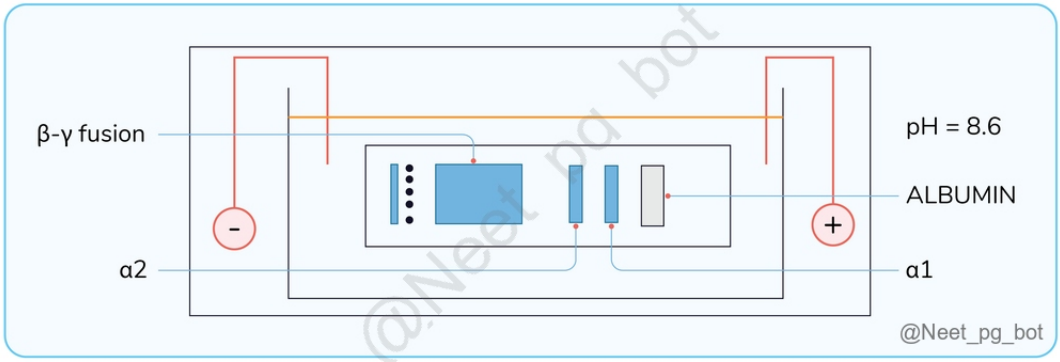


Correct Answer: A. Nephrotic syndrome

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Explanation: A prominent alpha-2 band on serum protein electrophoresis (SPEP) is a characteristic finding in nephrotic syndrome . In nephrotic syndrome: Albumin is lost in the urine due to increased glomerular permeability. Alpha-2 macroglobulin remains elevated because it is too large to be filtered out, leading to an enhanced alpha-2 band . Other small proteins are lost, leading to a relative increase in alpha-2 globulins (alpha-2 macroglobulin, haptoglobin, and ceruloplasmin) . Disorder EPP Pattern Multiple Myeloma (Option B) It is characterised by a narrow and intense band in the gamma region, the M band. This band indicates the presence of a monoclonal protein (M protein), which is a hallmark of multiple myeloma. {{caption_text}} Nephrotic syndrome (Option A) It shows a relative prominence of the alpha-2 band. This pattern results from the selective loss of smaller proteins, such as albumin, in the urine while larger proteins, such as alpha-2 macroglobulin, remain in the blood. Decompensated chronic liver disease (DCLD) (Option C) It exhibits beta-gamma bridging. This occurs due to polyclonal immunoglobulin production, leading to a continuous smear between the beta and gamma regions. Nephritic Syndrome (Option D) involves glomerular inflammation leading to hematuria, proteinuria, and decreased kidney function. It may show increased alpha-1 and alpha-2 globulins due to acute phase reactants but not a prominent alpha-2 band.





References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 58,59

↑ *Back to this question in Part A*

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Q8: A laboratory technician prepares samples for electrophoresis as the person is suspected to auffer from multiple myeloma. Which sample type is preferred for this test to ensure accurate results? (Q843523)

A. Serum

B. Plasma

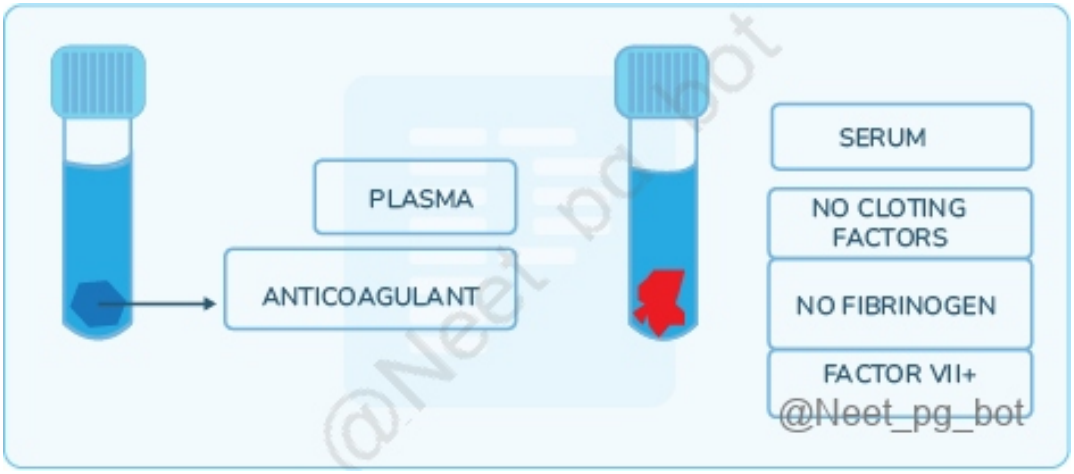
C. Whole blood

D. CSF

Correct Answer: A. Serum

■ *Listen to Audio 1*

Explanation: The preferred sample for electrophoresis is Serum due to the absence of fibrinogen. Serum (obtained after clotting) lacks clotting factors and fibrinogen. When a blood sample is collected in a tube with an anticoagulant, it separates into plasma at the top and blood cells below. Conversely, in a tube without an anticoagulant, blood clots due to the coagulation cascade, resulting in serum separation. Plasma (Option B) Fibrinogen present in plasma can migrate to the beta-gamma region, potentially masking monoclonal bands in multiple myeloma. Whole blood (Option C) contains cellular components (like red and white blood cells) that can interfere with the separation and analysis of proteins. Free of these cells, Serum provides a clearer and more accurate representation of the proteins in the blood. Urine (Option D) The protein concentration in urine is much lower compared to blood, which can lead to less reliable results. Additionally, urine proteins may vary based on hydration status and can include different proteins not present in serum.



References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 62,63

↑ *Back to this question in Part A*

@Neet_pg_bot

Q9: A 39-year-old male complains of unexplained fatigue and joint pain and visits his physician. The physician suspects chronic inflammatory disease and advises serum protein electrophoresis, which shows an increase in alpha 2 bands. Which of the following proteins shows an increase in alpha 2 bands? (Q843522)

A. Albumin

B. Macroglobulin

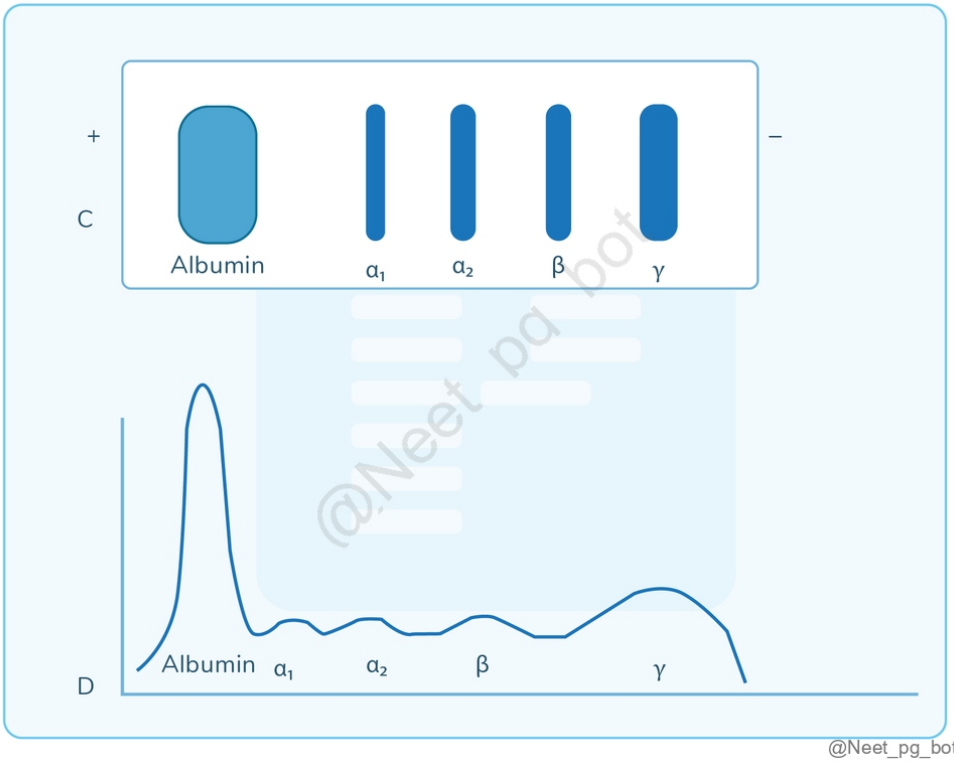
C. Gamma globulin

D. Transferrin

Correct Answer: B. Macroglobulin

■ *Listen to Audio 1*

Explanation: Macroglobulin is the protein that increases the alpha 2 band during inflammatory diseases. Serum protein electrophoresis: It is a diagnostic technique used to separate and analyse proteins in the blood based on their size, shape, and charge, helping to identify abnormalities in protein levels. It is commonly used to diagnose and monitor conditions like chronic inflammatory diseases, multiple myeloma, liver diseases, and nephrotic syndrome by evaluating specific protein bands. Electrophoresis band Proteins Albumin band Albumin (Option A ruled out) Alpha 1 band Alpha 1 antitrypsin,alpha-fetoprotein, transthyretin Alpha 2 band Macroglobulin (Option B) , haptoglobin, ceruloplasmin Beta band Transferrin (Option D) , low-density lipoproteins Gamma band Gamma immunoglobulin (Option C) ,fibrinogen



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References:

https://www.ncbi.nlm.nih.gov/books/NBK204/

Harper's Illustrated Biochemistry, 32nd edition, Page 636

↑ *Back to this question in Part A*

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Q10: Which protein migrates the farthest from the application point towards the anode in serum protein electrophoresis? (Q843520)

A. Albumin

B. Gamma globulin

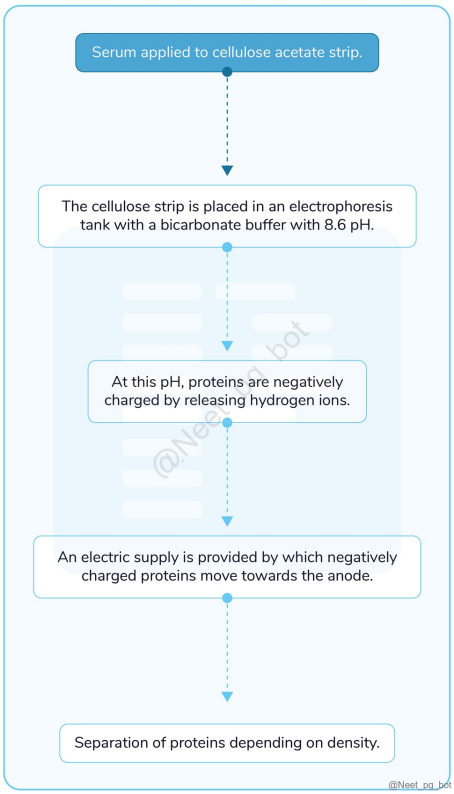
C. Alpha-1 globulin

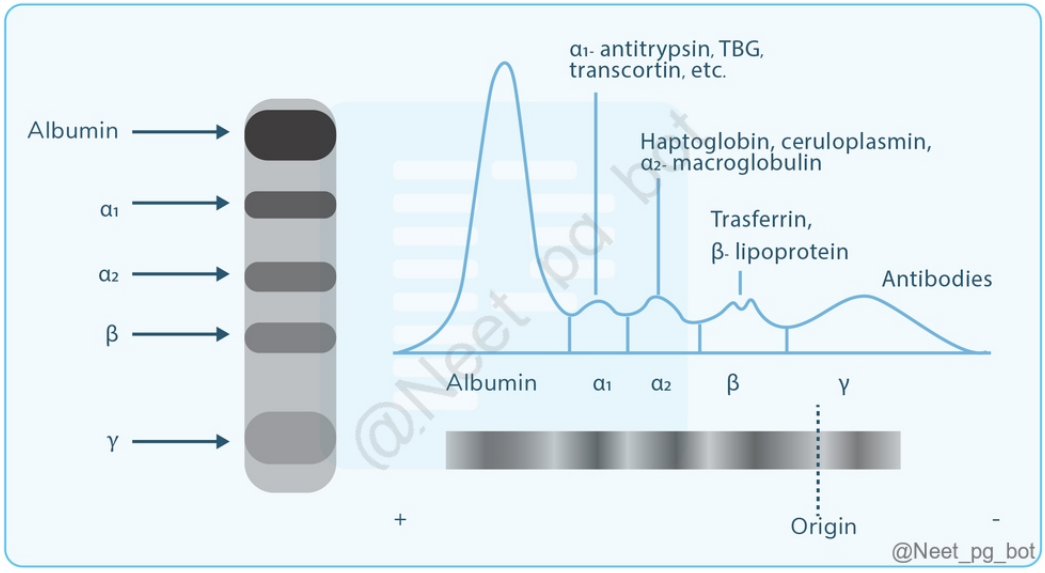
D. Beta globulin

Correct Answer: A. Albumin

■ *Listen to Audio 1*

Explanation: Albumin has the highest net negative charge at physiological pH (pH ~8.6 in electrophoresis). It has a low molecular weight and is the smallest major serum protein , allowing it to migrate the farthest toward the anode (positive electrode) in serum protein electrophoresis (SPEP) . Other globulin fractions (α, β, and γ) migrate slower due to lower net charge and larger size . Protein separation on Electrophoresis: Serum Protein Electrophoresis Migration Pattern Protein Fraction Charge Migration Distance (From Cathode to Anode) Albumin Most negative Farthest migration Alpha-1 globulin (Option C ruled out) Less negative Closer to origin Alpha-2 globulin Less negative Closer to origin Beta globulin (Option D ruled out) Intermediate Closer to albumin Gamma globulin (Option B ruled out) Least negative Closest to the cathode





References:

Textbook of Biochemistry by DM Vasudevan, 7th edition, Page 448.

Harper's Illustrated Biochemistry, 32nd edition, Page 636

↑ *Back to this question in Part A*

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Q11: All the following factors determine the mobility of proteins in electrophoresis except? (Q843519)

A. Charge

B. Isoelectric pH

C. Molecular weight

D. Shape

Correct Answer: B. Isoelectric pH

■ *Listen to Audio 1*

Explanation: The isoelectric pH is the point at which molecules have no net charge and, therefore, do not migrate during electrophoresis. The mobility of proteins in electrophoresis depends on Charge: Positively charged particles move towards negative electrodes and vice versa. (Option A ruled out) Molecular weight: Heavier particles move slowly when compared to lighter particles. (Option C ruled out) Shape of the particles: Globular proteins, being more compact, move faster than fibrous proteins, which are elongated and encounter resistance. (Option D ruled

out) Strength of electric field: A stronger electric field enhances the movement of molecules. Properties of the supporting medium: Denser gel decreases the mobility of large molecules. Temperature: Higher temperature increases the rate of movement.

References:

Textbook of Biochemistry by DM Vasudevan, 7th edition, Page 448

↑ *Back to this question in Part A*

@Neet_pg_bot

Q12: What is the most likely cause of the rightward shift of the oxyhemoglobin dissociation curve? (Q843517)

A. Decreased 2,3 -BPG

B. Decreased CO2

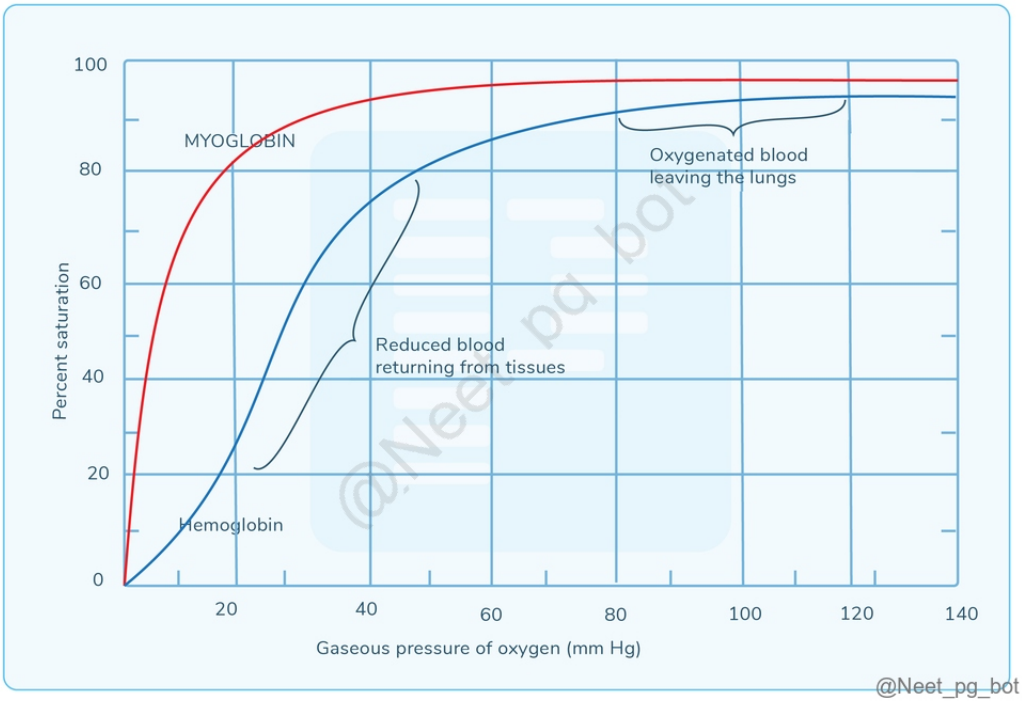
C. Increased pH

D. Increased temperature

Correct Answer: D. Increased temperature

■ *Listen to Audio 1*

Explanation: At higher temperatures, the oxyhemoglobin dissociation curve shifts towards the right, indicating decreased haemoglobin affinity for oxygen and facilitating the release of oxygen to tissues. The shape of oxyhemoglobin dissociation is sigmoid due to positive cooperativity. Oxyhemoglobin Dissociation Curve Shifts Factor Shifts Curve to the Right (↓ O2 Affinity, ↑ O2 Release) Shifts Curve to the Left (↑ O2 Affinity, ↓ O2 Release) pH (Bohr Effect) Decreased pH (Acidosis) Increased pH (Alkalosis) (Option C ruled out) Carbon Dioxide (CO■) Increased CO■ (Hypercapnia) Decreased CO■ (Hypocapnia) (Option B ruled out) Temperature Increased temperature (e.g., fever, exercise) Decreased temperature (e.g., hypothermia) 2,3-Bisphosphoglycerate (2,3-BPG) Increased 2,3-BPG (e.g., chronic hypoxia, anemia) Decreased 2,3-BPG (Option A ruled out) Carbon Monoxide (CO) - Presence of CO (Increased oxygen affinity, prevents release) Fetal Hemoglobin (HbF) - Presence of HbF (Higher oxygen affinity) Exercise Increased exercise - Altitude Acclimatization to high altitude - Growth Hormone Increases O■ release (Shifts right) - Thyroxine (T■) Increases O■ release (Shifts right) - `



References:

Textbook of Biochemistry by DM Vasudevan, 7th edition, Page 28

Harper's Illustrated Biochemistry, 32nd edition, Page 51

↑ *Back to this question in Part A*

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Q13: A newborn is diagnosed with a condition where their haemoglobin has an unusually high affinity for oxygen. What is the physiological basis for the increased oxygen affinity of fetal haemoglobin compared to adult haemoglobin? (Q843516)

A. Binds more 2,3-BPG, increasing oxygen affinity.

B. HbF contains serine that reduces the positive charge of the globin side chain.

C. Higher histidine content, reducing oxygen affinity.

D. Binds oxygen more loosely than adult Hb

Correct Answer: B. HbF contains serine that reduces the positive charge of the globin side chain.

■ *Listen to Audio 1*

Explanation: Correct answer B) HbF contains serine that reduces the positive charge of the globin side chain. Fetal haemoglobin (HbF) contains α■ and γ■ globin chains, where γ chains have serine instead of histidine. (Beta chains have more of histidine and lysine). This reduces the positive charge in the heme's central cavity, leading to decreased binding of 2,3-Bisphosphoglycerate (2,3-BPG). Difference between Adult Hemoglobin and Fetal Hemoglobin" Feature Adult Hemoglobin (HbA) Fetal Hemoglobin (HbF) Globin Side Chains α■β■ α■γ■ Oxygen Affinity Lower Higher 2,3-BPG Binding Higher Lower Physiological Role Tissue oxygen delivery Oxygen extraction from maternal blood Charge in Central Cavity More positive Less positive pH Sensitivity More sensitive Less sensitive Binds more 2,3-BPG, increasing oxygen affinity (Option A): Incorrect- More 2,3-BPG binding decreases oxygen affinity , stabilizing the taut form. HbF binds less 2,3-BPG , increasing its affinity for oxygen. Higher histidine content, reducing oxygen affinity (Option C): Incorrect- HbF has serine instead of histidine in its γ-chain, which actually increases oxygen affinity. Binds oxygen more loosely than adult Hb (Option D): Incorrect - HbF binds oxygen more tightly than HbA to facilitate oxygen transfer from maternal blood.

References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 53,56

↑ *Back to this question in Part A*

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Q14: Which of the following statements is TRUE regarding pKa and its implications in biochemistry? (Q843474)

A. The lower the pKa of the amino group, the weaker the acid.

B. pKa remains constant and does not change with the medium.

C. The net charge of a protein does not depend on the pKa value of the functional group.

D. When pH =pKa +/- 1, substances act as buffers.

Correct Answer: D. When pH =pKa +/- 1, substances act as buffers.

■ *Listen to Audio 1*

Explanation: A buffer resists changes in pH when small amounts of acid or base are added. This is explained by the Henderson-Hasselbalch equation : pH=pKa+log■([A –]/[HA]) pH = Acidity of the solution pKa = The pH at which 50% of the acid is dissociated [A■] = Concentration of the conjugate base [HA] = Concentration of the weak acid When pH = pKa , the concentrations of the acid and its conjugate base are equal , maximizing buffering capacity. The buffering effect is strongest within ±1 pH unit of the pKa value . pKa and Biochemical Implications Concept

References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 20,21

↑ *Back to this question in Part A*

Q15: An athlete undergoing high-altitude training adapts to decreased oxygen availability by altering haemoglobin to enhance oxygen delivery. Which structural form of haemoglobin is predominantly favoured to enhance oxygen release to tissues? (Q843493)

A. Taut structure

B. Relaxed structure

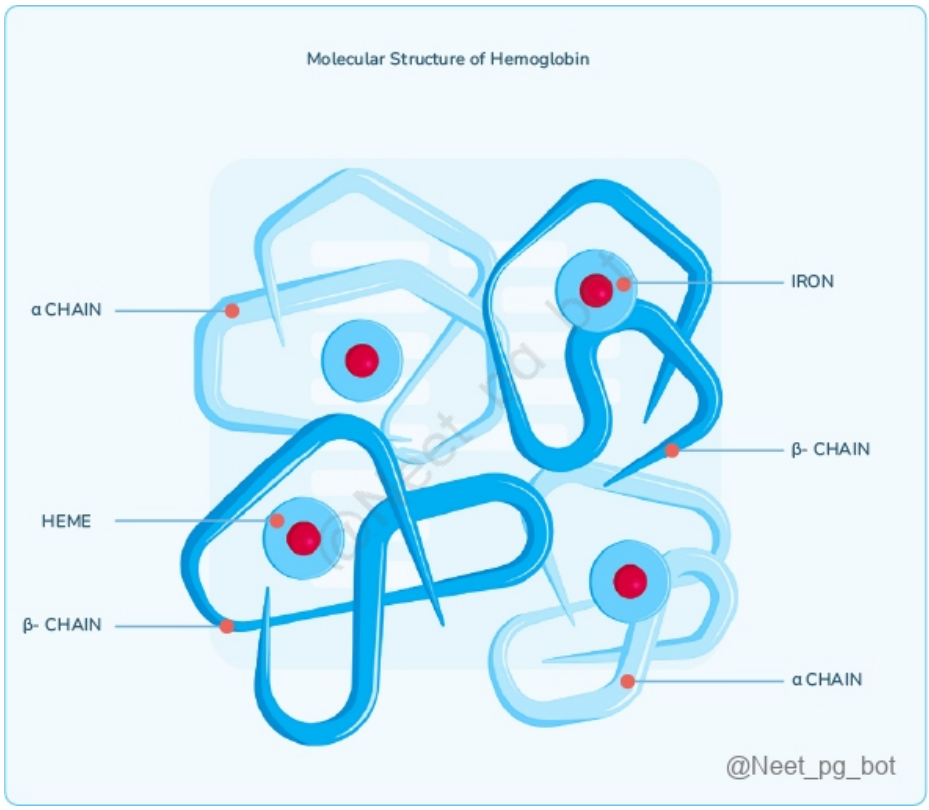
C. Globular structure

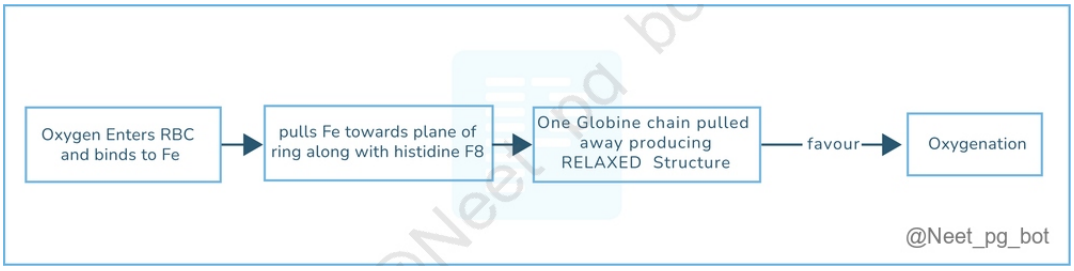
D. Tertiary structure

Correct Answer: A. Taut structure

■ *Listen to Audio 1*

Explanation: Taut form of hemoglobin has a lower affinity for oxygen, facilitating oxygen release to tissues under conditions of low oxygen and high CO■/H■ (Bohr effect). Structure of Haemoglobin: Hemoglobin consists of four globin chains, each with an associated heme group containing Fe²■. Strong polypeptide chain interaction: Taut structure favours deoxygenation. Weak polypeptide chain interaction: Relaxed structure favours oxygenation. Haemoglobin Structure and Oxygen Binding Structural Form Description Effect on Oxygen Binding Taut (T) form Low oxygen affinity, stabilized by CO■, H■, and 2,3-BPG Favours oxygen release to tissues Relaxed (R) form High oxygen affinity, stabilized by oxygen binding Favours oxygen uptake in lungs At the Tissue site: In the Lung:







References:

Harper's Illustrated Biochemistry, 32nd edition, Page 52

↑ *Back to this question in Part A*

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Q16: Which of the following correctly describes the role of histidine residues in myoglobin in relation to carbon monoxide (CO) binding? (Q843492)

A. Prevent CO binding by increasing the heme's affinity for oxygen.

B. Enhance CO binding, similar to oxygen.

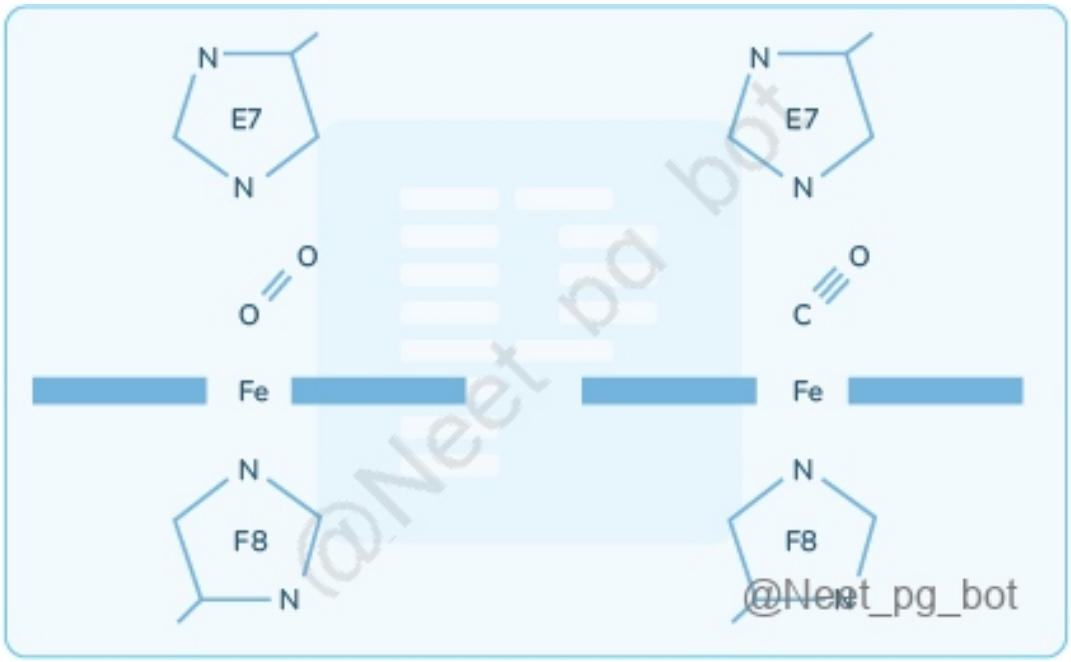
C. Inhibit CO binding by sterically hindering the heme iron.

D. It does not influence the binding of CO to the heme.

Correct Answer: C. Inhibit CO binding by sterically hindering the heme iron.

■ *Listen to Audio 1*

Explanation: Histidines E7 and F8 in myoglobin are crucial in regulating CO binding. Histidine E7 (distal histidine) particularly creates steric hindrance, which reduces CO's ability to bind effectively to the heme iron. Myoglobin Structure: Heme: Fe^{2}■ in the center of four pyrrole rings, slightly below the heme plane. Globin: Single polypeptide (~150 AA) with 8 α-helices (A–H). Histidine F8: Binds Fe^{2}■ (proximal). Histidine E7: Creates steric hindrance, reducing CO binding. CO vs O■: CO binds Fe^{2}■ ~2000× stronger; E7 limits this by hindering linear CO, while angled O■ fits easily. Prevent CO binding by increasing the heme's affinity for oxygen (Option A): Histidine does not directly increase oxygen affinity but stabilizes O■ binding via hydrogen bonding . Enhance CO binding, similar to oxygen (Option B): CO has a higher intrinsic affinity for heme than O■, but histidine hinders CO binding , not enhances it. It does not influence the binding of CO to the heme (Option D): Incorrect, as distal histidine (His E7) significantly reduces CO binding affinity by enforcing an angled binding geometry.



References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 50,51

↑ *Back to this question in Part A*

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Q17: Which cellular component is primarily responsible for degrading intracellular proteins tagged with ubiquitin? (Q843490)

A. Lysosomes

B. Proteasomes

C. Cathepsins

D. Endosomes

Correct Answer: B. Proteasomes

■ *Listen to Audio 1*

Explanation: Proteasomes are large protein complexes that degrade ubiquitin-tagged intracellular proteins through a process called ubiquitin-proteasome pathway (UPP) . This system is ATP-dependent and ensures the regulated degradation of proteins to maintain cellular homeostasis. The 26S proteasome consists of: 20S core particle (catalytic chamber for protein degradation). 19S regulatory particle (recognizes ubiquitinated proteins and unfolds them for degradation).. Ubiquitin–Proteasome Pathway: Main system for degrading misfolded or damaged proteins. Ubiquitination: Proteins tagged with ubiquitin via E1, E2, E3 enzymes. Proteasomal degradation: Tagged proteins broken down into peptides by the proteasome. Lysosomes (Option A): Degrade extracellular and some intracellular proteins via autophagy , but not ubiquitin-tagged proteins . Function mainly in bulk degradation rather than targeted proteolysis. Cathepsins (Option C): A group of lysosomal proteases involved in protein degradation inside lysosomes . They do not participate in ubiquitin-proteasome degradation . Endosomes (Option D): Involved in vesicular transport , sorting proteins to lysosomes , plasma membrane recycling , or exocytosis . They do not degrade ubiquitinated proteins directly.

References:

Harper's Illustrated Biochemistry, 32nd edition, Page 280

https://www.ncbi.nlm.nih.gov/books/NBK9957/

↑ *Back to this question in Part A*

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Q18: Which protein assists in proper folding by binding to hydrophobic regions of other proteins during the folding process? (Q843488)

A. Proteases

B. Chaperonins

C. Ubiquitin

D. Prions

Correct Answer: B. Chaperonins

■ *Listen to Audio 1*

Explanation: Protein folding is the process by which a protein structure assumes its functional three-dimensional shape from a linear chain of amino acids. Chaperonins, are hsp60 family of chaperones, whih are a class of molecular chaperones that assist in protein folding. They form large barrel-shaped complexes that can encapsulate unfolded or partially folded proteins, providing an isolated environment for proper folding. Chaperonins bind to hydrophobic regions of unfolded proteins, preventing inappropriate interactions and aggregation. Role of Chaperonins in Protein Folding Function Chaperonin Action Prevents misfolding Shields hydrophobic regions to avoid aggregation Assists refolding Helps denatured proteins regain functional shape Provides an isolated chamber Forms a cavity where proteins fold properly ATP-dependent process Uses ATP to facilitate conformational changes Heat shock proteins are molecular chaperones that play crucial roles in the folding and unfolding of complex polypeptides within the cellular system. These molecules are involved in various processes, including vesicular transport, prevention of protein aggregation in the cytosol, and cell signaling. Proteases (Option A): Enzymes that hydrolyze peptide bonds, degrading misfolded proteins. Ubiquitin(Option C): Tags damaged or misfolded proteins for proteasomal degradation. Prions(Option D): Misfolded proteins that induce misfolding in others, causing neurodegenerative diseases.

References:

Harper's Illustrated Biochemistry, 32nd edition, Page 42

↑ *Back to this question in Part A*

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Q19: Which of the following techniques provides the highest resolution for determining the primary structure of proteins? (Q843487)

A. Nuclear Magnetic Resonance (NMR) spectroscopy

B. X-ray crystallography

C. Sanger’s sequencing

D. Optical rotatory dispersion

Correct Answer: C. Sanger’s sequencing

■ *Listen to Audio 1*

Explanation: Correct answer: C) Sanger’s sequencing Sanger sequencing is typically used for DNA, but it can be adapted for protein sequencing in the form of Edman degradation, which determines the primary structure of

proteins. This method provides the exact sequence of amino acids, giving the highest resolution of the primary structure. Protein Structure and Methods of Determination Structure Methods of Determination Primary Edman sequencing Sanger’s sequencing (Option C) Reverse sequencing Secondary Optical rotatory dispersion (Option D) Ocular dichroism Tertiary X-ray crystallography (Option B) UV Spectroscopy NMR spectroscopy (Option A) Quaternary SDS PAGE (Sodium Dodecyl Sulfate-Polyacrylamide Gel Electrophoresis)

References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 27, 28, 29

↑ *Back to this question in Part A*

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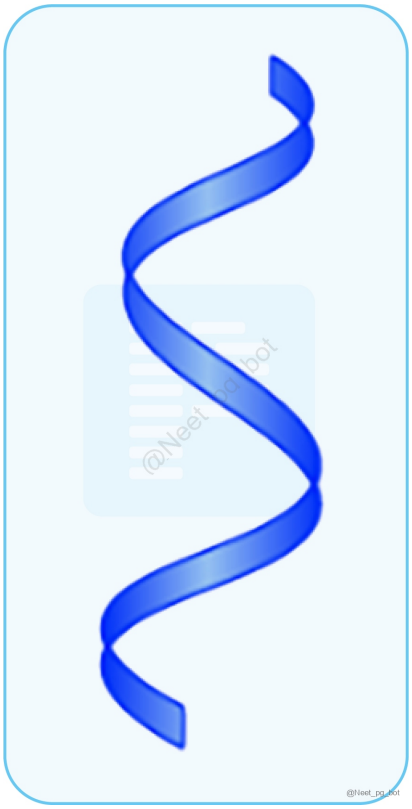
Q20: What type of bond is primarily responsible for stabilising the structure in proteins as shown in the image? (Q843486)

A. Covalent bonds between side chains

B. Ionic bonds between charged amino acids

C. Hydrogen bonds between backbone atoms

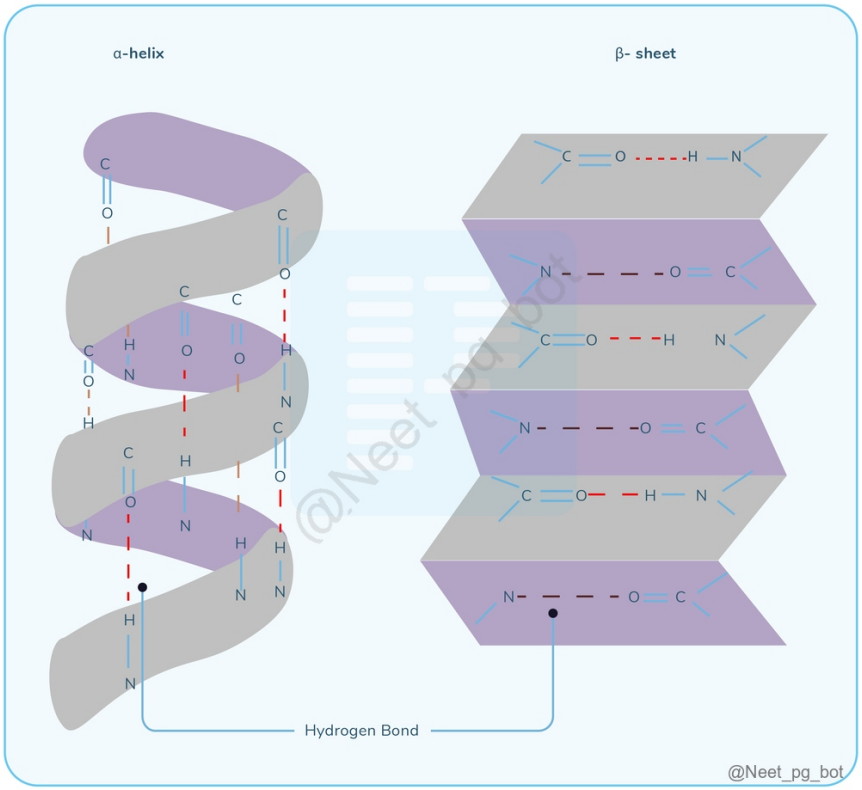
D. Disulfide bridges between cysteine residues



Correct Answer: C. Hydrogen bonds between backbone atoms

Listen to Audio 1

Explanation: The alpha-helix is a secondary structure of protein, stabilized primarily by hydrogen bonds between the carbonyl oxygen (C=O) of one amino acid and the amide hydrogen (N-H) of another amino acid, located four residues ahead in the chain. This forms a regular helical structure with 3.6 amino acids per turn . Secondary structure of Protein: α-Helix: Coiled, compact, flexible; intrastrand H-bonds (e.g., keratin). β-Sheet: Zigzag, extended, rigid; interstrand H-bonds (e.g., silk fibroin). Covalent bonds between side chains (Option A): Incorrect because covalent (peptide) bonds link amino acids but do not stabilize secondary structures. Ionic bonds between charged amino acids (Option B): Incorrect because ionic bonds contribute to tertiary and quaternary structures , not the stabilization of an alpha-helix. Disulfide bridges between cysteine residues (Option D): Incorrect because disulfide bonds stabilize tertiary and quaternary structures but are not responsible for alpha-helix formation .



References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 36, 37

↑ *Back to this question in Part A*

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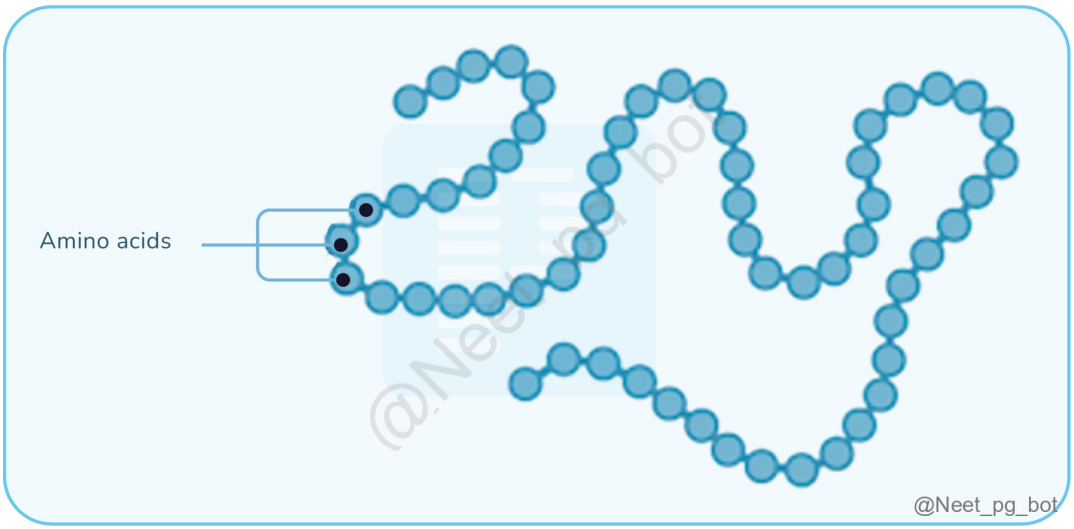
Q21: Which of the following interactions is primarily responsible for maintaining the structure of proteins, as shown in the image? (Q843485)

A. Peptide bonds

B. Hydrogen bonds

C. Disulfide bridges

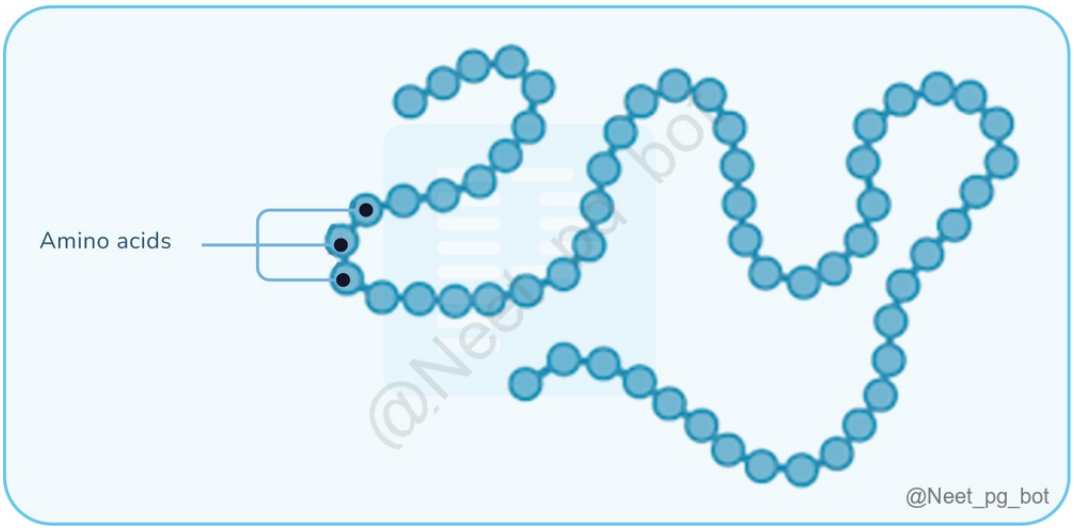
D. Van der Waals interactions

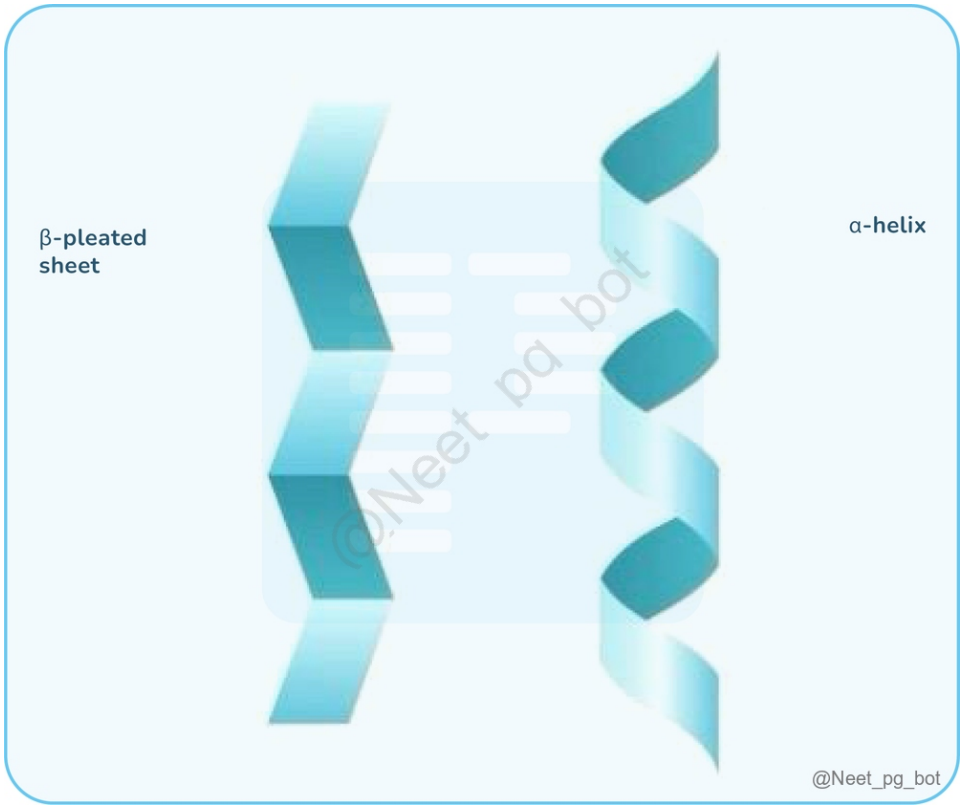


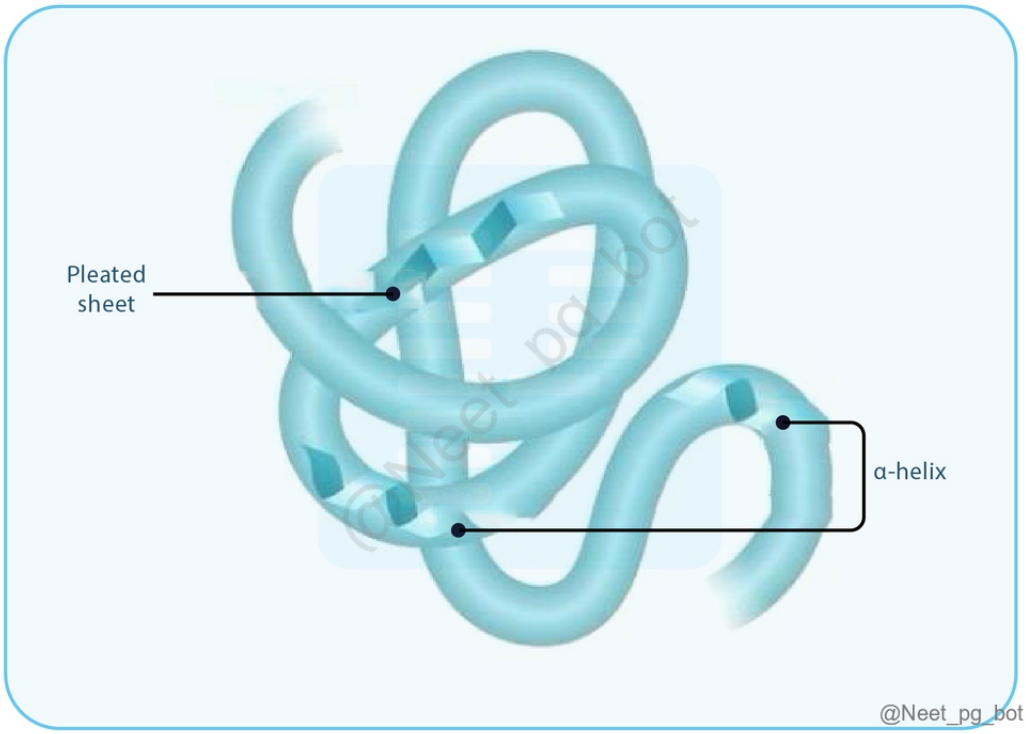
Correct Answer: A. Peptide bonds

■ *Listen to Audio 1*

Explanation: The linear sequence of amino acids in a polypeptide chain represents the Primary structure of the protein. Peptide bonds are covalent bonds formed between the carboxyl group (-COOH) of one amino acid and the amino group (-NH2) of the next amino acid. These bonds are responsible for linking amino acids together in a specific sequence Protein Structure Linkage Primary Structure: Linear sequence of amino acids encoded by mRNA. Peptide bonds (Covalent, via dehydration reaction) (Option A) Secondary structure: Local folding into α-helices & β-sheets due to backbone interactions and is of two types: Alpha-helix Beta-pleated sheet Hydrogen bonds (Between carbonyl and amide groups) (Option B) Tertiary structure: Three-dimensional structure of a single polypeptide due to side-chain interactions. Disulfide bonds (Option C) (Covalent), Hydrophobic interactions, Hydrogen bonds, Ionic bonds, Van der Waals forces Quaternary structure: Assembly of multiple polypeptide subunits into a functional protein Non-covalent interactions (Hydrogen bonds, Ionic bonds, Hydrophobic interactions, Van der Waals forces); Rarely Disulfide bonds Van der Waals interactions (Option D) These weak interactions contribute to overall protein stability and folding in tertiary and quaternary structures but do not determine the primary structure.


Amino acids
@Neet_pg_bot


β-pleated sheet
α-helix
@Neet_pg_bot





References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 36, 37, 38

↑ *Back to this question in Part A*

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Q22: Match the following protein tests with their primary purpose or detection target: 1.Ninhydrin test a. Detects compouds containing 2 or more peptide bonds 2.Xanthoproteic test b. Detects aromatic amino acids 3.Sakaguchi test c. Detects arginine 4.Biuret test d. Detects alpha-amino acids (Q843483)

A. 1-b, 2-c, 3-d, 4-a

B. 1-d, 2-b, 3-c, 4-a

C. 1-a, 2-d, 3-b, 4-c

D. 1-c, 2-a, 3-d, 4-b

Correct Answer: B. 1-d, 2-b, 3-c, 4-a

■ *Listen to Audio 1*

Explanation: Types of Tests for the Detection of Proteins Test Principle Detection Target Positive Result Biuret Test Peptide bonds react with Cu^{2}■ in an alkaline medium Peptide bonds (proteins and polypeptides) Violet color Ninhydrin Test Ninhydrin reacts with free α-amino groups Free amino acids and proteins with free NH■ Blue/Purple color Xanthoproteic Test Nitration of aromatic rings with HNO■ Aromatic amino acids (tyrosine, tryptophan, phenylalanine) Yellow color Sakaguchi Test Reaction of α-naphthol with guanidino group in the presence of NaOBr Arginine Red color Lead Acetate Test Reaction of sulfur-containing amino acids with lead acetate Sulfur-containing amino acids (cysteine, methionine) Black precipitate Millon's Test Reaction of phenol groups with mercuric nitrate in acidic medium Tyrosine Red color Nitroprusside Test Reaction of cysteine with sodium nitroprusside Cysteine Red color Test for Casein Acid precipitation of casein Casein (milk protein) White precipitate

References:

Textbook of Biochemistry by DM Vasudevan, 7th edition, Page 31

↑ *Back to this question in Part A*

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Q23: Which of the following is true regarding the properties of glycine? (Q843482)

A. Has asymmetric carbon atoms

B. Doesn't exhibit stereoisomerism

C. Optically active

D. Complex amino acid

Correct Answer: B. Doesn't exhibit stereoisomerism

■ *Listen to Audio 1*

Explanation: Doesn't exhibit stereoisomerism Glycine is the only amino acid that lacks a chiral center because its side chain is a hydrogen atom (-H), making both groups attached to the α-carbon identical. Due to the absence of a chiral center, glycine does not exhibit stereoisomerism and does not exist in D- or L-forms. Properties of Glycine: Property Description Simplest Amino Acid (Option D) Has two hydrogen atoms attached to the α-carbon. Polarity Non-polar; fits into both hydrophilic and hydrophobic environments due to its small size. Chirality Lacks a chiral center, making it optically inactive and incapable of stereoisomerism. (Option C) Glycine Cleavage System Produces ammonia (NH■), carbon dioxide (CO■), and N,N-methylene tetrahydrofolate. Biosynthetic Role Precursor for porphyrins, creatine, glutathione, serine, purines, and heme. Has symmetrical carbon atoms (Option A): Incorrect because glycine has only one carbon bonded to two identical hydrogen atoms, making it symmetrical.

References:

https://pubchem.ncbi.nlm.nih.gov/compound/Glycine

↑ *Back to this question in Part A*

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Q24: A 22-year-old woman presents with complaints of chronic fatigue and frequent infections. She follows a strict vegetarian diet and consumes less protein-rich food. Laboratory tests reveal a decreased level of a particular amino acid. Which of the following amino acids is likely deficient in this patient? (Q843477)

A. Threonine and Lysine

B. Tyrosine and Tryptophan

C. Alanine and Glycine

D. Cysteine and Methionine

Correct Answer: A. Threonine and Lysine

■ *Listen to Audio 1*

Explanation: Correct answer: A) Threonine and Lysine Threonine and Lysine are essential amino acids , meaning they must be obtained from the diet. A strict vegetarian diet, especially one low in protein-rich foods like legumes, dairy, and soy, can lead to deficiencies in essential amino acids. Lysine deficiency can result in fatigue, impaired immune function, and decreased protein synthesis , which explains the patient’s symptoms. Threonine deficiency affects immune function and protein metabolism , further contributing to fatigue and increased susceptibility to infections. Classification of Amino Acids: Essential vs. Non-Essential vs. Conditionally Essential Essential Amino Acids (Must be obtained from the diet) Amino Acid Dietary Sources Deficiency Effects Threonine Meat, dairy,

eggs, legumes Impaired immune response, fatigue Lysine Meat, fish, dairy, legumes Poor wound healing, frequent infections, fatigue Tryptophan (Option B) Dairy, turkey, nuts, seeds Mood disturbances, pellagra (niacin deficiency) Methionine (Option D) Meat, eggs, dairy Fatty liver, slow growth Phenylalanine Meat, dairy, soy, fish Growth retardation, mental impairment Leucine Meat, dairy, eggs, soybeans Muscle wasting, hypoglycemia Isoleucine Meat, fish, eggs, soy Muscle weakness, dizziness Valine Meat, dairy, legumes Neurological symptoms, muscle weakness Histidine Meat, poultry, fish, dairy Anemia, impaired immunity Non-Essential Amino Acids (Synthesized by the body) Amino Acid Synthesized From Deficiency Effects Tyrosine (Option B ruled out) Phenylalanine Rare deficiency, linked to metabolic disorders Cysteine (Option D ruled out) Methionine Rare deficiency, oxidative stress Alanine (Option C ruled out) Pyruvate No significant deficiency Glycine (Option C ruled out) Serine No significant deficiency Serine 3-Phosphoglycerate No significant deficiency Aspartate Oxaloacetate No significant deficiency Asparagine Aspartate No significant deficiency Glutamate α-Ketoglutarate No significant deficiency Proline Glutamate No significant deficiency Conditionally essential amino acids (needed during stress, illness, or rapid growth): Arginine, Glutamine.

References:

Harper's Illustrated Biochemistry, 32nd edition, Page 274

↑ *Back to this question in Part A*

@Neet_pg_bot

Q25: Which of the following amino acids contains a hydroxyl group (OH) in its side chain? (Q843476)

A. Phenylalanine

B. Serine

C. Cysteine

D. Glutamine

Correct Answer: B. Serine

■ *Listen to Audio 1*

Explanation: Serine contains a hydroxyl (-OH) group in its side chain, making it a polar, uncharged amino acid. This hydroxyl group participates in hydrogen bonding and is a target for phosphorylation in protein regulation. Structure of serine: COO - | CH-NH 3 + | CH 2 -OH Classification of Amino Acids Based on Functional Groups Amino acids are classified based on the chemical nature of their R (side chain) groups , which determine their biochemical properties. Below is a classification based on their functional groups: Category Amino Acids Functional Group Properties Hydroxyl (-OH) group Serine, Threonine, Tyrosine Hydroxyl (-OH) Polar, involved in hydrogen bonding and phosphorylation. Sulfur-containing Cysteine (Option B ruled out), Methionine Thiol (-SH), Thioether (-S-) Cysteine forms disulfide bonds; Methionine is involved in methyl transfer reactions. Aromatic Phenylalanine (Option A ruled out) , Tyrosine, Tryptophan Benzene or Indole rings Hydrophobic, absorb UV light, involved in protein-protein interactions. Amide (-CONH■) group Asparagine, Glutamine

(Option D ruled out) Amide (-CONH■) Polar, involved in hydrogen bonding. Carboxyl (-COOH) group (Acidic) Aspartic acid, Glutamic acid Carboxyl (-COOH) Negatively charged at physiological pH, important in enzyme active sites. Amino (-NH■■) group (Basic) Lysine, Arginine, Histidine Amine (-NH■, -C=NH, -NH■) Positively charged at physiological pH, involved in salt bridge formation. Aliphatic (Non-polar hydrocarbon chains) Glycine, Alanine, Valine, Leucine, Isoleucine, Proline Hydrocarbon (-CH, -CH■, -CH■) Hydrophobic, contribute to protein stability.

References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 18,19

↑ *Back to this question in Part A*

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Q26: Which of the following is not a characteristic of the isoelectric pH (pI) of a protein? (Q843475)

A. High solubility

B. Low buffering capacity

C. No net charge

D. Minimal mobility

Correct Answer: A. High solubility

■ *Listen to Audio 1*

Explanation: The pH at which an amino acid exists predominantly as a zwitterion and has no net charge is called the isoelectric point (pI) . At this pH, the molecule does not migrate in an electric field. At pI, proteins tend to aggregate due to reduced electrostatic repulsion, leading to low solubility in aqueous solutions. Concept of Isoelectric Point (pI): Characteristic

References:

Harper's Illustrated Biochemistry, 32nd edition, Page 20

Textbook of Biochemistry by DM Vasudevan, 7th edition, Page 28

↑ *Back to this question in Part A*

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• [04_Amino Acids and Proteins Chemistry] 014 Amino Acid Catabolism Urea Cycle And Hyperammonemia

« Back to Folder Directory

[04_Amino Acids and Proteins Chemistry] 014 Amino Acid Catabolism Urea Cycle And Hyperammonemia — Quick Jump

Q1 (p. 768)	*Q2 (p. 769)*	*Q3 (p. 770)*	*Q4 (p. 772)*	*Q5 (p. 773)*	*Q6 (p. 774)*
Q7 (p. 776)	*Q8 (p. 777)*	*Q9 (p. 779)*	*Q10 (p. 780)*	*Q11 (p. 781)*	*Q12 (p. 782)*
Q13 (p. 783)	*Q14 (p. 784)*	*Q15 (p. 785)*	*Q16 (p. 785)*	*Q17 (p. 786)*	*Q18 (p. 787)*
Q19 (p. 788)	*Q20 (p. 789)*	*Q21 (p. 790)*	*Q22 (p. 791)*	*Q23 (p. 792)*	*Q24 (p. 793)*
Q25 (p. 793)	*Q26 (p. 794)*				

Q1: A one-year-old boy presents with lethargy, poor muscle tone, and developmental delays. Blood tests reveal elevated levels of glycine. The physician suspects a defect in the glycine cleavage system. Which of the following metabolites, if found LOW, would support the diagnosis of a defective glycine cleavage system? (Q845049)

A. Serine

B. N5,N10 methylene tetrahydrofolate

C. Threonine

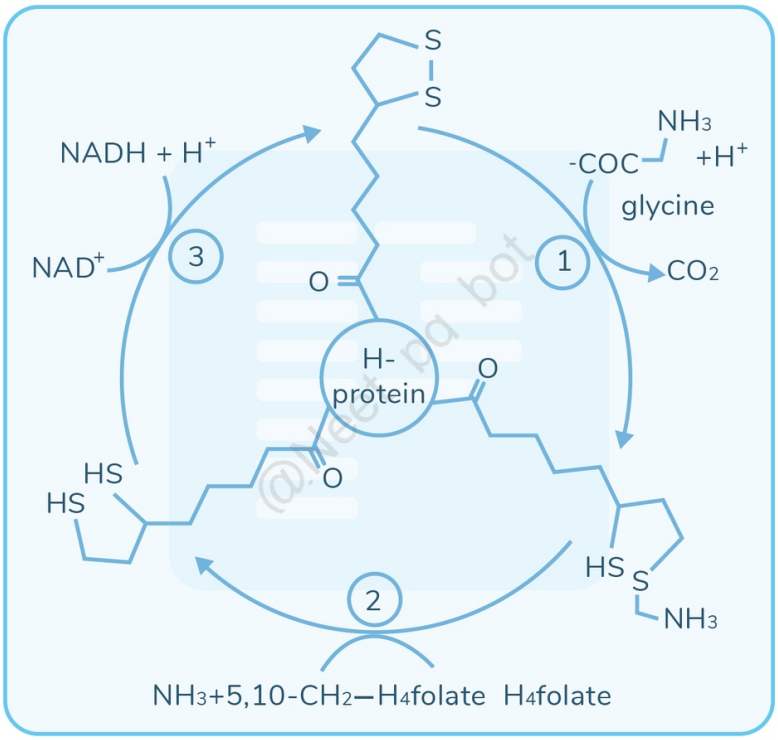
D. Succinyl CoA

Correct Answer: B. N5,N10 methylene tetrahydrofolate

■ *Listen to Audio 1*

Explanation: The glycine cleavage system directly produces N5,N10-methylene tetrahydrofolate when it breaks down glycine. A defect in this system would lead to a decreased production of this metabolite . Glycine Cleavage System (GCS): Location: Liver mitochondria Function: Splits glycine into CO■, NH■, and transfers a methylene group to THF → forms N■,N¹■-methylene-THF. Components: T: THF-dependent aminotransferase P: PLP-dependent glycine decarboxylase L: Lipoamide dehydrogenase H: Electron carrier protein Coenzymes: THF, PLP, Lipoamide Reaction: Glycine + H■-folate + NAD■ → CO■ + NH■ + 5,10-CH■-H■-folate + NADH + H■ Disorders: Nonketotic hyperglycinemia – caused due to a defect in the glycine decarboxylase complex, which results in the accumulation of glycine in blood, urine, and cerebrospinal fluid, causing neonatal lethargy, hypotonia, seizures, apnea and developmental delay. It can be managed by sodium benzoate (which combines with glycine to form hippurate and is excreted in urine), NMDA receptor antagonists (e.g., dextromethorphan or ketamine), anticonvulsants and supportive therapy. Primary hyperoxaluria – Defect in glyoxylate metabolism causing excess formation of oxalate, which combines with calcium to form calcium oxalate kidney stones. It

presents as recurrent renal stones, nephrocalcinosis and progressive renal failure. Treatment includes high fluid intake, pyridoxine (Vitamin B6), alkalinization of urine with potassium citrate, dialysis in advanced renal failure and liver or combined liver-kidney transplantation as definitive treatment in severe cases. Glycinuria – Defective renal tubular reabsorption of glycine, leading to increased urinary glycine excretion. It is usually asymptomatic and occasionally associated with kidney stone formation. Often no specific treatment required except adequate hydration to prevent renal stone formation. Serine levels might even increase as the body tries to compensate for the glycine deficiency. (Option A) A defective glycine cleavage system is unlikely to directly impact threonine levels. (Option C) Succinyl CoA is involved in heme synthesis ; defects in glycine breakdown wouldn't impact succinyl CoA levels. (Option D)



References:

Harper's Illustrated Biochemistry, 32nd edition, Page. 293

DM Vasudevan- Textbook of Biochemistry for Medical Students, 7th edition, Pages 210,211

↑ *Back to this question in Part A*

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Q2: A patient with a urea cycle disorder is being evaluated. Which urea cycle intermediate serves as the link between the urea cycle and the TCA cycle? (Q845061)

A. Argininosuccinate

B. Fumarate

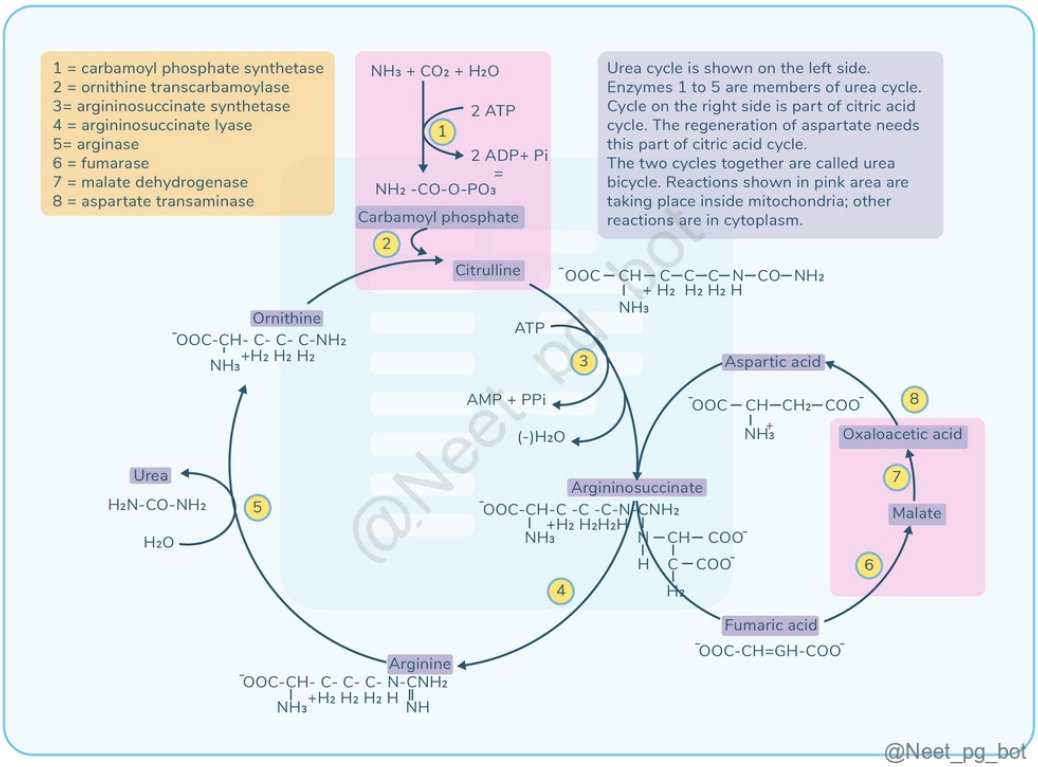
C. Oxaloacetate

D. Succinate

Correct Answer: B. Fumarate

■ *Listen to Audio 1*

Explanation: Argininosuccinate lyase cleaves argininosuccinate to arginine and fumarate. Fumarate is converted to malate and then to oxaloacetate in the TCA cycle , which is transaminated to aspartate . This links the urea cycle with the TCA cycle. The reaction can be summarised as Citrulline + aspartate → Arginine + fumarate.



References:

DM Vasudevan- Textbook of Biochemistry for Medical Students, 7th edition, Pages 204,205

↑ *Back to this question in Part A*

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Q3: A 3-week-old infant presents with poor feeding, lethargy, and vomiting. Lab results show elevated ammonia levels, increased glutamine and increased orotic acid in the blood, with no evidence of megaloblastic anaemia. Which enzyme deficiency is likely responsible for this condition? (Q845060)

A. Ornithine transcarbamylase (OTC)

B. Ornithine aminotransferase

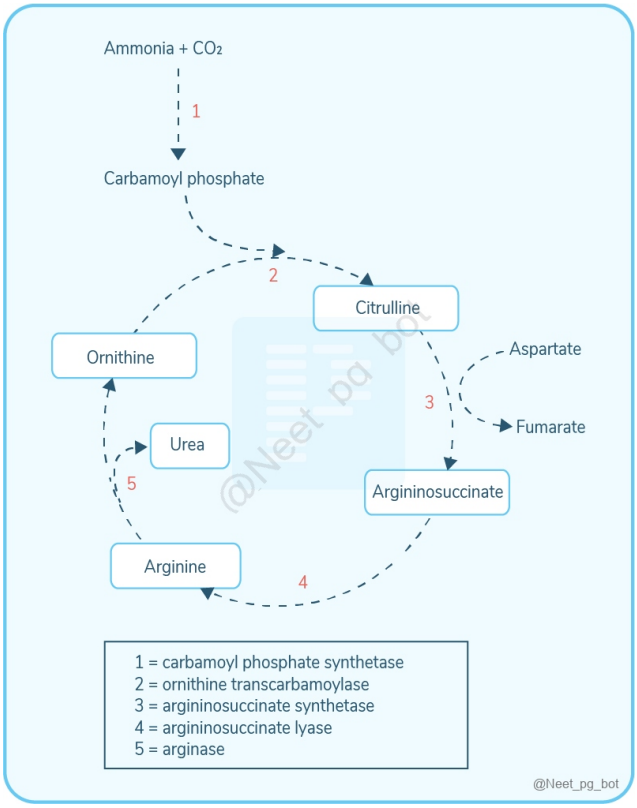
C. Uridine monophosphate (UMP) synthase

D. Hypoxanthine guanine phosphoribosyltransferase (HGPRT)

Correct Answer: A. Ornithine transcarbamylase (OTC)

■ *Listen to Audio 1*

Explanation: Hyperammonemia type 2 is an X-linked deficiency caused by a defect in ornithine transcarbamoylase (this reaction occurs in mitochondria), which forms citrulline in the Krebs cycle. Mothers with this condition may have hyperammonemia and avoid high-protein foods. Elevated blood, cerebrospinal fluid, and urine glutamine levels are due to increased glutamine synthesis in response to high ammonia levels. Orotic aciduria - Due to the channelling of carbamoyl phosphate into pyrimidine synthesis. Features OTC Deficiency UMP Synthase Deficiency Disorder Ornithine Transcarbamylase Deficiency Orotic Aciduria Metabolic pathway affected Urea cycle Pyrimidine synthesis pathway Enzyme deficiency Ornithine Transcarbamylase UMP Synthase Genetic inheritance X-linked recessive Autosomal recessive Main biochemical defect Impaired conversion of carbamoyl phosphate + ornithine → citrulline Impaired conversion of orotic acid → UMP Ammonia level Increased (hyperammonemia) Normal ammonia level Orotic acid in urine Increased Markedly increased Blood urea level Decreased Normal clinical features Vomiting, lethargy, cerebral edema, encephalopathy in neonates Megaloblastic anemia, growth retardation, developmental delay Treatment Protein restriction, ammonia-lowering therapy (e.g., sodium benzoate) Uridine supplementation



References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 287-288.

DM Vasudevan- Textbook of Biochemistry for Medical Students, 7th Edition, Pages 204, 206

↑ *Back to this question in Part A*

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Q4: Which enzyme in the histidine catabolism pathway facilitates incorporating the formimino group into the one-carbon pool, forming N5-formimino tetrahydrofolate? (Q845059)

A. Histidase

B. Urocanase

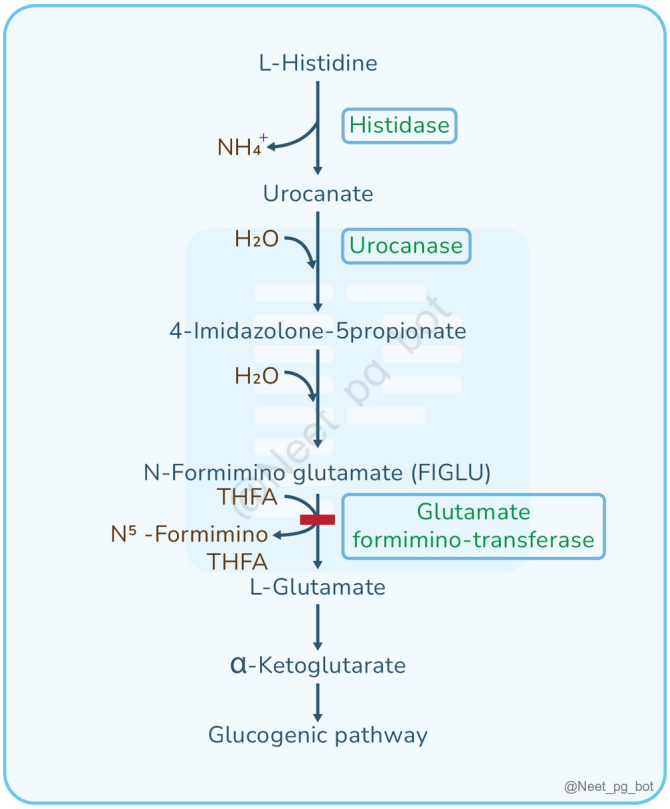
C. Glutamate formimino transferase

D. Glutamate dehydrogenase

Correct Answer: C. Glutamate formimino transferase

■ *Listen to Audio 1*

Explanation: Glutamate Formimino transferase catalyzes the transfer of the formimino group from formiminoglutamate (FIGLU) to THFA, forming N5-formimino THFA■. Catabolism of Histidine to α-ketoglutarate: Reaction Substrate Enzyme Product 1. Histidine Histidase (Option A) Urocanate 2. Urocanate Urocanase (Option B) Imidazole propionic acid 3. Imidazole propionic acid Hydrolase Formiminoglutamate (FIGLU) 4. FIGLU Glutamate Formimino transferase (Option C) L-Glutamate 5. Glutamate Glutamate dehydrogenase (Option D) α-Ketoglutarate The FIGLU test is a biochemical test used to detect folic acid deficiency by measuring the urinary excretion of Formiminoglutamic acid (FIGLU). The conversion of FIGLU to glutamate requires folic acid in the form of tetrahydrofolate (THF) which accepts the formimino group from FIGLU. If folate is deficient, this reaction cannot occur. As a result, FIGLU accumulates and is excreted in urine. It is used to diagnose megaloblastic anemia caused by folate deficiency from other deficiencies.



References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 292 - 293.

DM Vasudevan- Textbook of Biochemistry for Medical Students, 7th edition, Pages 244,207

↑ *Back to this question in Part A*

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Q5: In the kynurenine pathway of tryptophan catabolism, which of the following compounds directly contributes to the formation of N10-formyl tetrahydrofolate in the one-carbon pool? (Q845058)

A. Formyl kynurenine

B. Kynurenine

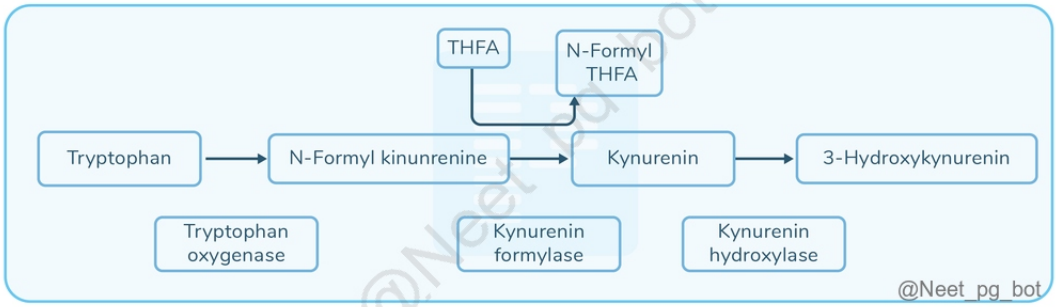
C. 3- Hydroxy kynurenine

D. THFA

Correct Answer: A. Formyl kynurenine

■ *Listen to Audio 1*

Explanation: Formyl kynurenine is a key intermediate in the kynurenine pathway. It is directly involved in transferring a formyl group, which leads to the production of N10-formyl THFA, an important one-carbon donor in nucleotide biosynthesis■. Tryptophan Catabolism and One-Carbon Pool Contributions Compound Role in Tryptophan Catabolism Contribution Formylkynurenine (Option A) Intermediate; releases formate for N10-formyl THFA Directly contributes to the formation of N10-formyl THFA Kynurenine (Option B) Intermediate; precursor for other metabolites Participates in the synthesis of other metabolites like quinolinic acid and nicotinic acid■. 3-Hydroxy kynurenine (Option C) Intermediate; precursor for other metabolites The product formed from formate derived from formylkynurenine THFA (Option D) Carrier of one-carbon units Not directly derived from tryptophan catabolism Note: The liver synthesises niacin from essential amino acids such as tryptophan, requiring 60mg of it to produce 1 mg of niacin (B2 and B6 are required for this synthesis).



References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 299.

↑ *Back to this question in Part A*

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Q6: A 62-year-old female patient presents with fatigue, pallor, and glossitis. Laboratory tests indicate macrocytic anaemia, and elevated homocysteine and methylmalonic acid levels. Given these findings, what is the most likely metabolic pathway disrupted by this patient's condition, leading to the observed anaemia? (Q845057)

A. Conversion of homocysteine to methionine

B. Reduction of folic acid to dihydrofolate

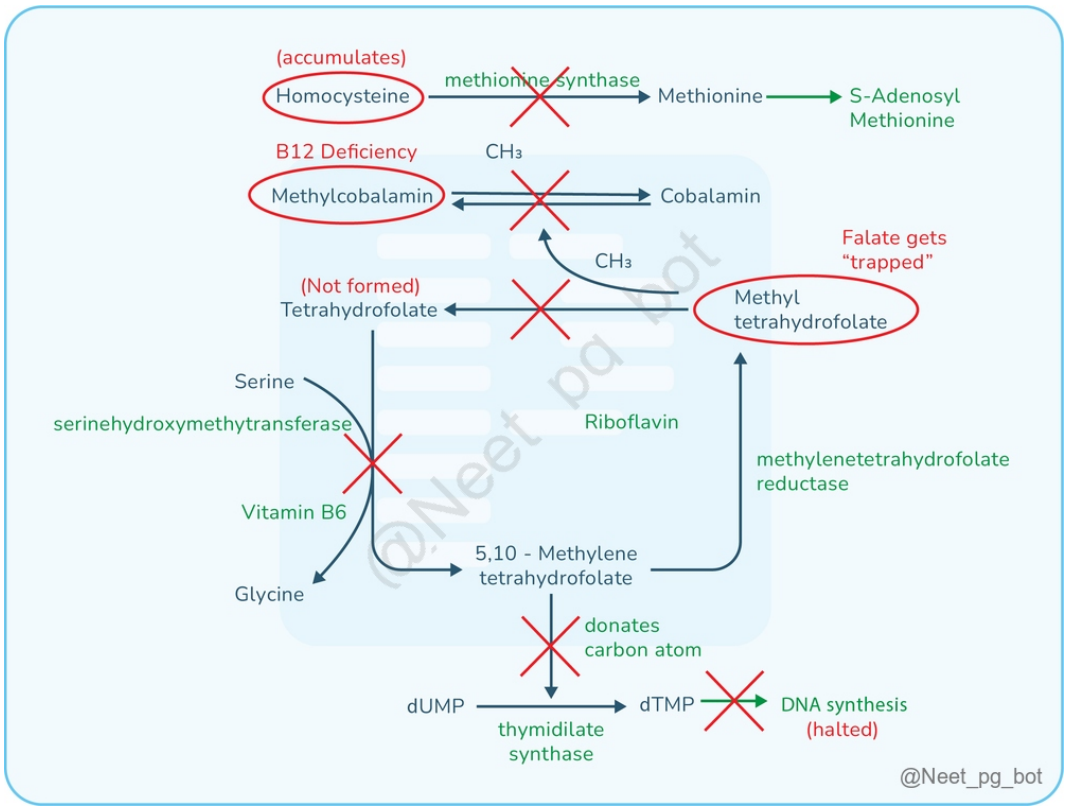
C. Deamination of histidine to formimino glutamate

D. Conversion of Phenylalanine to Tyrosine

Correct Answer: A. Conversion of homocysteine to methionine

■ *Listen to Audio 1*

Explanation: Macrocytic anaemia, elevated homocysteine and methylmalonic acid levels are consistent with Vitamin B12 deficiency. Conversion of homocysteine to methionine is disrupted in vitamin B12 deficiency. Vitamin B12 is a cofactor for methionine synthase, the enzyme responsible for converting homocysteine to methionine. This, in turn, forms S-adenosylmethionine, which is necessary for the synthesis of nitrogenous bases in DNA and RNA. In the absence of vitamin B12, the N5-methyltetrahydrofolate (N5-methyl THF) is trapped, leading to a functional folate deficiency and, consequently, macrocytic anaemia■. This " methyl folate trap " hypothesis explains why folate becomes metabolically inactive. Folate Trap: Reduction of folic acid to dihydrofolate (Option B) is catalyzed by dihydrofolate reductase and is unrelated to the vitamin B12-dependent processes. It is involved in the conversion of folic acid (the synthetic form) into its active form. Deamination of histidine to formimino glutamate (Option C) is related to folate metabolism but is more directly associated with diagnosing folate deficiency by the presence of FIGLU in the urine. Conversion of Phenylalanine to Tyrosine (Option D) is catalyzed by Phenylalanine hydroxylase, which does not require vitamin B12 as a cofactor.



References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 546.

↑ *Back to this question in Part A*

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Q7: Which of the following statements regarding one-carbon derivatives of tetrahydrofolic acid (THFA) and their associated enzymes and reactions is incorrect? (Q845056)

A. N5-formimino-THFA converts formiminoglutamate (FIGLU) to glutamate.

B. N5-methyl-THFA carries the methyl group for converting homocysteine to methionine.

C. N5N10-methylene-THFA is required to synthesise thymidylate (dTMP) from deoxyuridylate (dUMP).

D. N10-formyl-THFA transfers formyl groups in pyrimidine synthesis.

Correct Answer: D. N10-formyl-THFA transfers formyl groups in pyrimidine synthesis.

■ *Listen to Audio 1*

Explanation: N10-formyl-THFA transfers formyl groups in purine synthesis , forming C2 of the purine ring. One-Carbon Compounds: One-Carbon Derivatives of THFA Description Source Utilization N5-formyl-THFA and N10-formyl-THFA (Option D) Carry a formyl group (-CHO) Tryptophan Synthesis of purine nucleotides, forming C2 of the purine ring. N5-formimino-THFA (Option A) Carries a formimino group (-CH=NH) Histidine via FIGLU Involved in the conversion of formiminoglutamate (FIGLU) to glutamate, a step in histidine catabolism; Enzyme: Formiminoglutamate hydrolase N10-hydroxymethyl-THFA Carries a hydroxymethyl group (-CH2OH) Choline and betaine N5, N10-methylene-THFA (Option C) Carries a methylene group (-CH2-) Serine via serine hydroxymethyl transferase, glycine Glycine synthesis (conversion of serine to glycine), Thymidine synthesis (conversion of dUMP to dTMP); Enzyme: Thymidylate synthase N5-methyl-THFA (Option B) Carries a methyl group (-CH3) All one-carbon units ultimately form N5-methyl-THFA. Methionine synthesis (conversion of homocysteine to methionine); Enzyme: Methionine synthase Precursor for S-adenosylmethionine (SAM) for various transmethylation reactions.

References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 545.

DM Vasudevan- Textbook of Biochemistry for Medical Students, 7th edition, Pages 207,208.

↑ *Back to this question in Part A*

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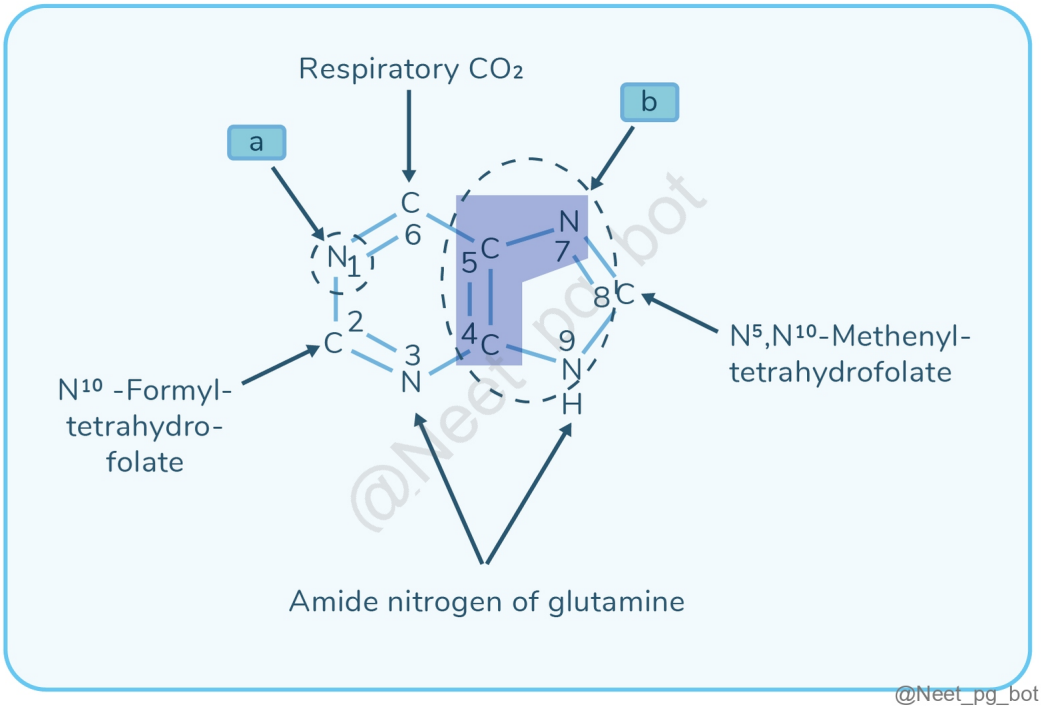
Q8: In the following figure, identify the sources of the marked components. (Q845055)

A. a- aspartate, b- glycine

B. a- glycine, b-aspartate

C. a- methionine, b-alanine

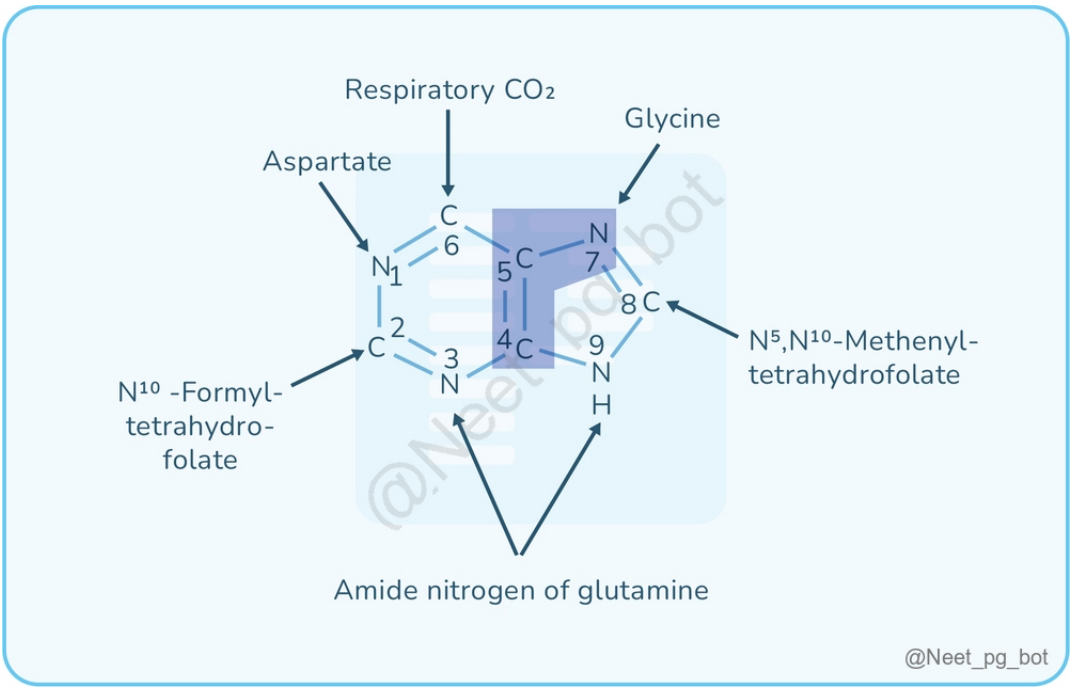
D. a- alanine, b- methionine



Correct Answer: A. a- aspartate, b- glycine

■ *Listen to Audio 1*

Explanation: The above-marked nitrogen atom is derived from the amino acid aspartate and the carbon atoms from glycine . The major site is the liver . Requires three amino acids - Glycine, Aspartate, Glutamine and the cofactor N10-formyl tetrahydrofolate (N10- formyl THF) for purine ring formation. The sources of each atom in a purine were found by feeding isotopic precursors of uric acid to pigeons. The C-2 atom is from formyl THF The C-8 atom is from N5N10 methenyl THF The C-6 atom is from respiratory CO2 . N1 is from aspartate N3 and N9 are from Glutamine C4, C5 and N7 are from Glycine Methionine and alanine have no relation to purine synthesis.



References:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7869020/

Harper's Illustrated Biochemistry, 32nd edition, Pages 338, 339

DM Vasudevan- Textbook of Biochemistry for Medical Students, 7th edition, Page 208

↑ *Back to this question in Part A*

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Q9: Which of the following diseases is caused by the deficiency of the enzyme ■ ALA synthase? (Q845054)

A. Porphyria cutanea tarda

B. X linked sideroblastic anaemia

C. Acute intermittent porphyria

D. Congenital erythropoietic porphyria

Correct Answer: B. X linked sideroblastic anaemia

■ *Listen to Audio 1*

Explanation: X-Linked Sideroblastic Anemia: Caused by ALA synthase deficiency , impairing heme synthesis in bone marrow and liver. First step: In mitochondria, glycine + succinyl-CoA → δ-ALA (via ALA synthase , PLP-dependent). Transporter: Glycine enters mitochondria via SLC25A38 . Enzyme Defect Name of the Disorder ALA synthase II X linked sideroblastic anaemia (Option B) ALA Dehydratase ALA dehydratase deficient porphyria (ADP) Porphobilinogen deaminase (Hydroxymethylbilane synthase) Acute intermittent porphyria (AIP) (Option C) Uroporphyrinogen III synthase Congenital erythropoietic porphyria (most severe) (Option D) Uroporphyrinogen decarboxylase defect Porphyria cutanea tarda (PCT) (Option A) Coproporphyrinogen oxidase Hereditary coproporphyria Protoporphyrinogen oxidase Variegate porphyria Ferrochelatase Erythropoietic protoporphyria (EPP)

References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 316, 317.

↑ *Back to this question in Part A*

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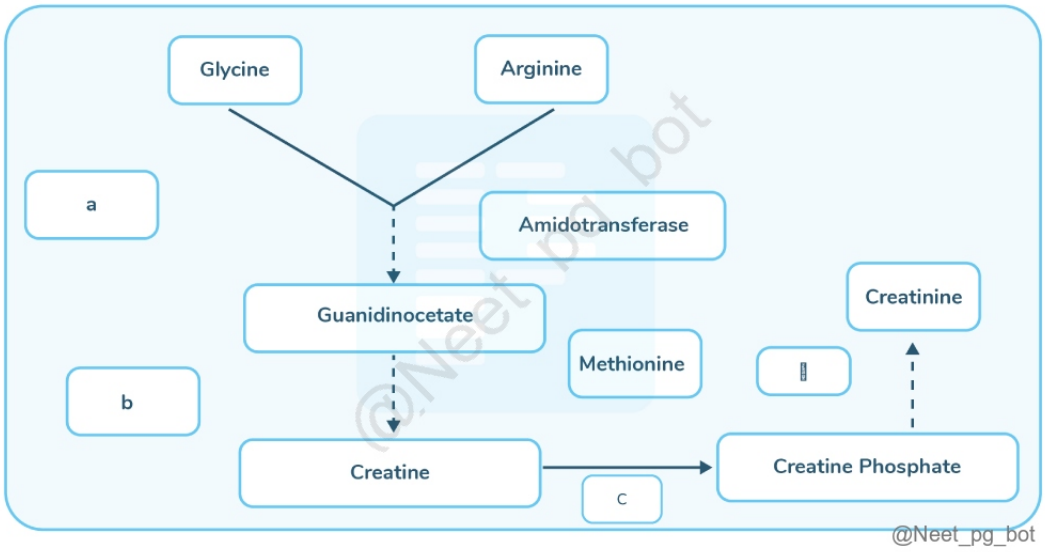
Q10: Based on the image given below, what are the sites at which the marked steps of creatinine synthesis occur? (Q845053)

A. a- brain, b- liver, c- kidney

B. a- kidney, b- liver, c- muscle

C. a- liver, b- kidney, c- muscle

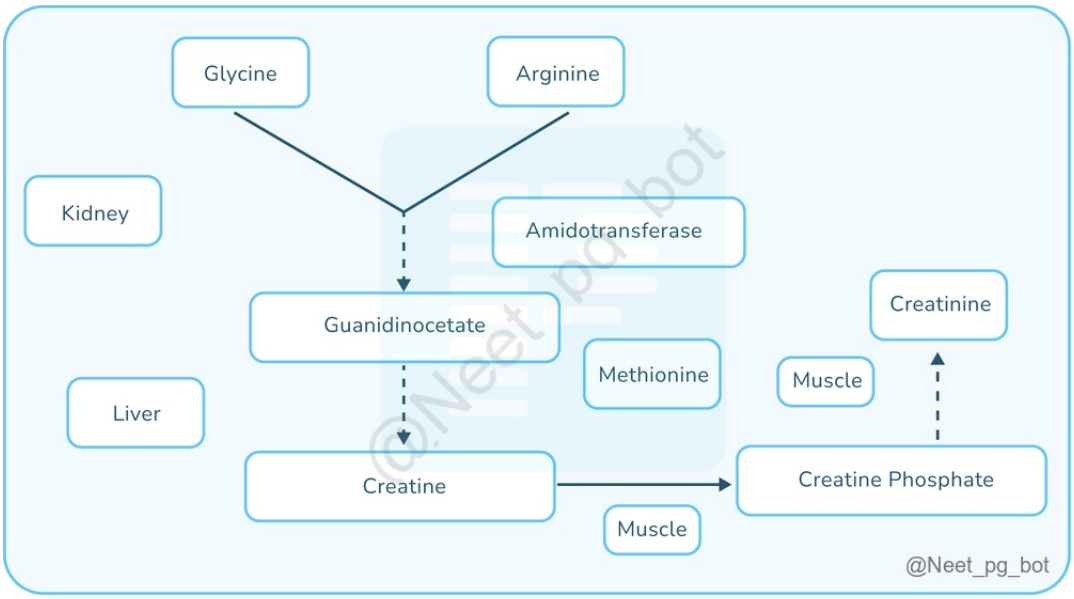
D. a- muscle, b- liver, c-kidney



Correct Answer: B. a- kidney, b- liver, c- muscle

■ *Listen to Audio 1*

Explanation: Creatinine synthesis requires the enzyme creatine kinase which is found in the liver, kidney and muscle. Steps Enzyme Site 1. The Amidino group of arginine is transferred to glycine to form guanidinoacetic acid. Amidotransferase Kidney (a) and pancreas 2. Guanidoacetic acid is Methylated by S-adenosyl methionine to form creatine. Methyl transferase Liver (b) 3. Creatine is phosphorylated to creatine phosphate. Creatine kinase Muscle, brain and liver. 4. Creatine phosphate may be converted to its anhydride, creatinine Non- enzymatic Muscle (c)



References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 311, 312.

DM Vasudevan- Textbook of Biochemistry for Medical Students, 7th edition, Pages 211.

↑ *Back to this question in Part A*

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Q11: Which of the following statement is not a known use of glycine in humans? (Q845052)

A. Precursor for metabolites like creatine, glutathione, and heme.

B. Enhances the quality of sleep and supports neurological functions.

C. Inhibits synthesis of bile acid, reducing lipid absorption.

D. Provide structural integrity for collagen structure.

Correct Answer: C. Inhibits synthesis of bile acid, reducing lipid absorption.

■ *Listen to Audio 1*

Explanation: Uses of Glycine 1) Metabolic Functions : Precursor for metabolites such as creatine, glutathione, heme , purines, and porphyrins. (Option A) Vital for protein synthesis. Plays a crucial role in collagen structure by occupying every third position in the collagen triple helix. (Option D) 2) Health and Disease Prevention : Dietary supplementation can prevent or treat metabolic disorders in patients with cardiovascular diseases, inflammatory diseases, obesity, cancers, and diabetes. Supports overall health, growth, and well-being in humans. 3) Neurological and Sleep Benefits : Acts as a neurotransmitter in the central nervous system, influencing food intake, behaviour, and body homeostasis. Enhances sleep quality and neurological functions. (Option B) 4) Immune Function : Regulates immune responses by altering intracellular calcium levels. Modulates the production of superoxide and cytokines. 5) Digestive Health : Facilitates the conjugation of bile acids. Aid in the absorption and digestion of lipid-soluble vitamins and lipids. (Option C)

References:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5350494/

↑ *Back to this question in Part A*

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Q12: The glycine cleavage system in the body produces CO2, ammonia and a one-carbon molecule. Which of the following is a subunit in the system? (Q845051)

A. Tetrahydrofolate

B. Lipoamide

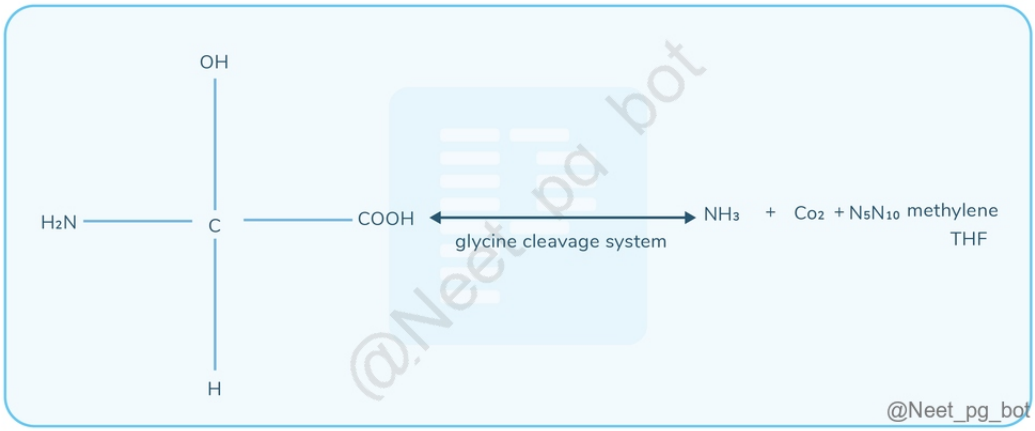
C. Tetrahydrofolate-dependent aminomethyltransferase (THFAAMT)

D. Pyridoxal phosphate

Correct Answer: C. Tetrahydrofolate-dependent aminomethyltransferase (THFAAMT)

■ *Listen to Audio 1*

Explanation: The subunit of the glycine cleavage system here is THFAAMT, and the others are the coenzymes that participate in the system. The glycine cleavage system takes place in the liver mitochondria. In this system, the simple amino acid glycine is split into ammonia and CO2, and the remaining methylene group (–CH2–) is transferred by Tetrahydrofolate and converted into N5N10 methylene tetrahydrofolate. Subunits of the glycine cleavage system Co-enzymes of glycine cleavage system Tetrahydrofolate dependant aminotransferase (THFAAMT) (Option C) THF (Option A) Pyridoxal phosphate-dependant (PLP) glycine decarboxylase Pyridoxal phosphate (PLP) (Option D) Lipoamide dependent dehydrogenase Lipoamide (Option B) Hydrogen or electron transport system



References:

Harper's Illustrated Biochemistry, 32nd edition, Page 293

↑ *Back to this question in Part A*

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Q13: A 6-month-old infant presented with lethargy, vomiting, and irritability. Blood tests revealed elevated ammonia levels. The infant was diagnosed with a urea cycle disorder. Which of the following medications, acting as ammonia-scavenging agents, would be most effective in reducing the infant's blood ammonia levels? (Q845050)

A. Phenylacetate

B. N-acetyl glutamate

C. Lactulose

D. Neomycin

Correct Answer: A. Phenylacetate

■ *Listen to Audio 1*

Explanation: Reducing Blood Ammonia Levels: 1. Ammonia-Scavenging Agents: Sodium benzoate: Conjugates with glycine to form hippuric acid , excreted in urine. This lowers glycine levels, promoting ammonia use for new glycine synthesis. Phenylacetate / Phenylbutyrate: Combine with glutamine to form phenylacetylglutamine , excreted in urine. This depletes glutamine and indirectly uses two ammonia molecules per reaction. Phenylbutyrate acts as a prodrug of phenylacetate. 2. Protein Breakdown Control: Adequate caloric intake prevents excessive protein catabolism and ammonia release. 3. Gut Ammonia Reduction: Neomycin: Suppresses urease-producing gut bacteria. (Option D) Lactulose: Converts ammonia to non-absorbable ammonium (NH■■), reducing systemic ammonia levels. (Option C) N-acetyl glutamate is used to treat hyperammonemia but does not

directly scavenge ammonia . (Option B) N-acetylglutamate is the essential allosteric activator of Carbamoyl Phosphate Synthetase I (CPS-I) which allows ammonia to be converted into carbamoyl phosphate, required for urea synthesis. In N-Acetylglutamate Synthase Deficiency, carglumic acid, a synthetic analogue of NAG is used.

References:

https://www.uptodate.com/contents/urea-cycle-disorders-management

https://www.ncbi.nlm.nih.gov/books/NBK547972/

↑ *Back to this question in Part A*

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Q14: A 45-year-old man presents to the clinic with complaints of fatigue and abdominal discomfort. Blood tests reveal elevated levels of alanine aminotransferase (ALT) and aspartate aminotransferase (AST). The physician suspects liver disease. Which of the following statements best describes the role of transamination reactions in amino acid metabolism? (Q843343)

A. Transfer an amino group to one keto acid, producing new amino and keto acids.

B. Specific to lysine, threonine, and proline.

C. Elevated AST and ALT indicate myocardial infarction.

D. Occur exclusively in the liver mitochondria.

Correct Answer: A. Transfer an amino group to one keto acid, producing new amino and keto acids.

■ *Listen to Audio 1*

Explanation: Transamination reactions transfer an amino group from an amino acid to a keto acid, catalysed by aminotransferases with Pyridoxal Phosphate (PLP) as a coenzyme. Transamination Process Transfer of an α-amino group from an amino acid to one keto acid, producing a new α-keto acid and a new amino acid. (Option A) The first step is the catabolism of most amino acids. All amino acids, except Proline, hydroxyproline, threonine, and lysine, participate in transamination . (Option B) Biological Importance Amino acid balance: Transfers amino groups to form non-essential AAs. Ammonia disposal: Channels amino groups into glutamate for deamination and urea excretion. Glutamate's Role α-Ketoglutarate: Accepts amino groups to form glutamate (cytoplasmic transamination). In mitochondria, glutamate undergoes oxidative deamination back to α-ketoglutarate, regenerating the amino acceptor. (Option D) Enzymes Aminotransferases (Transaminases): Catalyse the transfer of amino groups between carbon skeletons. They are not coenzymes. (Option C) Alanine Aminotransferase (ALT): Transfers amino group from alanine to α-ketoglutarate, forming pyruvate and glutamate. Aspartate Aminotransferase (AST): Transfers amino groups from glutamate to oxaloacetate, forming aspartate used in the urea cycle. Coenzyme Pyridoxal Phosphate (PLP): Required by all aminotransferases, derived from vitamin B6. Reversibility Transamination reactions are reversible, with equilibrium constants near 1.

References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 282,283,291

DM Vasudevan- Textbook of Biochemistry for Medical Students, 7th edition, Page 201

↑ *Back to this question in Part A*

@Neet_pg_bot

Q15: Which of the following is not a source of glycine? (Q845048)

A. Serine

B. Threonine

C. choline

D. Lysine

Correct Answer: D. Lysine

■ *Listen to Audio 1*

Explanation: Serine, Threonine and Alanine are sources of Glycine, whereas Lysine is not. Glycine Simple, non-essential amino acid. Acts as a precursor for metabolites like creatine, glutathione, heme, purines, and porphyrins. Vital for protein synthesis, collagen structure , neurotransmission, immune response, and lipid digestion. Sources of Glycine: Serine : Converted to glycine via serine hydroxymethyltransferase (SMT), present in mitochondria and cytoplasm. (Option A) Threonine : Converted to glycine by threonine aldolase. (Option B) Choline : Converted to glycine via sarcosine and betaine pathways. Glycine Synthase : Synthesises glycine from CO■, NH■■, and a one-carbon unit. This multi-enzyme complex requires NAD, lipoamide, tetrahydrofolic acid, and pyridoxal phosphate. This is a reversal of the glycine cleavage system. Glycine Aminotransferase : Catalyses glycine synthesis from glyoxylate and glutamate or alanine. (Option C)

References:

Harper's Illustrated Biochemistry, 32nd edition, Page 275

DM Vasudevan- Textbook of Biochemistry for Medical Students, 7th edition, Page 211

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5350494/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q16: A 45-year-old male with microcytic anaemia is found to have a deficiency of a vitamin essential for the rate-limiting step of heme synthesis. Which vitamin is most likely deficient? (Q843442)

A. Vitamin B6

B. Vitamin B12

C. Vitamin C

D. Vitamin D

Correct Answer: A. Vitamin B6

■ *Listen to Audio 1*

Explanation: Heme biosynthesis begins in mitochondria with the condensation of succinyl Co-A and glycine. This reaction produces 5'-aminolevulinic acid (ALA), a key heme intermediate. The enzyme aminolevulinic acid synthase (ALAS) requires pyridoxal phosphate (Vitamin B6) to catalyze this step. This reaction is the rate-limiting step in heme synthesis.

References:

https://www.ncbi.nlm.nih.gov/books/NBK537329/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: A 5-month-old boy presents with failure to thrive, developmental delay, and intermittent episodes of vomiting and lethargy. The episodes coincide with the introduction of solid foods. Blood tests reveal elevated ammonia levels and high levels of ornithine in blood. Urine analysis shows increased excretion of homocitrulline. What is the most likely diagnosis for this patient? (Q843441)

A. Citrullinemia

B. Argininosuccinate aciduria

C. HHH Syndrome

D. Argininemia

Correct Answer: C. HHH Syndrome

■ *Listen to Audio 1*

Explanation: HHH Syndrome Explanaion: This child with hyperammonemia triggered after solid food introduction is most likely to have HHH syndrome. Hyperornithinemia-Hyperammonemia-Homocitrullinuria Syndrome Inheritance Autosomal recessive Defective Gene ORC1 Defective Protein Mitochondrial Ornithine Transporter: This transporter moves ornithine into the mitochondria and citrulline out of the mitochondria. Mechanism Prevents ornithine from entering the mitochondria, which is necessary for the urea cycle. The buildup of ammonia in the blood (hyperammonemia). Ornithine also accumulates in the cytoplasm (hyperornithinemia). Carbamoyl phosphate reacts with lysine to form homocitrulline. Symptoms Failure to thrive. Developmental delay. Intermittent episodes of vomiting and lethargy (triggered by protein intake). Seizures. Intellectual disability. Diagnosis Elevated blood ammonia Elevated blood ornithine Elevated urinary homocitrulline. Treatment Low-protein diet Nitrogen scavenger medications Liver transplant in severe cases. Citrullinemia (Option A) leads to the accumulation of citrulline, which would be elevated in blood tests, unlike in this scenario. Argininosuccinate aciduria (Option B) causes the characteristic symptom of trichorrhexis nodosa (brittle, easily broken hair), which is not mentioned in this scenario. Argininemia (Option D) involves hyperammonemia . Compared to other urea cycle disorders, its presentation is typically delayed, appearing around 2 years of age, not at 5 months.

References:

Lippincott Illustrated Reviews: Biochemistry, 7th Edition, Page 287, 288.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q18: A 5-month-old male infant with a history of vomiting, irritability, and developmental delay. The onset of symptoms coincided with the introduction of formula feed. Physical examination revealed hepatomegaly and signs of encephalopathy. Blood tests revealed elevated ammonia levels and high glutamine, uracil, and orotic acid concentrations. Citrulline levels were normal. What is the most likely diagnosis? (Q843352)

A. Type 1 Hyperammonemia

B. Type 2 Hyperammonemia

C. Citrullinemia

D. Argininosuccinate aciduria

Correct Answer: B. Type 2 Hyperammonemia

■ *Listen to Audio 1*

Explanation: The infant's symptoms and laboratory findings with the timing of symptom onset after the introduction of the formula are highly suggestive of Type 2 Hyperammonemia . Feature Type 1 Hyperammonemia Type 2 Hyperammonemia Defective Enzyme Carbamoyl Phosphate Synthetase I (CPS1) Ornithine Transcarbamylase (OTC) Inheritance Autosomal recessive X-linked recessive Mechanism The inability to

synthesize carbamoyl phosphate blocks the urea cycle. Accumulation of carbamoyl phosphate in the mitochondria, which then spills over into the cytoplasm. Metabolic Consequences Elevated ammonia and glutamate Normal orotic acid and uracil Low citrulline. (Option A) Elevated ammonia, glutamate, and glutamine. High uracil and orotic acid (excess carbamoyl phosphate enters pyrimidine synthesis). Normal citrulline. Clinical Presentation Symptoms coincide with increased protein intake. CNS : Lethargy, drowsiness, coma, Convulsions, vomiting, respiratory stimulation, hyperventilation. Similar to Type 1. Hepatomegaly. Citrullinemia and Argininosuccinate aciduria (Option C and D) would result in elevated citrulline levels, contradicting the normal citrulline finding in this scenario.

References:

Lippincott Illustrated Reviews: Biochemistry, Edition 7, Pages 287-288.

↑ *Back to this question in Part A*

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Q19: Which of the following statements about carbamoyl phosphate synthetases is correct? (Q843351)

A. CPS-I is activated by PRPP, and CPS-II is inhibited by UTP.

B. CPS-I is located in cytosol, and CPS-II in mitochondria.

C. CPS-I is involved in urea cycle, and CPS-II in pyrimidine synthesis.

D. Source of nitrogen for CPS-I is glutamine, and CPS-II is ammonia.

Correct Answer: C. CPS-I is involved in urea cycle, and CPS-II in pyrimidine synthesis.

■ *Listen to Audio 1*

Explanation: CPS-I is involved in the urea cycle , whereas CPS-II is involved in pyrimidine synthesis . Aspect Carbamoyl Phosphate Synthetase-I (CPS-I) Carbamoyl Phosphate Synthetase-II (CPS-II) Site (Option B) Mitochondria Cytosol Pathway involved (Option C) Urea cycle Pyrimidine synthesis Source of Nitrogen (Option D) Ammonia ■-Amide group of glutamine Positive effector (Option A) N-acetyl glutamate Phosphoribosyl pyrophosphate (PRPP) Inhibitor Nil Uridine triphosphate(UTP)

References:

Lippincott Illustrated Reviews: Biochemistry, Edition 7,Chapter 22: Nucleotide Metabolism

DM Vasudevan- Textbook of Biochemistry for Medical Students, 7th edition, Page 203

https://pubmed.ncbi.nlm.nih.gov/9748664/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q20: Which of the following is true about the rate-limiting step of the urea cycle? (Q843350)

A. It involves the formation of citrulline from ornithine.

B. It is catalysed by carbamoyl phosphate synthetase I (CPS I).

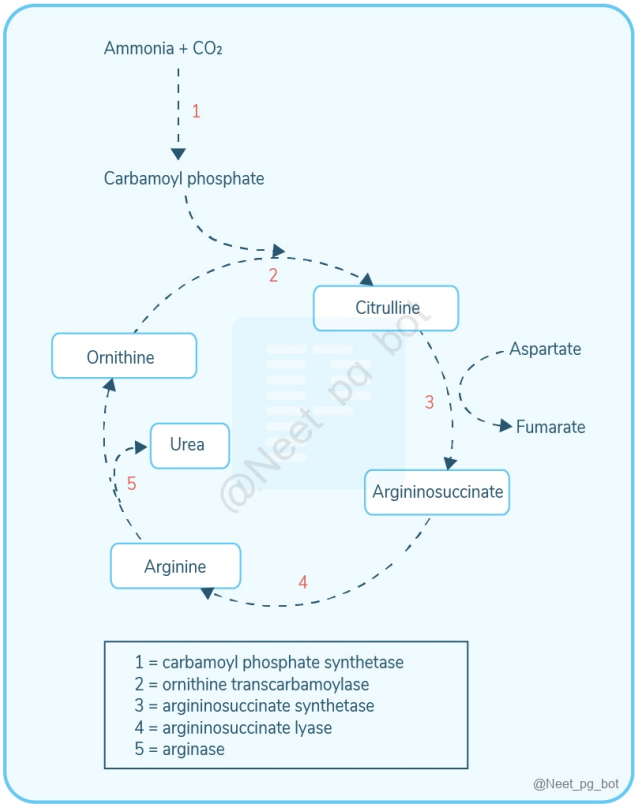
C. It is regulated by argininosuccinate synthetase.

D. It is controlled by the enzyme argininosuccinate lyase.

Correct Answer: B. It is catalysed by carbamoyl phosphate synthetase I (CPS I).

■ *Listen to Audio 1*

Explanation: The carbamoyl phosphate synthetase I (CPS I) reaction is the rate-limiting step in the urea cycle. The urea cycle is a cycle of biochemical reactions that produce urea from ammonia. It is also known as the Krebs-Henseleit urea cycle or the Ornithine cycle. It starts in the mitochondria of hepatocytes and ends in the cytoplasm . Reactions in the Urea Cycle Step Reaction Details Enzyme Involved and Location Key Points Carbamoyl Phosphate Formation Formation of carbamoyl phosphate from ammonia and CO■ using ATP. Carbamoyl Phosphate Synthetase I (CPS I) Mitochondria The rate-limiting step of the urea cycle. Irreversible and allosterically regulated by N-acetyl glutamate (NAG). Citrulline Formation (Option A) Transfer of carbamoyl phosphate to ornithine to form citrulline. Ornithine Transcarbamylase (OTC) Mitochondria Citrulline moves from mitochondria to cytosol for further processing in the urea cycle. Argininosuccinate Formation (Option C) Citrulline combines with aspartate to form argininosuccinate. Argininosuccinate Synthetase Cytoplasm Requires ATP. Links the urea cycle with the TCA cycle via aspartate. Argininosuccinate Cleavage (Option D) Cleavage of argininosuccinate into arginine and fumarate. Argininosuccinate Lyase Cytoplasm Fumarate enters the TCA cycle, providing a connection between the urea cycle and other metabolic pathways. Arginine Cleavage to Ornithine and Urea Hydrolysis of arginine to produce ornithine and urea. Arginase Cytoplasm Ornithine is recycled back into the urea cycle. Urea is excreted by the kidneys, with some urea also entering the intestine. Fate of Urea Urea is transported to the kidneys, filtered, and excreted in urine. Kidneys/ Intestine Urea excretion is the final step, removing excess nitrogen from the body.



References:

DM Vasudevan- Textbook of Biochemistry for Medical Students, 7th Edition, Pages 203, 204, 205.

https://www.ncbi.nlm.nih.gov/books/NBK513323/

https://www.ncbi.nlm.nih.gov/books/NBK27982/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q21: Which of the following statements about ammonia transport is correct? (Q843349)

A. Glutamine moves ammonia from liver to brain.

B. Alanine transports ammonia from liver to muscles.

C. Glutamine and alanine transport ammonia to the liver.

D. Alanine does not participate in ammonia transport.

Correct Answer: C. Glutamine and alanine transport ammonia to the liver.

■ *Listen to Audio 1*

Explanation: Ammonia is transported in a non-toxic form in the circulation as glutamine or alanine. Glutamine is formed from glutamate and ammonia by glutamine synthetase, primarily in skeletal muscle, liver, and CNS. Glutamine transports ammonia from the brain and intestine to the liver. (Option A) Glutamine is transported in the blood to the liver, where it is cleaved by glutaminase to glutamate and ammonia. The glutamate is oxidatively deaminated to ammonia and α-ketoglutarate by glutamate dehydrogenase (GDH), and the ammonia is converted to urea. Alanine is the transport form of ammonia from muscle to the liver. (Option B) Alanine transports ammonia to the liver via the glucose-alanine cycle. (Option D) Alanine is transported in the blood to the liver, where it is transaminated by alanine transaminase(ALT) to pyruvate. The pyruvate is used to synthesise glucose, which can enter the blood and be used by muscles. The glutamate product of ALT can be deaminated by GDH, generating ammonia. Thus, both alanine and glutamine carry ammonia to the liver. Transporter Formation Process Source Destination Mechanism/Role Glutamine Formed from glutamate and ammonia by glutamine synthetase . Skeletal muscle, liver, CNS Liver, kidneys Glutamine Transport: Carries ammonia from brain and intestine to liver; glutaminase releases NH■ and glutamate, which GDH further deaminates—ammonia enters the urea cycle . Alanine Formed from pyruvate and ammonia in muscles during the glucose-alanine cycle . Muscles Liver Alanine Cycle: Transports ammonia from muscle to liver; ALT converts alanine → pyruvate + glutamate. Pyruvate forms glucose, while glutamate releases NH■ for the urea cycle.

References:

Lippincott Illustrated Reviews: Biochemistry, Edition 7, Chapter 19: Amino Acids: Nitrogen Disposal

↑ *Back to this question in Part A*

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Q22: A 45-year-old man with chronic liver disease is noted to have elevated blood ammonia on laboratory testing. Which of the following is least likely to result from ammonia neurotoxicity? (Q843348)

A. Slurred speech

B. Tremors

C. Muscle rigidity

D. Blurred vision

Correct Answer: C. Muscle rigidity

■ *Listen to Audio 1*

Explanation: Elevated ammonia crosses the blood–brain barrier and is detoxified to glutamine in astrocytes. Excess glutamine increases osmotic pressure , leading to astrocyte swelling, cerebral edema, and impaired neurotransmission . Clinical features of ammonia neurotoxicity: CNS dysfunction manifests as tremors, slurred speech, drowsiness, incoordination, visual disturbances , and in severe cases, coma or death . Rigidity involves

extrapyramidal/motor pathways , which are not primarily affected by ammonia. Muscle tone is usually preserved in hyperammonemia, helping differentiate it from conditions like Parkinsonism or tetanus.

References:

Harper's Illustrated Biochemistry, 32nd edition, Page 162

Lippincott Illustrated Reviews: Biochemistry, Edition 7, Page 741.

DM Vasudevan- Textbook of Biochemistry for Medical Students, 7th edition, Page 207

↑ *Back to this question in Part A*

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Q23: Which of the following pathways is primarily involved in the detoxification of ammonia in the brain? (Q843347)

A. Conversion of ammonia to urea in neurons

B. Direct excretion of ammonia through the blood-brain barrier

C. Conversion of ammonia to glutamine via glutamine synthetase in astrocytes

D. Storage of ammonia in neurons as glutamate

Correct Answer: C. Conversion of ammonia to glutamine via glutamine synthetase in astrocytes

■ *Listen to Audio 1*

Explanation: Glutamine synthetase in astrocytes converts glutamate and ammonia to glutamine, effectively detoxifying ammonia in the brain. Ammonia Management in Neurons: Ammonia can be neurotoxic if accumulated. Glutamine Synthesis: Produced in neurons as a byproduct of amino acid metabolism and neurotransmitter turnover. Ammonia reacts with α-ketoglutarate and NADH to form glutamate, catalyzed by glutamate dehydrogenase. α-ketoglutarate + NH 3 ■ ■+ NADH → Glutamate + NAD + + H2■O Glutamate is converted to glutamine by glutamine synthetase, adding another ammonia molecule. Glutamine synthetase is predominantly found in astrocytes. Glutamate + NH 3 ■ + ATP → Glutamine + ADP + Pi This conversion reduces ammonia toxicity by storing it in a non-toxic form. The fate of glutamine: Release into the Bloodstream: Glutamine is released into the extracellular space and enters the bloodstream. This transport helps maintain nitrogen balance and prevents ammonia accumulation. Transport to the Liver: Glutamine carries ammonia from the brain to the liver, where it is deaminated to release ammonia. Ammonia enters the urea cycle in the liver for detoxification. Importance of Glutamine in Neuronal Health: Acts as a non-toxic ammonia carrier, preventing neurotoxicity. Serves as a precursor for neurotransmitters like GABA , maintaining neurotransmitter balance.

References:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8244593/

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4133642/

https://www.sciencedirect.com/science/article/pii/S019701862030200X

↑ *Back to this question in Part A*

@Neet_pg_bot

Q24: Which of the following reactions is not catalysed by glutamate dehydrogenase (GDH)? (Q843346)

A. Conversion of α-ketoglutarate and ammonia to glutamate

B. Oxidative deamination of glutamate

C. Formation of aspartate from oxaloacetate and ammonia

D. None of the above

Correct Answer: C. Formation of aspartate from oxaloacetate and ammonia

■ *Listen to Audio 1*

Explanation: The formation of aspartate from oxaloacetate and ammonia is catalyzed by aspartate transaminase , not GDH. In the liver, ammonia transforms into the less toxic form, urea, using glutamate and the enzyme glutamate dehydrogenase. Location: Found exclusively in liver mitochondria. Reactions Catalyzed by Glutamate Dehydrogenase: Oxidative Deamination (Conversion of Glutamate to α-Ketoglutarate) L-Glutamate + NAD + → α-Ketoglutarate + NH 3 ■ + NADH + H + Catalyzed by: GDH (Option A) Coenzyme: NAD■ Reductive Amination (Synthesis of Glutamate from α-Ketoglutarate) α-Ketoglutarate + NH 3 ■ + NADPH + H + → L-Glutamate + NADP + Catalyzed by: GDH (Option B) Coenzyme: NAD■ Regulation: Activation: ADP activates GDH. Inhibition: GTP, ATP, and NADH allosterically inhibit GDH.

References:

Harper's Illustrated Biochemistry, 32nd edition, Page 283

DM Vasudevan- Textbook of Biochemistry for Medical Students, 7th edition, Page 202

↑ *Back to this question in Part A*

@Neet_pg_bot

Q25: Which of the following pairs of amino acids do not undergo transamination reactions? (Q843345)

A. Serine and threonine

B. Lysine and threonine

C. Proline and alanine

D. Hydroxyproline and asparagine

Correct Answer: B. Lysine and threonine

■ *Listen to Audio 1*

Explanation: Lysine and Threonine: These amino acids do not undergo typical transamination. Lysine has an epsilon-amino group that can interfere with the transamination mechanism, and threonine undergoes deamination rather than transamination. Transamination is essential for amino acid catabolism and urea formation. Most amino acids participate in transamination reactions. Exceptions: Amino Acid Reason Proline Has a secondary amino group within its pyrrolidine ring structure. It cannot participate in standard transamination due to its structural characteristics. Hydroxyproline Derived from proline through post-translational modification; shares the same structural limitation. Lysine Has an epsilon-amino group in its side chain, which can form Schiff bases, potentially interfering with transamination. Threonine Loses its α-amino group through deamination rather than transamination. Direct transamination does not occur; instead, it is converted to alpha-ketobutyric acid.

References:

DM Vasudevan- Textbook of Biochemistry for Medical Students, 7th edition, Pages 24-25;215;226

↑ *Back to this question in Part A*

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Q26: A 50-year-old woman presents with confusion and lethargy. Blood tests show elevated levels of ammonia, suggesting hyperammonemia. Further tests indicate liver dysfunction. Which of the following best describes the process of oxidative deamination in amino acid metabolism? (Q843344)

A. Removal of an amino group from an amino acid, resulting in the corresponding keto acid and ammonia.

B. Involves only transamination reactions in the cytoplasm.

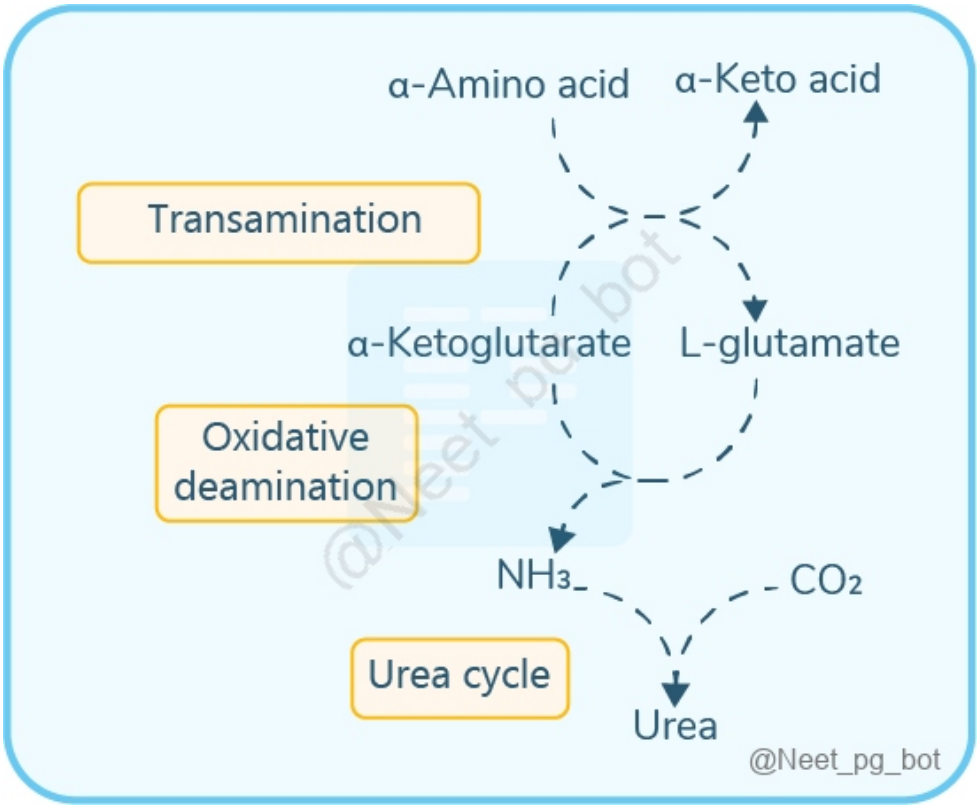
C. Generates ammonia, which is directly excreted by the kidneys.

D. Predominantly occurs in the liver cytoplasm, producing glutamine.

Correct Answer: A. Removal of an amino group from an amino acid, resulting in the corresponding keto acid and ammonia.

■ *Listen to Audio 1*

Explanation: Oxidative deamination removes the alpha-amino group from an amino acid, producing a keto acid and ammonia. This reaction, catalysed by glutamate dehydrogenase in liver mitochondria, is essential for ammonia detoxification and nitrogen balance. The ammonia formed is detoxified in the form of glutamate. The ammonia generated is processed through the urea cycle for excretion. (Option C) Oxidative Deamination: Process: Removes alpha-amino group from an amino acid, producing a keto acid and ammonia. Common Amino Acid: Glutamic acid. Enzyme: Glutamate dehydrogenase (GDH). Transdeamination: Step 1: Transamination (all organs) in cytoplasm forms glutamate. (Option D) Step 2 (Oxidative decarboxylation): In liver mitochondria, GDH removes the amino group from glutamate, producing alpha-ketoglutarate and ammonia. (Option B) Significance: It ensures efficient ammonia disposal, funnelling ammonia into the liver for detoxification via the urea cycle. Minor Deamination Pathways: Use L- and D-amino acid oxidases ; non-oxidative forms release ammonia directly. L-Amino Acid Oxidase: Acts in kidneys, producing NH■ and H■O■ ; catalase decomposes H■O■ to prevent ROS damage.



References:

Harper's Illustrated Biochemistry, 32nd edition, Page 282

DM Vasudevan- Textbook of Biochemistry for Medical Students, 7th edition, Page 202

↑ *Back to this question in Part A*

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• [04_Amino Acids and Proteins Chemistry] 015 Amino Acid Metabolic Disorder - I

« Back to Folder Directory

[04_Amino Acids and Proteins Chemistry] 015 Amino Acid Metabolic Disorder - I — Quick Jump

Q1 (p. 796)	*Q2 (p. 797)*	*Q3 (p. 798)*	*Q4 (p. 799)*	*Q5 (p. 800)*	*Q6 (p. 800)*
Q7 (p. 802)	*Q8 (p. 803)*	*Q9 (p. 804)*	*Q10 (p. 805)*	*Q11 (p. 806)*	*Q12 (p. 807)*
Q13 (p. 808)	*Q14 (p. 809)*	*Q15 (p. 810)*	*Q16 (p. 812)*	*Q17 (p. 812)*	*Q18 (p. 813)*
Q19 (p. 815)	*Q20 (p. 816)*	*Q21 (p. 817)*	*Q22 (p. 818)*	*Q23 (p. 819)*	*Q24 (p. 820)*
Q25 (p. 821)	*Q26 (p. 822)*	*Q27 (p. 823)*			

Q1: A 5-year-old boy is brought to recurrent kidney stones and abdominal pain. Urinalysis reveals hexagonal crystals, and a 24-hour urine test shows elevated levels of cystine. Which condition is the most likely diagnosis? (Q844923)

A. Cystinuria

B. Cystinosis

C. Hyperoxaluria

D. Nephrocalcinosis

Correct Answer: A. Cystinuria

■ *Listen to Audio 1*

Explanation: Cystinuria Explantion: Cystinuria is caused by the defect of a transporter that reabsorbs cysteine, ornithine, lysine and arginine in PCT and it causes recurrent kidney stones and hexagonal cystine crystals Cystinosis (Option B) is a lysosomal storage disorder caused by a defect in the transport protein cystinosin, leading to cystine accumulation within cells and formation of crystals in various tissues, including the cornea, causing photophobia. This condition also results in kidney damage, causing symptoms like polyuria and polydipsia, and recurrent kidney stones. The presence of characteristic crystals in the cornea on slit-lamp examination and hexagonal crystals in the urine supports the diagnosis of cystinosis. Hyperoxaluria (Option C) is incorrect because it leads to calcium oxalate stones (not cystine stones) and does not cause corneal deposits or Fanconi syndrome. Nephrocalcinosis (Option D) is incorrect because it refers to calcium deposition in the kidney and does not explain cystine crystals, Fanconi syndrome, or corneal involvement .



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 294, 295

↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: A 10-year-old child presents with an acute stroke. He is tall and thin with long fingers. Ocular examination shows downward dislocation of the lens. The most likely diagnosis is: (Q885184)

A. Marfan syndrome

B. Homocystinuria

C. Ehlers-Danlos syndrome

D. Osteogenesis imperfecta

Correct Answer: B. Homocystinuria

Explanation: Homocystinuria (most commonly due to cystathionine β-synthase deficiency) classically mimics Marfan syndrome in body habitus but is distinguished by severe thromboembolic tendency and the direction of lens dislocation . Clinical Features: Marfanoid habitus: Tall, thin build with arachnodactyly and chest wall deformities - similar to Marfan syndrome. Ectopia lentis (lens dislocation): In homocystinuria, the lens is displaced downward and inward. In Marfan syndrome, the lens is displaced upward and outward. Vascular risk: Markedly elevated homocysteine causes endothelial injury and a hypercoagulable state, leading to early arterial and venous thrombosis, including stroke or myocardial infarction in children. Neurological features: Intellectual disability / developmental delay is common if untreated (not a feature of Marfan syndrome). Diagnosis: ↑ Plasma homocysteine and methionine (in classic CBS deficiency) Positive cyanide-nitroprusside test in urine (detects homocystine) Management: Low-methionine diet Vitamin B■ (pyridoxine) in responsive patients, plus folate and vitamin B■■ Cysteine supplementation (becomes conditionally essential) Marfan syndrome (Option A): Although Marfan syndrome also presents with a tall, thin habitus and arachnodactyly, the lens dislocation is upward and outward, and it is not a prothrombotic disorder causing strokes in childhood. Its major risk is aortic root dilation and dissection. Ehlers-Danlos syndrome (Option C): Characterised by joint hypermobility and skin hyperextensibility. The vascular type can cause arterial rupture, but it does not typically present with marfanoid habitus plus lens dislocation. Osteogenesis imperfecta (Option D): Marked by recurrent fractures, blue sclerae, and hearing loss. It does not cause lens dislocation or the hypercoagulable state seen in this case.

References:

Nelson Textbook of Paediatrics, 20th Edition, Pages 644, 645

↑ *Back to this question in Part A*

@Neet_pg_bot

Q3: Deficiency of cystathionine β-synthase leads to the accumulation of which amino acid, increasing cardiovascular risk? (Q885183)

A. Methionine

B. Homocysteine

C. Cysteine

D. Taurine

Correct Answer: B. Homocysteine

Explanation: Classic homocystinuria is most commonly caused by deficiency of cystathionine β-synthase (CBS) , a vitamin B■ (pyridoxine/PLP)-dependent enzyme in the transsulfuration pathway. Biochemical Consequences: ↑ Homocysteine (key toxic metabolite) ↑ Methionine (shunted via remethylation pathway) ↓ Cysteine (becomes conditionally essential) The accumulation of homocysteine is particularly important because it is toxic to vascular endothelium and prothrombotic , leading to: Venous and arterial thrombosis Stroke and myocardial infarction at a young age Clinical Features: Ectopia lentis (lens dislocation downward and inward) Marfanoid habitus (tall, long limbs, arachnodactyly, pectus deformity) Early osteoporosis Intellectual disability (if untreated) Management:

Low-methionine diet Vitamin B■ (in responsive patients), plus folate and vitamin B■■ Cysteine supplementation (as it becomes essential) Methionine (Option A): Methionine levels are often elevated in CBS deficiency, but it is not the primary toxic metabolite responsible for endothelial injury and thrombosis. Cysteine (Option C): Cysteine is the product of the CBS-catalysed reaction. In CBS deficiency, cysteine levels are reduced, not increased, and it becomes a conditionally essential amino acid. Taurine (Option D): Taurine is a downstream metabolite of cysteine. Because the transsulfuration pathway is blocked, taurine synthesis is reduced, not increased.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 294, 295

↑ *Back to this question in Part A*

@Neet_pg_bot

Q4: Excessive urinary excretion of 5-HIAA (5-hydroxyindoleacetic acid) is characteristic of: (Q885182)

A. Pheochromocytoma

B. Carcinoid syndrome

C. Neuroblastoma

D. Mastocytosis

Correct Answer: B. Carcinoid syndrome

Explanation: Carcinoid tumours (neuroendocrine tumours), especially midgut tumours (e.g., ileum) with liver metastases , secrete serotonin (5-HT) into the systemic circulation. Normally, serotonin released into the portal circulation is inactivated by the liver, but hepatic metastases allow it to bypass first-pass metabolism , leading to carcinoid syndrome . Biochemical Basis: Serotonin is metabolised by monoamine oxidase (MAO) and aldehyde dehydrogenase to form 5-HIAA. 5-HIAA is excreted in urine and is measured in a 24-hour urine collection. Markedly elevated urinary 5-HIAA is the classic biochemical marker of carcinoid syndrome. Clinical Features: Episodic flushing Secretory diarrhea Bronchospasm/wheezing Carcinoid heart disease: fibrous plaque deposition on right-sided valves (tricuspid, pulmonary) Patients should avoid serotonin-rich foods (e.g., bananas, avocados, walnuts, tomatoes, pineapple) for 24-48 hours before testing to prevent false positives . Normal urinary 5-HIAA is low; markedly elevated levels strongly suggest carcinoid syndrome. Pheochromocytoma (Option A): This tumour secretes catecholamines (epinephrine/norepinephrine). Diagnosis is made using plasma or urinary metanephrines and VMA , not serotonin metabolites. Neuroblastoma (Option C): A childhood tumour of neural crest origin that also produces catecholamines . Its urinary markers are VMA and homovanillic acid (HVA) , not 5-HIAA. Mastocytosis (Option D): Characterised by mast cell proliferation and degranulation . Laboratory markers include serum tryptase and urinary N-methylhistamine , not 5-HIAA.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 310, 543

↑ *Back to this question in Part A*

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Q5: Xanthurenic aciduria after a tryptophan load test indicates deficiency of which vitamin? (Q885181)

A. Vitamin B■ (Thiamine)

B. Vitamin B■ (Riboflavin)

C. Vitamin B■ (Pyridoxine)

D. Niacin

Correct Answer: C. Vitamin B■ (Pyridoxine)

Explanation: Vitamin B■ (pyridoxine) is converted to its active form, pyridoxal 5′-phosphate (PLP) , which is an essential cofactor in amino acid metabolism , including the tryptophan → kynurenine → niacin pathway. A key PLP-dependent enzyme in this pathway is kynureninase . In vitamin B■ deficiency , kynureninase activity is reduced, causing the pathway to be diverted toward the formation of xanthurenic acid , which is then excreted in increased amounts in urine . This forms the basis of the tryptophan load test for B■ deficiency. Clinical features of Vitamin B■ Deficiency: Sideroblastic anemia Peripheral neuropathy Seborrheic dermatitis, cheilosis, glossitis Seizures (in severe deficiency) Common Causes: Chronic alcoholism Isoniazid therapy (binds and inactivates PLP) Malnutrition, malabsorption Laboratory findings: Increased urinary xanthurenic acid after tryptophan load Low serum PLP levels Reduced erythrocyte transaminase activity Vitamin B■ (Thiamine) (Option A): Deficiency causes beriberi and Wernicke-Korsakoff syndrome . It is assessed by erythrocyte transketolase activity , not by the tryptophan load test. Vitamin B■ (Riboflavin) (Option B): Riboflavin is a precursor of FAD and FMN used in redox reactions. Its deficiency is assessed by erythrocyte glutathione reductase activity . It does not cause xanthurenic aciduria. Niacin (Option D): Niacin (Vitamin B■) is the end product of the tryptophan-kynurenine pathway. Its deficiency causes pellagra . The tryptophan load test identifies a block before niacin formation , which occurs in vitamin B■ deficiency , not niacin deficiency.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 298

↑ *Back to this question in Part A*

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Q6: A middle-aged patient presents with bluish-black discolouration of the ears and sclera, along with low back pain. X-ray shows calcification of intervertebral discs. Which enzyme is deficient? (Q885180)

A. Phenylalanine hydroxylase

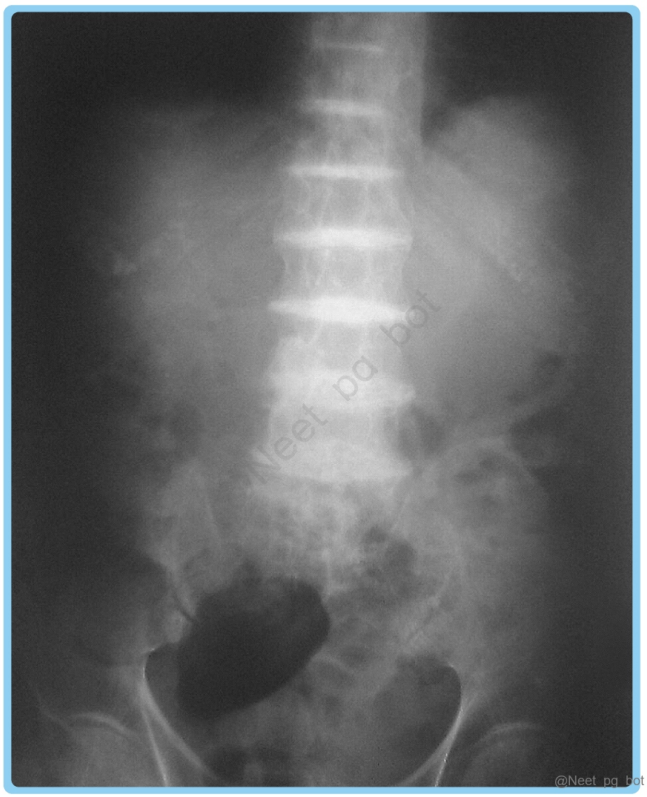
B. Homogentisate oxidase

C. Tyrosinase

D. Cystathionine β-synthase

Correct Answer: B. Homogentisate oxidase

Explanation: This presentation is classic for alkaptonuria with ochronosis (deposition of dark pigment in connective tissues) and ochronotic spondyloarthropathy . Biochemical defect: Caused by mutations in the HGD gene , leading to deficiency of homogentisate oxidase (homogentisate 1,2-dioxygenase) . This blocks degradation of homogentisic acid (HGA) in the phenylalanine/tyrosine catabolic pathway. HGA accumulates , undergoes oxidation and polymerisation , and deposits as a bluish-black pigment in connective tissues. Clinical Features: Ochronosis: Bluish-black pigmentation of ears, sclera, cartilage, and heart valves Ochronotic arthropathy: Deposition in hyaline cartilage , especially the spine , causing early-onset low back pain and intervertebral disc calcification on X-ray Urine turns black on standing or with alkalinization due to oxidation of HGA Progressive degenerative joint disease may require joint replacement ; spinal changes can mimic ankylosing spondylitis Management: Largely supportive (pain control, physiotherapy, orthopaedic care) Dietary reduction of phenylalanine/tyrosine may help Nitisinone can be used to reduce HGA production by inhibiting an upstream step in the pathway Phenylalanine hydroxylase (Option A): Deficiency causes Phenylketonuria (PKU), presenting in infancy with intellectual disability, seizures, and a musty/mousy odor, not with ochronosis or disc calcification in adulthood. Tyrosinase (Option C): This enzyme is required for melanin synthesis. Deficiency causes oculocutaneous albinism with hypopigmentation, which is the opposite of the dark ochronotic pigmentation seen here. Cystathionine β-synthase (Option D): Deficiency causes Homocystinuria , characterized by Marfanoid habitus, ectopia lentis, and thromboembolism. It does not cause bluish-black pigmentation or intervertebral disc calcification.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 296

↑ *Back to this question in Part A*

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Q7: Neonatal tyrosinemia (transient) is due to a deficiency of which enzyme? (Q885179)

A. Tyrosine aminotransferase

B. Fumarylacetoacetate hydrolase

C. 4-Hydroxyphenylpyruvate dioxygenase

D. Phenylalanine hydroxylase

Correct Answer: C. 4-Hydroxyphenylpyruvate dioxygenase

Explanation: Transient neonatal tyrosinemia is a non-genetic, self-limited metabolic condition seen mainly in premature or sick neonates . It is caused by delayed maturation (functional immaturity) of the hepatic enzyme

4-hydroxyphenylpyruvate dioxygenase (4-HPPD) , an important enzyme in tyrosine degradation . Biochemical Basis: 4-HPPD is an iron-dependent dioxygenase that also requires ascorbate (vitamin C) for optimal activity. It converts 4-hydroxyphenylpyruvate → homogentisate in the tyrosine catabolic pathway. In premature infants, immature enzyme activity leads to accumulation of tyrosine and increased urinary excretion of p-hydroxyphenyl metabolites . Clinical Features: Usually asymptomatic or mildly symptomatic Detected on newborn screening or biochemical testing Occurs more often in premature infants or those on high-protein feeds Course and Management: Benign and self-limiting Improves as the liver matures May be accelerated by vitamin C supplementation and temporary protein restriction Tyrosine aminotransferase (Option A): Deficiency of this enzyme causes Tyrosinemia Type II (Richner-Hanhart syndrome), a permanent genetic disorder characterized by painful corneal erosions, palmoplantar hyperkeratosis, and intellectual disability, not a transient neonatal condition. Fumarylacetoacetate hydrolase (Option B): This enzyme is deficient in Tyrosinemia Type I (hepatorenal tyrosinemia), the most severe form, presenting with acute liver failure, Fanconi syndrome, and a “boiled cabbage” odour. It is a life-threatening inherited disorder, not a transient neonatal problem. Phenylalanine hydroxylase (Option D): Deficiency of this enzyme causes Phenylketonuria (PKU), leading to elevated phenylalanine and low tyrosine, which is the opposite biochemical pattern of tyrosinemia.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 296, 304

↑ *Back to this question in Part A*

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Q8: A child presents with hepatomegaly, jaundice, abdominal distension, and a "boiled cabbage" odour. Laboratory findings show elevated succinylacetone. The most likely diagnosis is: (Q885177)

A. Phenylketonuria

B. Alkaptonuria

C. Type I tyrosinemia

D. Homocystinuria

Correct Answer: C. Type I tyrosinemia

Explanation: Type I tyrosinemia is caused by a deficiency of fumarylacetoacetate hydrolase (FAH) , the final enzyme in tyrosine degradation. This leads to the accumulation of fumarylacetoacetate and maleylacetoacetate , which are highly hepatotoxic and nephrotoxic , and to the formation of succinylacetone . Clinical Features: Severe hepatic disease : hepatomegaly, jaundice, ascites, acute or chronic liver failure Renal Fanconi syndrome : proximal tubular loss of phosphate, glucose, amino acids → vitamin D-resistant rickets Characteristic “boiled cabbage” odour due to sulfur-containing compounds/methionine metabolites Porphyria-like crises : abdominal pain, neuropathy (due to succinylacetone inhibiting porphobilinogen synthase) Diagnostic Hallmark: Elevated succinylacetone in blood or urine - pathognomonic and distinguishes Type I from other tyrosinemias. Treatment:

Nitisinone (NTBC) - inhibits 4-hydroxyphenylpyruvate dioxygenase, preventing formation of toxic downstream metabolites Dietary restriction of tyrosine and phenylalanine (since phenylalanine → tyrosine) Early treatment is crucial to prevent liver failure and neurologic complications. Phenylketonuria (Option A): Caused by phenylalanine hydroxylase deficiency . Presents with intellectual disability, seizures, hypopigmentation, and a "mousy/musty" odour. It does not cause severe liver failure and does not show elevated succinylacetone. Alkaptonuria (Option B): Due to homogentisate 1,2-dioxygenase deficiency . Presents later with ochronosis, arthritis, and urine that turns black on standing. It is not a cause of infantile liver failure. Homocystinuria (Option D): A disorder of methionine metabolism presenting with Marfanoid habitus, ectopia lentis, and thromboembolism. It may have a sweet/musty odour but does not cause this severe hepatic picture and does not show succinylacetone.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 296

↑ *Back to this question in Part A*

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Q9: A child with developmental delay and seizures is found to have elevated serum phenylalanine. Which test detects the abnormal urinary metabolite in this condition? (Q885174)

A. Cyanide-nitroprusside test

B. Ferric chloride test

C. Obermeyer test

D. Nitrosonaphthol test

Correct Answer: B. Ferric chloride test

Explanation: In Phenylketonuria (PKU) , deficiency of phenylalanine hydroxylase leads to accumulation of phenylalanine, which is shunted into alternative pathways producing phenylpyruvic acid (phenylpyruvate) , a phenylketone excreted in urine. The Ferric chloride (FeCl■) test detects phenylpyruvic acid in urine. When 10% ferric chloride is added, it produces a characteristic blue-green / emerald-green colour , which is the classic screening reaction in PKU. The musty/mousy odour of PKU is due mainly to phenylacetate , which does not give the FeCl■ reaction. Clinical correlation (untreated PKU): Developmental delay / intellectual disability Seizures Musty body odour Hypopigmentation (fair skin, light hair, blue eyes) due to reduced tyrosine → ↓ melanin Management (high-yield): Lifelong low-phenylalanine diet Tyrosine supplementation Early treatment is critical to prevent irreversible brain damage Modern diagnosis: Newborn screening by tandem mass spectrometry (MS/MS) and quantitative plasma amino acid analysis The ferric chloride test is not specific and may be positive in other disorders with phenolic/keto acids (e.g., tyrosinemia), so confirmatory testing is required . Cyanide-nitroprusside test (Option A): This test detects cystine or homocystine in urine and is used for cystinuria and homocystinuria , not for disorders of phenylalanine metabolism. Obermeyer test (Option C): This test detects indican in urine,

which is related to tryptophan metabolism and conditions such as Hartnup disease , not PKU. Nitrosonaphthol test (Option D): This test detects tyrosine metabolites (e.g., p-hydroxyphenylpyruvic acid) and is used as a screening test for tyrosinemia , not PKU

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 296

↑ *Back to this question in Part A*

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Q10: Match the following urinary odours with their corresponding diagnoses: Urinary Odour Diagnosis 1. Mousy odour a. DKA (Diabetic Ketoacidosis) 2. Fruity odour b. Hawkinsinuria 3. Cabbage odour c. Methionine malabsorption 4. Swimming pool odour d. PKU (Phenylketonuria) 5. Oast house odour e. Type 1 Tyrosinemia (Q844928)

A. 1-e, 2-a, 3-d, 4-b, 5-c

B. 1-d, 2-a, 3-e, 4-c, 5-b

C. 1-e, 2-a, 3-d, 4-c, 5-b

D. 1-d, 2-a, 3-e, 4-b, 5-c

Correct Answer: D. 1-d, 2-a, 3-e, 4-b, 5-c

■ *Listen to Audio 1*

Explanation:

Urine Odor and Diagnosis

Odor	Disorders
Mousy odour	PKU
Fruity odour	DKA (diabetic ketoacidosis)
Cabbage odour	Type 1 Tyrosinemia
Boiled cabbage odour	Hypermethioninemia
Oast house (urine and sweat smell as oasthouse) or beer baby syndrome	Methionine malabsorption
Swimming pool odour	Hawkinsinuria
Sweaty feet odour	Isovaleric acidemia
Tom cat Urine odour	Multiple carboxylase deficiency
Fish odor odour	Trimethylamine urea

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References:

www.researchgate.net/publication/329966890_Common_metabolic_disorder_inborn_errors_of_metabolism_concerns_in_primary_care_practice

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: A 45-year-old patient presents with episodes of palpitations, sweating, and severe headaches. These episodes occur intermittently and are associated with hypertension. Laboratory tests reveal elevated levels of plasma free metanephrines and urinary Vanillyl Mandelic Acid. Which of the following enzymes are directly involved in the synthesis of these metabolites? (Q844927)

A. Monoamine oxidase, Catechol-O-methyltransferase

B. Glutamate decarboxylase, Tyrosine hydroxylase

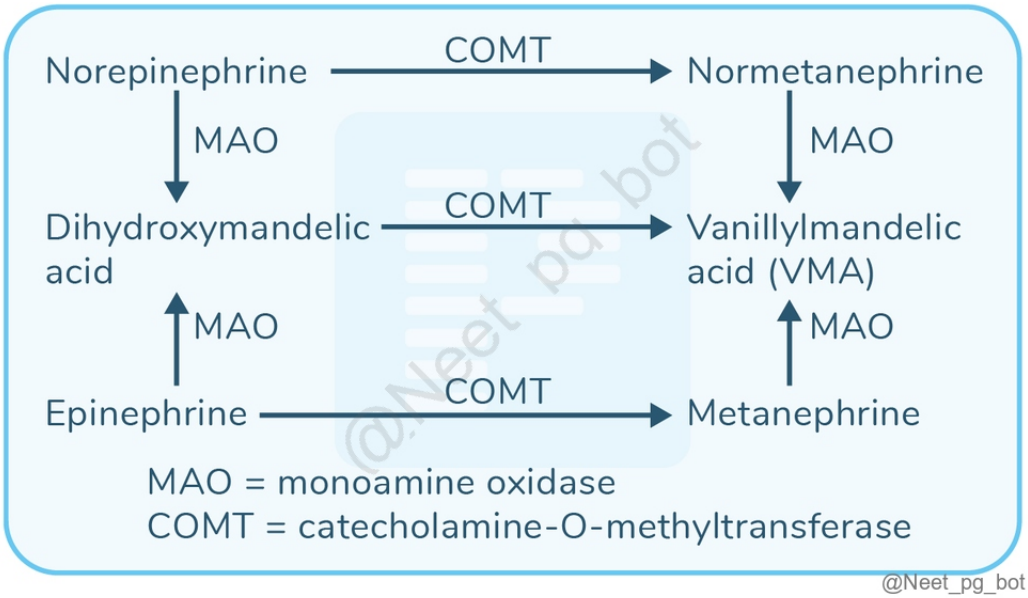
C. Tyrosine hydroxylase, Catechol-O-methyltransferase

D. Tyrosine hydroxylase, Monoamine oxidase

Correct Answer: A. Monoamine oxidase, Catechol-O-methyltransferase

■ *Listen to Audio 1*

Explanation: Monoamine oxidase & Catechol-O-methyltransferase are directly involved in the metabolism of catecholamines, leading to the formation of metanephrines and vanillylmandelic acid (VMA). In Pheochromocytoma, there is elevation of catecholamines leading to increased breakdown of epinephrine and norepinephrine, consequently increased levels of metabolites such as metanephrines and vanillylmandelic acid.



References:

Vasudevan Textbook of Biochemistry, 7th Edition, Page 236.

www.ncbi.nlm.nih.gov/books/NBK545235/

↑ *Back to this question in Part A*

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Q12: Which of the following statements about Segawa syndrome is incorrect? (Q844926)

A. Segawa syndrome is autosomal dominant.

B. Patients have a defect in the GTP cyclohydrolase enzyme.

C. CSF Biopterin levels are elevated.

D. Females are more commonly affected than males.

Correct Answer: C. CSF Biopterin levels are elevated.

■ *Listen to Audio 1*

Explanation: In Segawa syndrome, blood phenylalanine levels are high . The syndrome is characterized by a defect in the GTP cyclohydrolase enzyme , which leads to reduced levels of tetrahydrobiopterin (BH4) . Segawa Syndrome / Dopa-responsive dystonia (DRD) Inheritance Pattern Autosomal dominant (Option A) Clinical Features Dystonia with diurnal variation (worsens in the afternoon and evening) Incidence based on sex More common in females than males (Option D) Genetic Mutation Defect in the GTP cyclohydrolase enzyme (Option B) Biochemical Findings Reduced tetrahydrobiopterin (BH4) levels Normal or high blood phenylalanine levels CSF Biopterin levels are low Diagnosis Clinical observation of dystonia patterns Genetic testing for GTP cyclohydrolase enzyme mutation

References:

Pubmed

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4061475/

↑ *Back to this question in Part A*

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Q13: A newborn screening test reveals elevated phenylalanine levels, prompting further investigation. Genetic testing indicates a defect in dihydrobiopterin reductase. Based on this information, Which type of phenylketonuria does the patient most likely have? (Q844925)

A. Classic phenylketonuria

B. Non-classic phenylketonuria Type II

C. Non-classic phenylketonuria Type IV

D. Non-classic phenylketonuria Type V

Correct Answer: B. Non-classic phenylketonuria Type II

■ *Listen to Audio 1*

Explanation: Non-classic phenylketonuria Type II is characterized by a defect in dihydrobiopterin reductase. Type Defective Enzyme / Factor Pathway Defect Key Features Treatment Type I PKU Phenylalanine Hydroxylase deficiency Failure to convert phenylalanine → tyrosine Most common form; ↑ phenylalanine, intellectual disability, musty odor Low phenylalanine diet Type II PKU Dihydropteridine Reductase deficiency Failure to regenerate Tetrahydrobiopterin Severe neurological symptoms due to ↓ dopamine & serotonin Low phenylalanine diet + BH■ + neurotransmitter precursors Type III PKU Defect in BH■ synthesis enzymes (e.g., GTP cyclohydrolase) Decreased BH■ production Hyperphenylalaninemia with neurotransmitter deficiency BH■

supplementation + neurotransmitter therapy Type IV PKU Defect in 6-pyruvoyl-tetrahydropterin synthase Impaired BH■ synthesis Severe neurological deficits BH■ + neurotransmitter precursors Type V PKU Defect in sepiapterin reductase Final step of BH■ synthesis affected Neurological dysfunction with hyperphenylalaninemia BH■ supplementation + dopamine/serotonin precursors

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 296

↑ *Back to this question in Part A*

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Q14: A 12-year-old boy is brought to the ophthalmologist by his parents due to progressive vision loss, particularly at night. Fundoscopic examination reveals circular areas of chorioretinal atrophy and peripheral retinal degeneration. Laboratory tests indicate markedly elevated plasma ornithine levels. What is the primary biochemical defect in this patient's condition? (Q844924)

A. Deficiency of ornithine transcarbamylase

B. Deficiency of ornithine aminotransferase

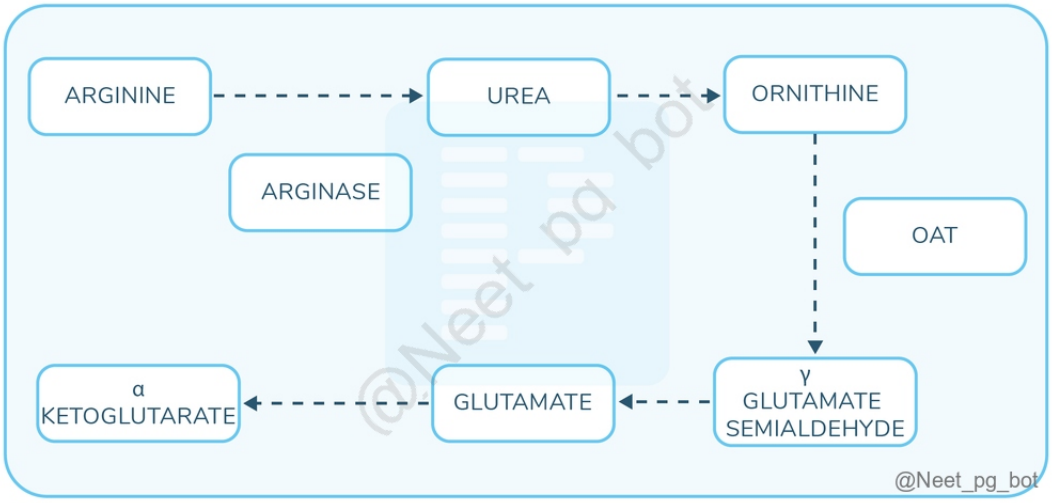
C. Deficiency of ornithine decarboxylase

D. Deficiency of ornithine cyclodeaminase

Correct Answer: B. Deficiency of ornithine aminotransferase

■ *Listen to Audio 1*

Explanation: Gyrate atrophy of the retina is caused by a deficiency in the enzyme ornithine aminotransferase (OAT), which leads to the accumulation of ornithine in the plasma. Ornithine is toxic to the retinal pigment epithelium and causes gyrate atrophy of the retina. This condition manifests with progressive vision loss, especially night blindness, and characteristic fundoscopic findings of chorioretinal atrophy. Management includes a Arginine restricted diet and vitamin B6 supplementation, as OAT activity can be B6-dependent in some patients. Ornithine transcarbamylase (Option A) is involved in the urea cycle, not directly related to gyrate atrophy. Ornithine decarboxylase (Option C) is involved in polyamine synthesis, not related to ornithine accumulation or retinal degeneration. Ornithine cyclodeaminase (Option D) is not related to the metabolic pathway implicated in gyrate atrophy.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 292

↑ *Back to this question in Part A*

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Q15: Which of the following statements about the metabolism of phenylalanine is incorrect? (Q844910)

A. Phenylalanine is converted to tyrosine catalyzed by phenylalanine hydroxylase.

B. Phenylalanine on transamination gets converted to phenylpyruvate

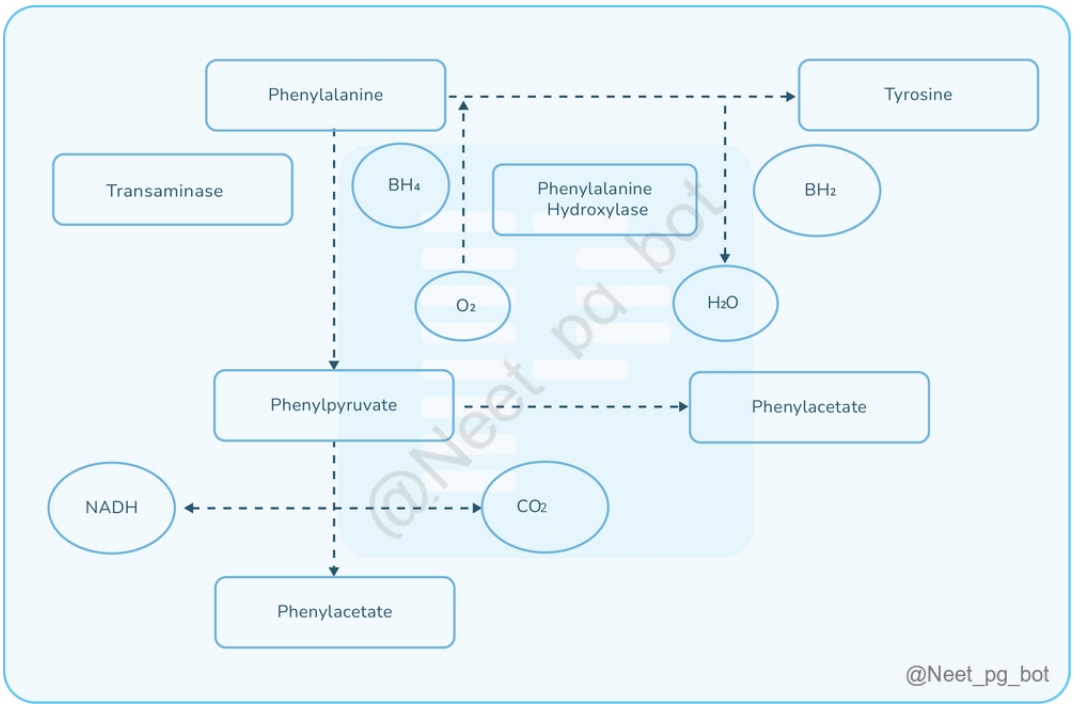
C. Phenylpyruvate on oxidation yields phenyl lactate

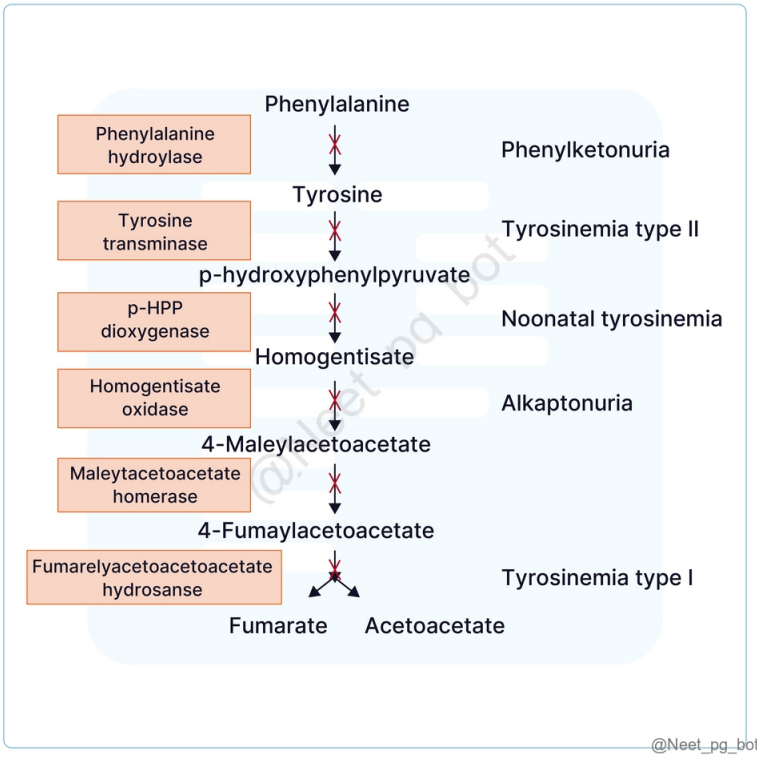
D. Phenylpyruvate on oxidative decarboxylation forms phenylacetate.

Correct Answer: C. Phenylpyruvate on oxidation yields phenyl lactate

■ *Listen to Audio 1*

Explanation: Phenylpyruvate on reduction, not oxidation, yields phenyl lactate. Phenylalanine has 2 metabolic fates in the body: Major Fate Phenylalanine is converted into tyrosine by the enzyme phenylalanine hydroxylase. Deficiency of phenylalanine hydroxylase results in Phenylketonuria. It requires tetrahydrobiopterin (BH4) as a cofactor Minor Fate Phenylalanine is converted into phenylpyruvate by transamination . Phenylpyruvate can further undergo different pathways: Phenylpyruvate can be reduced to phenyl lactate Phenylpyruvate undergoes oxidative decarboxylation, forming phenylacetate Phenylacetate combines with glutamine to form phenylacetylglutamine , which is excreted in the urine.





References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 277, 298

↑ *Back to this question in Part A*

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Q16: A 10-year-old girl is evaluated for developmental delay. Physical examination reveals a tall, thin build, scoliosis, and ectopia lentis. Laboratory tests show elevated levels of homocysteine and methionine in the plasma. Treatment is initiated with vitamin B6 (pyridoxine) and folate. Homocysteine levels improve, but methionine levels remain elevated. Based on this scenario, what type of defect does the patient most likely have? (Q844922)

A. Type 1 Homocystinuria

B. Type 2 Homocystinuria

C. Type 3 Homocystinuria

D. Marfan Syndrome

Correct Answer: A. Type 1 Homocystinuria

■ *Listen to Audio 1*

Explanation: Type 1 vs Type 2 Homocystinuria: Feature Type - I Homocystinuria Type - II Homocystinuria Enzyme Deficiency Cystathionine beta-synthase Methionine synthase Metabolic Pathway Affected Homocysteine to cystathionine Homocysteine to methionine Elevated Metabolites Homocysteine and methionine Homocysteine and elevated levels of methionine Biochemical Diagnosis Low levels of cysteine. High levels of methionine. High levels of cysteine. Low levels of methionine. Treatment Vitamin B6 (pyridoxine) supplementation; low-methionine diet; cysteine supplementation Vitamin B12 and folate supplementation; low-methionine diet Type 3 Homocystinuria (Option C) is a rare form not commonly linked with elevated methionine and does not match the patient's response to treatment. Deficiency of MTHFR (Methylenetetrahydrofolate reductase) prevents conversion of homocysteine back to methionine. Characterized by severe neurologic symptoms and elevated plasma homocysteine with low methionine levels . Marfan Syndrome (Option D) shares some physical traits (tall stature, scoliosis), but it does not include elevated homocysteine levels or a vitamin B6 response.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 294, 295

Vasudevan Textbook of Biochemistry, 7th Edition, Page 431.

↑ *Back to this question in Part A*

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Q17: A 7-year-old boy is brought with recurrent episodes of a red, scaly rash that worsens with sun exposure, as well as intermittent episodes of diarrhea and mood changes. A urine sample reveals an abnormal presence of neutral amino acids. The patient's diet is balanced, and there is no history of similar symptoms in the family. Laboratory tests indicate a deficiency in niacin despite adequate dietary intake. What is the underlying cause of the patient's condition? (Q844921)

A. Deficiency in tryptophan hydroxylase

B. Deficiency in the neutral amino acid transporter

C. Overactivity of serotonin decarboxylase

D. Excessive dietary leucine

Correct Answer: B. Deficiency in the neutral amino acid transporter

■ *Listen to Audio 1*

Explanation: Hartnup’s disease: It is caused by a defect in the neutral amino acid transporter in the kidneys and intestines, leading to poor absorption and increased excretion of neutral amino acids like tryptophan. This results in decreased availability of tryptophan for niacin synthesis, leading to symptoms similar to pellagra despite adequate dietary intake. The characteristic skin rash, diarrhea, and mood changes are consistent with this diagnosis. Obermeyer Test: A qualitative urine test to detect Indican . In Hartnup disease, tryptophan malabsorption leads to bacterial degradation in the gut into indole, which is absorbed, metabolized in the liver, and excreted as indican in urine. A positive Obermeyer test (deep blue color upon oxidation) indicates the presence of excessive indican, confirming tryptophan malabsorption and intestinal transport failure. Tryptophan hydroxylase (Option A) is involved in serotonin synthesis, not amino acid transport. Overactivity of serotonin decarboxylase (Option C) would affect serotonin levels, not amino acid transport. Excessive dietary leucine (Option D) can interfere with tryptophan metabolism, but it is not the primary cause of Hartnup's disease.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 298,543

↑ *Back to this question in Part A*

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Q18: A 45-year-old man presents with symptoms of diarrhea, dermatitis, and dementia. On further investigation, his diet is found to be predominantly corn-based, and he has a history of alcohol use disorder. Laboratory tests reveal low levels of tryptophan and niacin in his blood. Which enzyme deficiency is most likely contributing to the patient's condition? (Q844920)

A. Tryptophan hydroxylase

B. Kynureninase

C. Serotonin decarboxylase

D. Niacinamide adenine dinucleotide synthetase

Correct Answer: B. Kynureninase

■ *Listen to Audio 1*

Explanation: Pellagra is a condition that results from a deficiency of niacin (vitamin B3) or tryptophan. It is characterized by three main symptoms: dermatitis, diarrhea, and dementia. Kynureninase is an enzyme in the kynurenine pathway, responsible for converting tryptophan to niacin. A low activity of this enzyme is observed in B6 deficiency, which is caused by alcoholism. This can exacerbate pellagra caused by maize or corn intake. Causes of Pellagra: Diet with Niacin deficiency. Tryptophan (60 mg Tryptophan = 1 mg Niacin) deficiency in the maize-based diet. Tryptophan malabsorption - Hartnup's disease. Carcinoid Syndrome - Tryptophan is used to form Serotonin leading to niacin deficiency Vitamin B6 deficiency - inactivates Kynenurinase. Leucine pellagra - Quinolinate phosphoribosyltransferase (QPRTASE) enzyme is inhibited in a leucine-rich diet. Example: sorghum diet which is rich in leucine. Tryptophan hydroxylase (Option A) converts tryptophan to 5-hydroxytryptophan in the serotonin synthesis pathway, not involved in niacin synthesis. Serotonin decarboxylase (Option C) converts 5-hydroxytryptophan to serotonin, not in niacin synthesis. Niacinamide adenine dinucleotide synthetase (Option D) converts niacinamide to NAD (nicotinamide adenine dinucleotide), a form of niacin. It is not directly related to the metabolism of tryptophan into niacin.



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 542

Vasudevan Textbook of Biochemistry, 7th Edition, Page 482.

↑ *Back to this question in Part A*

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Q19: Which of the following statements is true? (Q844919)

A. Tyrosine is the amino acid forming Serotonin

B. Niacin deficiency is seen in Carcinoid syndrome

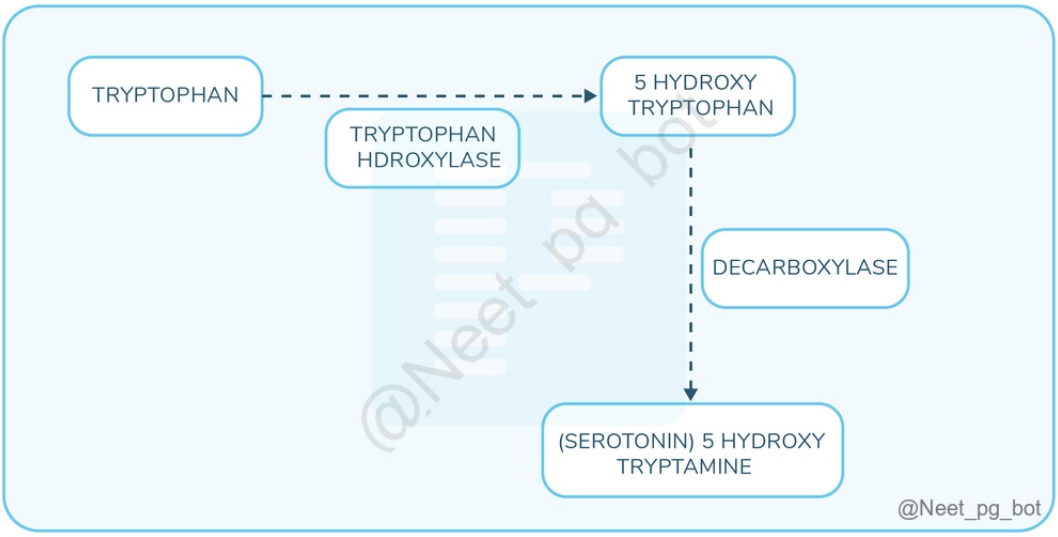
C. Tryptophan hydroxylase is required for Niacin synthesis

D. Decarboxylation of 5 hydroxy tryptophan forms niacin.

Correct Answer: B. Niacin deficiency is seen in Carcinoid syndrome

■ *Listen to Audio 1*

Explanation: Tryptophan forms both niacin and serotonin. (Option A) Carcinoid syndrome causes excess production of serotonin by carcinoid tumors and tryptophan is deficient for the synthesis of niacin causing niacin deficiency. 60 mg Tryptophan is required for the synthesis of 1 mg of Niacin, in the presence of vitamin B2 and B6. Synthesis of Serotonin Other causes of niacin deficiency include Hartnup's disease, in maize eaters, and drugs like Isoniazid Tryptophan hydroxylase is required for Niacin synthesis (Option C): Tryptophan hydroxylase is required for the synthesis of serotonin Decarboxylation of 5 hydroxy tryptophan forms niacin (Option D): Decarboxylation of 5 hydroxy tryptophan forms serotonin



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 310, 543

↑ *Back to this question in Part A*

@Neet_pg_bot

Q20: Which of the following TCA intermediates is not formed by glucogenic amino acids? (Q844918)

A. Alpha Ketoglutarate

B. Isocitrate

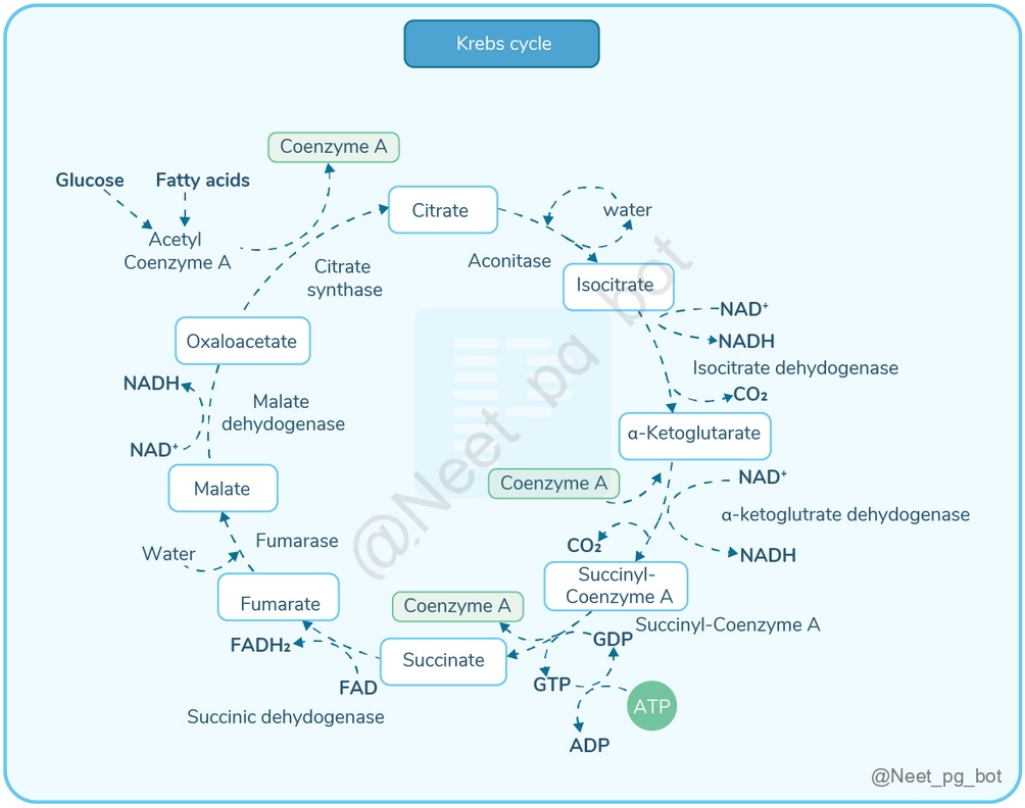
C. Fumarate

D. Oxaloacetate

Correct Answer: B. Isocitrate

■ *Listen to Audio 1*

Explanation: Glucogenic amino acids give rise to 4 intermediates of TCA Alpha Ketoglutarate Succinyl CoA Fumarate Oxaloacetate Amino acids give rise to: Alpha-ketoglutarate (on Transamination) Glutamate Glutamine Histidine Proline Arginine Succinyl CoA V- Valine I- Isoleucine M- Methionine (Mnemonic- VIM) Fumarate Phenylalanine Tyrosine Oxaloacetate (on Transamination) Aspartate Asparagine



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 160

↑ *Back to this question in Part A*

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Q21: Which of the following amino acids are both glucogenic and ketogenic? (Q844917)

A. Leucine

B. Tryptophan

C. Lysine

D. Alanine

Correct Answer: B. Tryptophan

■ *Listen to Audio 1*

Explanation: Tryptophan is both glucogenic and ketogenic. Tryptophan on catabolism gives one of the glycolytic intermediate - Pyruvate or TCA cycle intermediate other than Acetyl CoA. Classification of Amino Acid Catabolism Purely Ketogenic Purely Glucogenic Both Ketogenic and Glucogenic On catabolism gives only acetyl CoA On catabolism gives only pyruvate Substance on catabolism gives one of the glycolytic intermediate -Pyruvate or TCA cycle intermediate other than Acetyl CoA Amino acids are: Leucine Lysine Amino acids are: Alanine: On transamination yields pyruvate. Glycine: Simplest amino acid. 3-hydroxyl group- containing amino acids Serine Threonine Hydroxyproline Sulphur-containing amino acid Cysteine Amino acids are: Aromatic amino acids Phenylalanine Tyrosine Tryptophan Branched-chain amino acid Isoleucine Threonine

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 291

↑ *Back to this question in Part A*

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Q22: A 6-month-old infant has been having developmental delays. Further examination reveals a defect in the enzyme called phenylalanine hydroxylase. Which of the following urine odor is seen in this infant? (Q844916)

A. Fruity odour

B. Musty odour

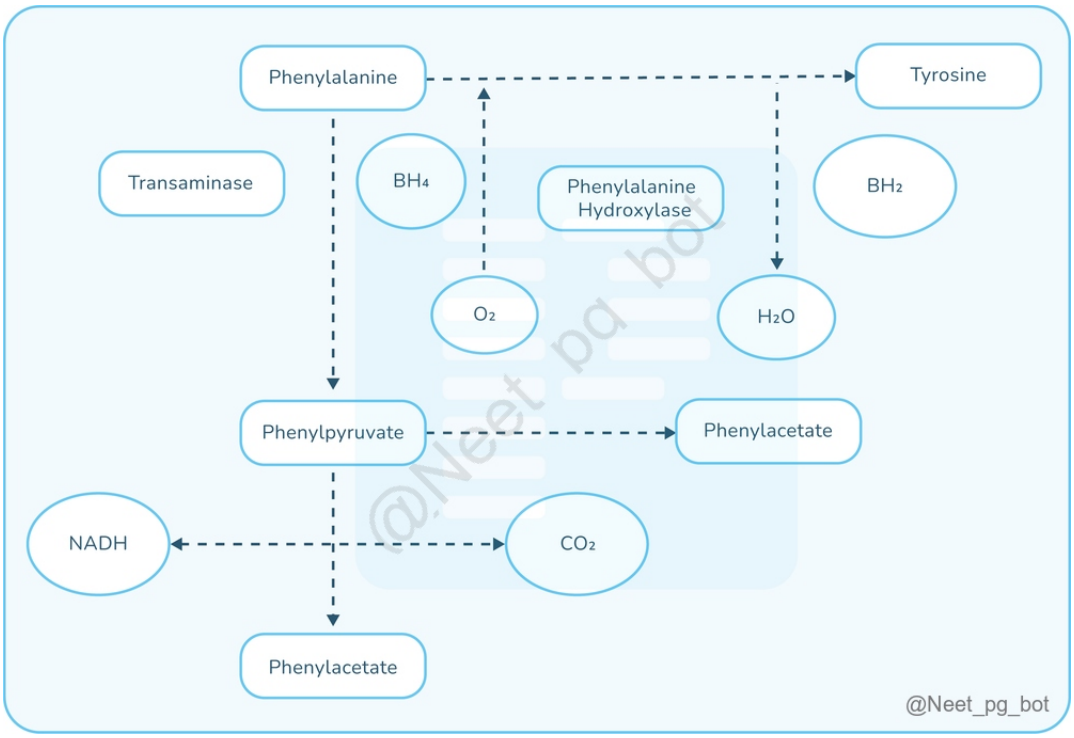
C. Cabbage odour

D. Burnt sugar

Correct Answer: B. Musty odour

■ *Listen to Audio 1*

Explanation: The clinical scenario is a case of phenylketonuria which causes 3 main clinical features: Mental retardation Hypopigmentation Mousy odour Phenylketonuria (PKU): Phenylketonuria (PKU) is an autosomal recessive inborn error of metabolism caused by a deficiency of phenylalanine hydroxylase or its cofactor tetrahydrobiopterin (BH4). This deficiency leads to an accumulation of phenylalanine and its metabolites, including phenylpyruvic acid. Mental Retardation: Cause: Mental retardation in phenylketonuria (PKU) is due to the accumulation of phenylketones, which compete with other neutral amino acids for transport across the blood-brain barrier. Management: To prevent mental retardation, a phenylalanine-restricted diet should be followed, along with tyrosine supplements. Hypopigmentation: Cause: Reduced melanin production in PKU results from a deficiency in tyrosine, a crucial component for melanin synthesis. This leads to lighter skin and hair color compared to individuals without PKU. Musty/ Mousy urine odor: Cause: Due to the accumulation of phenylpyruvic acid (a metabolite of excess phenylalanine) in the urine. Management: Dietary: Lifelong restriction of Phenylalanine. Tyrosine becomes a conditionally essential amino acid. Pharmacotherapy: Sapropterin (BH4 supplementation) is used for patients with residual enzyme activity (BH4-responsive PKU). Monitoring: Regular serum phenylalanine level monitoring is vital to prevent permanent intellectual disability.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 296, 573, 574,

www.ncbi.nlm.nih.gov/books/NBK535378/

www.msdmanuals.com/en-in/home/children-s-health-issues/hereditary-metabolic-disorders/tyrosinemia

↑ *Back to this question in Part A*

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Q23: A 6-month-old infant presents with a lack of pigmentation in their skin, hair, and eyes. The parents are concerned about the child's sensitivity to sunlight and the involuntary back-and-forth eye movements that the child exhibits. A significant decrease in melanin pigmentation is present. Which of the following enzymes is absent or deficient in this condition? (Q844915)

A. Alanine aminotransferase (ALT)

B. Tyrosinase

C. Phenylalanine hydroxylase

D. S-adenosylmethionine

Correct Answer: B. Tyrosinase

■ *Listen to Audio 1*

Explanation: Melanin is a pigment produced by cells called melanocytes, and it plays a critical role in determining the colour of our skin, hair, and eyes. Tyrosinase catalyzes the first two steps in the conversion of the amino acid tyrosine to melanin . Tyrosine → L-Dopa → Dopaquinone Without enough functional tyrosinase, melanin production is reduced or completely absent, leading to the characteristic features of albinism. Reaction Enzyme Tyrosine to DOPA Tyrosine hydroxylase/ Tyrosinase DOPA to Melanin Non-enzyme catalyzed chemical reaction Alanine aminotransferase (ALT) (Option A) is a liver enzyme that is released into the bloodstream when the liver is damaged. Phenylalanine hydroxylase (Option C) is an enzyme that converts phenylalanine to tyrosine. A deficiency in this enzyme leads to phenylketonuria (PKU). S-adenosylmethionine (Option D) required for: Norepinephrine to Epinephrine: Enzyme: Phenylethanolamine N-methyltransferase (PNMT). Conversion of Homocysteine to Methionine: Enzyme: Methionine synthase, using methylcobalamin as a cofactor. Methylation of DNA: SAM donates methyl groups to cytosine residues, affecting gene expression. Synthesis of Creatine: SAM is involved in the methylation of guanidinoacetate to form creatine. Methylation of Neurotransmitters: SAM is also involved in the methylation of other neurotransmitters, including serotonin to melatonin. Lipid Metabolism: Involved in the synthesis of phosphatidylcholine from phosphatidylethanolamine.

References:

pubmed.ncbi.nlm.nih.gov/1642278/

www.researchgate.net/figure/Pathway-of-melanin-synthesis

↑ *Back to this question in Part A*

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Q24: Tyrosine is involved in the synthesis of multiple neurotransmitters. Match the following neurotransmitters with the corresponding enzymes involved in their synthesis? Neurotransmitter Enzyme 1. Dihydroxy phenylalanine (DOPA) a. Dopamine beta-hydroxylase 2. Dopamine b. N-methyl transferase 3. Norepinephrine c. Tyrosine hydroxylase 4. Epinephrine d. DOPA Decarboxylase (Q844914)

A. 1-d, 2-c, 3-b, 4-a

B. 1-c, 2-b, 3-a, 4-d

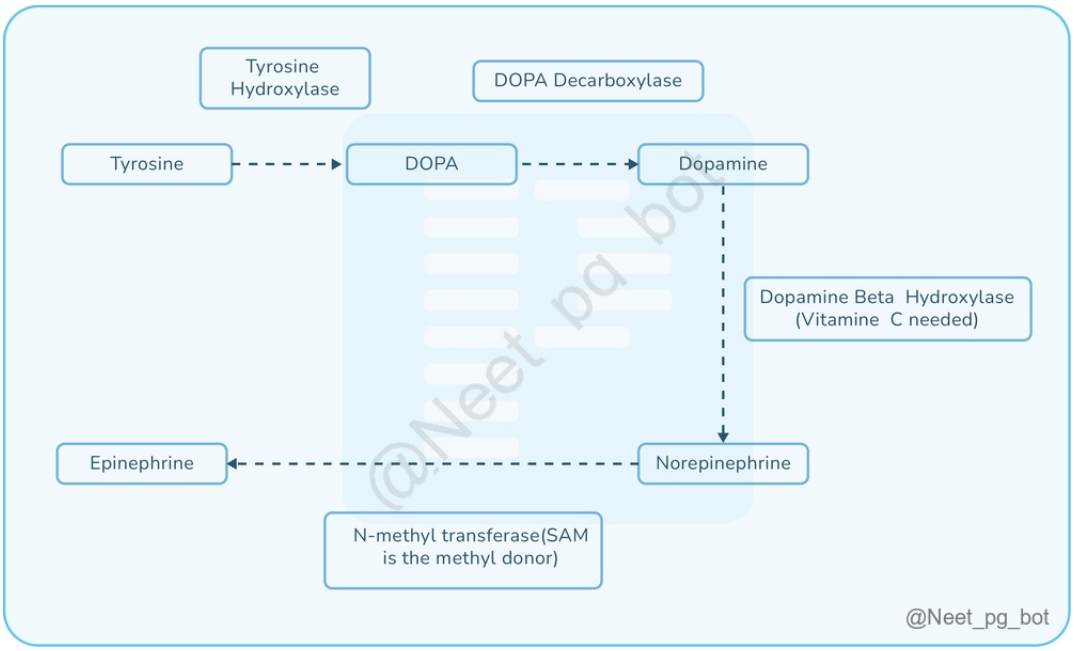
C. 1-d, 2-b, 3-c, 4-a

D. 1-c, 2-d, 3-a, 4-b

Correct Answer: D. 1-c, 2-d, 3-a, 4-b

■ *Listen to Audio 1*

Explanation: Reaction Enzyme Tyrosine to dihydroxy phenylalanine (DOPA) Tyrosine hydroxylase DOPA to Dopamine DOPA Decarboxylase (uses PLP as a coenzyme) Dopamine to Norepinephrine Dopamine beta-hydroxylase (depends on Vitamin C) Norepinephrine to epinephrine N-methyl transferase (S-adenosylmethionine (SAM) is the methyl donor) Synthesis of Neurotransmitters from Tyrosine- Dopamine, Norepinephrine and Epinephrine



References:

Vasudevan Textbook of Biochemistry, 7th Edition, Pages 234, 235

↑ *Back to this question in Part A*

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Q25: A 5-month-old infant is brought to the pediatrician due to poor weight gain, vomiting, and yellowing of the skin and eyes. Genetic testing confirms a deficiency in fumarylacetoacetate hydrolase. The physician recommends a low-protein diet and the drug Nitisinone. Which of the following is not an indication of Nitisinone? (Q844913)

A. Tyrosinemia type I

B. Alkaptonuria

C. Hawkinsinuria

D. Tyrosinemia type II

Correct Answer: D. Tyrosinemia type II

■ *Listen to Audio 1*

Explanation: Nitisinone: Inhibits 4-Hydroxyphenylpyruvate dioxygenase (HPPD). In Tyrosinemia Type 1, the metabolic block at Fumarylacetoacetate hydrolase causes toxic succinylacetone buildup. Nitisinone blocks the pathway upstream, preventing the formation of these toxic metabolites. Not indicated for Type 2 Tyrosinemia: Type 2 Tyrosinemia is caused by Tyrosine Aminotransferase deficiency. This occurs upstream of the HPPD enzyme. Therefore, inhibiting HPPD with Nitisinone is ineffective, as it does not address the metabolic bottleneck causing the Tyrosine buildup in Type 2. Indications: Tyrosinemia type I Alkaptonuria Hawkinsinuria

References:

usiena-air.unisi.it/bitstream/11365/1006035/7/

www.ncbi.nlm.nih.gov/books/NBK589678/

https://link.springer.com/article/10.1007/

↑ *Back to this question in Part A*

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Q26: A 4-month-old infant presents with jaundice, hepatomegaly, and failure to thrive. The parents also report that the infant has episodes of vomiting and lethargy. Laboratory tests reveal elevated levels of tyrosine and succinylacetone in the blood and urine, along with signs of coagulopathy and renal tubular dysfunction. What is the most likely underlying metabolic disorder? (Q844912)

A. Phenylketonuria

B. Tyrosinemia type II

C. Tyrosinemia type I

D. Alkaptonuria

Correct Answer: C. Tyrosinemia type I

■ *Listen to Audio 1*

Explanation: The clinical scenario described, including jaundice, hepatomegaly, failure to thrive, and elevated levels of tyrosine and succinylacetone , is characteristic of Tyrosinemia type I , which is caused by a deficiency in fumarylacetoacetate hydrolase. Disorders of Tyrosine Metabolism: Disorder Deficient Enzyme Key Clinical Features Tyrosinemia type I Fumarylacetoacetate hydrolase Jaundice, hepatomegaly, hypoglycemia, HCC, liver cirrhosis Often presents with Fanconi syndrome and renal failure Elevated tyrosine and succinylacetone (pathognomonic) Tyrosinemia type II Tyrosine aminotransferase Painful corneal erosions, palmoplantar hyperkeratosis Also known as oculocutaneous tyrosinemia Elevated tyrosine Tyrosinemia type III

p-Hydroxyphenylpyruvate dioxygenase Recurrent seizures, intermittent ataxia Elevated tyrosine and 4-hydroxyphenylpyruvate Alkaptonuria (Black urine disease) Homogentisate 1,2-dioxygenase Dark urine, ochronosis, arthritis Black or brown pigmentation of sclera (Ochronosis) and ear cartilage Elevated homogentisic acid

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 296

Pubmed

↑ *Back to this question in Part A*

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Q27: A 2-year-old child is brought to the clinic by his parents due to darkening of urine upon standing and brownish discolouration of the sclera and ear cartilage. The parents also report that their child has been experiencing joint pain. Upon further investigation, the physician suspects a tyrosine metabolic disorder and orders urine tests. Which of the following enzymes is most likely deficient in this patient? (Q844911)

A. Parahydroxy phenylpyruvate dioxygenase

B. Tyrosine transaminase

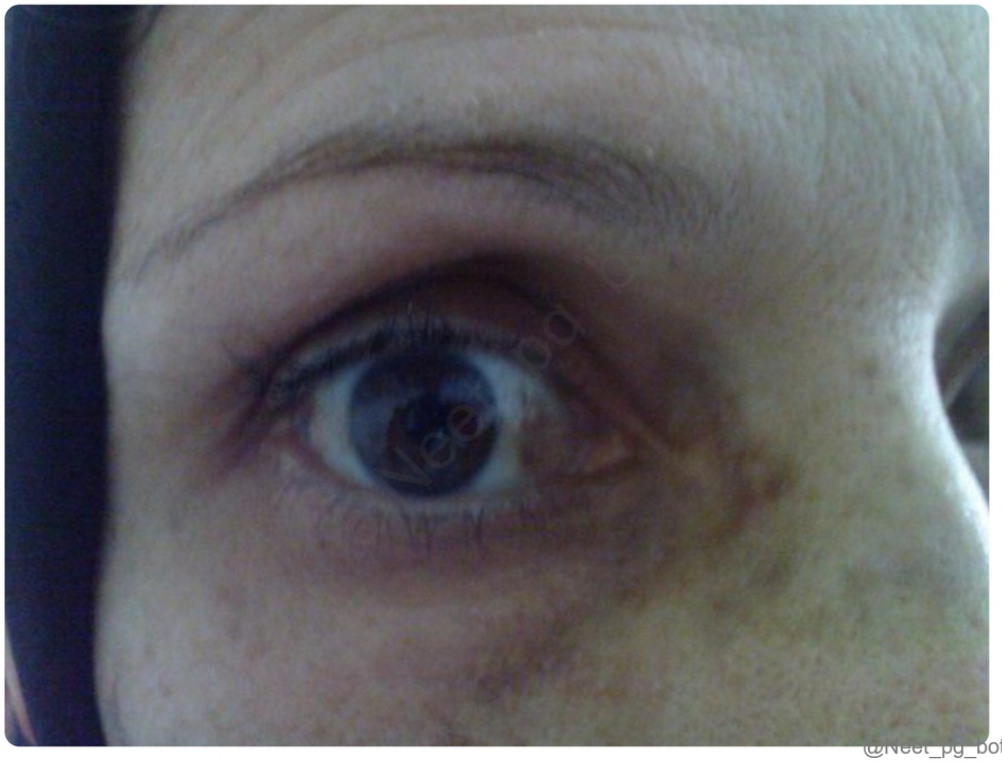
C. Homogentisate oxidase

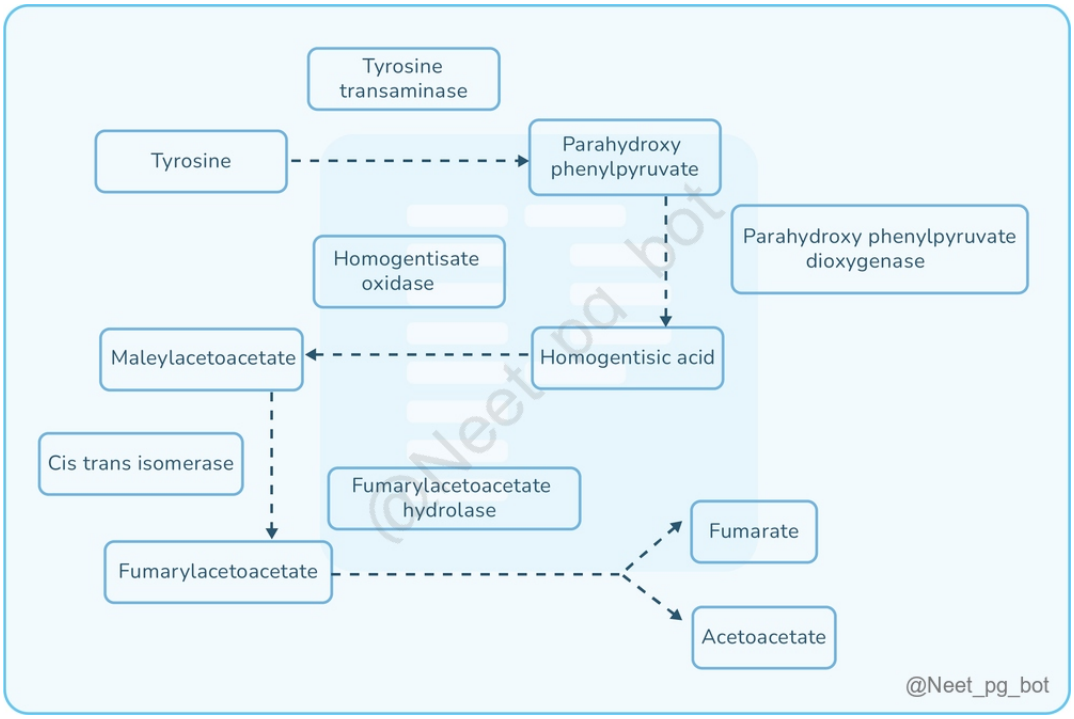
D. Fumarylacetoacetate hydrolase

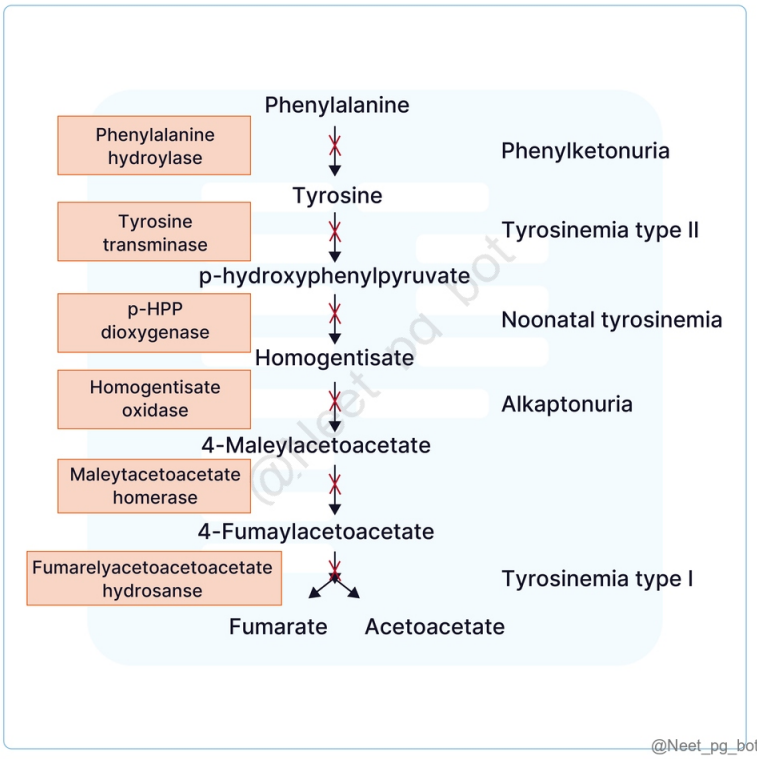
Correct Answer: C. Homogentisate oxidase

■ *Listen to Audio 1*

Explanation: The clinical scenario described, including darkening of urine upon standing, brownish discolouration of the sclera and ear cartilage, and joint pain, is characteristic of Alkaptonuria , which is caused by a deficiency of the enzyme homogentisate 1,2-dioxygenase . Garrod's tetrad in alkaptonuria 1. Homogentisic Aciduria Presence of homogentisic acid in the urine, which turns dark upon standing or when exposed to air. 2. Ochronosis Deposition of dark pigment in connective tissues, particularly in the ear cartilage, sclera, and other connective tissues, leading to bluish-black discolouration. 3. Arthropathy Degenerative joint disease, especially affecting the spine and large joints, leading to chronic pain and stiffness. 4. Cardiac Involvement Includes heart valve calcification and atherosclerosis due to the deposition of pigment in cardiac tissues. 5. Ochronotic Spondylosis Spinal X-rays reveal intervertebral disc calcification and narrowing


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Tyrosine transaminase
Tyrosine
Parahydroxy phenylpyruvate
Parahydroxy phenylpyruvate dioxygenase
Homogentisate oxidase
Maleylacetoacetate
Homogentisic acid
Cis trans isomerase
Fumarylacetoacetate hydrolase
Fumarate
Fumarylacetoacetate
Acetoacetate
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References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 296

↑ *Back to this question in Part A*

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• [04_Amino Acids and Proteins Chemistry] 016 Glycine And One Carbon Pool

« Back to Folder Directory

[04_Amino Acids and Proteins Chemistry] 016 Glycine And One Carbon Pool — Quick Jump

Q1 (p. 826)	*Q2 (p. 827)*	*Q3 (p. 828)*	*Q4 (p. 829)*	*Q5 (p. 830)*	*Q6 (p. 831)*
Q7 (p. 832)	*Q8 (p. 833)*	*Q9 (p. 834)*	*Q10 (p. 836)*	*Q11 (p. 837)*	*Q12 (p. 838)*
Q13 (p. 839)	*Q14 (p. 841)*	*Q15 (p. 841)*			

Q1: Which of the following is not a source of glycine? (Q843353)

A. Serine

B. Threonine

C. Alanine

D. Lysine

Correct Answer: D. Lysine

Explanation: Glycine Simple, non-essential amino acid. Acts as a precursor for metabolites like creatine, glutathione, heme, purines, and porphyrins. Vital for protein synthesis, collagen structure, neurotransmission, immune response, and lipid digestion. Sources of Glycine: Serine : Converted to glycine via serine hydroxymethyltransferase (SMT), present in mitochondria and cytoplasm. (Option A) Threonine : Converted to glycine by threonine aldolase. (Option B) Choline : Converted to glycine via sarcosine and betaine pathways. Glycine Synthase : Synthesises glycine from CO■, NH■■, and a one-carbon unit. This multi-enzyme complex requires NAD, lipoamide, tetrahydrofolic acid, and pyridoxal phosphate. This is a reversal of the glycine cleavage system. 5.Glycine Aminotransferase : Catalyses glycine synthesis from glyoxylate and glutamate or alanine. (Option C)

References:

Harper's Illustrated Biochemistry, Edition 32, Page 275

Vasudevan Textbook of Biochemistry for Medical Students, Edition 7, Page 211

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5350494/

↑ *Back to this question in Part A*

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Q2: A 1 year old boy presents with lethargy, poor muscle tone, and developmental delays. Blood tests reveal elevated levels of glycine. The physician suspects a defect in the glycine cleavage system. Which of the following metabolites if found LOW, would support the diagnosis of a defective glycine cleavage system? (Q843443)

A. Serine

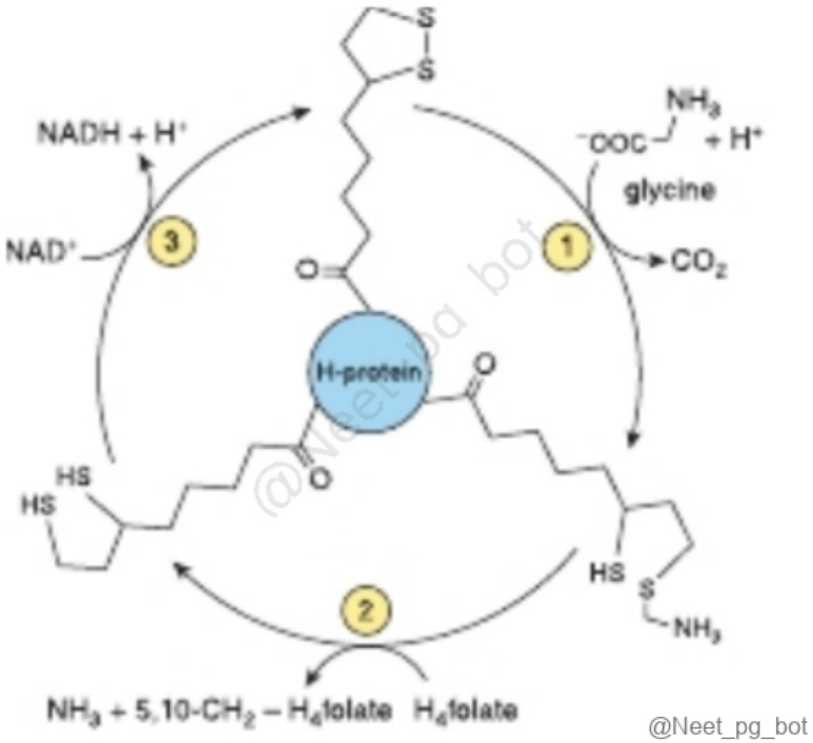
B. N5,N10 methylene tetrahydrofolate

C. Threonine

D. Succinyl CoA

Correct Answer: B. N5,N10 methylene tetrahydrofolate

Explanation: The glycine cleavage system directly produces N5,N10-methylene tetrahydrofolate when it breaks down glycine. A defect in this system would lead to a decreased production of this metabolite. Glycine Cleavage System: Located in liver mitochondria Splits glycine into carbon dioxide, ammonia and Forms N■, N¹■-methylenetetrahydrofolate (THFA). This process is the major catabolic route for glycine. Glycine Cleavage Complex : composed of three enzymes and an H-protein attached to dihydrolipoyl moiety. Subunits : 4 subunits. T: Tetrahydrofolate-dependent aminotransferase. P: Pyridoxal phosphate-dependent glycine decarboxylase. L: Lipoamide dependent dehydrogenase. H: Hydrogen or electron transport system. Co-enzymes of glycine cleavage system Tetrahydrofolate (THF) Pyridoxal phosphate (PLP) Lipoamide Mechanism : Glycine is broken down into CO2, NH4+, and a methylene group (–CH2–). Formation of N5, N10 methylene tetrahydrofolate : Methylene group from glycine is transferred to tetrahydrofolate (THF). Forms N5, N10 methylene tetrahydrofolate. Reaction : Glycine + H■-folate + NAD■ → CO■ + NH■ + 5,10-CH■-H■-folate + NADH + H■ Defects in glycine cleavage system : Nonketotic Hyperglycinemia: A rare inborn disorder where glycine accumulates in all body tissues, including the central nervous system. Primary Hyperoxaluria: Failure to break down glyoxylate from glycine deamination leads to oxalate formation, causing kidney stones, nephrocalcinosis, and potentially early death from renal failure or hypertension. Glycinuria: A defect in kidney reabsorption leads to excessive glycine in urine. Option A: Serine levels might even increase as the body tries to compensate for the glycine deficiency. Option C: A defective glycine cleavage system is unlikely to directly impact threonine levels. Option D: Succinyl CoA is involved in heme synthesis; defects in glycine breakdown wouldn't impact succinyl CoA levels.



References:

Harper 32nd edition Pg no. 293 Vasudevan Edition 7 page no:210,211 Image: Harper 32nd edition

↑ *Back to this question in Part A*

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Q3: A 6-month-old infant presented with lethargy, vomiting, and irritability. Blood tests revealed elevated ammonia levels. The infant was diagnosed with a urea cycle disorder. Which of the following medications, acting as ammonia-scavenging agents, would be most effective in reducing the infant's blood ammonia levels? (Q843444)

A. Phenyl acetate

B. N-acetyl glutamate

C. Lactulose

D. Neomycin

Correct Answer: A. Phenyl acetate

Explanation: Reducing blood ammonia levels: Ammonia-Scavenging Agent: Sodium benzoate : Reacts with glycine in the liver to form hippuric acid, which is then excreted in the urine reducing the availability of glycine. Reducing the availability of glycine shifts the equilibrium to favor glycine formation. Ammonia is used to produce glycine, therefore reducing blood ammonia levels. Phenylacetate : Conjugates with glutamine to form phenylacetylglutamine (PGA), which is then excreted in the urine, depleting glutamine levels. Glutamate production from ammonia and alpha-ketoglutarate increases to compensate for depletion. Each molecule of phenyl acetate utilizes two molecules of ammonia: one for the initial glutamine conjugation and one for subsequent glutamate-to-glutamine conversion. Protein degradation : Adequate caloric intake can help prevent the breakdown of protein and, subsequently, the production of ammonia. Urease-producing microorganisms : Certain bacteria in the gut produce urease, an enzyme that breaks down urea into ammonia. To prevent this, Low-dose neomycin: Helps suppress the growth of urease-producing bacteria. (Option D) Lactulose: Converts ammonia into ammonium ions which cannot be easily absorbed, thus reducing the amount of ammonia. (Option C) N-acetyl glutamate (Option B) is used in the treatment of hyperammonemia but does not directly scavenge ammonia. N-acetyl glutamate is a obligatory allosteric activator of Carbamoyl Phosphate Synthetase I (CPS-I), the rate-limiting enzyme of the urea cycle. NAG is synthesized from Acetyl-CoA and Glutamate by N-Acetylglutamate synthase. High levels of Arginine activate this synthase, providing feed-forward regulation to prime the urea cycle for protein-rich meals.

References:

www.uptodate.com/contents/urea-cycle-disorders-management

↑ *Back to this question in Part A*

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Q4: The glycine cleavage system in the body produces CO2, ammonia and a one carbon molecule. Which of the following is a subunit in the system? (Q843446)

A. Tetrahydrofolate

B. Lipoamide

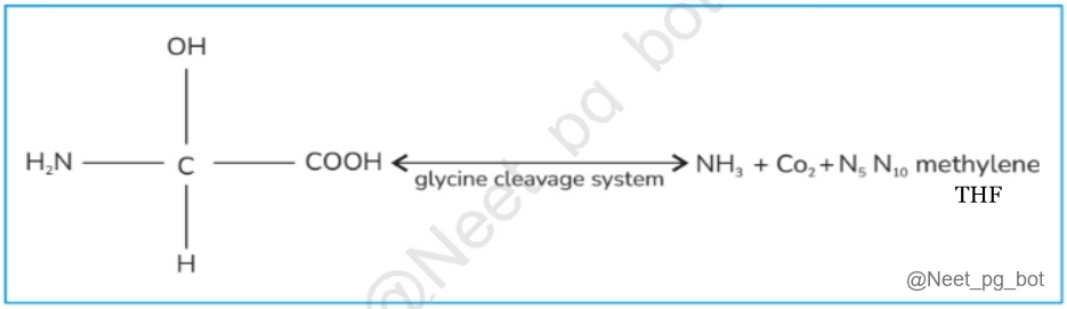
C. Tetrahydrofolate dependent aminomethyltransferase (THFAAMT)

D. Pyridoxal phosphate

Correct Answer: C. Tetrahydrofolate dependent aminomethyltransferase (THFAAMT)

Explanation: The subunit of the glycine cleavage system here is THFAAMT and the others are the coenzymes that take part in the system. The glycine cleavage system takes place in the liver mitochondria . In this system the simple amino acid glycine is split into ammonia, CO2 and the remaining methylene group (–CH2–) is transferred by Tetrahydrofolate (Option A) and is converted into N5N10 methylene tetrahydrofolate. Subunits of glycine cleavage system Co- enzymes of glycine cleavage system Tetrahydrofolate dependant aminotransferase (THFAAMT) (Option C) THF (Option A) Pyridoxal phosphate dependant(PLP) glycine decarboxylase Pyridoxal phosphate (PLP) (Option D) Lipoamide dependent dehydrogenase Lipoamide (Option B) Hydrogen or electron

transport system



References:

Harper's Illustrated biochemistry, 32nd edition, pg:293

↑ *Back to this question in Part A*

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Q5: Which of the following is not a known use of glycine in humans ? (Q843447)

A. Precursor for metabolites like creatine, glutathione, and heme.

B. Enhances the quality of sleep and supports neurological functions.

C. Inhibits synthesis of bile acid,reducing lipid absorption.

D. Provide structural integrity for collagen structure.

Correct Answer: C. Inhibits synthesis of bile acid,reducing lipid absorption.

Explanation: Uses of Glycine : Metabolic Functions : Precursor for metabolites such as creatine, glutathione, heme , purines, and porphyrins. (Option A) Vital for protein synthesis. Plays a crucial role in collagen structure by occupying every third position in the collagen triple helix. (Option D) Health and Disease Prevention : Dietary supplementation can prevent or treat metabolic disorders in patients with cardiovascular diseases, inflammatory diseases, obesity, cancers, and diabetes. Supports overall health, growth, and well-being in humans. Neurological and Sleep Benefits : Acts as a neurotransmitter in the central nervous system, influencing food intake, behavior, and body homeostasis. Enhances sleep quality and neurological functions. (Option B) Immune Function : Regulates immune responses by altering intracellular calcium levels. Modulates the production of superoxide and cytokines. Digestive Health : Facilitates the conjugation of bile acids. Aid in the absorption and digestion of lipid-soluble vitamins and lipids. (Option C)

References:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5350494/

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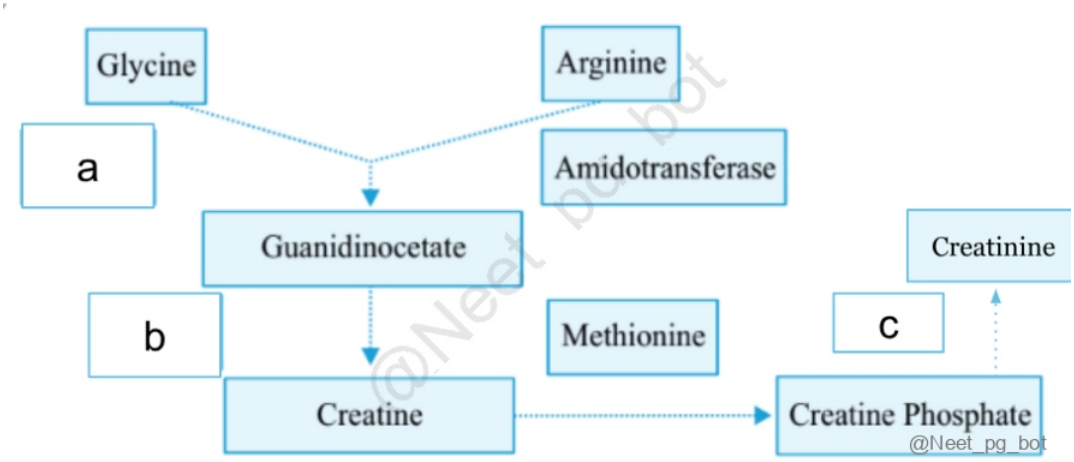
Q6: Based on the image given below, what are the sites at which the marked steps of creatinine synthesis occur? (Q843456)

A. a- brain, b- liver, c- kidney

B. a- kidney,b- liver, c- muscle

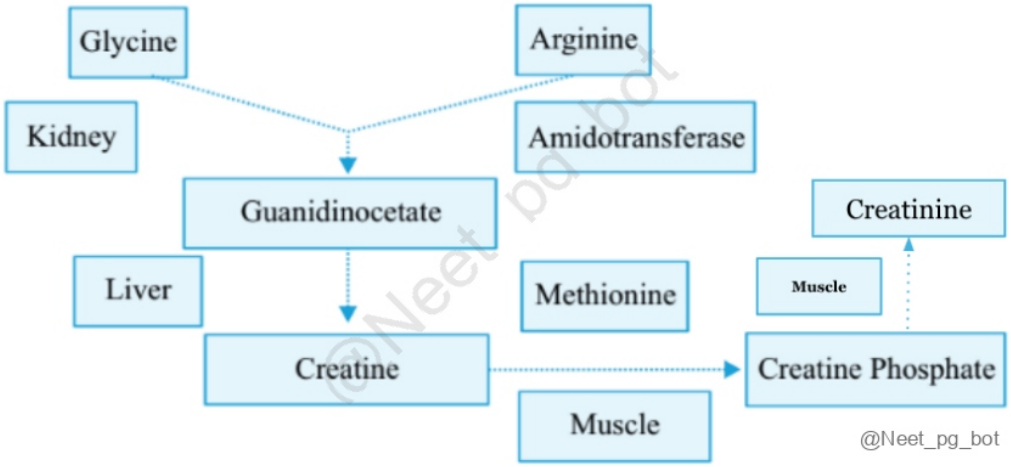
C. a- liver, b- kidney, c- muscle

D. a- muscle, b- liver, c-kidney



Correct Answer: B. a- kidney,b- liver, c- muscle

Explanation: Creatinine synthesis requires the enzyme creatine kinase which is found in the liver, kidney and muscle. Steps Enzyme Site 1. The Amidino group of arginine is transferred to glycine to form guanidinoacetic acid. Amidotransferase Kidney and pancreas (a) 2. Guanidoacetic acid is Methylated by S-adenosyl methionine to form creatine. Methyl transferase Liver (b) 3. Creatine is phosphorylated to creatine phosphate Creatine kinase Muscle, brain and liver. 4. Creatine phosphate may be converted to its anhydride, creatinine Non- enzymatic Muscle (c)



References:

Harper's Illustrated Biochemistry, 32nd edition, Page 311, 312.

Vasudevan Textbook of Biochemistry, 7th edition, Page 211.

↑ *Back to this question in Part A*

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Q7: Which of the following diseases is caused by the deficiency of the enzyme ■ ALA synthase? (Q843457)

A. Porphyria cutanea tarda

B. X linked sideroblastic anemia

C. Acute intermittent porphyria

D. Congenital erythropoietic porphyria

Correct Answer: B. X linked sideroblastic anemia

Explanation: The deficiency of ALA synthase causes X-linked sideroblastic anemia. Heme synthesis takes place in the bone marrow(85%) and hepatocytes . It starts with the condensation of succinyl-CoA and glycine . First step: Reaction takes place in mitochondria. Substrates used: Glycine. Glycine react with succinyl CoA (intermediate of citric acid cycle) in the presence of ■ ALA synthase to form ■ ALA. It is a decarboxylation reaction. Coenzyme : Pyridoxal phosphate. Prerequisite : Glycine should enter into mitochondria. Transporter : SLC25A38 Enzyme Defect Name of the Disorder ALA synthase II X linked sideroblastic anaemia (Option B) ALA Dehydratase ALA dehydratase deficient porphyria (ADP) Porphobilinogen deaminase (Hydroxymethylbilane synthase) Acute intermittent porphyria (AIP) (Option C) Uroporphyrinogen III synthase Congenital erythropoietic porphyria (most severe) (Option D) Uroporphyrinogen decarboxylase defect Porphyria cutanea tarda (PCT) (Option A)

Coproporphyrinogen oxidase Hereditary coproporphyria Protoporphyrinogen oxidase Variegate porphyria Ferrochelatase Erythropoietic protoporphyria (EPP)

References:

Harper's Illustrated Biochemistry, 32nd edition, pg:316, 317.

↑ *Back to this question in Part A*

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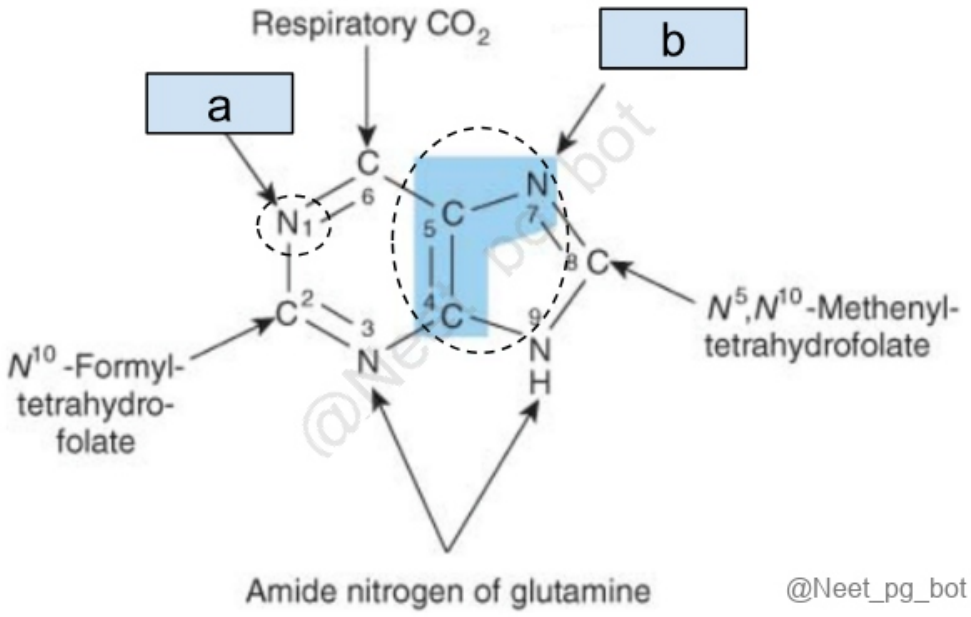
Q8: In the following figure, identify the sources of the marked components. (Q843458)

A. a- aspartate, b- glycine

B. a- glycine, b-aspartate

C. a- methionine, b-alanine

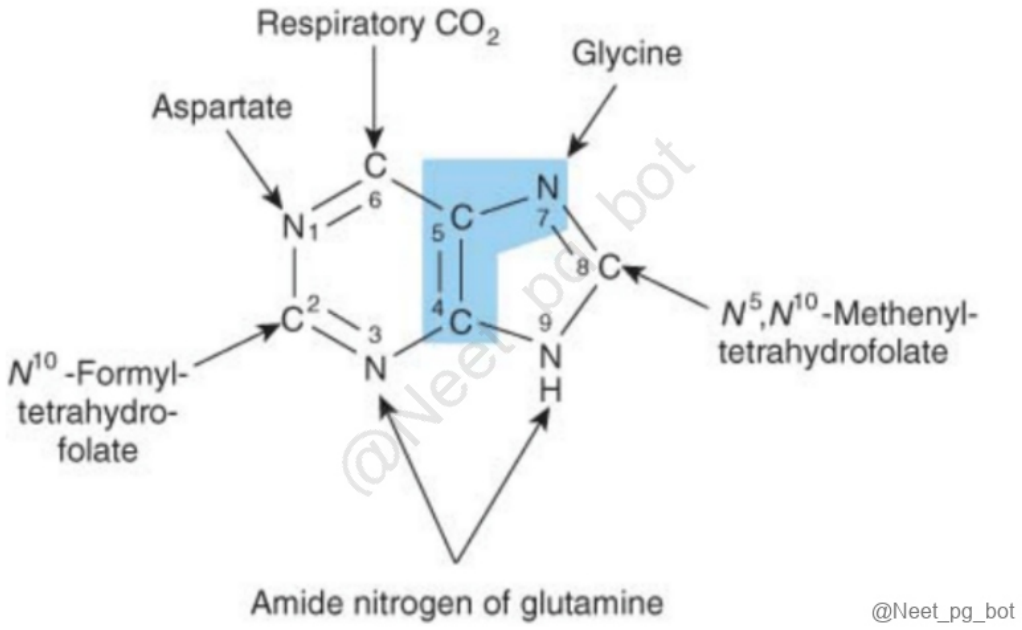
D. a- alanine, b- methionine



Correct Answer: A. a- aspartate, b- glycine

Explanation: Correct Answer: Option A) a- aspartate, b- glycine The above marked nitrogen atom is derived from the amino acid aspartate and the carbon atoms from glycine . The major site is liver . Requires three amino acids - Glycine, Aspartate, Glutamine and the cofactor N10-formyl tetrahydrofolate (N10- formyl THF) for purine ring formation. The sources of each atom in a purine was found by feeding isotopic precursors of uric acid to pigeons.

The C-2 atom is from formyl THF The C-8 atom is from N5N10 methenyl THF The C-6 atom is from respiratory CO2 . N1 is from aspartate N3 and N9 are from Glutamine C4, C5 and N7 are from Glycine Methionine and alanine have no relation to purine synthesis.



References:

Harper's Illustrated biochemistry, 32nd edition, Page 338, 339

Vasudevan textbook of biochemistry, 7th edition, Page 208

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7869020/

https://basicmedicalkey.com/metabolism-of-purine-pyrimidine-nucleotides/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q9: Which of the following statements is incorrect regarding one-carbon derivatives of tetrahydrofolic acid (THFA) and their associated enzymes and reactions? (Q843459)

A. N5-formimino-THFA converts formiminoglutamate (FIGLU) to glutamate.

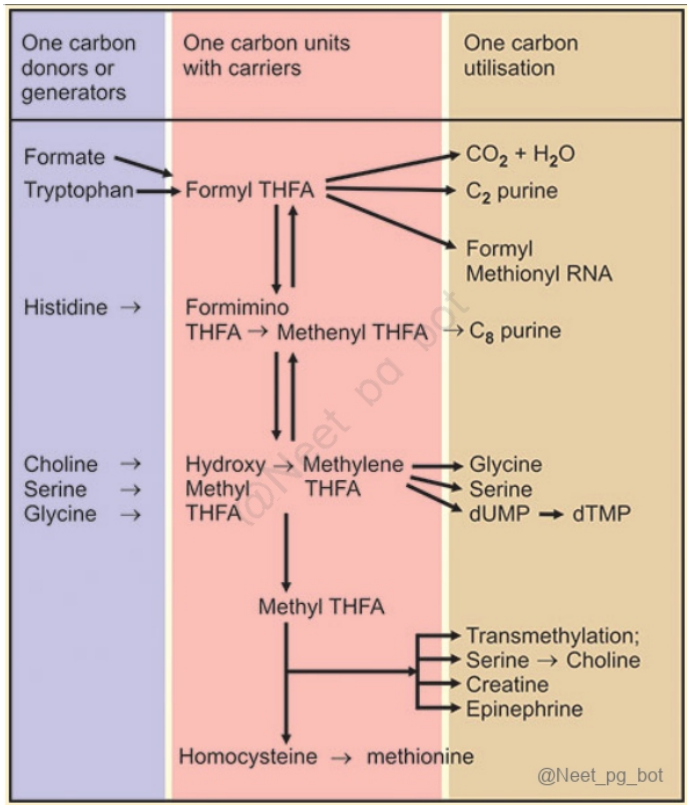
B. N5-methyl-THFA carries the methyl group for converting homocysteine to methionine.

C. N5N10-methylene-THFA is required for the synthesis of thymidylate (dTMP) from deoxyuridylate (dUMP).

D. N10-formyl-THFA transfers formyl groups in pyrimidine synthesis.

Correct Answer: D. N10-formyl-THFA transfers formyl groups in pyrimidine synthesis.

Explanation: N10-formyl-THFA transfers formyl groups in purine synthesis , forming C2 of the purine ring. One-Carbon Compounds: One-Carbon Derivatives of THFA Description Source Utilization N5-formyl-THFA and N10-formyl-THFA (Option D) Carry a formyl group (-CHO) Tryptophan Synthesis of purine nucleotides, forming C2 of the purine ring. N5-formimino-THFA (Option A) Carries a formimino group (-CH=NH) Histidine via FIGLU Involved in the conversion of formiminoglutamate (FIGLU) to glutamate, a step in histidine catabolism; Enzyme: Formiminoglutamate hydrolase N10-hydroxymethyl-THFA Carries a hydroxymethyl group (-CH2OH) Choline and betaine N5, N10-methylene-THFA (Option C) Carries a methylene group (-CH2-) Serine via serine hydroxymethyl transferase, glycine Glycine synthesis (conversion of serine to glycine), Thymidine synthesis (conversion of dUMP to dTMP); Enzyme: Thymidylate synthase N5-methyl-THFA (Option B) Carries a methyl group (-CH3) All one-carbon units ultimately form N5-methyl-THFA Methionine synthesis (conversion of homocysteine to methionine); Enzyme: Methionine synthase Precursor for S-adenosylmethionine (SAM) for various transmethylation reactions.



References:

Reference: Harper's Illustrated Biochemistry (32nd Edition), Page 545. Vasudevan Edition 7 Pg 207, 208. Images: Vasudevan Edition 7 Pg 208.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q10: A 62-year-old female patient presents with fatigue, pallor, and glossitis. Laboratory tests indicate macrocytic anemia, elevated homocysteine and methylmalonic acid levels. Given these findings, what is the most likely metabolic pathway disrupted by this patient's condition, leading to the observed anemia? (Q843462)

A. Conversion of homocysteine to methionine

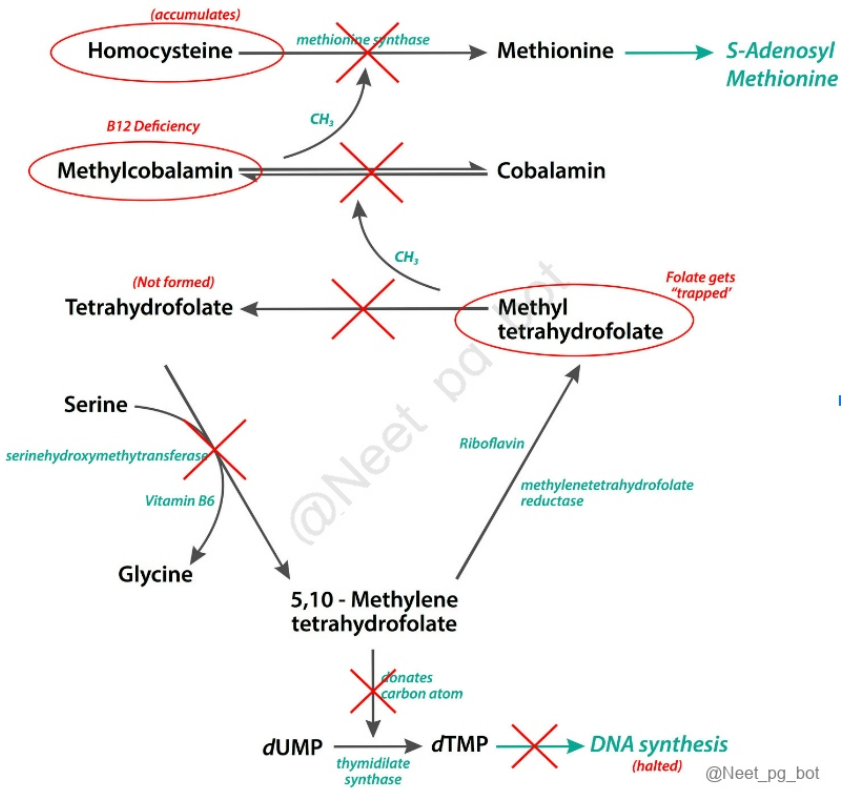
B. Reduction of folic acid to dihydrofolate

C. Deamination of histidine to formimino glutamate

D. Conversion of Phenylalanine to Tyrosine

Correct Answer: A. Conversion of homocysteine to methionine

Explanation: Macrocytic anemia, elevated homocysteine and methylmalonic acid levels are consistent with Vitamin B12 deficiency. Conversion of homocysteine to methionine is disrupted in vitamin B12 deficiency. Vitamin B12 is a cofactor for methionine synthase, the enzyme responsible for converting homocysteine to methionine, which in turn forms S-Adenosyl methionine, necessary for the synthesis of nitrogenous bases in DNA and RNA. In the absence of vitamin B12, the N5-methyl tetrahydrofolate (N5-methyl THF) is trapped, leading to a functional folate deficiency and consequently, macrocytic anemia■. This " methyl folate trap " hypothesis explains why folate becomes metabolically inactive. Administering folate to a B12-deficient patient bypasses the need for B12 in DNA synthesis (correcting anemia), but leaves the neurological damage untreated . This worsens the undiagnosed B12 deficiency. Neurological Hallmark: Subacute Combined Degeneration (SACD) of the spinal cord (dorsal column & lateral corticospinal tract involvement), leading to sensory loss and spasticity. Folate Trap Outline: Option B: Reduction of folic acid to dihydrofolate is catalyzed by dihydrofolate reductase and is unrelated to the vitamin B12-dependent processes. It is involved in the conversion of folic acid (the synthetic form) into its active forms. Option C: Deamination of histidine to formimino glutamate is related to folate metabolism but is more directly associated with diagnosing folate deficiency by the presence of FIGLU in the urine. Option D: Conversion of Phenylalanine to Tyrosine is catalyzed by Phenylalanine hydroxylase, which does not requires vitamin B12 as a cofactor. Deficiency Primary Effect Biochemical Marker Clinical Outcome Vitamin B12 Impaired conversion of N5-methyl THF to THF Elevated homocysteine and Methylmalonic acid Megaloblastic anemia, peripheral neuropathy Folate Reduced synthesis of THF from dietary folate forms Accumulation of FIGLU in urine, Elevated homocysteine and normal Methylmalonic acid Megaloblastic anemia



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 546.

https://www.firstclassmed.com/articles/2017/folate-trap

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: In the kynurenine pathway of tryptophan catabolism, which of the following compounds directly contributes to the formation of N10-formyl tetrahydrofolate in the one-carbon pool? (Q843463)

A. Formyl kynurenine

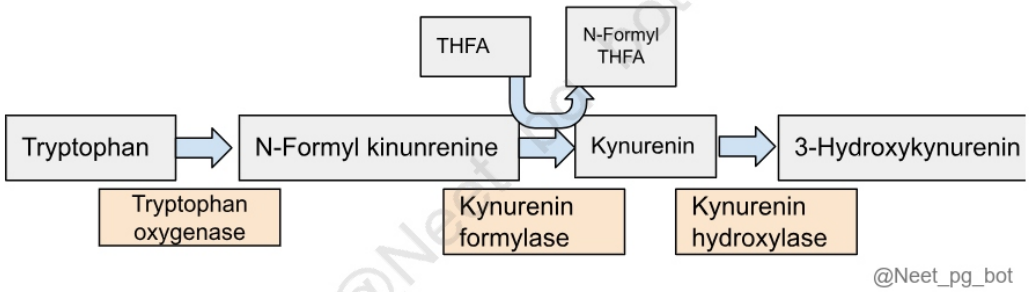
B. Kynurenine

C. 3- Hydroxy kynurenine

D. THFA

Correct Answer: A. Formyl kynurenine

Explanation: Formylkynurenine is a key intermediate in the kynurenine pathway. It is directly involved in transferring a formyl group, which leads to the production of N10-formyl THFA, an important one-carbon donor in nucleotide biosynthesis■. Tryptophan Catabolism and One-Carbon Pool Contributions Compound Role in Tryptophan Catabolism Contribution Formylkynurenine (Option A) Intermediate; releases formate for N10-formyl THFA Directly contributes to the formation of N10-formyl THFA Kynurenine (Option B) Intermediate; precursor for other metabolites Participates in the synthesis of other metabolites like quinolinic acid and nicotinic acid■. 3-Hydroxy kynurenine (Option C) Intermediate; precursor for other metabolites Product formed from formate derived from formylkynurenine THFA (Option D) Carrier of one-carbon units Not directly derived from tryptophan catabolism



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 299.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q12: Which enzyme in the histidine catabolism pathway facilitates the incorporation of the formimino group into the one-carbon pool, forming N5-formimino tetrahydrofolate? (Q843464)

A. Histidase

B. Urocanase

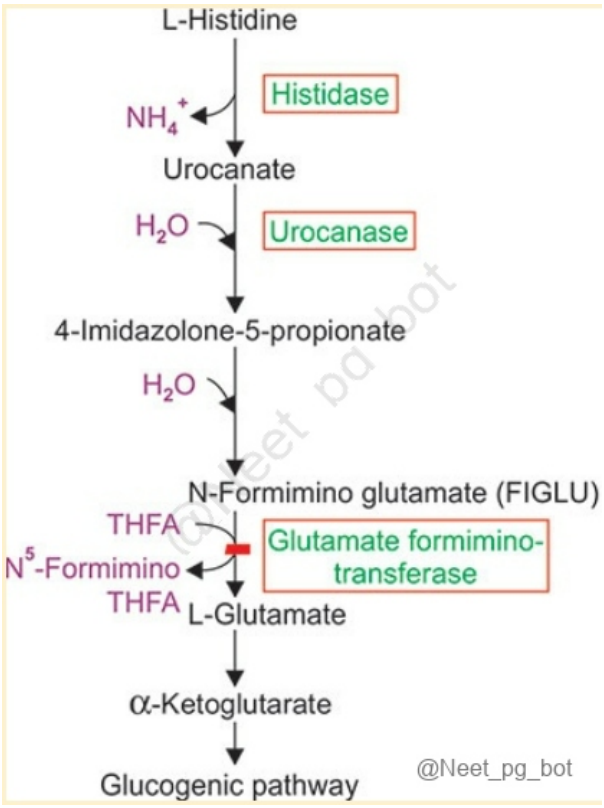
C. Glutamate formimino transferase

D. Glutamate dehydrogenase

Correct Answer: C. Glutamate formimino transferase

Explanation: Glutamate Formimino transferase catalyzes the transfer of the formimino group from formiminoglutamate (FIGLU) to THFA, forming N5-formimino THFA■. Catabolism of Histidine to α-ketoglutarate: Reaction Substrate Enzyme Product 1. Histidine Histidase (Option A) Urocanate 2. Urocanate Urocanase (Option B) Imidazole propionic acid 3. Imidazole propionic acid Hydrolase Formiminoglutamate

(FIGLU) 4. FIGLU Glutamate Formimino transferase (Option C) L-Glutamate 5. Glutamate Glutamate dehydrogenase (Option D) α-Ketoglutarate Histidine to α-Ketoglutarate Pathway: Role of Tetrahydrofolate (THF) Histidine is metabolised to α-ketoglutarate, which subsequently enters the TCA cycle. Role of Tetrahydrofolate (THF): Histidine is broken down into formiminoglutamate (FIGLU). THF plays a critical role in converting FIGLU to glutamate. THF Function: THF accepts the formimino group (FIG) from FIGLU to become 5-formimino tetrahydrofolate (FITHFA). As a result of losing formimino group (FIG), FIGLU becomes glutamate. Folate Deficiency Consequences: Without sufficient THF, FIGLU cannot transfer its formimino group. FIGLU builds up and is excreted in urine, especially after a histidine load. Histidine Load Test: Used to diagnose folate deficiency by detecting elevated levels of FIGLU in urine after a histidine load.



References:

Harper's Illustrated Biochemistry (32nd Edition), Page 292 - 293. Vasudevan 7th Edition, Pg 244, 207. Images: https://www.jaypeedigital.com/eReader/chapter/9789386322401/ch1

↑ *Back to this question in Part A*

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Q13: A 3-week-old infant presents with poor feeding, lethargy, and vomiting. Lab results show elevated ammonia levels, increased glutamine and increased orotic acid in the blood, with no evidence of megaloblastic anemia. Which enzyme deficiency is likely responsible for this condition? (Q845009)

A. Ornithine transcarbamylase (OTC)

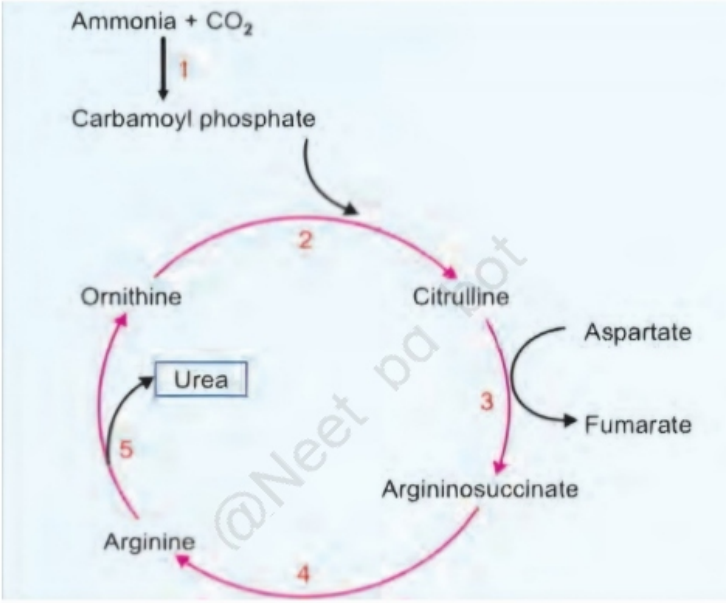
B. Ornithine aminotransferase

C. Uridine monophosphate (UMP) synthase

D. Hypoxanthine guanine phosphoribosyltransferase (HGPRT)

Correct Answer: A. Ornithine transcarbamylase (OTC)

Explanation: Hyperammonemia type 2 is an X-linked deficiency caused by a defect in ornithine transcarbamoylase (this reaction occurs in mitochondria), which forms citrulline in the Krebs cycle. Mothers with this condition may have hyperammonemia and avoid high-protein foods. Elevated blood, cerebrospinal fluid, and urine glutamine levels are due to increased glutamine synthesis in response to high ammonia levels. Orotic aciduria - Due to the channelling of carbamoyl phosphate into pyrimidine synthesis.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 287-288.

Vasudevan Textbook of Biochemistry for Students, Edition 7, Page 204,206

↑ *Back to this question in Part A*

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Q14: A patient with a urea cycle disorder is being evaluated. Which urea cycle intermediate serves as the link between the urea cycle and the TCA cycle? (Q845010)

A. Argininosuccinate

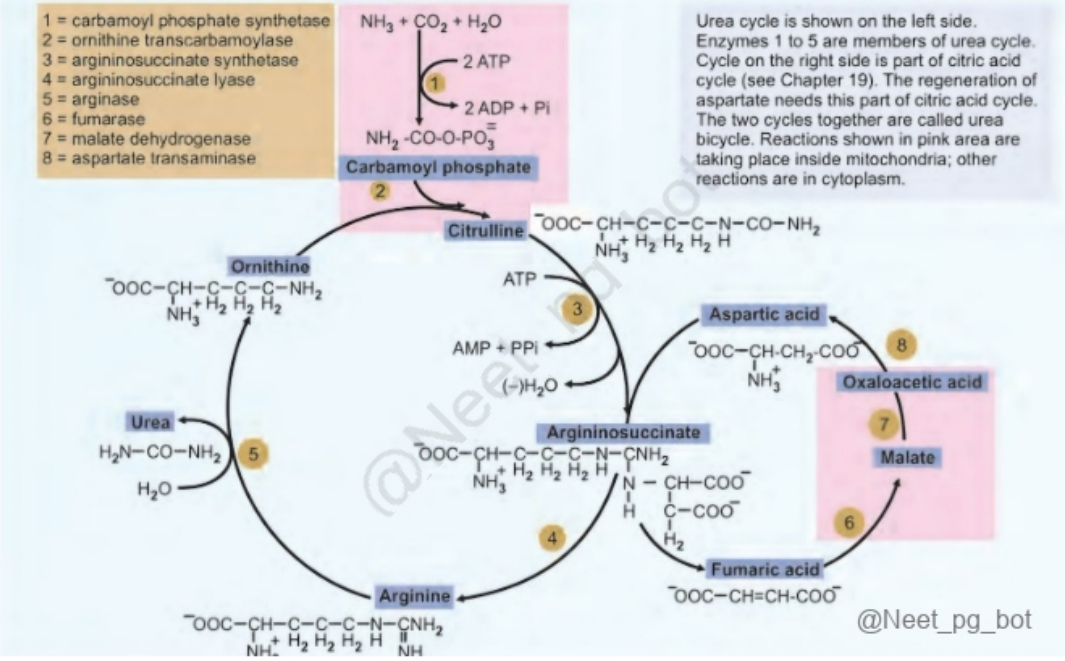
B. Fumarate

C. Oxaloacetate

D. Succinate

Correct Answer: B. Fumarate

Explanation: Argininosuccinate lyase cleaves argininosuccinate to arginine and fumarate. Fumarate is converted to malate and then to oxaloacetate in the TCA cycle , which is transaminated to aspartate . This links the urea cycle with the TCA cycle. The reaction can be summarised as: Citrulline + aspartate → Arginine + fumarate.



References:

Vasudevan Edition 7 page no:204,205

↑ *Back to this question in Part A*

@Neet_pg_bot

Q15: Arrange the following steps of the synthesis of creatinine from glycine in the correct order: A. Transport of creatine from the liver to muscle via blood B. Formation of guanidino acetic acid from arginine and glycine C. Formation of creatine by methyl transferase D. Formation of creatine phosphate E. Conversion of creatine phosphate to creatinine (Q875957)

A. B→ C→A→ D → E

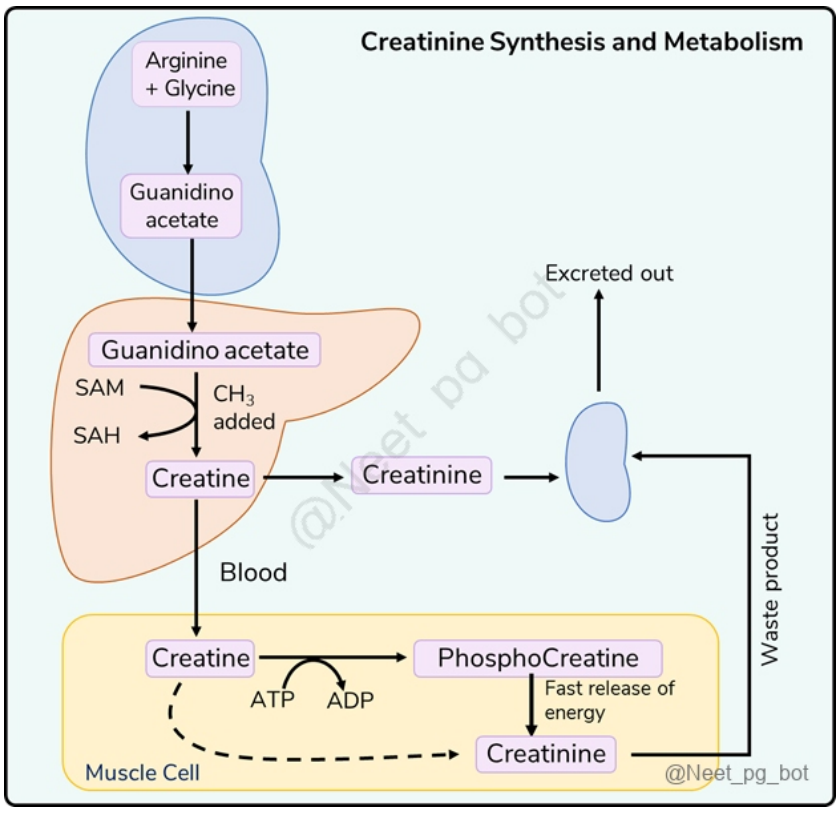
B. A→ D→C→ E → B

C. D→ A→E→ C → B

D. C→ D→B→ E → A

Correct Answer: A. B→ C→A→ D → E

Explanation: Correct Option A - B→ C→A→ D → E: Creatine phosphate serves as a rapidly mobilized reserve of high-energy phosphate molecules, transferring phosphate groups to ADP to maintain ATP levels during intense muscle contraction. Creatine synthesis involves: Formation of guanidinoacetate from arginine and glycine by amidinotransferase in the kidneys. Transfer of a methyl group from S-Adenosylmethionine by methyltransferase in the liver to guanidinoacetate, forming creatine. Transport of creatine from the liver to muscles. Reversible phosphorylation of creatine to creatine phosphate by creatine kinase, using ATP. Spontaneous cyclization of creatine phosphate to form creatinine, excreted in urine.



References:

Harper 31st/e pg. 52

↑ *Back to this question in Part A*

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• [04_Amino Acids and Proteins Chemistry] 017 Previous Year Questions

« Back to Folder Directory

[04_Amino Acids and Proteins Chemistry] 017 Previous Year Questions — Quick Jump

Q1 (p. 844)	*Q2 (p. 845)*	*Q3 (p. 846)*	*Q4 (p. 847)*	*Q5 (p. 848)*	*Q6 (p. 849)*
Q7 (p. 850)	*Q8 (p. 851)*	*Q9 (p. 852)*	*Q10 (p. 852)*	*Q11 (p. 854)*	*Q12 (p. 855)*
Q13 (p. 856)	*Q14 (p. 857)*	*Q15 (p. 857)*	*Q16 (p. 858)*	*Q17 (p. 859)*	*Q18 (p. 859)*
Q19 (p. 860)	*Q20 (p. 861)*	*Q21 (p. 864)*	*Q22 (p. 865)*	*Q23 (p. 866)*	*Q24 (p. 866)*
Q25 (p. 867)	*Q26 (p. 868)*	*Q27 (p. 869)*	*Q28 (p. 870)*	*Q29 (p. 870)*	*Q30 (p. 872)*
Q31 (p. 873)	*Q32 (p. 874)*	*Q33 (p. 874)*	*Q34 (p. 877)*	*Q35 (p. 877)*	*Q36 (p. 878)*
Q37 (p. 879)	*Q38 (p. 880)*	*Q39 (p. 881)*	*Q40 (p. 881)*	*Q41 (p. 882)*	*Q42 (p. 883)*
Q43 (p. 884)	*Q44 (p. 885)*	*Q45 (p. 888)*	*Q46 (p. 888)*	*Q47 (p. 889)*	*Q48 (p. 890)*
Q49 (p. 890)	*Q50 (p. 892)*	*Q51 (p. 892)*	*Q52 (p. 893)*	*Q53 (p. 894)*	*Q54 (p. 894)*
Q55 (p. 895)	*Q56 (p. 896)*	*Q57 (p. 897)*	*Q58 (p. 898)*	*Q59 (p. 898)*	*Q60 (p. 899)*
Q61 (p. 900)	*Q62 (p. 901)*	*Q63 (p. 902)*	*Q64 (p. 903)*	*Q65 (p. 904)*	*Q66 (p. 905)*
Q67 (p. 906)	*Q68 (p. 907)*	*Q69 (p. 908)*	*Q70 (p. 908)*	*Q71 (p. 909)*	*Q72 (p. 910)*
Q73 (p. 911)	*Q74 (p. 912)*	*Q75 (p. 913)*	*Q76 (p. 913)*	*Q77 (p. 914)*	*Q78 (p. 915)*
Q79 (p. 916)	*Q80 (p. 916)*	*Q81 (p. 917)*			

Q1: Identify the test: (Q854626)

A. Rothera’s test

B. Benedict’s test

C. Fehling’s test

D. Barfoed’s test



Correct Answer: A. Rothera’s test

Explanation: Rothera's test: The presence of ketosis can be established by the detection of ketone bodies in urine by Rothera's test. Method-Saturate 5 mL of urine with solid ammonium sulfate. Add a few drops of freshly prepared sodium nitroprusside followed by 2 mL of liquor ammonia along the sides of the test tube. Development of a purple ring indicates the presence of ketone bodies in urine.

References:

Vasudevan’s Textbook of Biochemistry for Medical Students, 7th Edition, Page 166

↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: A chronic alcoholic patient is diagnosed with gouty arthritis. What biochemical changes will be seen in this patient? (Q855230)

A. Decreased NADH/NAD+ ratio

B. Increased urea and urate levels

C. Increased urate and lactate levels

D. Increased level of alkaline phosphatase

Correct Answer: C. Increased urate and lactate levels

Explanation: Increased Uric Acid (Hyperuricemia): Chronic alcohol consumption is a significant contributor to hyperuricemia, a condition characterized by elevated levels of uric acid in the blood. This occurs due to: Increased turnover of purines from alcohol metabolism. Decreased renal excretion of uric acid as lactate competes with uric acid for excretion through the kidneys. These factors increase the likelihood of uric acid crystal deposition in joints, triggering gouty arthritis. Increased Lactate Levels (Lactic Acidosis): Alcohol metabolism, particularly through the enzyme alcohol dehydrogenase, generates an increased NADH/NAD+ ratio. This shift favors the conversion of pyruvate to lactate, leading to lactic acidosis. Elevated lactate levels further impair the kidney's ability to excrete uric acid, exacerbating hyperuricemia and contributing to gout attacks. Decreased NADH/NAD+ ratio (Option A): Alcohol metabolism increases the NADH/NAD+ ratio, which indirectly contributes to lactate production and hyperuricemia. Increased urea and urate levels (Option B): While urate levels increase, chronic alcohol use does not typically elevate urea levels unless there is concurrent kidney damage or protein metabolism disturbances. Increased level of alkaline phosphatase (Option D): This is associated with conditions like bone or liver disease but is not a key feature of gouty arthritis linked to alcohol consumption.

References:

Is lactic acid a misunderstood trigger of gout attack for a century? - ScienceDirect Gout Symptoms, Causes & Diet Recommendations | NIAMS

↑ *Back to this question in Part A*

@Neet_pg_bot

Q3: Which of the following does not contribute to the repair of DNA double-stranded breaks? (Q857361)

A. ATP

B. RAD-51

C. ATM

D. Ku 70

Correct Answer: A. ATP

Explanation: ATP is not directly involved in the repair of DNA double-stranded breaks (DSBs), but it provides the energy necessary for many cellular processes, including DNA repair. Homologous DNA Repair (HR) HR is a type of Double-strand DNA break repair mechanism used to fix double-strand breaks (DSBs) by utilizing a homologous DNA template for error-free repair. HR is active during the S and G2 phases of the cell cycle. RAD 51 and ATM are involved in the G2 phase Nonhomologous End Joining (NHEJ) NHEJ is a DNA repair mechanism that fixes double-strand breaks (DSBs) by directly joining the broken ends without requiring a homologous template. Active throughout the entire cell cycle, including G0/G1 phases. Ku70 is involved in the G1

phase

References:

Harper 32nd edition, pg 380

Lippincott 7th edition, 1219-1222

Holliday junction

↑ *Back to this question in Part A*

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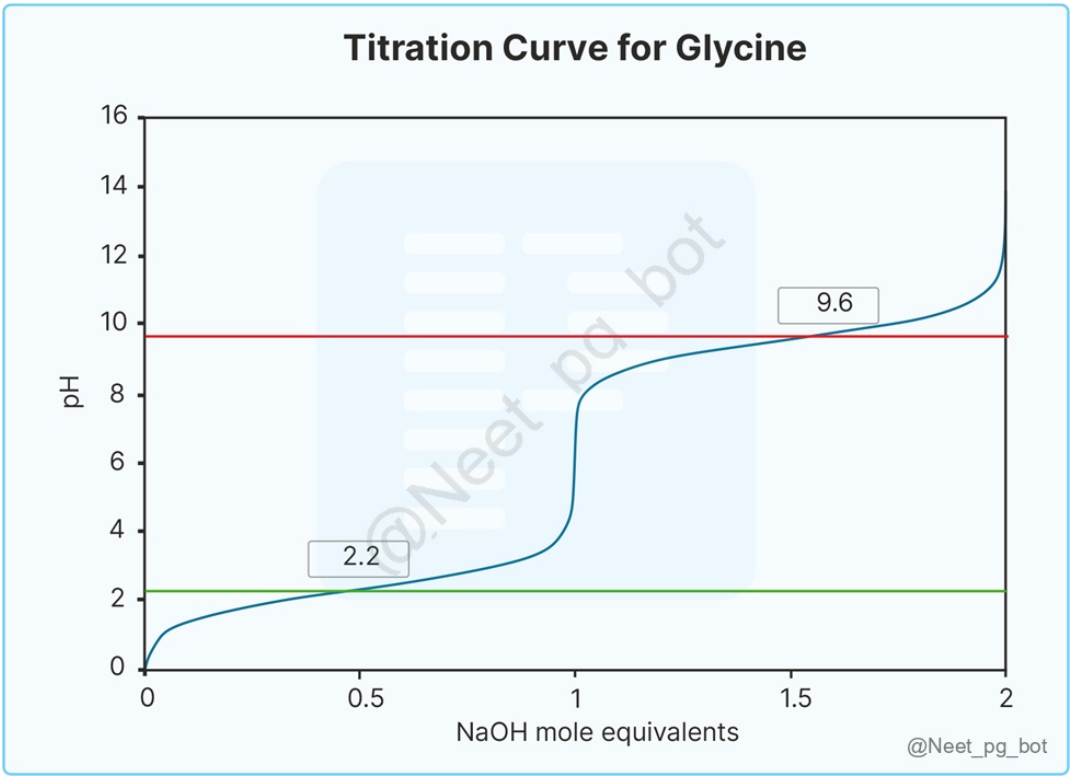
Q4: In the given glycine titration curve, calculate or identify the pH at which molecules exist in a zwitter ion form? (Q865428)

A. 4.4

B. 6

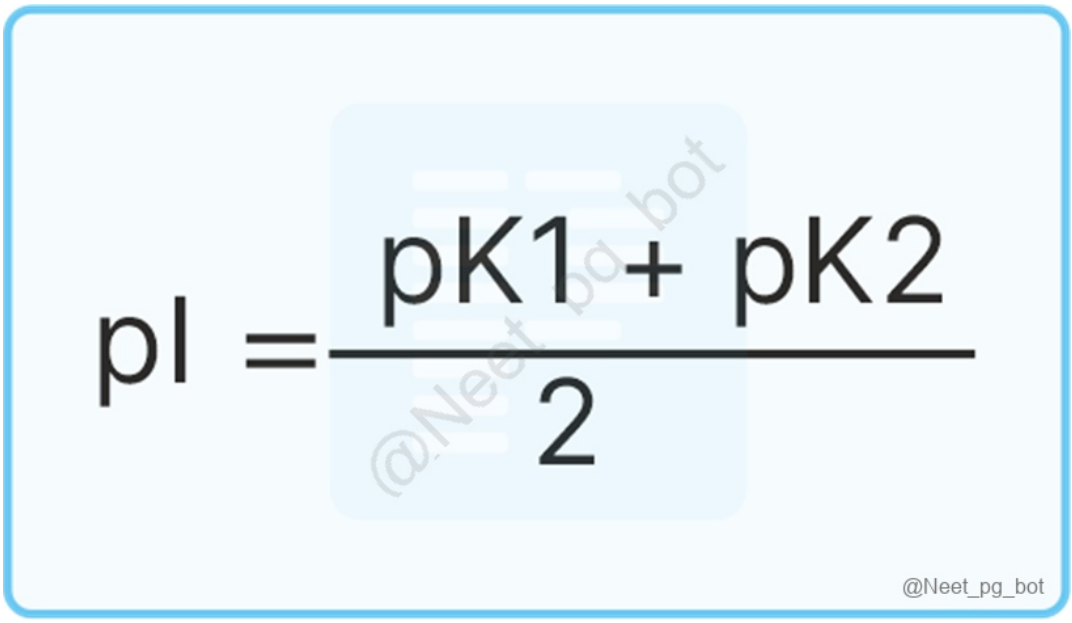
C. 9

D. 2.2



Correct Answer: B. 6

Explanation: In the given glycine titration curve, the pH at which the molecules predominantly exist in their zwitterionic form is 6. Isoelectric pH It is the pH at which a molecule carries no net electrical charge. For proteins and amino acids, this is the pH where the molecule exists as a “zwitter ion” with equal numbers of positive and negative charges, resulting in a net zero charge and no migration in an electric field. The isoelectric pH (pI) for monoamino monocarboxylic amino acids can be calculated: In the above question, it will be = (2.2 + 9.6)/2 = 5.9 ■ 6 .



References:

Vasudevan’s Textbook of Biochemistry, 7th Edition, Page 28.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q5: A female patient presented with pain and redness in the great toe. The serum uric acid level is 9.4mg/dl. The physician prescribed a drug for the treatment. Which of the following enzyme is likely to be inhibited by this drug? (Q859691)

A. Phosphoribosyl transferase

B. Thymidylate synthase

C. Xanthine oxidase

D. Dihydrofolate reductase

Correct Answer: C. Xanthine oxidase

Explanation: The clinical scenario describes a female patient with pain and redness in the great toe, which suggests gout, a condition caused by elevated uric acid levels. The patient's serum uric acid level of 9.4 mg/dl is above the normal range, indicating hyperuricemia, which can lead to the formation of uric acid crystals in the joints, causing pain and inflammation. The physician has prescribed a drug that helps manage gout by reducing uric acid levels. The drug prescribed is allopurinol, which is known to inhibit xanthine oxidase, the enzyme responsible for converting hypoxanthine to xanthine and xanthine to uric acid. By inhibiting xanthine oxidase, allopurinol decreases the production of uric acid, effectively managing gout by lowering uric acid levels in the body. Phosphoribosyl transferase (Option A): Involved in purine metabolism, but not the target of allopurinol. Thymidylate synthase (Option B): Involved in DNA synthesis, not related to gout treatment. Dihydrofolate reductase (Option D): Involved in folate metabolism, and inhibited by drugs like methotrexate, not relevant to gout treatment.

References:

Harper’s Illustrated Biochemistry (23rd Edition), Page 81,82.

Vasudevan Textbook of Biochemistry (7th Edition), Page 62.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: What is true about the Hayflick limit? (Q856475)

A. Human cells can divide a finite number of times only

B. Free radical oxidation level is unrestricted

C. Telomerase activity is unrestricted

D. RNA polymerase can function for a finite number of time only

Correct Answer: A. Human cells can divide a finite number of times only

Explanation: The Hayflick limit , discovered by Dr. Leonard Hayflick, states that normal human cells have a finite capacity for replication. Telomeres, protective caps at chromosome ends, shorten with each cell division due to incomplete DNA replication. Shortened telomeres eventually lead to cellular senescence, preventing uncontrolled cell division and potential cancer. Some cells, like stem cells and certain cancers, express telomerase, extending telomeres and bypassing the Hayflick limit. In normal human cells, telomerase activity is limited, leading to replicative senescence. The Hayflick limit , the maximum times cells can divide, is influenced by several factors: Telomere Length: Shortening telomeres indicate reduced division capacity. Telomerase Activity: Higher activity extends telomeres, bypassing the limit. DNA Damage: Accumulated damage triggers senescence, limiting division. Cellular Stress: Various stressors accelerate senescence, reducing the limit. Genetic Factors: Variations affect telomere maintenance and DNA repair. Environmental Factors: Lifestyle, pollutants, and stress impact cellular health, affecting the limit. Free radical oxidation level is unrestricted (Option B): The Hayflick limit is not

directly related to free radical oxidation levels. Telomerase activity is unrestricted (Option C): Telomerase activity is typically restricted in normal human cells, unlike stem cells and certain cancers. RNA polymerase can function for a finite number of times only (Option D): The Hayflick limit primarily involves telomere shortening, not RNA polymerase function.

References:

Hayflick limit

↑ *Back to this question in Part A*

@Neet_pg_bot

Q7: Which of the following post-translational modifications is common to collagen, hemoglobin, and immunoglobulins? (Q860100)

A. Hydroxylation

B. Subunit aggregation

C. Poly A tail addition

D. DNA mismatch repair

Correct Answer: B. Subunit aggregation

Explanation: Collagen forms a functional triple helix through aggregation of three polypeptide chains, stabilized by hydrogen bonds and hydroxyproline residues. Hemoglobin is a functional tetramer, composed of two α-globin and two β-globin chains, which aggregate to carry oxygen efficiently. Immunoglobulins (antibodies) consist of two heavy and two light chains, which aggregate to form the functional Y-shaped molecule. In all three proteins, subunit aggregation is a crucial post-translational event for proper folding, stability, and biological function. Incorrect Options: Hydroxylation (Option A): This is a post-translational modification involving the addition of hydroxyl groups to amino acids. While hydroxylation is important in collagen synthesis, it is not a common modification for hemoglobin and immunoglobulins. Poly A tail addition (Option C): This is a post-transcriptional modification that involves adding a polyadenine tail to the 3' end of mRNA. It helps in mRNA stability and translation efficiency but does not pertain to the protein structures in question. DNA mismatch repair (Option D): DNA mismatch repair is a genomic maintenance process that corrects errors in DNA replication. It is unrelated to protein post-translational modifications.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg. 35-40

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg. 604

↑ *Back to this question in Part A*

@Neet_pg_bot

Q8: A patient presents with dermatitis, dementia, and cognitive decline. Which micronutrient deficiency is most likely responsible? (Q844235)

A. Niacin

B. Thiamine

C. Tryptophan

D. Riboflavin

Correct Answer: A. Niacin

Explanation: The Clinical scenario of Dermatitis, Dementia and Cognitive decline suggests Niacin deficiency (Pellagra) Pellagra is characterized by the classic triad of symptoms: Dermatitis Bright red erythema- in sun-exposed areas (face, ankles, feet) Casal's necklace- ↑pigmentation around the neck Diarrhoea Dementia {{caption_text}} Incorrect Options: Thiamine (Option B) (Vitamin B1) deficiency typically presents with symptoms related to the nervous system, such as peripheral neuropathy, cardiovascular abnormalities, and Wernicke-Korsakoff syndrome. Tryptophan (Option C) plays a crucial role in various metabolic processes. It serves as a precursor for Niacin (Vitamin B3) and Serotonin. Thus symptoms like mood alteration, decreased/increased appetite is also present. Riboflavin (Option D) deficiency causes cheilosis, glossitis, seborrheic dermatitis and proliferation of bulbar conjunctival capillaries.



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg.542,543 Textbook of Biochemistry- Vasudevan, 7th Edition, Pg. 239-242,477,479,481,482

↑ *Back to this question in Part A*

@Neet_pg_bot

Q9: Which of the following is deficient in a patient with phenylketonuria with normal phenyl alanine hydroxylase levels? (Q844228)

A. Tyrosine transaminase

B. Dopa decarboxylase

C. Tyrosine hydroxylase

D. Dihydrobiopterine reductase

Correct Answer: D. Dihydrobiopterine reductase

Explanation: In malignant phenylketonuria , the enzyme phenylalanine hydroxylase is normal, but there is a deficiency of tetrahydrobiopterin (BH■) due to lack of dihydrobiopterin reductase . Without BH■, phenylalanine cannot be converted to tyrosine, leading to high phenylalanine levels despite normal PAH. Classic phenylketonuria (PKU) results from a deficiency of phenylalanine hydroxylase (PAH) , which converts phenylalanine → tyrosine using tetrahydrobiopterin (BH■) as a cofactor. However, in some cases, PAH enzyme activity is normal , but there is a deficiency of dihydrobiopterin reductase (DHPR) , which regenerates BH■ from BH■ . This leads to a functional deficiency of BH■ , resulting in accumulation of phenylalanine and reduced synthesis of dopamine, norepinephrine, and serotonin , since BH■ is also a cofactor for tyrosine hydroxylase and tryptophan hydroxylase . Clinical features: Intellectual disability (if untreated) Movement disorders Seizures and hypomyelination (due to neurotransmitter deficiency) Tyrosine transaminase (Option A): Deficiency causes tyrosinemia type II , not PKU. Dopa decarboxylase (Option B): Converts L-DOPA to dopamine; not linked to PKU pathology. Tyrosine hydroxylase (Option C): Catalyzes dopamine synthesis, but its deficiency leads to catecholamine deficiency, not phenylalanine accumulation

References:

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↑ *Back to this question in Part A*

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Q10: A 2-month-old infant presents with poor feeding, vomiting, and lethargy. Laboratory studies show hyperammonemia and elevated levels of orotic acid in the urine. Which of the following is the most likely enzyme deficiency? (Q866671)

A. Carbamoyl phosphate synthetase I deficiency

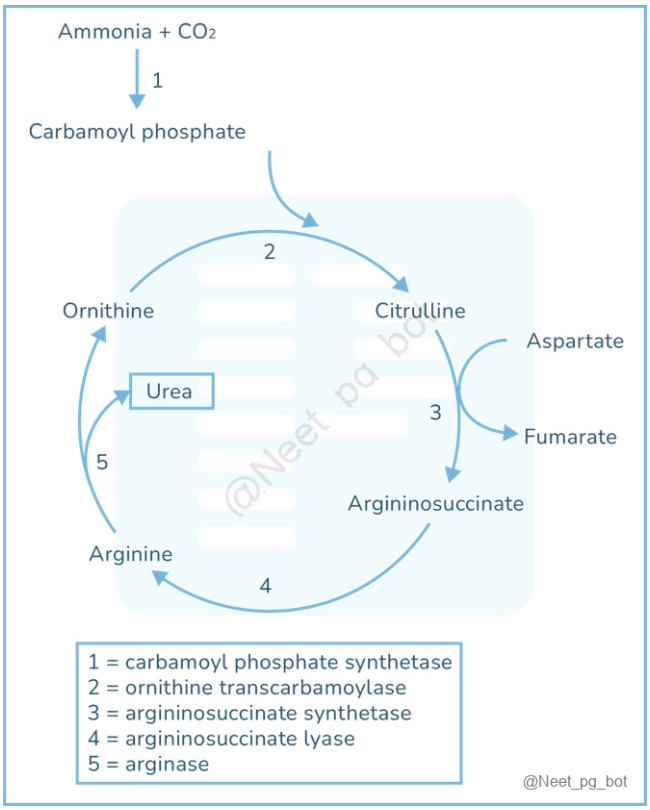
B. Ornithine transcarbamylase (OTC) deficiency

C. Orotidine 5'-phosphate decarboxylase deficiency

D. Argininosuccinate lyase deficiency

Correct Answer: B. Ornithine transcarbamylase (OTC) deficiency

Explanation: The infant's symptoms and laboratory findings are highly suggestive of Type 2 Hyperammonemia , or Ornithine Transcarbamylase (OTC) Deficiency. Hyperammonemia type 2 is an X-linked deficiency caused by a defect in ornithine transcarbamoylase (this reaction occurs in mitochondria), which forms citrulline in the Krebs cycle. Mothers with this condition may have hyperammonemia and avoid high-protein foods. Elevated blood, cerebrospinal fluid, and urine glutamine levels are due to increased glutamine synthesis in response to high ammonia levels. Orotic aciduria - Due to the channelling of carbamoyl phosphate into pyrimidine synthesis. Feature Type 1 Hyperammonemia Type 2 Hyperammonemia Defective Enzyme Carbamoyl Phosphate Synthetase I (CPS1) (Option A) Ornithine Transcarbamylase (OTC) Inheritance Autosomal recessive X-linked recessive Mechanism The inability to synthesize carbamoyl phosphate blocks the urea cycle. Accumulation of carbamoyl phosphate in the mitochondria, which then spills over into the cytoplasm. Metabolic Consequences Elevated ammonia and glutamate Normal orotic acid and uracil Low citrulline. Elevated ammonia, glutamate, and glutamine. High uracil and/or orotic acid (excess carbamoyl phosphate enters pyrimidine synthesis). Normal citrulline. Clinical Presentation Symptoms coincide with increased protein intake. CNS : Lethargy, drowsiness, coma, Convulsions, vomiting, respiratory stimulation, hyperventilation. Similar to Type 1. Hepatomegaly. Orotidine 5'-phosphate decarboxylase deficiency (Option C): this is orotic aciduria (megaloblastic anaemia, growth retardation), but ammonia is normal. Argininosuccinate lyase deficiency (Option D): causes hyperammonemia but with elevated citrulline/argininosuccinate, not orotic acid.



↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: On centrifugation of a cell, LDH will get deposited in: (Q859706)

A. Mitochondria

B. Lysosome

C. Cytoplasm

D. Golgi complex

Correct Answer: C. Cytoplasm

Explanation: Lactate dehydrogenase (LDH) is primarily located in the cytoplasm. The enzyme exists in various isoforms, with LDH-5 being the most abundant in the cytoplasm. About 99% of LDH activity is found in the cytoplasm, where it plays a critical role in the conversion of lactate to pyruvate and vice versa, part of the cell's metabolic processes. While a small fraction of LDH can be found in the nucleus (involved in DNA binding and transcription), the vast majority of LDH activity occurs in the cytoplasm. Mitochondria (Option A): LDH is not primarily located in the mitochondria. Mitochondria contain enzymes like pyruvate dehydrogenase and enzymes involved in the citric acid cycle, but LDH, which is involved in anaerobic metabolism, is predominantly found in

the cytoplasm. Lysosome (Option B): LDH is not found in significant amounts in the lysosome. Lysosomes are involved in cellular digestion and degradation processes, and while some enzymes may be active there, LDH is not typically one of them. It is primarily cytoplasmic Golgi complex (Option D) is responsible for modifying, sorting, and packaging proteins for secretion or use within the cell, but it is not a location where LDH is predominantly found. LDH is mainly active in the cytoplasm and plays its role in energy metabolism rather than processing or transporting cellular components like the Golgi.

References:

Phosphotyrosine-containing Lactate dehydrogenase - PubMed

Transport-exclusion pharmacology to localize lactate dehydrogenase activity within cells

↑ *Back to this question in Part A*

@Neet_pg_bot

Q12: An infant is brought by his parents with complaints that his urine turns black on standing. Which of the following metabolic disorders is most likely? (Q855263)

A. Phenylketonuria

B. Alkaptonuria

C. Homocystinuria

D. Maple syrup urine disease

Correct Answer: B. Alkaptonuria

Explanation: Alkaptonuria is Characterized by black urine upon standing due to the oxidation and polymerization of homogentisic acid (HGA). Alkaptonuria Definition Rare autosomal recessive genetic disorder caused by a deficiency of homogentisate 1,2 dioxygenase (HGD) , an enzyme in the tyrosine metabolism pathway. Mechanism and Pathophysiology Defective tyrosine metabolism → homogentisic acid (HGA) accumulation → ochronotic pigment deposition → systemic disease (ochronosis). Deficiency: HGD fails to convert HGA to maleylacetoacetate. Consequences: HGA oxidizes into melanin-like polymers → connective tissue deposition (ochronosis). Affects joints, heart valves, kidneys, and vessels. Clinical Features Triad: Homogentisic aciduria, ochronosis, ochronotic osteoarthropathy. Pediatric signs: Dark urine (oxidized HGA). Musculoskeletal : Arthritis (spine, hips, knees), kyphoscoliosis, spinal stenosis. Cardiac : A ortic stenosis, mitral valve disease, coronary artery disease. Renal/Metabolic: Stones (renal, gallbladder, prostate). Neurological : Peripheral neuropathy, stroke. Evaluation Clinical scoring: AKU Severity Score Index (AKUSSI). Diagnostic tests: Gold standard: Urinary HGA quantification via gas chromatography-mass spectrometry. Genetic testing for HGD mutations. Imaging (CT/MRI): Joint involvement, valvular calcification. Echocardiography: Valvular abnormalities. Management Symptomatic : Pain control, physiotherapy, joint surgery. Diet : Low-protein diet (limited efficacy). Medications: Vitamin C : Reduces HGA oxidation but not urinary excretion. Nitisinone : Inhibits tyrosine metabolism, reduces HGA >95%. Side effects: tyrosinemia, leukopenia, thrombocytopenia. Prognosis and Complications Prognosis :

Normal life expectancy; reduced quality of life (pain, sleep issues, disability). Complications : Stones (renal, gallbladder, salivary). Tendon/ligament ruptures. Osteopenia, fractures. Aortic calcification/stenosis. Phenylketonuria (Option A): Caused by phenylalanine hydroxylase deficiency, leading to the accumulation of phenylalanine. Symptoms include intellectual disability, seizures, and musty odor, but no black urine. Homocystinuria (Option C): Due to cystathionine beta-synthase deficiency. Presents with lens dislocation, thromboembolism, and developmental delay, but not black urine. Maple Syrup Urine Disease (Option D): Results from branched-chain alpha-keto acid dehydrogenase deficiency. Urine has a characteristic sweet odor but does not turn black on standing.

References:

Vasudevan Textbook of Biochemistry (7th Edition), Page 237, 238

https://www.ncbi.nlm.nih.gov/books/NBK560571/

Harper's Illustrated Biochemistry, 32nd Edition, Page 296, 304

↑ *Back to this question in Part A*

@Neet_pg_bot

Q13: What happens to chloride levels in sweat and pancreatic secretions in cystic fibrosis? (Q854625)

A. Chloride is raised in sweat

B. Chloride is decreased in sweat

C. Chloride is decreased in pancreatic secretions

D. None of the above

Correct Answer: A. Chloride is raised in sweat

Explanation: In cystic fibrosis, CFTR channels are defective. In sweat glands, CFTR normally reabsorbs chloride so its dysfunction leads to high chloride in sweat . In pancreatic ducts, CFTR normally secretes chloride and bicarbonate so its dysfunction causes low chloride and thick secretions , contributing to duct obstruction and pancreatic insufficiency. Cystic fibrosis: Inheritance Autosomal recessive disorder. Genetic Cause Mutations in the CFTR gene (cystic fibrosis transmembrane regulator). CFTR Function Cyclic AMP-regulated chloride (Cl^-) transporter. Symptoms Chronic bacterial infections in airways and sinuses. Fat maldigestion due to pancreatic exocrine insufficiency. Infertility in males due to abnormal development of the vas deferens. Diagnostic Feature Elevated chloride levels in sweat (>60 mmol/L). Note: Chloride decreased in pancreatic secretions (Option C) TRUE, but not the best single answer.

References:

Vasudevan's Textbook of Biochemistry for Medical Students, 7th edition, page 486

↑ *Back to this question in Part A*

@Neet_pg_bot

Q14: A 45-year-old man presents with deep labored breathing and altered sensorium. His arterial blood gas shows a metabolic acidosis. The following serum electrolyte values are obtained: Na■: 145 mmol/L K■: 4 mmol/L Cl■: 90 mmol/L HCO■■: 15 mmol/L Calculate the anion gap (AG) and interpret the finding. (Q854606)

A. 16 mmol/L – Normal anion gap metabolic acidosis

B. 40mmol/L – High anion gap metabolic acidosis

C. 14 mmol/L – Normal anion gap metabolic acidosis

D. 32 mmol/L – Mixed metabolic acidosis

Correct Answer: B. 40mmol/L – High anion gap metabolic acidosis

Explanation: Formula: Anion Gap (AG)=(Na++K+)–(Cl–+HCO3–) AG=(145+4)–(90+15)=149–105=44 mmol/L (Note: K■ is often omitted in clinical practice — if omitted, AG = 145 - (90 + 15) = 40 mmol/L, still markedly elevated.) A normal AG is approximately 8–12 mmol/L (without K■). Here, AG ≈ 40–44 mmol/L, indicating a high anion gap metabolic acidosis (HAGMA). Causes of High Anion Gap Metabolic Acidosis (Mnemonic: MUDPILES): M – Methanol U – Uremia (renal failure) D – Diabetic ketoacidosis P – Paraldehyde / Propylene glycol I – Isoniazid / Iron L – Lactic acidosis E – Ethylene glycol S – Salicylate toxicity

References:

Vasudevan’s Textbook of Biochemistry for Medical Students, 7th edition, page 399

↑ *Back to this question in Part A*

@Neet_pg_bot

Q15: Which of the following enzyme defects leads to Pompe’s disease? (Q857351)

A. Lysosomal alpha glucosidase

B. β-hexosaminidase -A

C. β-galactocerebrosidase

D. β-glucocerebrosidase

Correct Answer: A. Lysosomal alpha glucosidase

Explanation: Pompe's Disease (Glycogen storage disease Type II) Enzyme Deficiency Lysosomal α1→4 and α1→6 glucosidase (acid maltase) Affected Gene GAA Clinical Features Muscle hypotonia, death due to heart failure within 2 years of age (seen in juvenile) Muscle dystrophy (seen in adults) Mode of Inheritance Autosomal recessive Management Supportive care Enzyme replacement therapy Chest x-ray at regular intervals Obtain an initial echocardiogram to evaluate the extent of cardiomyopathy Medical management may be useful based on the stage of cardiomyopathy. Avoid drastic changes in fluid status, either through dehydration or fluid overload.

References:

Harper's Biochemistry 32e pg: 174

Vasudevan Textbook of Biochemistry (7th Edition), Page 129.

Pompe disease diagnosis and management guideline - PMC

↑ *Back to this question in Part A*

@Neet_pg_bot

Q16: Xanthurenic aciduria may be treated with high doses of which vitamin? (Q857350)

A. Vitamin B6

B. Vitamin B12

C. Vitamin B5

D. Vitamin B7

Correct Answer: A. Vitamin B6

Explanation: The metabolic disorders which respond to vitamin B6 therapy are: Xanthurenic aciduria Homocystinuria Vitamin B6 (Pyridoxal Phosphate - PLP): Amino Acid Metabolism PLP acts as a coenzyme for many reactions in amino acid metabolism. Transamination Catalyzed by amino transferases (e.g., Alanine transaminase), converting amino acids to keto acids. Decarboxylation Required for decarboxylation of amino acids, forming important neurotransmitters and metabolites. Neurotransmitter Synthesis GABA from glutamate (important for inhibitory neurotransmission). Histamine from histidine (mediator of allergy). Serotonin from 5-hydroxy tryptophan (mood regulation). Taurine from cysteine. Sulfur Containing Amino Acids Involved in methionine and cysteine metabolism. Homocysteine Metabolism Converts homocysteine to cystathionine; deficiency leads to increased homocysteine, associated with myocardial infarction risk. Heme Synthesis Required for ALA synthase, the rate-limiting enzyme in heme biosynthesis. Niacin Production Required for the conversion of tryptophan to niacin (via kynurenine). Glycogenolysis PLP is a coenzyme for phosphorylase, which converts glycogen to glucose-1-phosphate.

References:

Vasudevan’s Textbook of Biochemistry for Medical Students, 7th edition, page 483

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: Deficiency of which of the following causes Barth’s syndrome? (Q857325)

A. Cardiolipin

B. Sphingomyelin

C. Glycolipids

D. Cerebroside

Correct Answer: A. Cardiolipin

Explanation: Barth’s syndrome: Cause Mutations affecting cardiolipin metabolism (decreased or altered cardiolipin levels), causing defects in mitochondrial function. Clinical Features Cardioskeletal myopathy (affects both heart and muscle). Mitochondrial dysfunction in aging and pathologic conditions.

References:

Harper’s Biochemistry 32nd edition, page 211

↑ *Back to this question in Part A*

@Neet_pg_bot

Q18: Liver produces ketones but cannot use it due to the deficiency of which of the following? (Q857321)

A. Thiophorase

B. Alkaline phosphatase

C. Alanine transaminase

D. Thoiolase

Correct Answer: A. Thiophorase

Explanation: Liver cannot use ketone bodies due to the deficiency of Thiphorase KETOSIS Ketone Bodies Produced in the liver Utilized by extrahepatic tissues due to the absence of Thiophorase enzyme in the liver Tissues that use Ketone Bodies Almost all tissues except: Liver Red blood cells (RBCs) Tissues that prefer ketone bodies: Heart muscle Renal cortex Placenta Tissues that use ketone bodies only in the absence of glucose: Skeletal muscle Brain Additional Tissues Using Ketone Bodies Intestinal mucosal cells Adipocytes Enzyme Involved Thiophorase : Converts acetoacetate to acetoacetyl CoA Fatty Acids as Primary Energy Source During Starvation Skeletal muscles Heart Liver Energy Production Pathway Acetoacetyl CoA enters the beta-oxidation pathway to produce energy

References:

Vasudevan Biochemistry 7th edition. Pg.164

Harper Biochemistry 32nd edition. Pg. 221

↑ *Back to this question in Part A*

@Neet_pg_bot

Q19: A 16-year-old patient presents with pellagra-like skin eruptions during the spring season for the past 5 years. Laboratory tests reveal normal levels of 5-Hydroxyindoleacetic Acid (HIAA), high aminoaciduria, and normal urinary indole levels. What is the most likely diagnosis? (Q860099)

A. Carcinoid tumor

B. Nutritional pellagra

C. Hartnup disorder

D. Essential fructosuria

Correct Answer: C. Hartnup disorder

Explanation: The patient's presentation is consistent with Hartnup disease (Pellagra-like dermatosis) , a rare inborn error of metabolism. This condition is associated with impaired transport of monoamine monocarboxylic amino acids across the brush border epithelium of the intestine and renal tubules. Hartnup disorder Inheritance Autosomal recessive Pathogenesis Mutation in SLC6A19 gene; Impaired transport of neutral amino acids across intestinal and kidney brush border epithelium Clinical features Photosensitivity Pellagra-like rash (Decreased Niacin due to tryptophan deficiency) Neurologic symptoms (ataxia, emotional instability) - Decreased serotonin due to tryptophan deficiency Biochemical defect Deficiency of nicotinamide synthesis Diagnosis Aminoaciduria (neutral amino acids) in urine and plasma Demonstrate SLC6A19 defect Treatment Administration of nicotinamide/nicotinic acid Protection from sunlight Carcinoid tumor (Option A): Carcinoid tumors cause increased serotonin production, which is metabolized to 5-HIAA and excreted in urine. The normal 5-HIAA levels in this patient rule out carcinoid syndrome. Nutritional pellagra (Option B): Nutritional pellagra is caused by niacin (vitamin B3) deficiency and is characterized by the "4 Ds": diarrhea, dermatitis, dementia, and death.

However, this patient has high aminoaciduria, which is not typical of nutritional pellagra. Essential fructosuria (Option D): It is caused by a deficiency of fructokinase, leading to the excretion of unmetabolized fructose in the urine. It does not cause pellagra-like symptoms or high aminoaciduria.

References:

Nelson Textbook of Paediatrics Edition 21st Pg. 707, 708, 3501

↑ *Back to this question in Part A*

@Neet_pg_bot

Q20: Which of the following substances is used in a tube for collecting blood samples specifically for measuring alcohol concentration? (Q860092)

A. Heparin + EDTA

B. Sodium fluoride + Potassium dichromate

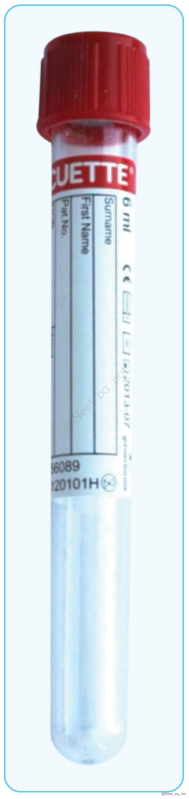
C. Heparin

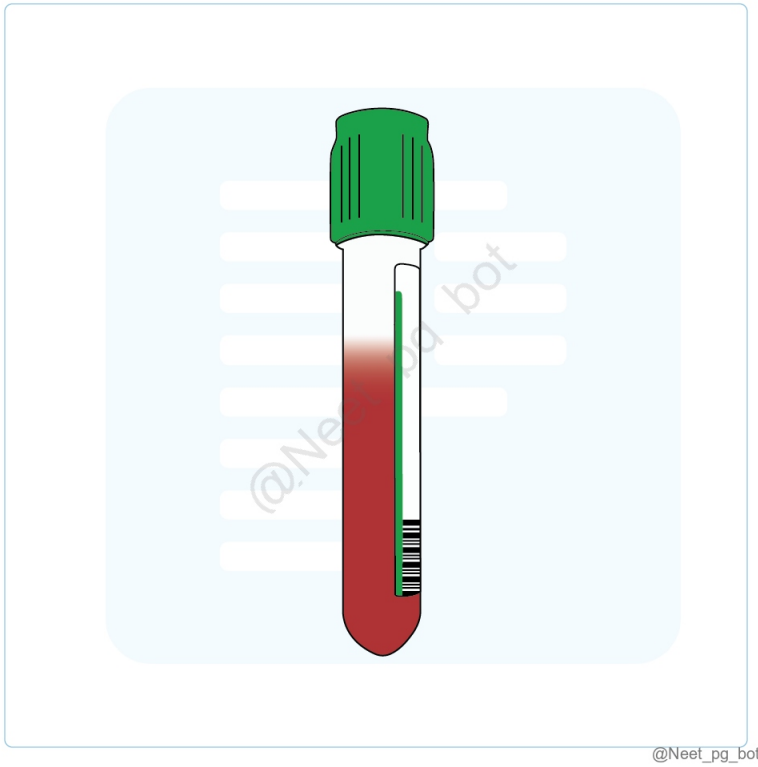
D. Sodium citrate

Correct Answer: B. Sodium fluoride + Potassium dichromate

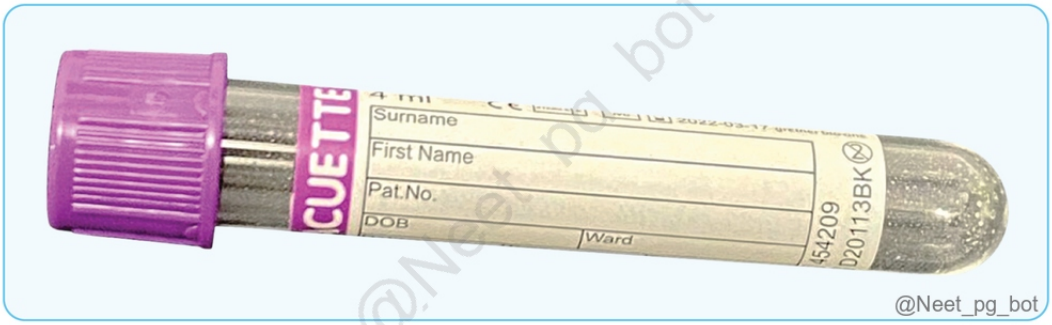
Explanation: Sodium fluoride is an antiglycolytic agent that inhibits the breakdown of glucose, and Potassium dichromate is a reagent that stabilizes the sample for accurate alcohol concentration measurement. This combination is specifically used for alcohol testing because it prevents the metabolism of alcohol in the sample. Blood Collection Tubes Tube Type Additives & Mechanism Key Applications Coagulation Tube {{caption_text}} Sodium citrate binds calcium (Factor IV) to prevent clotting; anticoagulation reversed by adding calcium. Used for coagulation studies. Must be filled completely. Serum Tube {{caption_text}} Glass/silica activates clotting (Factor XII) . Some include gel separators to prevent cellular contamination. Used for clinical chemistry, immunology, and serology. Heparin Tube Sodium/lithium heparin inhibits thrombin and Factor Xa to prevent clotting. Common in biochemical tests. EDTA Tube {{caption_text}} EDTA chelates calcium irreversibly, preventing clotting. Preferred for hematology. Stable for enzymatic studies. Glycolytic Inhibitor Tube {{caption_text}} Sodium fluoride inhibits glycolysis; may also include citrate, oxalate, or EDTA to prevent clotting. Used to measure glucose and lactate levels. Heparin + EDTA (Option A): Both heparin and EDTA are anticoagulants , they are not specifically used for preserving blood samples for alcohol concentration measurement. They lack the ability to inhibit glycolysis, which can alter alcohol levels in the sample. Heparin (Option C) : Heparin is an anticoagulant used to prevent blood clotting, but it is not suitable for alcohol concentration tests as it does not preserve the sample for this specific purpose. Sodium citrate (Option D) : Sodium citrate is an anticoagulant used mainly for coagulation studies, but it is not used for measuring blood alcohol concentration.


BD Vacutainer®
Buff. Na Citrate
0.109M, 3.2%
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LOT 21241322 2013-02
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Surname
First Name
6 ml
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References:

https://www.ncbi.nlm.nih.gov/books/NBK555991/

↑ *Back to this question in Part A*

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Q21: Which of the following uses CRISPR-Cas9 technology? (Q860081)

A. DNA Editing

B. Post-transcriptional Modification

C. DNA Induction

D. DNA Editing Solution

Correct Answer: A. DNA Editing

Explanation: CRISPR cas9 is used in editing parts of the genome by removing, adding or altering sections of the DNA sequence. It is currently the simplest, most versatile and precise method of genetic manipulation. C-clustered R-regulatory I-interspaced S-short P-palindromic R-repeats NHEJ and HEJ are the two mechanisms employed by the CRISPR-Cas9 System. It is a powerful tool for inducing precise double-stranded breaks in DNA.

CRISPR-Cas9 System Components: Guide RNA (gRNA): Directs the Cas9 enzyme to a specific DNA sequence. Cas9 Enzyme: Induces double-stranded breaks (DSBs) at the target site. Repair Mechanisms: Non-homologous end Joining (NHEJ): This technique directly ligates broken DNA ends without a template, often leading to small insertions or deletions. It can also cause gene knockout by disrupting the coding sequence. Homologous End Joining (HEJ): Uses a homologous template for precise repair of Double strand breaks and enables accurate gene knock-in or correction of mutations. Post-transcriptional Modification (Option B): This process involves changes to RNA after it has been transcribed from DNA, such as splicing, capping, and polyadenylation. It does not involve CRISPR-Cas9 technology, which primarily targets DNA rather than RNA. DNA Induction (Option C): This term is not commonly used in the context of CRISPR-Cas9 technology. CRISPR-Cas9 is focused on directly editing DNA sequences rather than inducing changes. DNA Editing Solution (Option D): While CRISPR-Cas9 is a powerful tool for DNA editing, the term "DNA Editing Solution" is vague and not specific to the CRISPR-Cas9 system itself.

References:

Brock Biology of Microorganisms 15th Edition, Page 298

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8215069/

Harper’s Illustrated Biochemistry 32nd Edition, Page 454

↑ *Back to this question in Part A*

@Neet_pg_bot

Q22: A young girl presented to the OPD for an examination. She is tall and thin with slender fingers, and dislocated lenses. Workup revealed cystathione beta-synthase deficiency. Which amino acid supplement is required in this case? (Q855999)

A. Cysteine

B. Methionine

C. Serine

D. Tyrosine

Correct Answer: A. Cysteine

Explanation: The Clinical scenario of tall, thin and dislocated lens with cystathione beta synthase deficiency indicates Type 1 Homocystinuria. Cysteine is deficient in this patient, thus supplement is required in this case. Type 1 vs Type 2 Homocystinuria: Feature Type - I Homocystinuria Type - II Homocystinuria Enzyme Deficiency Cystathionine beta-synthase Methionine synthase Metabolic Pathway Affected Homocysteine to cystathionine Homocysteine to methionine Elevated Metabolites Homocysteine and methionine Homocysteine and elevated levels of Cysteine Biochemical Diagnosis Low levels of cysteine. High levels of methionine. High levels of cysteine. Low levels of methionine. Treatment Vitamin B6 (pyridoxine) supplementation; low-methionine diet;

cysteine supplementation Vitamin B12 and folate supplementation; low-methionine diet

References:

Harper's Illustrated Biochemistry.32nd edition p.294, 295

Vasudevan Textbook of Biochemistry (7th Edition), Page 431.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q23: Match the following amino acids with their derivatives: A. Tyrosine i. Melatonin B. Tryptophan ii. Taurine C. Glycine iii. Thyroid hormones D. Cysteine iv. Creatinine (Q879286)

A. A-i, B-ii, C-iii, D-iv

B. A-iii, B-i, C-iv, D-ii

C. A-iv, B-ii, C-i, D-iii

D. A-ii, B-i, C-iv, D-iii

Correct Answer: B. A-iii, B-i, C-iv, D-ii

Explanation: Tyrosine → Thyroid hormone: Tyrosine residues in thyroglobulin are iodinated to form T3 and T4 . Tryptophan → Melatonin: Tryptophan → Serotonin → Melatonin in the pineal gland. Glycine → Creatinine: Creatine is synthesised from arginine + glycine + methionine , and its breakdown forms creatinine . Cysteine → Taurine: Cysteine undergoes decarboxylation and oxidation to form taurine , important in bile acid conjugation.

References:

DM Vasudevan’s Textbook of Biochemistry, 7th Edition, Pages 232-235

↑ *Back to this question in Part A*

@Neet_pg_bot

Q24: All of the following are lipotropic factors except? (Q859698)

A. Choline

B. Lecithine

C. Arginine

D. Methionine

Correct Answer: C. Arginine

Explanation: Arginine is an amino acid involved in nitric oxide production and other functions but is not considered a lipotropic factor. Lipotropic factors Lipotropic factors are nutrients such as choline, betaine, and methionine that help improve liver function and detoxification. (Option A & D ruled out) Lecithin contains choline as its primary active ingredient and is considered a lipotropic agent. (Option B ruled out) These compounds promote the flow of fat and bile to and from the liver, aiding fat metabolism and liver health. They are used to treat liver disorders like hepatitis, cirrhosis, and chemical-induced liver disease. Lipotropic formulas may increase the liver's levels of S-adenosyl-methionine and glutathione, key compounds for liver function and detoxification. Common lipotropic agents include choline, methionine, betaine, folic acid, and vitamin B12. These formulas support detoxification, protect liver function, and enhance fat metabolism. They may also play a role in methylation and epigenetics, influencing health at a molecular level. Carnitine , another lipotropic agent, helps prevent alcohol-induced fatty liver disease by improving fatty acid metabolism. Lipotropic formulas are also used in detoxification, especially in conditions like chronic candidiasis and liver damage. Daily recommended doses for choline and methionine are 1000 mg each . Lipotropic agents are often combined with other herbs and nutrients to support liver health and detoxification.

References:

Lipotropic Agent - an overview | ScienceDirect Topics

Lipotropic factors and oncogenesis - PubMed

https://www.healthline.com/health/lipotropic-injections

↑ *Back to this question in Part A*

@Neet_pg_bot

Q25: A patient came to the hospital with a complaint of abdominal pain. Routine investigations were unremarkable. On observation, the urine turned black on standing. Which of the following enzymes is likely to be defective in this patient? (Q858459)

A. Homogentisate oxidase

B. Xanthine oxidase

C. Phenylalanine hydroxylase

D. Dihydric orotate dehydrogenase

Correct Answer: A. Homogentisate oxidase

Explanation: The clinical presentation of black urine on standing, along with abdominal pain, points to alkaptonuria , a rare autosomal recessive metabolic disorder caused by a deficiency in the enzyme homogentisate oxidase. Alkaptonuria Homogentisate oxidase is an enzyme involved in the breakdown of the amino acids tyrosine and phenylalanine Its deficiency leads to the accumulation of homogentisic acid in the body Pathophysiology: Homogentisic acid is excreted in the urine. Upon exposure to air (oxidation), it polymerizes, causing the urine to turn black on standing Clinical Features Urine discoloration: The hallmark sign is black urine when exposed to air Abdominal pain: Likely due to deposition of homogentisic acid or associated complications like kidney stones Ochronosis : Long-term deposition of homogentisic acid in connective tissues, leading to bluish-black discoloration (typically seen in adulthood) Arthropathy : Degenerative joint disease due to ochronotic deposits in cartilage Diagnosis: Urine analysis: Elevated levels of homogentisic acid. Genetic testing: Mutations in the HGD gene (homogentisate oxidase gene) Management: Limit intake of tyrosine and phenylalanine. Vitamin C may slow ochronosis progression. Nitisinone : A newer drug that inhibits the production of homogentisic acid by blocking the enzyme upstream in the metabolic pathway Xanthine oxidase (Option B): Xanthine oxidase is an enzyme involved in purine metabolism , converting hypoxanthine to xanthine to uric acid . Deficiency leads to xanthinuria , which presents with low uric acid levels and renal stones , but does not cause black discoloration of urine on standing . Therefore, this enzyme defect does not explain the findings in this patient. Phenylalanine hydroxylase (Option C): Phenylalanine hydroxylase converts phenylalanine to tyrosine . Deficiency causes phenylketonuria (PKU) , characterized by intellectual disability, seizures, fair skin, eczema, and a musty/mousy odor of urine due to phenylketones. Urine does not turn black on standing , making this option inconsistent with the clinical scenario. Dihydroorotate dehydrogenase (Option D): Dihydroorotate dehydrogenase is involved in de novo pyrimidine synthesis , converting dihydroorotate to orotate . Defects in pyrimidine metabolism are associated with orotic aciduria , which presents with megaloblastic anemia and developmental delay . It does not cause black urine on standing , so it does not match the presentation.

References:

https://pmc.ncbi.nlm.nih.gov/articles/PMC3978898/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q26: In a patient with obstructive jaundice, which of the following outcomes may be observed in Van der Bergh's reaction? (Q854880)

A. Direct positive

B. Indirect positive

C. Both positive

D. Coproporphyrin

Correct Answer: A. Direct positive

Explanation: Van den Bergh's reaction is a biochemical test used to differentiate types of bilirubin in serum by observing their reactivity with diazotized sulfanilic acid . Types of Reaction: Type of Jaundice Type of Bilirubin Elevated Van den Bergh Reaction Interpretation Hemolytic (Pre-hepatic) Unconjugated (Indirect) Indirect positive (requires alcohol to react) Due to excessive RBC breakdown Hepatocellular (Hepatic) Both conjugated & unconjugated Biphasic (Both positive) Liver cell injury affects conjugation & excretion Obstructive (Post-hepatic) Conjugated (Direct) Direct positive (reacts immediately) Due to bile duct obstruction Mechanism in Obstructive Jaundice: In obstructive jaundice, conjugated bilirubin (water-soluble) regurgitates into the bloodstream due to biliary obstruction. Since conjugated bilirubin reacts directly with diazo reagent without the need for alcohol, it produces a direct positive Van den Bergh reaction.

References:

Obstructive jaundice

↑ *Back to this question in Part A*

@Neet_pg_bot

Q27: Nitric oxide is synthesized from: (Q854869)

A. L-Arginine

B. L-Citrulline

C. Glycine

D. Lysine

Correct Answer: A. L-Arginine

Explanation: Nitric oxide (NO) is synthesized from the amino acid L-arginine by a group of enzymes called nitric oxide synthases (NOS). The process involves the conversion of L-arginine to NO and L-citrulline. L-arginine: It is the primary precursor for the synthesis of nitric oxide in the body. Nitric oxide synthases convert L-arginine to nitric oxide and L-citrulline in a reaction that requires oxygen and cofactors. The synthesis of nitric oxide (NO) from L-arginine involves a reaction catalyzed by the enzyme nitric oxide synthase (NOS). The general equation for NO synthesis is as follows: L-arginine + NADPH + O 2 + H* → Nitric oxide + L-citrulline + NADP+ + H 2 O There are three isoforms of NOS: Endothelial NOS (eNOS) Neuronal NOS (NOS) Inducible NOS (iNOS). L-Citrulline (Option B): It is the product, not the substrate, of NO synthesis. Glycine (Option C) & Lysine (Option D): Not involved in NO production.

References:

NO

↑ *Back to this question in Part A*

@Neet_pg_bot

Q28: The maximum thermic effect is seen with a meal that has (Q856500)

A. Protein

B. Fat

C. Carbohydrates

D. Mixed meal having all 3 in equal amount

Correct Answer: A. Protein

Explanation: F) refers to the increase in energy expenditure due to digestion, absorption, and metabolism of nutrients. Protein: Has the highest thermic effect (~20–30% of its energy content), as it requires significant energy for metabolism and nitrogen disposal. Fat: Has the lowest thermic effect (~0–3%), as it is metabolized more efficiently with minimal energy expenditure. Carbohydrates: Moderate thermic effect (~5–10%) , primarily for digestion and conversion into glycogen or fat. Mixed meal: The thermic effect of a mixed meal depends on its macronutrient composition but will generally be lower than a protein-rich meal if all components are equal.

References:

TEF

↑ *Back to this question in Part A*

@Neet_pg_bot

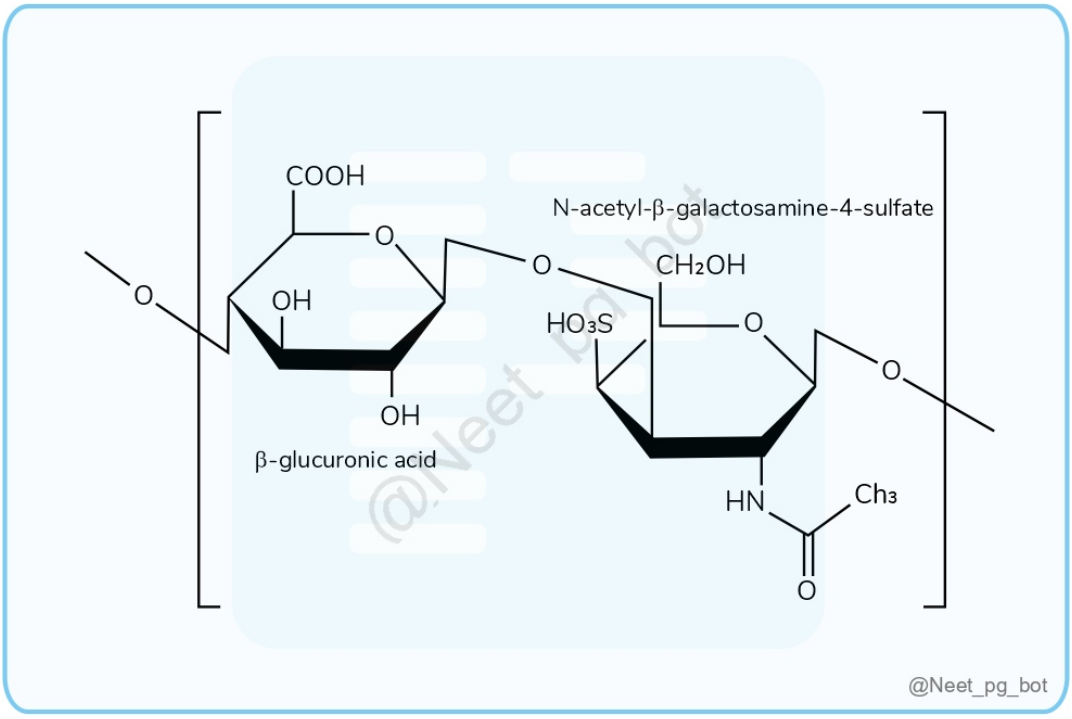
Q29: The given chemical structure is of: (Q856923)

A. Keratan sulfate

B. Chondroitin sulfate

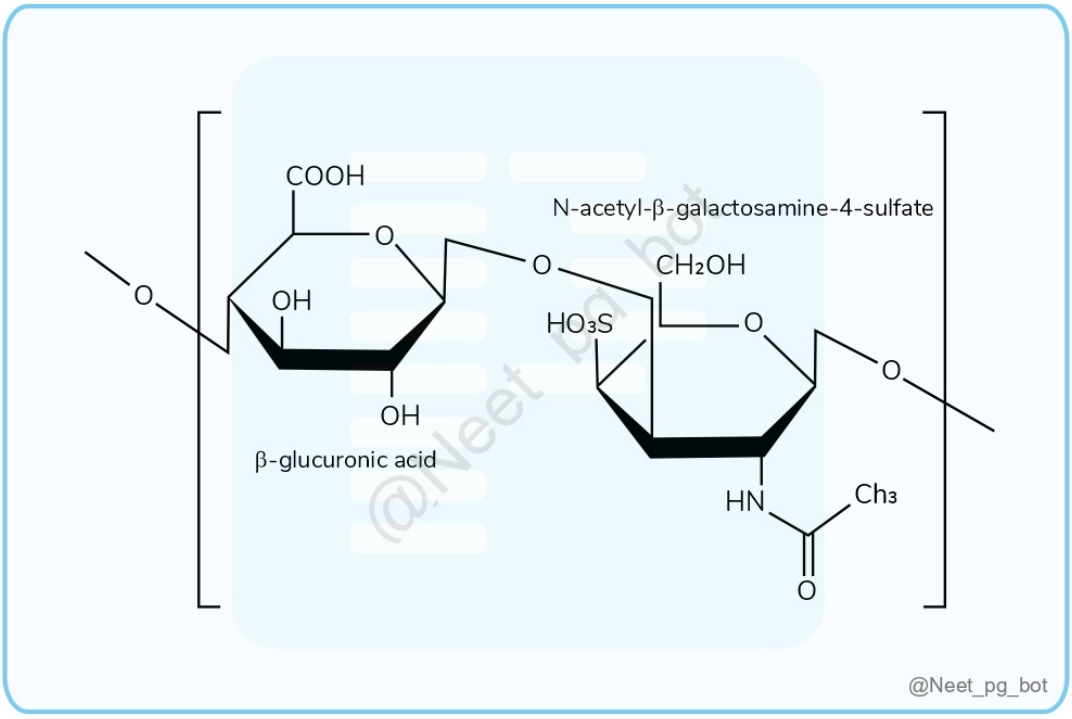
C. Heparin

D. Dermatan sulfate



Correct Answer: B. Chondroitin sulfate

Explanation: Chondroitin sulfate is a glycosaminoglycan (GAG) that is a major component of the extracellular matrix and connective tissues in the body. It consists of repeating units of N-acetylgalactosamine (GalNAc) and glucuronic acid (GIcA) or iduronic acid (Ido). Chondroitin sulfate provides structural support and elasticity to various tissues, including cartilage, tendons, ligaments, and skin. Keratan sulfate(Options A): Another type of glycosaminoglycan. It consists of repeating units of galactose and N-acetylglucosamine. It is commonly found in the cornea of the eye and cartilage, and it helps maintain the structural integrity of these tissues. Heparin(Options C): Different type of glycosaminoglycan that is highly sulfated. It consists of repeating units of glucuronic acid and N-acetylglucosamine. Heparin is an anticoagulant and is primarily produced and stored in mast cells. It plays a role in preventing blood clotting. Dermatan sulfate(Options D): Another glycosaminoglycan that is composed of repeating units of iduronic acid and N-acetylgalactosamine. It is found in various tissues, including skin, blood vessels, and heart valves.



References:

Chondritin sulfate

↑ *Back to this question in Part A*

@Neet_pg_bot

Q30: Which of the following is not a true statement regarding telomerase? (Q856880)

A. Has reverse transcriptase activity

B. Present only in Eukaryotes

C. Maintain the chromosome length

D. Involved in DNA repair

Correct Answer: D. Involved in DNA repair

Explanation: Telomerase is not involved in DNA repair process. Telomerase: An RNA-dependent DNA polymerase that counteracts telomere shortening. It is the enzyme responsible for telomere synthesis . Adds TTAGGG repeats to the 3′ ends of chromosomes. Contains its own RNA template, which is complementary to the telomeric DNA sequence, enabling it to extend telomeres. Has reverse transcriptase activity (Option A): Telomerase extends the telomeres using its reverse transcriptase activity. Present only in Eukaryotes (Option B): Telomerase is found in eukaryotes, as prokaryotes have circular DNA and do not require telomerase. Maintain the

chromosome length (Option C): Telomerase prevents the shortening of chromosomes during replication.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg. 365

Vasudevan, 7th Ed. Pg 585

Telomeres and telomere length

↑ *Back to this question in Part A*

@Neet_pg_bot

Q31: Ammonia in the brain is detoxified to form (Q856874)

A. Urea

B. Glutamine

C. Ammonium ions

D. Alanine

Correct Answer: B. Glutamine

Explanation: Ammonia in Brian is detoxified to form Glutamine. Glutamine synthetase in astrocytes converts glutamate and ammonia to glutamine, effectively detoxifying ammonia in the brain. Ammonia Management in Neurons: Ammonia can be neurotoxic if accumulated. Glutamine Synthesis: Produced in neurons as a byproduct of amino acid metabolism and neurotransmitter turnover. Ammonia reacts with α-ketoglutarate and NADH to form glutamate, catalyzed by glutamate dehydrogenase. α-ketoglutarate + NH 3 ■ ■+ NADH → Glutamate + NAD + + H2■O Glutamate is converted to glutamine by glutamine synthetase, adding another ammonia molecule. Glutamine synthetase is predominantly found in astrocytes. Glutamate + NH 3 ■ + ATP → Glutamine + ADP + Pi This conversion reduces ammonia toxicity by storing it in a non-toxic form. The fate of glutamine: Release into the Bloodstream: Glutamine is released into the extracellular space and enters the bloodstream. This transport helps maintain nitrogen balance and prevents ammonia accumulation. Transport to the Liver: Glutamine carries ammonia from the brain to the liver, where it is deaminated to release ammonia. Ammonia enters the urea cycle in the liver for detoxification. Importance of Glutamine in Neuronal Health: Acts as a non-toxic ammonia carrier, preventing neurotoxicity. Serves as a precursor for neurotransmitters like GABA , maintaining neurotransmitter balance.

References:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8244593/

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4133642/

https://www.sciencedirect.com/science/article/pii/S019701862030200X

↑ *Back to this question in Part A*

@Neet_pg_bot

Q32: Which of the following disorders is characterized by the presence of a cherry red spot on the retina? (Q860068)

A. Tay-Sachs disease

B. Fabry's disease

C. Gaucher's disease

D. Von Gierke's disease

Correct Answer: A. Tay-Sachs disease

Explanation: Tay-Sachs disease is a lysosomal storage disorder characterized by progressive neurodegeneration, developmental delay, and a cherry-red spot on the retina. Tay-Sachs Disease Inheritance Autosomal recessive Cause Deficiency of hexosaminidase A enzyme Accumulation GM2 ganglioside in ganglion cells and neurons Clinical Manifestations Initial symptoms include loss of head control, inability to sit, exaggerated startle response to sound, and a disproportionately large head. Progressive features include spasticity, deafness, and blindness. Not associated with organomegaly. Fundus Finding Cherry-red spot is caused by GM2 ganglioside accumulation in the ganglion cells of the retina, creating a contrast with the normal red fovea. Fabry's disease (Option B): Fabry's disease is caused by a deficiency of alpha-galactosidase A, leading to the accumulation of globotriaosylceramide. It presents with angiokeratomas, neuropathic pain, and renal involvement but does not typically feature a cherry-red spot on the retina. Gaucher's disease (Option C): Gaucher's disease is due to glucocerebrosidase deficiency, causing glucocerebroside accumulation in macrophages. It presents with hepatosplenomegaly, bone pain, and cytopenias but is not associated with a cherry-red spot. Von Gierke's disease (Option D): Von Gierke's disease (glycogen storage disease type I) results from a deficiency of glucose-6-phosphatase. It causes severe fasting hypoglycemia, hepatomegaly, and lactic acidosis but does not involve retinal findings.

References:

Ghai Essential Pediatrics, 10th Edition, Pg.693

Tay-sach disease

https://www.ncbi.nlm.nih.gov/books/NBK564432/#:~:text=Tay%20Sachs%20disease%20manifests%20in,as%20one%20week%20of%20life

↑ *Back to this question in Part A*

@Neet_pg_bot

Q33: A patient presented with joint pain and on evaluation, urine turns black on standing. What is the likely enzyme deficiency? (Q855951)

A. Homogentisic acid 1,2-dioxygenase

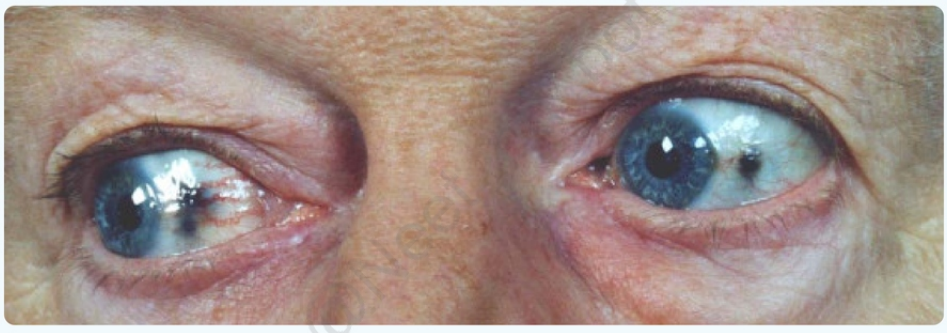
B. Tyrosine transaminase

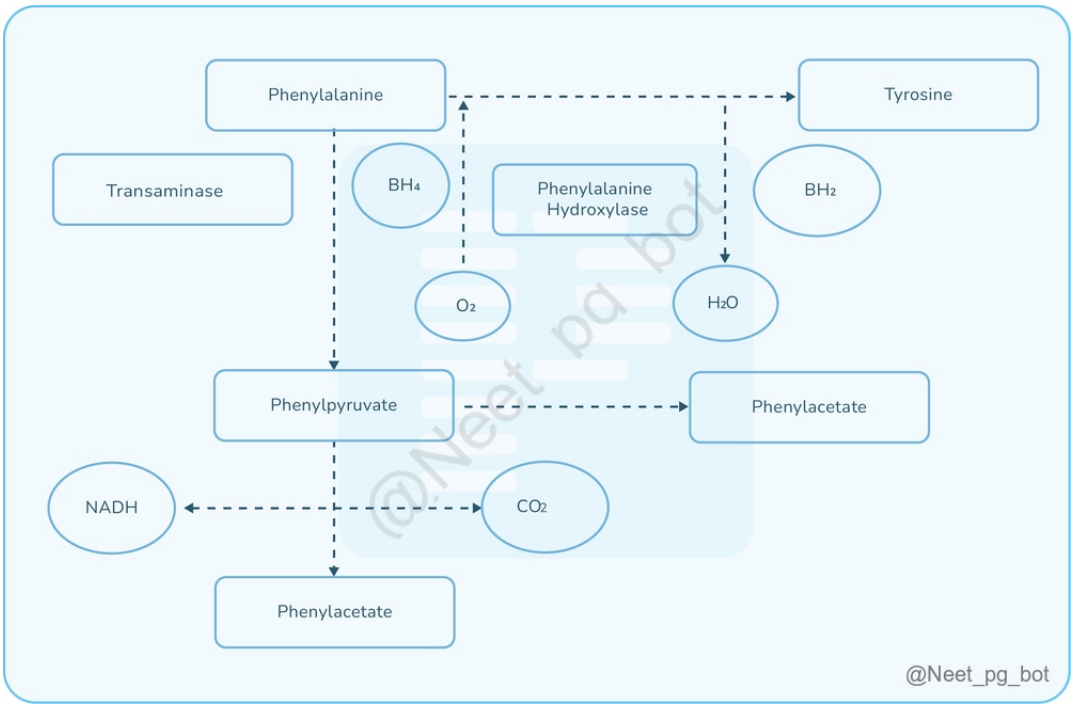
C. Fumarylacetoacetase hydrolase

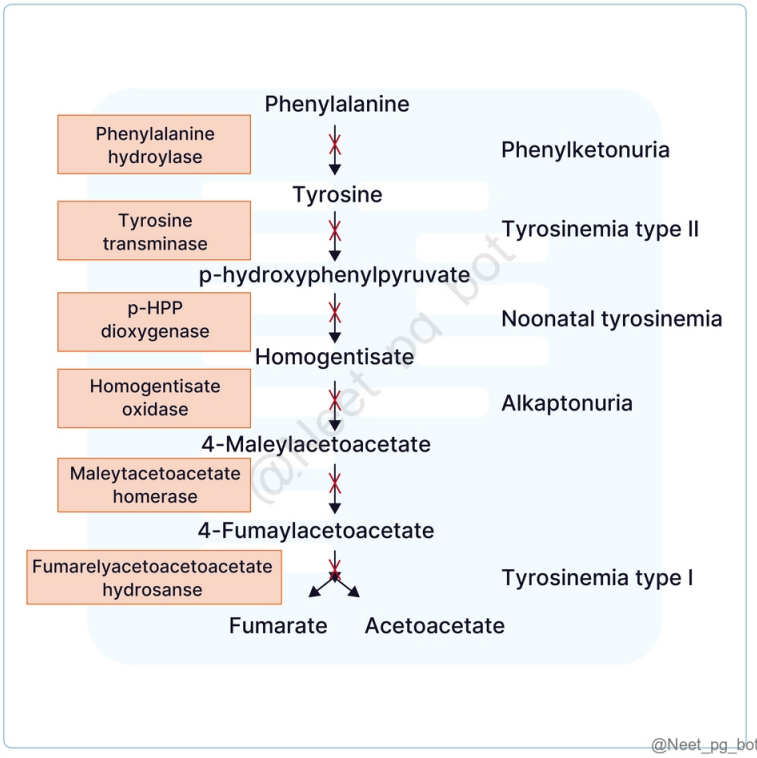
D. 4-hydroxy-phenyl pyruvate dioxygenase

Correct Answer: A. Homogentisic acid 1,2-dioxygenase

Explanation: The clinical scenario described, including darkening of urine upon standing and joint pain, is characteristic of Alkaptonuria , which is caused by a deficiency in the enzyme homogentisate oxidase. Garrod's tetrad in alkaptonuria 1. Homogentisic Aciduria Presence of homogentisic acid in the urine, which turns dark upon standing or when exposed to air. 2. Ochronosis {{caption_text}} Deposition of dark pigment in connective tissues, particularly noticeable in the ear cartilage, sclera, and other connective tissues, leading to a bluish-black discoloration.The image shows Osler’s sign. 3. Arthropathy Degenerative joint disease, especially affecting the spine and large joints, leading to chronic pain and stiffness. 4. Cardiac Involvement Includes heart valve calcification and atherosclerosis due to the deposition of pigment in cardiac tissues. Reaction Enzyme Associated Disorder Tyrosine → p-Hydroxyphenylpyruvate Tyrosine transaminase Tyrosinemia type II p-Hydroxyphenylpyruvate → Homogentisate p-Hydroxyphenylpyruvate dioxygenase Tyrosinemia type III Homogentisate → Maleylacetoacetate Homogentisate 1,2-dioxygenase Alkaptonuria Maleylacetoacetate → Fumarylacetoacetate Maleylacetoacetate isomerase - Fumarylacetoacetate → Fumarate + Acetoacetate Fumarylacetoacetate hydrolase Tyrosinemia type I







References:

Harper's Illustrated Biochemistry, 32nd Edition Pg. 296

↑ *Back to this question in Part A*

@Neet_pg_bot

Q34: Which of the following options is false in a patient with advanced liver disease? (Q856619)

A. Hyperammonemia

B. Decreased ATP synthesis

C. Oxidative carboxylation of alpha-ketoglutarate

D. Increased synthesis of glutamine and glutamine from alpha-ketoglutarate

Correct Answer: C. Oxidative carboxylation of alpha-ketoglutarate

Explanation: In advanced liver disease, Hyperammonemia arises from impaired urea cycle and defective hepatic ammonia detoxification. (Option A ruled out) Mitochondrial dysfunction reduces ATP synthesis and depletes alpha-ketoglutarate, which is diverted for glutamine synthesis to detoxify ammonia. (Option B ruled out) Oxidative carboxylation of alpha-ketoglutarate does not occur; instead, glutamate undergoes oxidative deamination to regenerate alpha-ketoglutarate. (Option C) (Option D ruled out) This metabolic disruption contributes to energy failure and ammonia accumulation. Glutamate dehydrogenase (GDH): Glutamate dehydrogenase (GDH) converts glutamate to alpha-ketoglutarate This reaction occurs only in the Liver Glutamate is the only AA, that can undergo oxidative deamination to release amino groups in the liver Activators of GDH Inhibitors of GDH NAD ATP ADP GTP GDP NADH

References:

Harper’s biochemistry 32nd edition, Pg 282,283,291

Vasudevan 7th edition, Pg 201

↑ *Back to this question in Part A*

@Neet_pg_bot

Q35: Which of the following consumes the highest energy during assimilation? (Q855919)

A. Proteins

B. Carbohydrates

C. Mono-unsaturated fatty acids

D. Polyunsaturated fatty acids

Correct Answer: A. Proteins

Explanation: Proteins consume the highest energy during assimilation as they have the highest thermogenic effect and require maximum energy. Carbohydrates (Option B) utilizes less energy for assimilation 5%. MUFA (Option C) has a single double bond and consumes the least energy to be broken down. PUFA (Option D) are essential amino acids required by our body and require the least energy to be broken down.

References:

Vasudevan Textbook of Biochemistry, 7th Edition, Page 529

↑ *Back to this question in Part A*

@Neet_pg_bot

Q36: Which enzyme is likely to be deficient in a 4-year-old child who presents with a habit of biting their lips and fingers, and has a missing portion of fingers on their left hand? Further evaluation shows delayed developmental milestones. The parents report that two male cousins on the mother's side have similar symptoms and have been diagnosed with a genetic disorder. Laboratory reports indicate hyperuricemia. (Q857544)

A. Purine nucleoside phosphorylase

B. Hypoxanthine -guanine phosphoribosyl transferase.

C. Adenine phosphoribosyl transferase

D. Adenosine deaminase

Correct Answer: B. Hypoxanthine -guanine phosphoribosyl transferase.

Explanation: Lesch-Nyhan Syndrome (X-linked recessive disorder) Enzyme deficiency: HGPRT deficiency impairs the purine salvage pathway. Excess uric acid production (hyperuricemia) due to increased purine breakdown. Neurological symptoms due to the lack of purine recycling, affecting brain development. Key symptoms: Self-mutilation behavior (lip and finger biting) ■■■■ ■■ ■■ ■■ ■■ ■■ ■ ■ ■ ■ ■■ ■■ ■■ ■■ {{caption_text}} Developmental delay Hyperuricemia → Gout, nephrolithiasis X-linked inheritance: Symptoms in males, family history consistent with maternal transmission. Purine nucleoside phosphorylase (Option A): Inherited in an autosomal recessive pattern. Adenine phosphoribosyl transferase (Option C): Deficiency is characterised by excessive production and renal excretion of 2,8-dihydroxyadenine (DHA), which leads to kidney stone formation and crystal-induced kidney damage (DHA crystal nephropathy). Adenosine deaminase (Option D): Deficiency damages the immune system and causes severe combined immunodeficiency (SCID).



References:

Lesch-Nyhan syndrome

↑ *Back to this question in Part A*

@Neet_pg_bot

Q37: Which of the following is considered a universal methyl group donor in various biochemical reactions? (Q856961)

A. Glucose-6-phosphate

B. Acetyl CoA

C. S-Adenosyl Methionine

D. S-Adenosyl Serine

Correct Answer: C. S-Adenosyl Methionine

Explanation: S-Adenosyl Methionine (SAM) is the universal methyl group donor in various biochemical reactions. Key Features of SAM: Formed from methionine and ATP. Transfers methyl groups (-CH■) to a variety of

molecules, including: DNA (methylation) Neurotransmitters (e.g., epinephrine synthesis) Phospholipids After donating its methyl group, it becomes S-adenosyl homocysteine (SAH), which can be recycled back to methionine via the methionine cycle. Glucose-6-phosphate (Option A): Involved in glycolysis and the HMP shunt, not methylation. Acetyl CoA (Option B): Donates acetyl groups (not methyl) in pathways like the TCA cycle and fatty acid synthesis. S-Adenosyl Serine (Option D): No significant role as a methyl donor.

References:

Methyl donor

↑ *Back to this question in Part A*

@Neet_pg_bot

Q38: What is the enzyme that is responsible for the defect causing Maple syrup urine disease? (Q856942)

A. Branched-chain a-ketoacid dehydrogenase

B. Isovaleryl CoA dehydrogenase

C. Pyruvate carboxylase

D. Acetyl CoA carboxylase

Correct Answer: A. Branched-chain a-ketoacid dehydrogenase

Explanation: Maple syrup urine disease (MSUD) is caused by a deficiency of the branched-chain α-ketoacid dehydrogenase (BCKD) complex , which is required for the oxidative decarboxylation of branched-chain amino acids (leucine, isoleucine, valine). Its deficiency leads to accumulation of these amino acids → sweet “maple syrup” odor in urine and severe neurotoxicity. Maple Syrup Urine Disease (MSUD): Autosomal recessive metabolic disorder caused by a deficiency of the enzyme branched-chain α-ketoacid dehydrogenase (BCKD) , Responsible for the breakdown of branched-chain amino acids (BCAAs): Leucine Isoleucine Valine Enzyme Function and Pathophysiology: BCKD is a mitochondrial multienzyme complex that catalyzes the oxidative decarboxylation of branched-chain α-keto acids. These α-keto acids are derived from the transamination of BCAAs. Deficiency Results In: Accumulation of BCAAs and their α-keto acid derivatives (e.g., α-ketoisocaproic acid). Toxic metabolites lead to severe neurological dysfunction due to their impact on brain metabolism. Clinical Features: Neonatal onset : Usually presents within the first few days of life. Poor feeding Vomiting Lethargy Maple syrup odor of urine (due to isoleucine metabolite sotolone) Seizures Neurological damage: Due to leucine accumulation affecting brain function. Treatment : Dietary restriction of BCAAs (low protein diet). Special medical formulas without leucine, isoleucine, and valine. Thiamine supplementation: As BCKD is a thiamine-dependent enzyme. Isovaleryl-CoA dehydrogenase (Option B): Defective in isovaleric acidemia. Pyruvate carboxylase (Option C): Defect causes lactic acidosis, not MSUD. Acetyl-CoA carboxylase(Option D): Involved in fatty acid synthesis, unrelated to BCAA metabolism.

References:

MSUD

↑ *Back to this question in Part A*

@Neet_pg_bot

Q39: In a patient with Maple Syrup Urine Disease (MSUD), all of the following amino acids should be restricted in diet except? (Q855044)

A. Methionine

B. Valine

C. Leucine

D. Isoleucine

Correct Answer: A. Methionine

Explanation: Methionine breaks down in maple syrup urine disease. Maple syrup urine disease is a rare genetic disorder that affects the breakdown of the branched-chain amino acids (BCAAs) leucine, isoleucine, and valine. In people with maple syrup urine disease, they cannot be metabolized properly due to a deficiency in the enzyme branched-chain ketoacid dehydrogenase. Burnt sugar odour from urine Ketosis Mental retardation Abnormal muscle tone

References:

www.ncbi.nlm.nih.gov/books/NBK557773/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q40: Which of the following options best describes the mutation seen in patients with cystic fibrosis? (Q855554)

A. Tryptophan at 708 position, chloride channel

B. Phenylalanine at 708 position, calcium channel

C. Phenylalanine at 508 position, chloride channel

D. Tryptophan at 508 position, calcium channel

Correct Answer: C. Phenylalanine at 508 position, chloride channel

Explanation: The mutation seen in cystic fibrosis (CF) is CFTR (Cystic Fibrosis Transmembrane Conductance Regulator) gene. Gene affected: CFTR gene, located on chromosome 7. Mutation type: The most common mutation is F508 (deletion of three nucleotides leading to the loss of phenylalanine at position 508). Effect : This mutation leads to defective CFTR protein, which functions as a chloride channel in epithelial cells. Pathophysiology : Defective chloride transport results in thick, sticky mucus affecting lungs, pancreas, and other organs. Inheritance : Autosomal recessive disorder. Clinical Features of Cystic Fibrosis (CF): Respiratory : Chronic cough, recurrent infections, bronchiectasis, nasal polyps. Gastrointestinal : Pancreatic insufficiency, malabsorption, meconium ileus, liver disease. Nutritional : Vitamin deficiencies (A, D, E, K), failure to thrive.

References:

www.ncbi.nlm.nih.gov/books/NBK493206/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q41: A 62-year-old male with CAD comes to the physician for a follow-up examination. His blood pressure is 130/80 mm Hg, height of 182 cm, weight of 85 kg, and a BMI of 25 kg/m². Serum studies show a glucose concentration of 95 mg/dL and homocysteine concentration of 19.3 μmol/L (N=5–15). Serum lipid concentrations are within the normal range. Which of the following enzyme deficiencies is most likely to be found in this patient? (Q857892)

A. Homogentisate oxidase

B. Cystathionine beta synthase

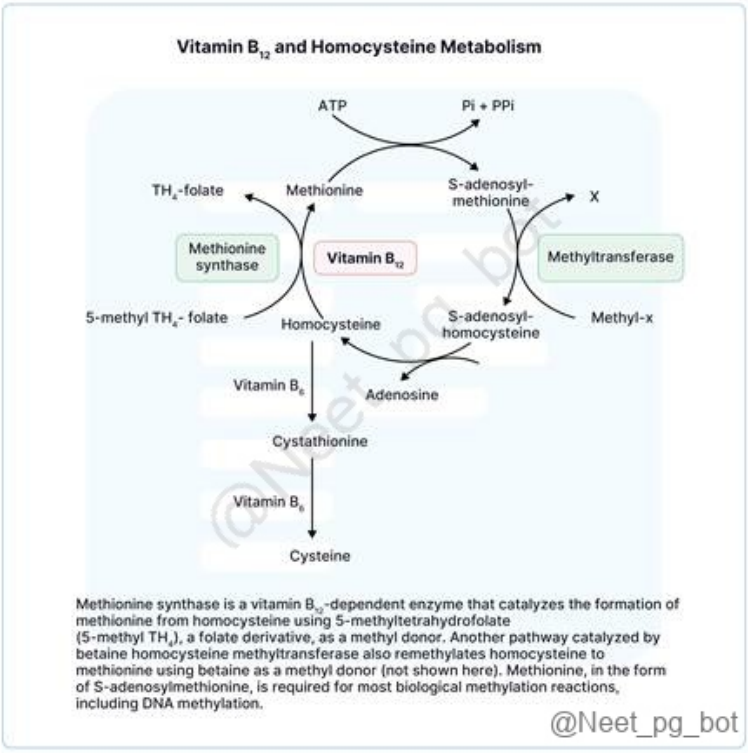
C. Cystathionine gamma synthase

D. HGPRTase

Correct Answer: B. Cystathionine beta synthase

Explanation: The clinical scenario CAD patient with high homocysteine levels is suggestive of homocystinuria caused by Cystathionine beta synthase deficiency is associated with homocystinuria. Homocystinuria: A genetic disorder characterized by elevated levels of homocysteine in the blood. The clinical manifestations of homocystinuria can include cardiovascular issues (such as early-onset atherosclerosis), skeletal abnormalities, intellectual disabilities, and other systemic complications. The patient in the question has an elevated homocysteine concentration, which is characteristic of homocystinuria. Types of Homocystinuria: Type 1 vs Type 2 Homocystinuria: Feature Type - I Homocystinuria Type - II Homocystinuria Enzyme Deficiency Cystathionine beta-synthase Methionine synthase Metabolic Pathway Affected Homocysteine to cystathionine Homocysteine to methionine Elevated Metabolites Homocysteine and methionine Homocysteine and elevated levels of methionine

Biochemical Diagnosis Low levels of cysteine. High levels of methionine. High levels of cysteine. Low levels of methionine. Treatment Vitamin B6 (pyridoxine) supplementation; low-methionine diet; cysteine supplementation Vitamin B12 and folate supplementation; low-methionine diet Homogentisate oxidase (Option A) deficiency leads to alkaptonuria, a condition characterized by the accumulation of homogentisic acid, causing darkening of urine and connective tissue. Cystathionine gamma synthase (Option C) deficiency is not a well-known enzyme deficiency associated with a specific clinical disorder. Cystathionine gamma synthase is involved in the transsulfuration pathway, converting cystathionine to cysteine. HGPRTase (Hypoxanthine-guanine phosphoribosyltransferase) (Option D) deficiency is seen in Lesch-Nyhan syndrome, which is characterized by neurological and behavioral abnormalities, along with uric acid overproduction leading to gout.



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg. 544

Vasudevan 7th edition , pg no 454,477,478,479,482,483,486

Adenosylcobalamin

Methylcobalamin

↑ *Back to this question in Part A*

@Neet_pg_bot

Q42: Which of the following neurotransmitter is not derived from amino acid? (Q839939)

A. GABA

B. Acetylcholine

C. Dopamine

D. 5 hydroxytryptamine

Correct Answer: B. Acetylcholine

Explanation: Most neurotransmitters are biogenic amines derived from amino acids , but acetylcholine is an exception — it is synthesized from acetyl-CoA and choline , not from any amino acid. Neurotransmitter Derived From Key Enzyme GABA Glutamate (Amino acid) Glutamate decarboxylase Dopamine Tyrosine (Amino acid) Tyrosine hydroxylase, DOPA decarboxylase 5-Hydroxytryptamine (Serotonin) Tryptophan (Amino acid) Tryptophan hydroxylase, decarboxylase Acetylcholine Choline + Acetyl-CoA Choline acetyltransferase .

References:

.Harper's Illustrated Biochemistry, 32nd Edition, Page 19, 308

↑ *Back to this question in Part A*

@Neet_pg_bot

Q43: Replacing alanine by which amino acid, will increase the UV absorbance of protein at 280nm wavelength? (Q854875)

A. Leucine

B. Proline

C. Arginine

D. Tryptophan

Correct Answer: D. Tryptophan

Explanation: Tryptophan has a strong UV absorbance at 280 nm due to its aromatic side chain. Replacing alanine (which has low absorbance at 280 nm) with tryptophan will significantly increase the protein’s UV absorbance at this wavelength. Tryptophan is non polar, hydrophobic and aromatic amino acid. Involved in protein synthesis and neurotransmitter synthesis. Absorption of UV light at different wavelengths: At 280nm: Aromatic amino acids like tryptophan and phenylalanine At 260nm: Purines and pyrimidines Leucine (Option A): Leucine is also a non-aromatic amino acid and does not significantly contribute to UV absorbance at 280nm. Proline (Option B):

Proline is an imino acid and absorbs UV light at 260nm. Therefore, it would not increase the UV absorbance at 280nm wavelength. Arginine (Option C): Arginine is a positively charged amino acid and does not possess an aromatic ring.It does not contribute significantly to UV absorbance at 280nm.

References:

Tryptophan

↑ *Back to this question in Part A*

@Neet_pg_bot

Q44: Tau protein inclusions are involved in: (Q866657)

A. Alzheimer’s disease

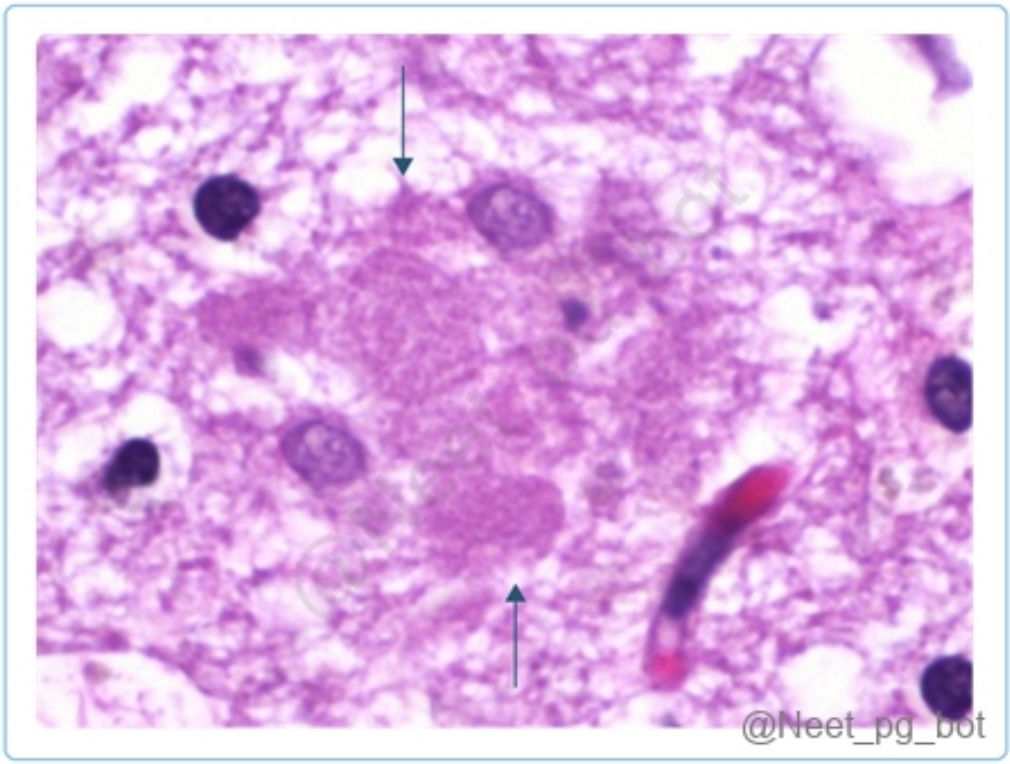
B. Huntington's disease

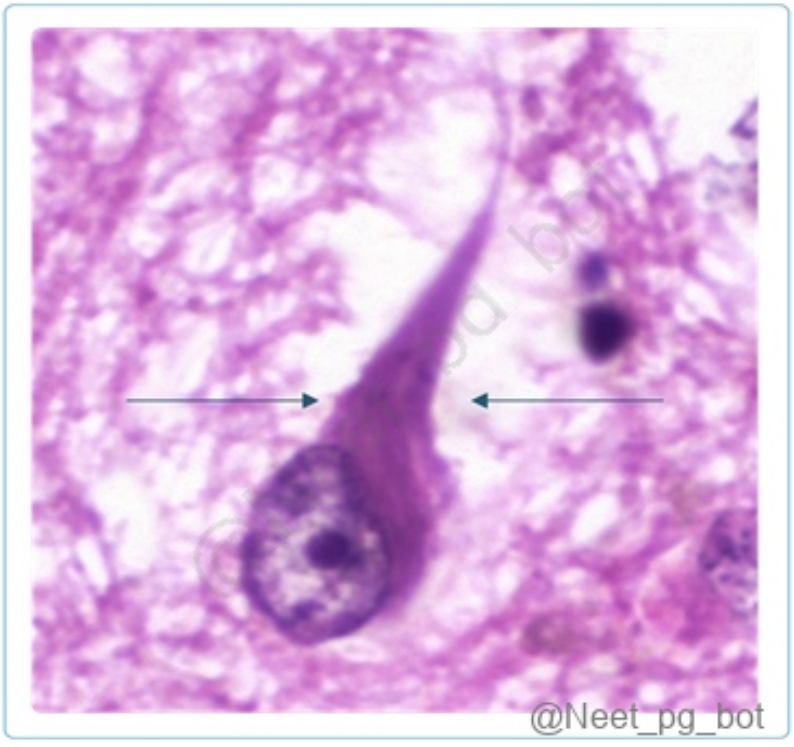
C. Amyotrophic lateral sclerosis

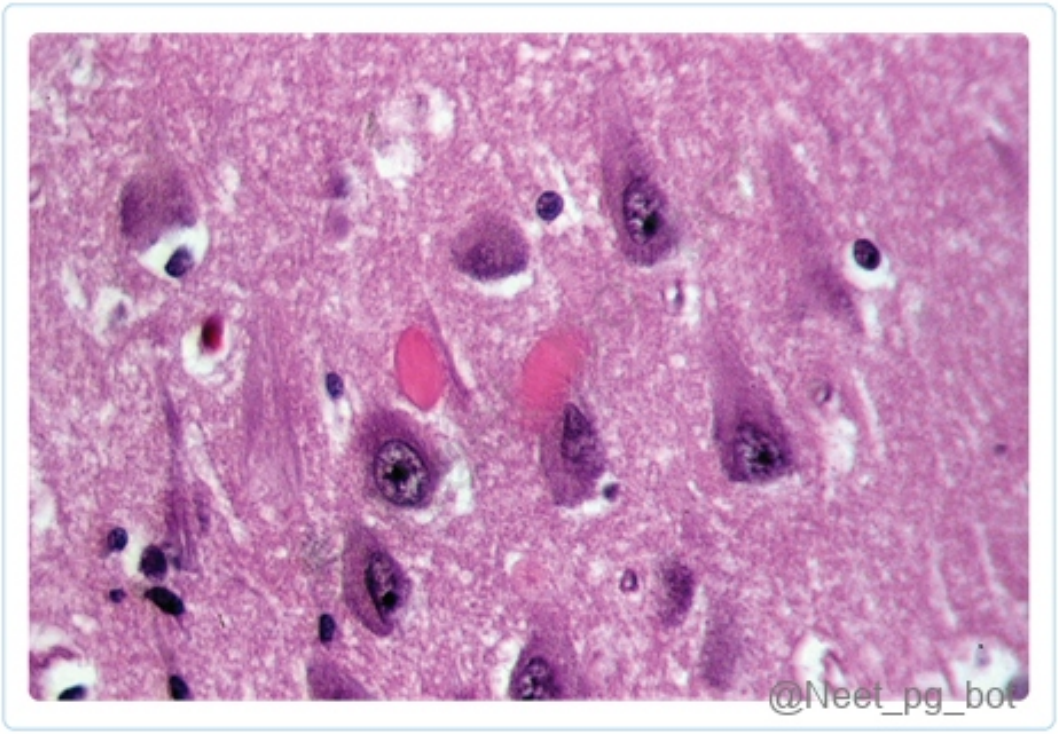
D. CNS lymphoma

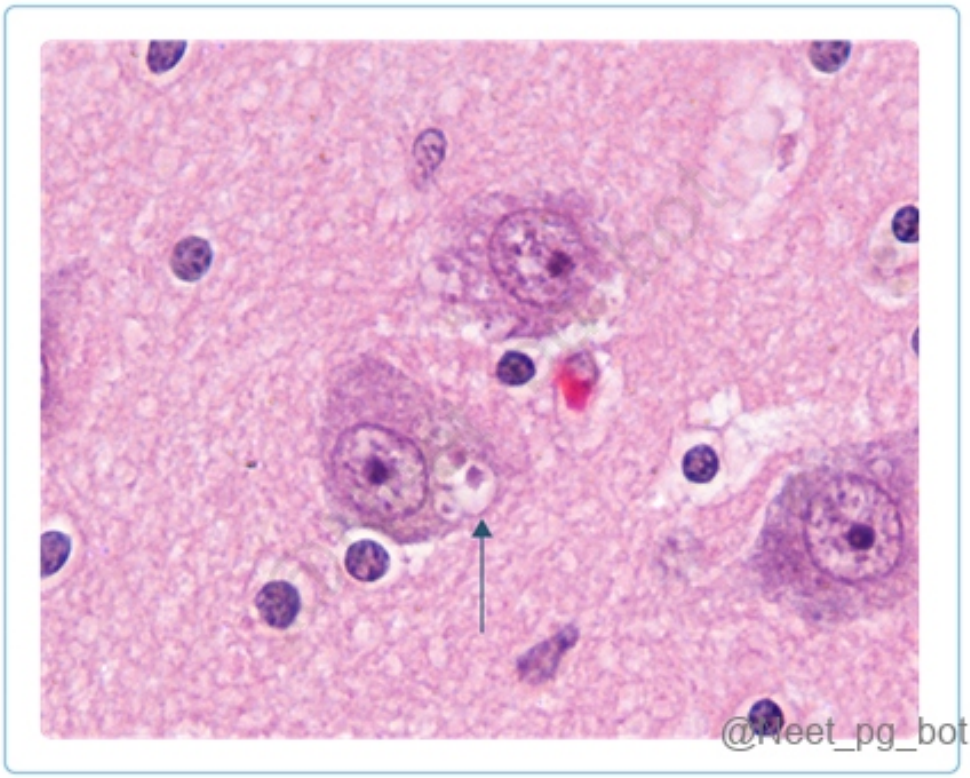
Correct Answer: A. Alzheimer’s disease

Explanation: The fundamental abnormality in Alzheimer’s disease is the accumulation of two proteins (Aβ and tau) in specific brain regions in the form of plaques and tangles. Morphology of Alzheimer’s disease: Gross: Cortical atrophy (narrowed gyri and widened sulci) is predominant in the frontal, temporal, and parietal lobes. Histology: Plaques Deposits of Aβ peptides in the neuropil. They are of two types: Diffuse and neuritic (senile). Neuritic plaques contain both Aβ 40 and Aβ 42. Diffuse plaques are predominantly made up of Aβ 42 and seen in trisomy 21 patients. Tangles Aggregates of the microtubule-binding protein tau. Demonstrated by silver (Bielschowsky) staining. Commonly found in cortical neurons, especially in the entorhinal cortex, and in other sites such as pyramidal cells of the hippocampus, amygdala, basal forebrain, and raphe nuclei. Hirano bodies Intracellular eosinophilic inclusions are typically found in conditions like Alzheimer's disease and Creutzfeldt–Jakob disease. Granulovacuolar degeneration Formation of small, clear vacuoles within the neurons. Plaques Neurofibrillary tangles Hirano bodies Granulovacuolar degeneration Incorrect Options: Huntington's Disease (Option B) is characterised by toxic gain-of-function of mutant huntingtin protein and neuronal loss in the striatum. Amyotrophic Lateral Sclerosis (ALS) (Option C) involves the degeneration of motor neurons, often linked to superoxide dismutase 1 (SOD1) mutations or TDP-43 protein inclusions. CNS Lymphoma (Option D) is a type of brain tumour originating from lymphocytes (B cells) and is unrelated to tau protein.


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↑ *Back to this question in Part A*

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Q45: Which of the following is the best method to detect HbA1c? (Q856564)

A. lon-exchange chromatography

B. Immunoassay

C. Electrophoresis

D. Affinity chromatography

Correct Answer: A. lon-exchange chromatography

Explanation: Glycated hemoglobin (HbA1c) is measured by ion exchange chromatography. This method is based on difference in charge. A mixture of charged proteins is analyzed over carboxymethylcellulose paper which is negatively charged. A positively charged resin bed separates negatively charged HbA1c. It has the advantage of separating hemoglobin variants into peaks. Some hemoglobin variants may interfere with results. Electrophoresis (Option B) : It is the movement of biomolecules in the electric field. It depends on charge, size and shape It is used to separate the components of a protein mixture It denatures the protein structure and coats all the protein with a negative charge. Immunoassay (Option C) measures the presence or concentration of a macromolecule in a solution through the use of an antibody (usually) or an antigen, e.g. ELISA Affinity chromatography (Option D) is based on affinity between the molecules. A mixture of enzymes and other proteins is analysed wherein the other protein elutes first. It is used to separate enzymes, vitamins, antibodies and hormones.

References:

Vasudevan 7th edition Pg no: 445-453

↑ *Back to this question in Part A*

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Q46: Thermogenin protein is mainly found in: (Q857488)

A. Golgi complex

B. Mitochondria

C. Endoplasmic reticulum

D. Nucleus

Correct Answer: B. Mitochondria

Explanation: Thermogenin is an uncoupling protein (UCP). It is mostly found in the inner mitochondrial membrane in the mitochondria of brown adipose tissue cells. Its main function is to produce heat to maintain the

body's temperature. It acts by blocking the formation of ATP and instead, the energy from cellular respiration is used in generating heat. Thermogenin plays a crucial role in thermogenesis and maintains the body temperature. Thermogenin is an uncoupling protein which is present in the mitochondria of brown adípose tissue cells Acts by preventing ATP synthesis. Dissipated energy is produced in cellular respiration as heat. Maintains body temperature by thermogenesis. Other uncouplers include: 2,4-dinitrophenol (DNP) Dinitrocresol Pentachlorophenol Trifluorocarbonylcyanide phenylhydrazone (FCCP)

References:

Thermogenin

↑ *Back to this question in Part A*

@Neet_pg_bot

Q47: Oxaloacetate is produced from which of the following amino acid? (Q857864)

A. Alanine

B. Lysine

C. Leucine

D. Aspartate

Correct Answer: D. Aspartate

Explanation: Aspartate is the amino acid that contributes to the production of oxaloacetate through transamination reactions in the citric acid cycle. Aspartic Acid (Aspartate): Isolation and Characteristics: Aspartic acid (Asp) was first isolated by Kreusler in 1869. It is a non-essential, glucogenic amino acid, meaning it can be synthesized by the body and can be converted to glucose. Transamination and TCA Cycle : Aspartate undergoes transamination to produce oxaloacetate, a key intermediate that initiates the TCA (Krebs) cycle. The enzyme Aspartate Aminotransferase (AST), which requires PLP (pyridoxal phosphate) as a coenzyme, transfers the amino group of aspartate to α-ketoglutarate, forming oxaloacetate and glutamate. Reaction: $\text{Aspartate} + \alpha\text{-Ketoglutarate} \rightarrow^{\text{AST/PLP}} \text{Oxaloacetate} + \text{Glutamate}$ Clinical Significance of AST: AST levels are clinically important: Elevated AST is seen in conditions like cardiac ischemia and hepatic diseases (such as liver damage or hepatitis). Malate-Aspartate Shuttle: Transfers cytoplasmic NADH into the mitochondria for oxidation in the electron transport chain, contributing to ATP production. Role in the Urea Cycle: Donating its alpha-amino group to form urea, which is excreted in urine. Glucogenic Pathway: The carbon skeleton of aspartate can also be converted to fumarate, which enters the glucogenic pathway, contributing to glucose synthesis. Reaction: $\text{Citrulline} + \text{Aspartate} \rightarrow \text{Arginine} + \text{Fumarate}$ Synthesis of Purines and Pyrimidines: Synthesis of purines: contributing the 1st nitrogen and 6th NH_2 group. Pyrimidine synthesis: Its entire structure is incorporated into the pyrimidine ring, contributing the 1st nitrogen and carbons 4, 5, 6. Alanine (Option A) can be involved in the glucose-alanine cycle and can be converted into pyruvate, not oxaloacetate. Lysine (Option B) is an essential amino acid, and it is not directly involved in the synthesis of oxaloacetate. Leucine (Option C) is also not directly involved in the synthesis of oxaloacetate. It is primarily involved in protein synthesis and can be catabolized to

generate acetyl-CoA, which can then enter the citric acid cycle.

References:

Vasudevan Edition 7 page no:226

↑ *Back to this question in Part A*

@Neet_pg_bot

Q48: A farmer sprayed a chemical in the pool which causes inhibition of complex-III of the electron transport chain. Identify the inhibitor? (Q857851)

A. Rotenone

B. TTFA

C. Cyanide

D. Antimycin

Correct Answer: D. Antimycin

Explanation: Antimycin is an inhibitor of complex III (cytochrome b-c1 complex) of the electron transport chain. It interrupts the transfer of electrons from ubiquinol to cytochrome c, thereby inhibiting the flow of electrons through this complex. Inhibitors of the Electron Transport Chain (ETC): Inhibitor Target Site Mechanism Rotenone (Option A) Amobarbital Piericidin A Complex I (NADH dehydrogenase) Blocks electron transfer from Complex I to ubiquinone■ TTFA (Thenoyltrifluoroacetone) (Option B) Carboxin Complex II (Succinate dehydrogenase) Blocks electron transfer from Complex II to ubiquinone■ Malonate Complex II Malonate is a competitive inhibitor of Complex II. Antimycin A (Option D) BAL Complex III Cytochrome bc1 complex Blocks electron transfer from Complex III to Cytochrome C. Cyanide (CN–) (Option C) Carbon monoxide (CO) Hydrogen Sulfide (H2S) Complex IV (Cytochrome c Oxidase) Prevents electron transfer from Complex IV to oxygen■ Oligomycin ATP Synthase (Complex V) Blocks the proton channel of ATP synthase, preventing ATP synthesis■ Atractyloside Adenine Nucleotide Translocase Blocks ADP/ATP exchange across the mitochondrial membrane■ 2,4-dinitrophenol (Option D) is an uncoupling agent that disrupts the coupling of electron transport and ATP synthesis in mitochondria. If DNP were added, it would uncouple oxidative phosphorylation from respiration but wouldn’t block respiration entirely.

References:

Harper’s Illustrated Biochemistry, 32nd edition, pg: 126

↑ *Back to this question in Part A*

@Neet_pg_bot

Q49: A patient presents with recurrent episodes of severe body pain triggered by vigorous exercise. Laboratory findings reveal anemia and the presence of sickle-shaped red blood cells. He is diagnosed with sickle cell anemia. What amino acid substitution occurs in sickle cell anemia? (Q857841)

A. Substitution of valine by glutamic acid at the 6th position

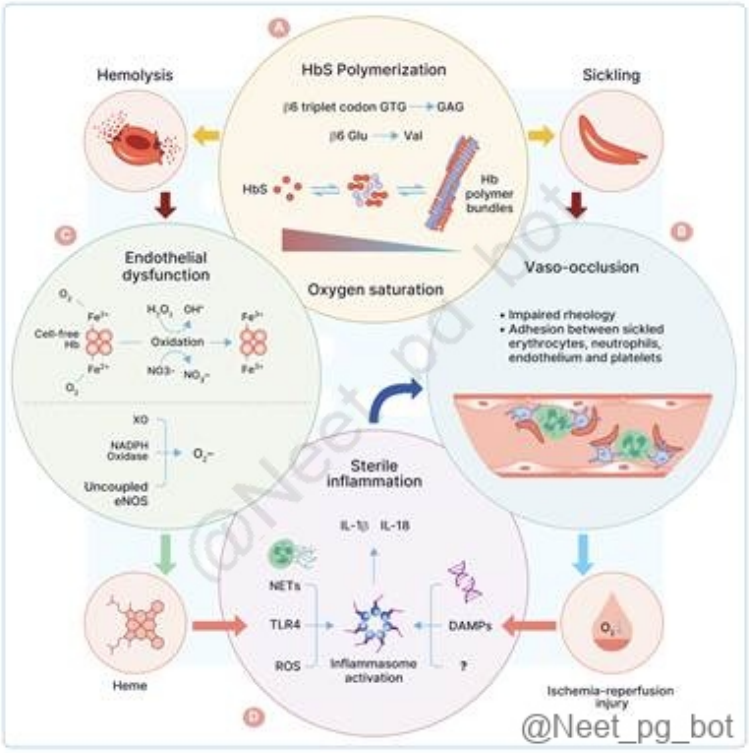
B. Substitution of glutamic acid by valine at the 6th position

C. Substitution of valine by glutamic acid at the 5th position

D. Substitution of glutamic acid by valine at the 5th position

Correct Answer: B. Substitution of glutamic acid by valine at the 6th position

Explanation: Sickle cell anemia is caused by a point mutation in the β-globin gene on chromosome 11 . The mutation replaces glutamic acid (polar, hydrophilic) with valine (nonpolar, hydrophobic) at the 6th position of the β-chain of hemoglobin (HbA → HbS) . This single substitution leads to hydrophobic interactions between hemoglobin molecules under low oxygen tension , causing polymerization of HbS and sickling of RBCs . Sickled cells are rigid and fragile → vaso-occlusive crises , hemolytic anemia , and ischemic pain episodes .



References:

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https://pmc.ncbi.nlm.nih.gov/articles/PMC7053558/#

↑ *Back to this question in Part A*

@Neet_pg_bot

Q50: What do chaperones assist in? (Q855214)

A. Ubiquitinization of proteins

B. Protein modification

C. Protein folding

D. Protein cleavage

Correct Answer: C. Protein folding

Explanation: Chaperones are essential proteins that aid in the folding of nascent or denatured proteins into their correct three-dimensional conformations. Prevent Misfolding: Chaperones prevent incorrect interactions between amino acid residues during protein synthesis or stress conditions (e.g., heat shock). Assist Refolding: In cases where proteins lose their structure due to stress (like heat shock or chemical damage), chaperones help refold them. Types of Chaperones: Heat shock proteins (HSPs): Act during stress (e.g., Hsp70, Hsp90). Chaperonins (e.g., GroEL-GroES complex): Create a safe environment for folding inside a barrel-like structure. Energy Dependency: Many chaperones require ATP for their function, especially during binding and release cycles (e.g., Hsp70).

References:

Chaperones

↑ *Back to this question in Part A*

@Neet_pg_bot

Q51: DNA sequence is determined by? (Q857323)

A. Flow cytometry

B. FISH

C. RFLP

D. PCR

Correct Answer: D. PCR

Explanation: Polymerase Chain Reaction (PCR) amplifies specific DNA sequences, and the amplified product can then be sequenced to determine the exact DNA sequence. PCR is the essential first step in most modern sequencing techniques. It allows targeted identification of DNA by generating many copies of the region of interest. Flow cytometry (Option A): Used for cell counting and analysis of cell characteristics. FISH (Fluorescence In Situ Hybridisation) (Option B): Used to detect and localise the presence of specific DNA sequences on chromosomes. RFLP (Restriction Fragment Length Polymorphism) (Option C): A method used to detect genetic variation based on the length of restriction enzyme-digested DNA fragments.

References:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3883366/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q52: Which of the following methods cannot be used to precipitate proteins? (Q856573)

A. Add alcohol and acetone

B. Using heavy metal ions

C. Moving pH away from isoelectric pH

D. Adding trichloroacetic acid

Correct Answer: C. Moving pH away from isoelectric pH

Explanation: At the isoelectric pH, proteins are least soluble because they have no net charge. This results in minimal repulsion between molecules, causing them to aggregate and precipitate. Hence, high solubility is not a characteristic of the isoelectric pH. For proteins and amino acids, this is the pH where the molecule exists as a zwitterion with equal numbers of positive and negative charges, resulting in a net zero charge and no migration in an electric field. Method Mechanism of precipitation 1. Heat 2. Strong mineral acids Cause precipitation by denaturation 3. Heavy metal salts in alkaline medium In alkaline medium Proteins are negatively are positively charged So, Neutralization of charges occur which leads to precipitation known as positive ion precipitation 4. Alkaloidal reagents (Trichloroacetic acid, phosphotungstic acid, sulfosalicylic acid) Alkaloidal reagents are

negatively charged In acidic medium , protein are positively charged So, Neutrialization of charges occur & leads to negative ion precipitation 5. Organic solvents e.g. alcohol & acetone 6. Neutral salts e.g. ammonium sulfate Both cause precipitation by dehydration of proteins Add alcohol and acetone (Option A) Alkaloidal reagents are negatively charged. In an acidic medium, proteins are positively charged. So, by neutralization of charges, negative ion precipitates out. Using heavy metals and ion (Option B) Neutralisation of charges occur which lead to precipitation of positive ion as heavy metals are positively charged. Adding trichloroacetic acid (Option D) It is negatively charged. In an acidic medium, proteins are positively charged. So, neutralization of charges occurs and the negative ion is separated.

References:

Harper’s Biochemistry, 32nd edition pg 20

Textbook of Biochemistry by DM Vasudevan, 7th edition, pg 28

↑ *Back to this question in Part A*

@Neet_pg_bot

Q53: Aromatic ring is seen in which of the following amino acids? (Q860441)

A. Phenylalanine, valine, tryptophan

B. Phenylalanine, tryptophan, tyrosine

C. Tyrosine, cysteine, tryptophan

D. Lysine, methionine, valine

Correct Answer: B. Phenylalanine, tryptophan, tyrosine

Explanation: Classification of amino acids based on the functional group: Amino acids Examples Basic amino acids Arginine, Lysine , Histidine (Option D ruled out) Acidic amino acids Aspartic acid, Glutamic acid OH group-containing amino acids Serine, Threonine, Tyrosine SH group-containing amino acids Cysteine , Methionine (Option C, D ruled out) NH2 group-containing amino acids Asparagine, Glutamine Aromatic amino acids (Option B) Phenylalanine, Tyrosine, Tryptophan, Histidine Aliphatic amino acid Glycine, Alanine, Branched chain AA (Valine , Leucine, Isoleucine) (Option A, D ruled out) Imino acid Proline

References:

Harper’s Biochemistry, 32nd edition pg 18,19, 291, 274

↑ *Back to this question in Part A*

@Neet_pg_bot

Q54: If the RNA sequence is CAAGGU, what is the sequence of the template strand of DNA? (Q860375)

A. CAAGGT

B. GTTCCA

C. ACCTTG

D. TGGAAC

Correct Answer: C. ACCTTG

Explanation: RNA Sequence given: CAAGGU RNA-DNA Relationship RNA is transcribed from the template strand of DNA. The template strand is complementary to the RNA sequence. Base-pairing rules: Cytosine (C) ↔ Guanine (G) Adenine (A) ↔ Thymine (T) Uracil (U in RNA) ↔ Adenine (A in DNA) Step-by-Step Conversion RNA: CAAGGU Template DNA strand (complementary and antiparallel): C ↔ G A ↔ T A ↔ T G ↔ C G ↔ C U ↔ A Template strand: GTTCCA However, DNA sequences are conventionally written in the 5' → 3' orientation. Reverse the 3' → 5' sequence to obtain its 5' → 3' orientation That is GTTCCA → ACCTTG.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg. 349, 355

↑ *Back to this question in Part A*

@Neet_pg_bot

Q55: Ammonia is transported from the brain to the liver in the form of which amino acid? (Q860348)

A. Glutamate

B. Alanine

C. Glycine

D. Glutamine

Correct Answer: D. Glutamine

Explanation: Glutamine synthetase in astrocytes converts glutamate and ammonia to glutamine, effectively detoxifying ammonia in the brain. Ammonia Management in Neurons: Ammonia can be neurotoxic if accumulated. Glutamine Synthesis: Produced in neurons as a byproduct of amino acid metabolism and neurotransmitter turnover. Ammonia reacts with α-ketoglutarate and NADH to form glutamate, catalyzed by glutamate dehydrogenase. α-ketoglutarate + NH 3 ■ ■+ NADH → Glutamate + NAD + + H2■O Glutamate is

converted to glutamine by glutamine synthetase, adding another ammonia molecule. Glutamine synthetase is predominantly found in astrocytes. Glutamate + NH 3 ■ + ATP → Glutamine + ADP + Pi This conversion reduces ammonia toxicity by storing it in a non-toxic form. (Option A ruled out) The fate of glutamine: Release into the Bloodstream: Glutamine is released into the extracellular space and enters the bloodstream. This transport helps maintain nitrogen balance and prevents ammonia accumulation. Transport to the Liver: Glutamine carries ammonia from the brain to the liver, where it is deaminated to release ammonia. Ammonia enters the urea cycle in the liver for detoxification. Importance of Glutamine in Neuronal Health: Acts as a non-toxic ammonia carrier, preventing neurotoxicity. Serves as a precursor for neurotransmitters like GABA , maintaining neurotransmitter balance. Glycine (Option C): Glycine is involved in various metabolic processes but does not play a role in ammonia transport from the brain to the liver.

References:

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https://www.sciencedirect.com/science/article/pii/S019701862030200X

Lippincott Edition 7: Chapter 9: Amino Acids: Nitrogen Disposal

↑ *Back to this question in Part A*

@Neet_pg_bot

Q56: A patient presents with arthritis, and arthroscopy reveals blackening of the joints. Which enzyme deficiency is most likely responsible for this condition? (Q852546)

A. Homogentisate oxidase

B. Branched-chain alpha-ketoacid dehydrogenase

C. Tyrosinase

D. Fumarase

Correct Answer: A. Homogentisate oxidase

Explanation: Arthritis with joint blackening suggests alkaptonuria , a disorder caused by homogentisate oxidase deficiency, leading to the accumulation of homogentisic acid, which deposits in joints and cartilage. Ochronosis (dark pigmentation): Homogentisic acid polymerizes and stains connective tissues, cartilage, and joints black , seen during arthroscopy. Other features: Black urine (on standing due to oxidation), ochronotic pigmentation in the sclera and ear cartilage. Alkaptonuria Feature Alkaptonuria (Homogentisate Oxidase Deficiency) Deficient Enzyme Homogentisate oxidase Accumulated Metabolite Homogentisic acid Key Features Black urine (on standing), ochronosis (dark connective tissue pigmentation), early-onset arthritis Affected Areas Joints, cartilage (ear, sclera, intervertebral discs) Diagnosis Urine darkening with alkalization, elevated homogentisic acid in urine

Treatment Low-protein diet (restricting phenylalanine & tyrosine), vitamin C, symptomatic management Branched-chain alpha-ketoacid dehydrogenase (Option B): Incorrect because its deficiency causes Maple Syrup Urine Disease (MSUD), characterized by neurological symptoms, metabolic acidosis, and sweet-smelling urine, not arthritis. Tyrosinase (Option C): Incorrect because its deficiency leads to albinism, causing hypopigmentation, not joint issues. Fumarase (Option D): Incorrect because its deficiency affects the Krebs cycle, leading to metabolic acidosis, hypotonia, and neurological dysfunction, not ochronosis or arthritis.

References:

Harper's Illustrated Biochemistry, 32nd Edition Pg. 296

↑ *Back to this question in Part A*

@Neet_pg_bot

Q57: Which of the following amino acids primarily acts as a buffer in blood due to its ability to accept and donate protons at physiological pH? (Q852550)

A. Histidine

B. Arginine

C. Tryptophan

D. Tyrosine

Correct Answer: A. Histidine

Explanation: Physiological pH & Buffering Role: Histidine has an imidazole side chain with a pKa of ~6.0 , which is close to physiological pH (7.4). This allows it to accept or donate protons , making it an effective buffer in blood . Role in Hemoglobin Buffering: Hemoglobin contains histidine residues that help maintain blood pH by binding or releasing H■ ions. This is essential in the Bohr effect , which regulates oxygen delivery. Amino Acids as Buffers Amino Acid Buffering Ability Reason Histidine Strong buffer Imidazole group has pKa ~6.0, close to physiological pH Arginine Poor buffer Guanidinium group has pKa ~12.5, too high to act as a buffer at pH 7.4 Tryptophan Minimal buffering Indole group is non-ionizable at physiological pH Tyrosine Minimal buffering Phenol group has pKa ~10.1, too high to be effective Arginine (Option B): Incorrect because its guanidinium group (pKa ~12.5) does not donate or accept protons at physiological pH. Tryptophan (Option C): Incorrect because its indole ring is not significantly ionizable at pH 7.4, so it lacks buffering ability. Tyrosine (Option D): Incorrect because its phenol group (pKa ~10.1) does not participate in blood buffering at physiological pH.

References:

Harper's Biochemistry, 32nd edition pg 18,19, 20, 21.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q58: Which amino acid donates nitrogen to sodium benzoate when given for urea cycle disorders? (Q853005)

A. Glutamine

B. Glutamate

C. Glycine

D. Arginine

Correct Answer: C. Glycine

Explanation: Sodium benzoate : It reacts with glycine in the liver to form hippuric acid, which is then excreted in the urine, reducing the availability of glycine. Reducing the availability of glycine shifts the equilibrium to favour glycine formation. Ammonia is used to produce glycine, therefore reducing blood ammonia levels.

References:

https://www.uptodate.com/contents/urea-cycle-disorders-management

https://www.ncbi.nlm.nih.gov/books/NBK547972/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q59: Match the following amino acid with derivatives: 1. Histidine a.Catecholamines 2. Tyrosine b. Histamine 3. Tryptophan c. Glutathione 4. Cysteine d. Melatonin (Q852999)

A. 1-b, 2-a, 3-d, 4-c

B. 1-a, 2-c, 3-b, 4-d

C. 1-d, 2-c, 3-b, 4-a

D. 1-b, 2-c, 3-d, 4-a

Correct Answer: A. 1-b, 2-a, 3-d, 4-c

Explanation: Amino Acid Derivative Function/Importance Glutamine Glutamate Neurotransmitter Tyrosine Dopamine Neurotransmitter Norepinephrine Neurotransmitter Epinephrine (Adrenaline) Hormone Tryptophan

Serotonin Neurotransmitter Melatonin Hormone Phenylalanine Tyrosine Precursor to dopamine, norepinephrine, and epinephrine. Histidine Histamine Involved in immune response, regulates gastric acid secretion, and neurotransmission. Glutamic Acid GABA (Gamma-Aminobutyric Acid) Inhibitory neurotransmitter Arginine Nitric Oxide Signaling molecule, involved in vasodilation, immune response, and neurotransmission. Cysteine Glutathione Antioxidant Methionine S-Adenosylmethionine (SAM) Methyl donor, involved in methylation reactions, DNA synthesis, and regulation of gene expression.

References:

Harper's Illustrated Biochemistry, 32nd edition, Pages 306-314

↑ *Back to this question in Part A*

@Neet_pg_bot

Q60: A patient has elevated phenylalanine levels (40 mg/dL), but phenylalanine hydroxylase enzyme levels are normal. Which cofactor deficiency is most likely responsible for this condition? (Q866320)

A. Tetrahydrofolate

B. Tetrahydrobiopterin (BH4)

C. Thiamine

D. Pyridoxine

Correct Answer: B. Tetrahydrobiopterin (BH4)

Explanation: The elevated phenylalanine levels despite normal phenylalanine hydroxylase indicate a deficiency of its cofactor, Tetrahydrobiopterin (BH■), which renders the enzyme phenylalanine hydroxylase (PAH) nonfunctional and leads to hyperphenylalaninemia. Phenylalanine has 2 metabolic fates in the body Major Phenylalanine gets converted into tyrosine with the help of enzyme phenylalanine hydroxylase Deficiency of phenylalanine hydroxylase results in Phenylketonuria. Requires tetrahydrobiopterin (BH4) as a cofactor (Option B) Minor Phenylalanine gets converted into Phenylpyruvate by transamination. Phenylpyruvate can further undergo different pathways: Phenylpyruvate can get reduced to phenyl lactate Phenylpyruvate undergoes oxidative decarboxylation to form phenylacetate Phenylacetate combines with glutamine to form phenylacetylglutamine, which is excreted in urine. Incorrect Options: Tetrahydrofolate (Option A) is used in 1-carbon transfers, particularly in nucleotide and amino acid metabolism — not involved in phenylalanine metabolism. Thiamine (Option C) is a cofactor for enzymes like pyruvate dehydrogenase and α-ketoglutarate dehydrogenase, not related to phenylalanine. Pyridoxine (Option D) is a cofactor for transamination reactions and neurotransmitter synthesis — not involved in the PAH reaction.

↑ *Back to this question in Part A*

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Q61: Which of the following is true about the structural organization of proteins? (Q858466)

A. Tertiary structure is three-dimensional

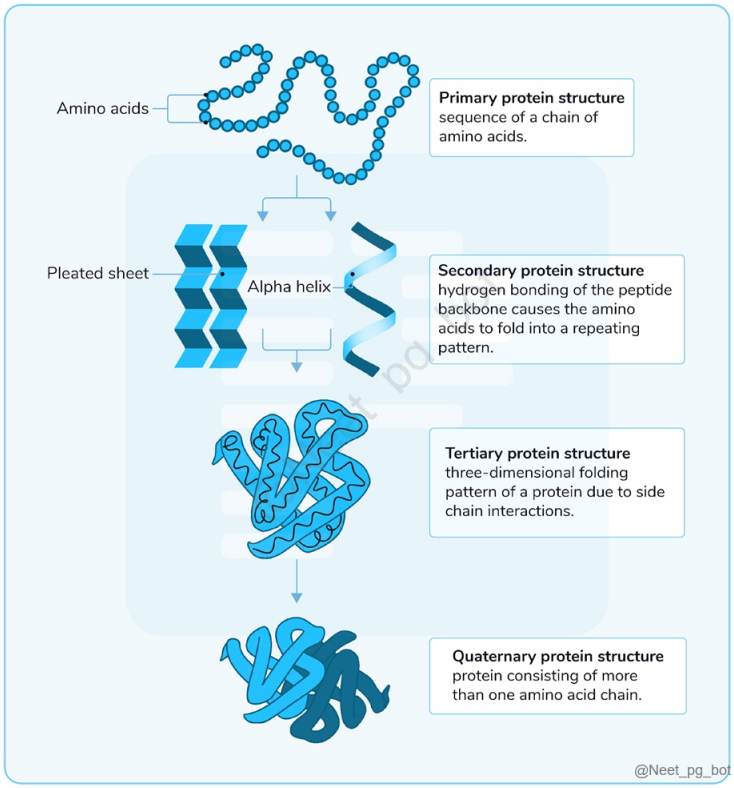
B. The secondary structure is stabilized by disulfide bonds.

C. Primary, secondary, and tertiary structures are destroyed by denaturation.

D. The secondary and tertiary structure depends upon amino acid sequence.

Correct Answer: A. Tertiary structure is three-dimensional

Explanation: The tertiary structure represents the overall three-dimensional folding of a single polypeptide chain. It is stabilized by interactions like hydrogen bonds, ionic bonds, hydrophobic interactions, and disulfide bridges. Functionality : The tertiary structure is crucial for the biological activity of the protein, as it determines the placement of active sites, binding sites, and interaction regions. This makes the three-dimensional nature of tertiary structure a key feature in protein organization. Incorrect Options: Primary, secondary, and tertiary structures are destroyed by denaturation (Option B): Denaturation affects only the secondary, tertiary, and quaternary structures, leaving the primary structure (amino acid sequence) intact. The secondary structure is stabilized by disulfide bonds (Option C): The secondary structure (e.g., α-helices and β-sheets) is primarily stabilized by hydrogen bonds between the carbonyl oxygen and amide hydrogen of the polypeptide backbone. The secondary and tertiary structure depends upon amino acid sequence (Option D): The amino acid sequence (primary structure) dictates how the protein folds into its secondary and tertiary structures, but this does not directly address the structural organization.



References:

Protein structures

↑ *Back to this question in Part A*

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Q62: A 40-year-old female working as a senior executive leading a sedentary lifestyle presents to the OPD with palmoplantar pigmentation. X-ray spine shows calcification of intervertebral discs. On adding Benedict's reagent to the urine sample, it gives a greenish-brown precipitate and comes positive. What is the most likely enzyme deficiency in this patient? (Q860064)

A. Phenylalanine hydroxylase

B. Tyrosine hydroxylase

C. Homogentisic acid oxidase

D. PHPP dioxygenase

Correct Answer: C. Homogentisic acid oxidase

Explanation: The patient is most likely suffering from alkaptonuria, a condition caused by homogentisic acid oxidase deficiency. This leads to the accumulation of homogentisic acid, which is excreted in the urine. When exposed to air, the urine darkens, and a positive Benedict's test (which detects reducing sugars) can produce a greenish-brown precipitate. The palmoplantar pigmentation and intervertebral disc calcification (also known as ochronosis) are characteristic of this condition. Alkaptonuria Definition Rare autosomal recessive genetic disorder caused by a deficiency of homogentisate 1,2 dioxygenase (HGD) , an enzyme in the tyrosine metabolism pathway. Mechanism and Pathophysiology Defective tyrosine metabolism → homogentisic acid (HGA) accumulation → ochronotic pigment deposition → systemic disease (ochronosis). Deficiency: HGD fails to convert HGA to maleylacetoacetate. Consequences: HGA oxidizes into melanin-like polymers → connective tissue deposition (ochronosis). Affects joints, heart valves, kidneys, and vessels. Clinical Features Triad: Homogentisic aciduria, ochronosis, ochronotic osteoarthropathy. Pediatric signs: Dark urine (oxidized HGA). Musculoskeletal : Arthritis (spine, hips, knees), kyphoscoliosis, spinal stenosis. Cardiac : A ortic stenosis, mitral valve disease, coronary artery disease. Renal/Metabolic: Stones (renal, gallbladder, prostate). Neurological : Peripheral neuropathy, stroke. Evaluation Clinical scoring: AKU Severity Score Index (AKUSSI). Diagnostic tests: Gold standard: Urinary HGA quantification via gas chromatography-mass spectrometry. Genetic testing for HGD mutations. Imaging (CT/MRI): Joint involvement, valvular calcification. Echocardiography: Valvular abnormalities. Management Symptomatic : Pain control, physiotherapy, joint surgery. Diet : Low-protein diet (limited efficacy). Medications: Vitamin C : Reduces HGA oxidation but not urinary excretion. Nitisinone : Inhibits tyrosine metabolism, reduces HGA >95%. Side effects: tyrosinemia, leukopenia, thrombocytopenia. Prognosis and Complications Prognosis : Normal life expectancy; reduced quality of life (pain, sleep issues, disability). Complications : Stones (renal, gallbladder, salivary). Tendon/ligament ruptures. Osteopenia, fractures. Aortic calcification/stenosis. Phenylalanine hydroxylase (Option A): Phenylalanine hydroxylase deficiency causes phenylketonuria (PKU), which is associated with intellectual disability, seizures, and a musty odor. The symptoms described here are not typical of PKU. Tyrosine hydroxylase (Option B): Tyrosine hydroxylase deficiency is associated with a condition called hereditary tyrosinemia type 2, which can cause eye and skin problems but does not present with the urine color change or intervertebral disc calcification seen in this case. PHPP dioxygenase (Option D): Deficiency in 4-hydroxyphenylpyruvate dioxygenase causes type III tyrosinemia, which is associated with elevated levels of tyrosine in the blood and urine, but does not typically present with palmoplantar pigmentation or calcification of intervertebral discs.

References:

Vasudevan Textbook of Biochemistry (7th Edition), Page 237, 238

https://www.ncbi.nlm.nih.gov/books/NBK560571/

Harper's Illustrated Biochemistry, 32nd Edition, Page 296, 304

https://pubmed.ncbi.nlm.nih.gov/10942115/#:~:text=Tyrosinemia%20type%20III%20(OMIM%20276710,of%20tyrosine%20and%20phenolic%20metabolites.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q63: A 3-month-old baby presents with severe hepatomegaly, cataracts in both eyes, lethargy, and hypotonia. Based on these symptoms, which enzyme deficiency is most likely involved? (Q860054)

A. Galactokinase

B. Hepatic Kinase

C. Hepatic Glucose-6-Phosphatase

D. Galactose-1-Phosphate Uridyl Transferase

Correct Answer: D. Galactose-1-Phosphate Uridyl Transferase

Explanation: Galactose-1-Phosphate Uridyl Transferase deficiency causes classic galactosemia, a severe metabolic disorder characterized by hepatomegaly, cataracts, lethargy, and hypotonia due to the accumulation of toxic metabolites like galactose-1-phosphate and galactitol. Galactosemia Enzyme Deficiency Galactose-1-phosphate uridylyltransferase (GALT) - Classical type Galactokinase- benign disease with only oil drop cataract. (Option A ruled out) Epimerase- SNHL + symptoms of classical type (Low galactose-based diet can be given which is contraindicated in the classical type) Pathophysiology Accumulation of galactose-1-phosphate (toxic causing jaundice, and liver damage) Inability to metabolize galactose from milk Damage to liver, kidney, and brain Clinical Features Hepatic: Jaundice, hepatomegaly GI: Vomiting, diarrhea, failure to thrive CNS: Seizures, intellectual disability Eyes: Oil drop cataracts Increased risk of E. coli sepsis Diagnosis Reducing substance in urine (positive)(Positive Benedict test but negative glucose oxidase test) Direct enzyme (GALT) assay Genetic testing (Perinatal diagnosis is possible) Management Lactose-free diet Breast Milk contraindicated Soy-based or elemental formulas Calcium supplementation Hepatic Glucose-6-Phosphatase (Option C) : Deficiency of it causes glycogen storage disease type I (Von Gierke disease), which presents with hepatomegaly, hypoglycemia, and lactic acidosis. Cataracts are not a feature of this condition.

References:

Nelson Textbook of Pediatrics, 21st Edition, Pg. 788 and 789

↑ *Back to this question in Part A*

@Neet_pg_bot

Q64: A couple brings their 4-year-old child to the outpatient department (OPD) due to self-mutilating behavior and poor growth. Which of the following associated enzyme deficiencies could explain these symptoms? (Q860052)

A. HGPRT deficiency

B. Homogentisic Aciduria

C. Phenylalanine Hydroxylase deficiency

D. Adenosine deaminase deficiency

Correct Answer: A. HGPRT deficiency

Explanation: Hypoxanthine-guanine phosphoribosyltransferase (HGPRT) deficiency causes Lesch-Nyhan syndrome, a rare X-linked recessive disorder. It is characterized by self-mutilating behavior, hyperuricemia, gout, neurological abnormalities, and poor growth. HGPRT is critical for p urine salvage , and its deficiency leads to an overproduction of uric acid. Lesch-Nyhan Syndrome Clinical Features Metabolic: Severe gout, sodium urate crystal deposition causing j oint swelling and kidney damage . Neurological: Poor muscle control. Involuntary movements (resembling Huntington's disease). Facial grimacing, repetitive limb movements. Behavioral: Self-mutilation (lip and finger biting, starting ~2nd year). Other: Moderate developmental delays , potential megaloblastic anemia (from poor vitamin B12 utilization). Management Metabolic: Allopurinol for gout; lithotripsy for kidney stones. Neurological: Symptomatic treatment with drugs like carbidopa/levodopa, diazepam, phenobarbital, or haloperidol. Homogentisic Aciduria (Option B): Homogentisic aciduria, or alkaptonuria, results from homogentisate 1,2-dioxygenase deficiency. It leads to darkened urine, ochronosis (dark pigmentation of connective tissues), and arthritis but does not cause self-mutilation or poor growth. Phenylalanine Hydroxylase deficiency (Option C): Phenylalanine hydroxylase deficiency causes phenylketonuria (PKU), which presents with intellectual disability, seizures, and hypopigmentation. Self-mutilation and hyperuricemia are not associated with this condition. Adenosine deaminase deficiency (Option D): A rare genetic disorder caused by ADA gene mutations, leading to a lack of B and T lymphocytes and severe immune deficiency. Symptoms, often appearing before 6 months, include recurrent infections, chronic diarrhea, rashes, and delayed growth. Without treatment, life-threatening infections and certain cancers may occur.

References:

https://www.ninds.nih.gov/health-information/disorders/lesch-nyhan-syndrome

Vasudevan Textbook of Biochemistry (7th Edition), Page 237, 238

Harper’s Illustrated Biochemistry, 32nd Edition, Pg 334-336

https://www.cancer.gov/publications/dictionaries/cancer-terms/def/adenosine-deaminase-deficient-severe-combined-immunodeficiency

↑ *Back to this question in Part A*

@Neet_pg_bot

Q65: Zellweger syndrome is associated with which cellular organelle? (Q860043)

A. Peroxisomes

B. Nucleosomes

C. Lysosomes

D. Ribosomes

Correct Answer: A. Peroxisomes

Explanation: Zellweger syndrome is a peroxisomal biogenesis disorder caused by mutations in PEX genes. It results in defective peroxisome formation, leading to an inability to metabolize very long-chain fatty acids (VLCFAs) and plasmalogens. Clinical features include hypotonia, seizures, hepatomegaly, and craniofacial abnormalities. Zellweger Spectrum Disorder Rare autosomal recessive disorder with defective peroxisome biogenesis affecting VLCFA metabolism. Includes Zellweger syndrome, neonatal adrenoleukodystrophy, infantile Refsum disease, and rhizomelic chondrodysplasia punctata. Etiology Caused by mutations in PEX genes encoding peroxins, primarily PEX1/PEX6 Functional peroxisomal disturbances: Zellweger spectrum: Generalized peroxisomal dysfunction. Adrenoleukodystrophy: Mutations in ABCD1, impacting VLCFA transport. Rhizomelic chondrodysplasia punctata: Multiple enzymatic defects. Pathophysiology Peroxisomes: Membrane-bound organelles with >50 enzymes for fatty acid metabolism, abundant in liver/kidney. Mutated PEX genes disrupt peroxisome formation, leading to VLCFA accumulation, reduced steroid biosynthesis, and neuronal damage. Major abnormalities: Brain: Demyelination, neuronal injury. Liver: Fibrosis. Kidney: Cortical cysts. Clinical Features Multisystem involvement with severity depending on age of onset: Neonatal-Infantile: Hypotonia, feeding issues, seizures, facial dysmorphism, ocular and hearing abnormalities, hepatomegaly. Childhood: Developmental delay, FTT, hepatic dysfunction, adrenal insufficiency, neuroregression. Adolescent-Adult: Neuroregression, ataxia, neuropathy, adrenal insufficiency, leukodystrophy. Diagnosis Initial: Clinical features + elevated VLCFA on newborn screening. Biochemical tests: Elevated VLCFA, bile acid intermediates, and phytanic/pristanic acid; reduced plasmalogens. Genetic testing: Confirms PEX mutations. Prenatal diagnosis and genetic counseling recommended. Management No cure; supportive care focuses on improving quality of life. Therapies : Docosahexaenoic acid Lorenzo oil: Reduces VLCFA but doesn't halt disease progression. Cholic acid : Approved for hepatic dysfunction. Supportive Measures: Hearing aids for hearing loss. Ophthalmologic care for vision issues. Antiepileptics for seizures. Vitamin supplementation: Fat-soluble vitamins (A, D, E, K). Cortisone for adrenal insufficiency. Gastrostomy for feeding difficulties. Nucleosomes (Option B): Disorders involving nucleosomes are linked to chromatin remodeling or epigenetic regulation, not Zellweger syndrome. Lysosomes (Option C): Lysosomal storage diseases (e.g., Tay-Sachs disease, Gaucher disease) result from enzyme deficiencies affecting lysosomal function. Zellweger syndrome does not involve lysosomes. Ribosomes (Option D): Diseases involving ribosomes include ribosomopathies like Diamond-Blackfan anemia, which are unrelated to Zellweger syndrome.

References:

https://www.ncbi.nlm.nih.gov/sites/books/NBK560676/

↑ *Back to this question in Part A*

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Q66: Which of the following is caused by the deficiency of the enzyme tyrosine transaminase? (Q860430)

A. Type I Tyrosinemia

B. Type II Tyrosinemia

C. Alkaptonuria

D. Type III Tyrosinemia

Correct Answer: B. Type II Tyrosinemia

Explanation: Type II tyrosinemia is caused by a deficiency of tyrosine transaminase (also known as tyrosine aminotransferase), which leads to the accumulation of tyrosine and its metabolites. This condition primarily affects the eyes, skin, and nervous system, with symptoms including corneal ulcers and intellectual disability. Disorders of Tyrosine Metabolism Disorder Deficiency Enzyme Key Clinical Features Tyrosinemia type I (Option A ruled out) Fumarylacetoacetate hydrolase Jaundice, hepatomegaly, hypoglycemia, HCC, liver cirrhosis Often presents with Fanconi syndrome and renal failure Elevated tyrosine and succinylacetone (pathognomic) Tyrosinemia type II (Option B) Tyrosine aminotransferase Painful corneal erosions, palmoplantar hyperkeratosis Also known as oculocutaneous tyrosinemia Elevated tyrosine Tyrosinemia type III (Option D ruled out) p-Hydroxyphenylpyruvate dioxygenase Recurrent seizures, intermittent ataxia Elevated tyrosine and p-hydroxyphenylpyruvate Alkaptonuria (Black urine disease) (Option C ruled out) Homogentisate 1,2-dioxygenase Dark urine, ochronosis, arthritis Black or brown pigmentation of sclera (Osler's sign) and ear cartilage Elevated homogentisic acid

References:

Harper's Illustrated Biochemistry, 32nd Edition Pg. 296

Pubmed

↑ *Back to this question in Part A*

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Q67: An infant presents to the Pediatrics OPD with a distended abdomen and jaundice. The mother reports that the baby has shown an increased appetite in recent months. During the clinical examination, the physician notes hepatomegaly. Which of the following enzyme deficiencies is most likely responsible for this condition? (Q860357)

A. Glycogen Synthase

B. Glycogen phosphorylase

C. Glucose-6-Phosphatase

D. Galactose-1-Phosphate Uridyl Transferase

Correct Answer: C. Glucose-6-Phosphatase

Explanation: Glucose-6-Phosphatase deficiency causes Von Gierke disease (Glycogen Storage Disease type I). It leads to the inability to convert glucose-6-phosphate to glucose, causing accumulation of glycogen in the liver and kidney. This results in hepatomegaly, marked abdominal distension, jaundice, chronic hunger (increased appetite), and hypoglycemia. Glycogen synthase (Option A): A deficiency in glycogen synthase leads to Glycogen Storage Disease Type 0 , characterized by inability to store glycogen , leading to hypoglycemia without hepatomegaly or jaundice . Glycogen Phosphorylase (Option B): Causes Hers disease (GSD Type VI), which presents with milder hypoglycemia and hepatomegaly, but no jaundice or severe symptoms . Galactose-1-Phosphate Uridyl Transferase (Option D) : It's deficiency causes Classic Galactosemia which presents with vomiting, cataracts, failure to thrive , and liver failure , not isolated hepatomegaly with increased appetite.

References:

Nelson Textbook of Pediatrics, 21st Edition, Pg. 788 and 789

↑ *Back to this question in Part A*

@Neet_pg_bot

Q68: A 1-month-old infant presents with recurrent vomiting, diarrhea, jaundice, and failure to thrive shortly after breastfeeding began. Examination shows hepatomegaly, and urine tests are positive for reducing substances but negative for glucose. What is the most likely diagnosis? (Q860334)

A. Lactose Intolerance

B. Duodenal Atresia

C. Galactosemia

D. Pyloric Stenosis

Correct Answer: C. Galactosemia

Explanation: This presentation is classic for galactosemia, caused by deficiency of galactose-1-phosphate uridyl transferase, leading to accumulation of galactose-1-phosphate in liver, kidneys, and brain. Symptoms begin soon after exposure to breast milk (contains lactose → glucose + galactose). Typical features include vomiting, diarrhea, jaundice, hepatomegaly, failure to thrive, and urine positive for reducing substances but glucose negative, confirming non-glucose sugar excretion. A) Lactose intolerance : Congenital form is rare, does not cause jaundice or hepatomegaly, and usually presents later. B) Duodenal atresia: Presents in first days of life with bilious vomiting, no diarrhea or hepatomegaly. D) Pyloric stenosis : Causes projectile non-bilious vomiting at 3–6 weeks, no jaundice or reducing substances, and no liver involvement.

References:

Nelson Textbook of Pediatrics, 21st Edition, Pg. 788 and 789

https://www.ncbi.nlm.nih.gov/books/NBK532285/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q69: An infant presents with distended abdomen, jaundice, increased appetite, and hepatomegaly. Which enzyme deficiency is most likely responsible for this condition? (Q860329)

A. Glucose-6-phosphatase

B. Glycogen phosphorylase

C. Galactose-1-phosphate uridyl transferase

D. Glycogen synthase

Correct Answer: C. Galactose-1-phosphate uridyl transferase

Explanation: This presentation is classic for Galactosemia , caused by deficiency of galactose-1-phosphate uridyl transferase (GALT) . The enzyme is essential for converting galactose-1-phosphate → UDP-galactose , allowing galactose to enter glycolysis. Its deficiency leads to accumulation of galactose-1-phosphate and galactitol in the liver, kidney, and brain. Clinical features include: Jaundice and hepatomegaly (due to hepatic injury) Failure to thrive and hypoglycemia Vomiting after milk intake (lactose → glucose + galactose) Cataracts (from galactitol accumulation) Increased appetite and abdominal distension from hepatomegaly Glucose-6-phosphatase (A): Causes Von Gierke's disease (GSD I) with severe fasting hypoglycemia, lactic acidosis, and hepatomegaly, but no jaundice or cataracts. Glycogen phosphorylase (B): Deficiency leads to Hers disease (GSD VI) — mild hypoglycemia and hepatomegaly, without jaundice. Glycogen synthase (D): Causes GSD 0, presenting with fasting hypoglycemia and no hepatomegaly.

References:

Harper's Illustrated Biochemistry (23rd Edition), Page 174.

Vasudevan Textbook of Biochemistry (7th Edition), Page 129.

↑ *Back to this question in Part A*

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Q70: A 7-year-old child of short stature presents with skin patches and learning difficulties. Diagnostic workup indicates a defect in DNA repair specifically related to double-strand breaks. What is the most likely diagnosis? (Q860325)

A. Bloom Syndrome

B. Xeroderma Pigmentosum

C. Cockayne Syndrome

D. Lynch Syndrome

Correct Answer: A. Bloom Syndrome

Explanation: Bloom Syndrome Bloom syndrome is caused by a defect in the BLM gene, which encodes a RecQ helicase essential for DNA repair, particularly double-strand break repair. Clinical Features : Physical: Short stature, narrow face, small jaw, large ears, and high-pitched voice. Skin: Red rash on sun-exposed areas (face, arms, hands) with dilated blood vessels; patches of lighter/darker skin. Development: Fertility and learning difficulties; growth and developmental delays. Infections: Frequent ear and lung infections. Comorbidities: Increased risk of diabetes and chronic obstructive pulmonary disease (COPD). Cancer Risk: Early-onset cancers, especially squamous cell skin cancer, leukemia, lymphoma, and gastrointestinal cancers. Xeroderma pigmentosum (Option B): It is an autosomal recessive disorder characterized by sun sensitivity, with approximately 50% of patients having acute burning on minimal sun exposure. UV radiation causes the cross-linking of pyrimidine residues , preventing normal DNA replication. Nucleotide excision repair (NER) prevents this DNA damage. Inherited loss-of-function mutations in any of these genes give rise to xeroderma pigmentosum. The onset of freckling (lentigines) on sun-exposed skin before age two years in most patients. At an early age, there is a significantly increased risk of sunlight-induced cutaneous neoplasms (basal cell carcinoma, squamous cell carcinoma, melanoma) . Cockayne Syndrome (Option C): Cockayne syndrome is associated with defective transcription-coupled nucleotide excision repair, resulting in photosensitivity, growth failure, and neurodevelopmental abnormalities. Double-strand break repair is not affected. Lynch Syndrome (Option D): Lynch syndrome, also known as hereditary non-polyposis colorectal cancer (HNPCC), involves a defect in mismatch repair genes (MLH1, MSH2, MSH6, PMS2). It is not associated with childhood growth failure or learning difficulties.

References:

https://www.cancer.gov/publications/dictionaries/cancer-terms/def/bloom-syndrome

Robbins and Cotran, Pathologic Basis of Disease, 10th edition, Page no. 314

Fitzpatrick’s Dermatology, 9th edition, Page no. 2343, 2347

↑ *Back to this question in Part A*

@Neet_pg_bot

Q71: A neonate presents with seizures and is found to have a cherry red spot on fundus examination. Enzyme assay reveals deficiency of hexosaminidase A. Which of the following substances is most likely to be accumulated in this patient? (Q866323)

A. GM1 ganglioside

B. GM2 ganglioside

C. Sphingomyelin

D. Glucocerebroside

Correct Answer: B. GM2 ganglioside

Explanation: A neonate presenting with seizures and a cherry red spot likely has Tay-Sachs Disease, a lysosomal storage disorder caused by hexosaminidase A deficiency, leading to the accumulation of GM2 ganglioside in neurons. Tay-Sachs Disease Inheritance Autosomal recessive Cause Deficiency of hexosaminidase A enzyme Accumulation GM2 ganglioside in ganglion cells and neurons Clinical Manifestations Initial symptoms include loss of head control, inability to sit, exaggerated startle response to sound, and a disproportionately large head. Progressive features include spasticity, deafness, and blindness. Not associated with organomegaly. Fundus Finding Cherry-red spot is caused by GM2 ganglioside accumulation in the ganglion cells of the retina, creating a contrast with the normal red fovea. Incorrect Options: GM1 ganglioside (Option A): Accumulates in GM1 gangliosidosis, caused by β-galactosidase deficiency. Sphingomyelin (Option C): Accumulates in Niemann-Pick disease, caused by sphingomyelinase deficiency. Glucocerebroside (Option D): Accumulates in Gaucher disease, caused by glucocerebrosidase deficiency.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q72: In uncontrolled diabetes, in which of the following cells there is decreased glucose uptake due to insulin deficiency: (Q865446)

A. RBC

B. Skeletal muscle

C. Liver

D. Brain

Correct Answer: B. Skeletal muscle

Explanation: In uncontrolled diabetes, insulin deficiency leads to decreased glucose uptake in cells that rely on insulin for glucose transport. Skeletal muscle cells are insulin-dependent, meaning they require insulin to facilitate the uptake of glucose via glucose transporters (e.g., GLUT4). In the absence of sufficient insulin, glucose uptake in skeletal muscle decreases, leading to elevated blood glucose levels. RBC (Option A) RBCs do not depend on insulin for glucose uptake. They use GLUT1 transporters, which allow glucose to enter the cells independently of insulin levels. Therefore, their glucose uptake is not significantly affected by insulin deficiency. Liver (Option C) The liver can take up glucose independently of insulin to some extent, especially under the influence of other

hormones like glucagon. While insulin does regulate hepatic glucose uptake and production, the primary effect of insulin deficiency in the liver is increased glucose output (e.g., gluconeogenesis), not a significant decrease in uptake. Brain (Option D) Brain cells primarily use GLUT3 and GLUT1 transporters, which are insulin-independent. The brain can continue to take up glucose even in the absence of insulin.

References:

Harper's Illustrated Biochemistry, 31st Edition, Page 576.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q73: For mycobacterium species differentiation, technique used is/are? Line probe assay MIRU (Mycobacterium Interspersed Rapatitive Units) Spoligotyping RFLP (Q865405)

A. 1, 2, 3

B. 2, 3, 4

C. 1, 2, 4

D. 1, 2, 3, 4

Correct Answer: D. 1, 2, 3, 4

Explanation: All of the listed techniques can be used for Mycobacterium species differentiation , particularly for Mycobacterium tuberculosis complex (MTBC) and non-tuberculous mycobacteria (NTM). TB lab investigations: Investigations Purpose Advantages Limitations Sputum smear Initial screening for pulmonary TB. Samples are stained using the Ziehl-Neelsen method or Auramine-O stain to visualize acid-fast bacilli (AFB) under a microscope. Quick and inexpensive. Can provide results within a few hours. Low sensitivity, especially in cases of extrapulmonary TB or in patients with HIV. Cannot differentiate between Mycobacterium tuberculosis and non-tuberculous mycobacteria (NTM). Culture Methods Confirm the diagnosis of TB and assess drug susceptibility. Solid Media: Sputum is inoculated onto solid culture media like Löwenstein-Jensen or Middlebrook agar. Liquid Media: Automated systems like BACTEC or MGIT are used to detect growth faster. The gold standard for TB diagnosis. Allows for drug susceptibility testing. Time-consuming; results can take weeks. Requires specialized laboratory facilities. Xpert MTB/RIF Detects Mycobacterium tuberculosis and rifampicin resistance. Method : A cartridge-based system that performs nucleic acid amplification (NAAT). Sputum or other specimens are processed directly in the cartridge. Rapid results (within 2 hours). High sensitivity and specificity. Simultaneously detects rifampicin resistance. More expensive than smear microscopy. Does not provide full drug susceptibility testing. LAMP (Loop-Mediated Isothermal Amplification) Detects DNA of Mycobacterium tuberculosis. Method : Amplifies DNA at a constant temperature without the need for thermal cycling. Sputum or other specimens are used. Rapid results (within 1-2 hours). High specificity and sensitivity. Can be performed at the point of care. Used for patients with HIV with CD4+ counts of less than 100 cells/µL irrespective of signs and symptoms of TB. Less widely available than PCR-based tests. Cannot detect drug resistance directly. TrueNat

Point-of-care test for TB detection. Method : Uses a micro-PCR technology to amplify DNA. Results are available within 60 minutes. Portable and easy to use in peripheral health facilities. Provides results quickly, facilitating immediate treatment decisions. Line Probe Assay (LPA) (Option 1) Detects TB and drug resistance (rifampicin and isoniazid). Method : Sputum samples are processed, and DNA is extracted. Amplified DNA is hybridized to specific probes on a membrane strip. Rapid results (within 1-2 days). Can detect multiple mutations associated with drug resistance. Requires a laboratory setup and trained personnel. Not suitable for all strains of TB. RFLP (Option 4) IS6110 restriction fragment length polymorphism (RFLP) analysis is widely used for molecular epidemiological studies of tuberculosis. Restriction enzymes Nru I, EcoN I, Pst I, and Pvu II were used to analyze IS6110 fingerprints in Mycobacterium tuberculosis strains H37Rv (virulent) and H37Ra (avirulent). MIRU (Mycobacterial Interspersed Repetitive Units) (Option 2) is a genotyping method based on variable-number tandem repeats (VNTRs) found at specific loci in the Mycobacterium genome. By analyzing the number of repeat units at these loci, MIRU provides a unique fingerprint for different strains. Spoligotyping (Option 3) is a molecular technique that targets the direct repeat (DR) region of the Mycobacterium tuberculosis complex (MTBC) genome. This region consists of short, conserved repeats interspersed with unique spacer sequences. By detecting the presence or absence of these spacers, spoligotyping can differentiate among various strains of MTBC.

References:

Genotyping Mycobacterium tuberculosis isolates with few copies of IS6110: Value of additional genetic markers - ScienceDirect

IS6110-based restriction fragment length polymorphism (RFLP) analysis of Mycobacterium tuberculosis H37Rv and H37Ra - PubMed

TB lab investigations

↑ *Back to this question in Part A*

@Neet_pg_bot

Q74: A child presents with abdominal distension and an enlarged liver by 8 cm below the costal margin. The liver is smooth on palpation. Which of the following is the most likely diagnosis? (Q866675)

A. Glycogen Storage Disease (GSD)

B. Lysosomal Storage Disease (LSD)

C. Hepatocellular Carcinoma (HCC)

D. Autoimmune Hepatitis

Correct Answer: A. Glycogen Storage Disease (GSD)

Explanation: A child with abdominal distension and massive hepatomegaly (8 cm below the costal margin) points to a metabolic or infiltrative cause. Smooth liver suggests uniform enlargement without focal mass or nodularity. In GSD (e.g., Von Gierke disease, Pompe, Cori, Anderson), hepatomegaly results from excessive glycogen accumulation in hepatocytes. Often presents in infancy/early childhood with hypoglycemia, lactic acidosis, hyperlipidemia (depending on subtype), but in some types, the main clue is a large smooth liver . Incorrect Options: Lysosomal Storage Disease (LSD) (Option B): Also causes hepatomegaly, but often with coexisting splenomegaly, coarse facial features, and developmental regression (e.g., Gaucher, Niemann–Pick). Hepatocellular carcinoma (HCC) (Option C): Rare in a young child without pre-existing liver disease; typically causes a firm, irregular nodular liver. Autoimmune hepatitis (Option D): Usually causes mild to moderate hepatomegaly with jaundice, fatigue, elevated transaminases; not massive smooth enlargement.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q75: A patient presents with fatigue, anemia, and hepatomegaly. Hemoglobin electrophoresis reveals the presence of HbS. What will be the appropriate treatment? (Q866656)

A. Hydroxyurea

B. Blood transfusion

C. Iron supplementation

D. Vitamin B12 supplementation

Correct Answer: A. Hydroxyurea

Explanation: The presence of HbS confirms Sickle Cell Anemia , and hydroxyurea is the primary treatment as it boosts fetal hemoglobin (HbF) levels, reducing red blood cell sickling and related complications. Cause: Mutation in β-globin gene → production of abnormal hemoglobin S (HbS). Pathophysiology: HbS polymerizes under low oxygen → sickling of RBCs → vaso-occlusion and hemolysis. Symptoms: Anemia, fatigue, pain crises, splenomegaly/hepatomegaly, increased infection risk. Diagnosis: Hemoglobin electrophoresis showing HbS. Treatment: Hydroxyurea: Increases fetal hemoglobin (HbF), reducing sickling episodes. Blood transfusions for severe anemia/crisis. Supportive care: pain management, infection prevention. Incorrect Options: Blood transfusion (Option B) may be used in severe cases but are not the first-line chronic therapy. Iron supplementation (Option C) and Vitamin B12 supplementation (Option D) are not specific treatments for sickle cell anemia.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q76: The biochemical form of amyloid in hemodialysis-associated amyloidosis is: (Q859718)

A. AL

B. AA

C. Aß2m

D. ATTR

Correct Answer: C. Aß2m

Explanation: Hemodialysis-associated amyloidosis is caused by the accumulation of β2-microglobulin (Aβ2M) in tissues, particularly in osteoarticular tissue, the gastrointestinal tract, and the circulatory system. β2-microglobulin is a small protein that accumulates in the blood during long-term hemodialysis, as it is not easily filtered by conventional dialysis membranes. Over time, this leads to the formation of amyloid deposits, which can cause joint pain, stiffness, and other complications in patients undergoing long-term dialysis. Type of Amyloidosis Responsible Protein Organs Affected Primary (AL) (Option A ruled out) Monoclonal light chains Heart, kidneys, liver, peripheral and autonomic nervous system, GI tract Senile systemic (ATTR) (Option D ruled out) Wild-type transthyretin Heart Hereditary (ATTR) Mutant transthyretin Heart Hereditary (AApoA1) Apolipoprotein A1 Heart, kidneys, liver, peripheral nervous system, skin Hereditary (AFib) Mutant fibrinogen A alpha Kidneys, liver Hereditary (ALys) Lysozyme Kidneys, liver Isolated atrial (AANF) Atrial natriuretic factor Heart Secondary (AA) (Option B ruled out) Serum amyloid A Kidneys, GI tract, heart Dialysis-related (Aβ2M) β2-microglobulin Osteoarticular tissue, GI tract, circulatory system Finnish-type (AGel) Gelsolin Lattice dystrophy of cornea, corneal neuropathy

References:

The Amyloidoses: Clinical Features, Diagnosis and Treatment - PMC

↑ *Back to this question in Part A*

@Neet_pg_bot

Q77: What do chaperones assist in? (Q859701)

A. Ubiquitinization of proteins

B. Protein modification

C. Protein folding

D. Protein cleavage

Correct Answer: C. Protein folding

Explanation: Chaperones are proteins that assist in the correct folding of other proteins. When a protein is synthesized, it may adopt various three-dimensional structures, and only one of these structures will have biological activity. Chaperones prevent the incorrect folding of proteins and ensure they achieve the correct structure necessary for proper function. They are crucial in the assembly of tertiary and quaternary protein structures. Chaperones, such as heat shock proteins (HSPs), are also produced in response to stress, including heat, toxins, or radiation, and help in maintaining protein integrity under these conditions. Misfolded proteins can lead to diseases, including prion diseases and chaperonopathies. Incorrect Options: Ubiquitinization of proteins (Option A): Ubiquitinization refers to the process where a small protein called ubiquitin is attached to other proteins, typically marking them for degradation by the proteasome. This is unrelated to the function of chaperones, which focus on helping proteins fold correctly, not tagging them for destruction. Protein modification (Option B) involves chemical changes to a protein after its synthesis, such as phosphorylation, glycosylation, or acetylation. While chaperones help proteins fold correctly, they do not directly modify them in terms of adding chemical groups or altering their structure after they are fully folded. Protein cleavage (Option D) is the process of breaking down proteins into smaller peptides, often by enzymes like proteases. This process is not related to the role of chaperones, which help proteins fold into their correct structures rather than breaking them down or cleaving them into smaller parts.

References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page no: 604

↑ *Back to this question in Part A*

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Q78: What is the function of proteasomes? (Q858454)

A. Protein folding

B. Post-translation modification

C. Protein degradation

D. Protein sorting

Correct Answer: C. Protein degradation

Explanation: Proteasomes are large protein complexes that degrade damaged, misfolded, or unneeded proteins tagged with ubiquitin, maintaining cellular protein quality and regulating various biological processes. Proteasomes are large protein complexes found in cells that are responsible for protein degradation. They play a vital role in maintaining cellular homeostasis by selectively breaking down proteins that are damaged, misfolded, or no longer needed. Proteasomes consist of multiple subunits that work together to recognize and degrade target proteins. The main function of proteasomes is to break down proteins into smaller peptide fragments, which can then be further processed and recycled by the cell. The degradation of proteins by proteasomes is essential for various cellular processes, including the regulation of cell cycle progression, removal of misfolded or damaged proteins, antigen presentation in the immune system and control of protein levels and turnover.

References:

Proteasomes

↑ *Back to this question in Part A*

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Q79: A farmer sprayed a chemical in the pool which causes inhibition of complex-III of the electron transport chain. Identify the inhibitor? (Q820323)

A. Rotenone

B. TTFA

C. Cyanide

D. Antimycin

Correct Answer: D. Antimycin

Explanation: Correct Option D - Antimycin: Antimycin is an inhibitor of complex III (cytochrome b-c1 complex) of the electron transport chain. It interrupts the transfer of electrons from ubiquinol to cytochrome c, thereby inhibiting the flow of electrons through this complex. Incorrect Option: Option A - Rotenone: Rotenone is an inhibitor of complex I of the electron transport chain. It blocks the transfer of electrons from NADH to ubiquinone in the mitochondrial respiratory chain. Option B - TTFA: TTFA(Thenoyltrifluoroacetone is an inhibitor of complex II of the electron transport chain. Option C - Cyanide: Cyanide is an inhibitor of complex IV (cytochrome c oxidase) of the electron transport chain. It prevents the transfer of electrons to oxygen, disrupting the final step of electron transport and therefore blocking the production of ATP.

References:

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↑ *Back to this question in Part A*

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Q80: Oxaloacetate is produced from which of the following amino acid? (Q820367)

A. Alanine

B. Lysine

C. Leucine

D. Aspartate

Correct Answer: D. Aspartate

Explanation: Correct Option D - Aspartate: Aspartate is the amino acid that contributes to the production of oxaloacetate through transamination reactions in the citric acid cycle. Incorrect Options: Option A - Alanine: Alanine can be involved in the glucose-alanine cycle and can be converted into pyruvate, not oxaloacetate. Option B - Lysine: Lysine is an essential amino acid, and it is not directly involved in the synthesis of oxaloacetate. Option C - Leucine: Leucine is also not directly involved in the synthesis of oxaloacetate. It is primarily involved in protein synthesis and can be catabolized to generate acetyl-CoA, which can then enter the citric acid cycle.

↑ *Back to this question in Part A*

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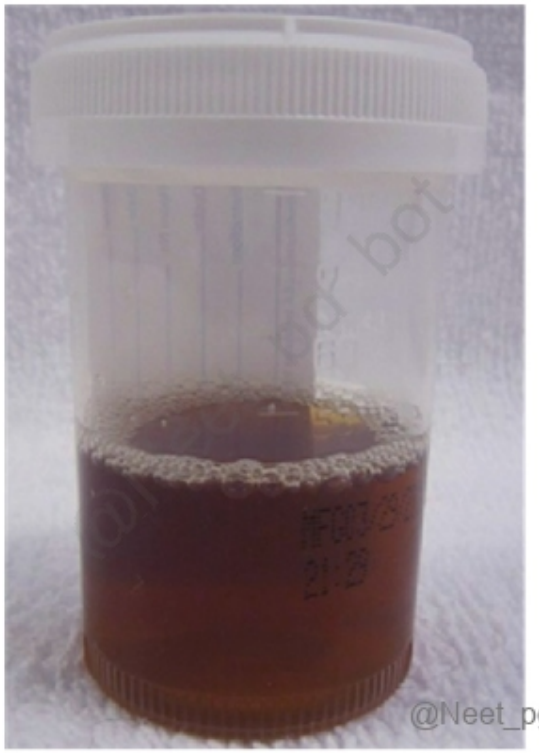
Q81: The lab technician observes that a urine sample obtained from a 4-year-old male appeared normal initially, but underwent a change in color as depicted in the image after a few hours. Subsequently, the color intensified over the following hours. Which deficiency can potentially lead to this condition? (Q793373)

A. Beta-carotene

B. Tyrosine aminotransferase

C. L-ascorbic acid

D. Homogentisic acid oxidase



Correct Answer: D. Homogentisic acid oxidase

Explanation: Correct Option D - Homogentisic acid oxidase: Homogentisic acid (HGA) is a metabolite that is used as the diagnostic substance in the tissues for alkaptonuria. Alkaptonuria is an autosomal recessive disease that occurs rarely. When Homogentisic-acid oxidase is found deficient in the tissue, then normal procedure of phenylalanine metabolism gets blocked and Homogentisic acid (HGA) becomes unable to break. Hence, urine appears as black and dark-stained nappies, and can be seen as the earliest signs of it. Incorrect Options: Option A - Beta -carotene is a type of carotenoid that is present in carrots, apricots, and sweet potatoes. It is a provitamin and helps the body to make vitamin A. High doses of beta carotene might turn urine bright orange or yellow. Option B - Tyrosine aminotransferase deficiency in children causes tyrosine to break incompletely. . Ocular manifestations- Painful corneal erosions and Cutaneous manifestation-: Palmar hyperkeratosis Option C - Excess vitamin C can change urine colour into dark yellow or orange and vice-versa.

↑ *Back to this question in Part A*

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• [05_Enzymes] 018 Enzymes

« Back to Folder Directory

[05_Enzymes] 018 Enzymes — Quick Jump

Q1 (p. 919)	*Q2 (p. 921)*	*Q3 (p. 922)*	*Q4 (p. 923)*	*Q5 (p. 924)*	*Q6 (p. 925)*
Q7 (p. 926)	*Q8 (p. 927)*	*Q9 (p. 927)*	*Q10 (p. 929)*	*Q11 (p. 929)*	*Q12 (p. 930)*
Q13 (p. 932)	*Q14 (p. 933)*	*Q15 (p. 936)*	*Q16 (p. 937)*	*Q17 (p. 938)*	*Q18 (p. 938)*
Q19 (p. 939)	*Q20 (p. 940)*	*Q21 (p. 941)*	*Q22 (p. 942)*	*Q23 (p. 943)*	*Q24 (p. 944)*
Q25 (p. 945)	*Q26 (p. 946)*				

Q1: Which of the following statements accurately describes the changes in Km (Michaelis constant) and Vmax (maximum reaction rate) in the presence of an uncompetitive inhibitor? (Q843311)

A. Km increases, Vmax increases

B. Km decreases, Vmax decreases

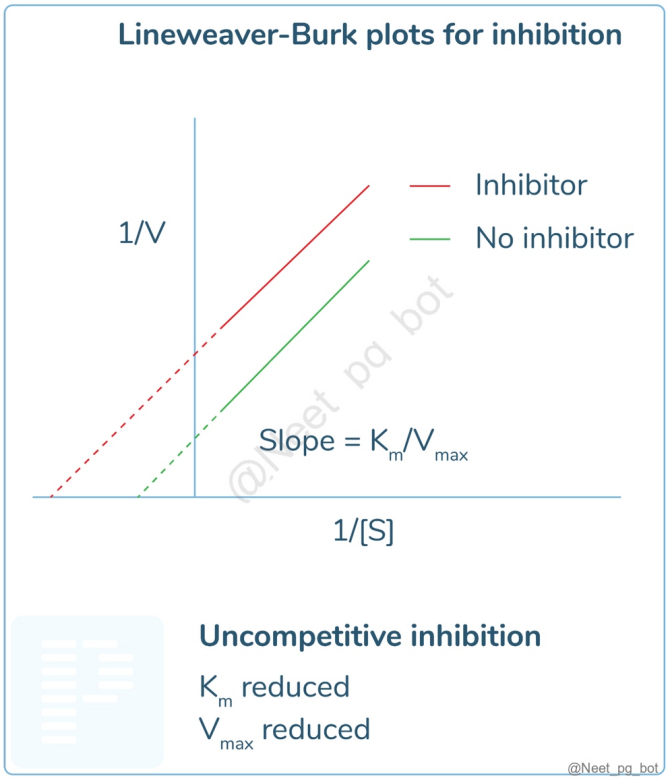
C. Km stays the same, Vmax decreases

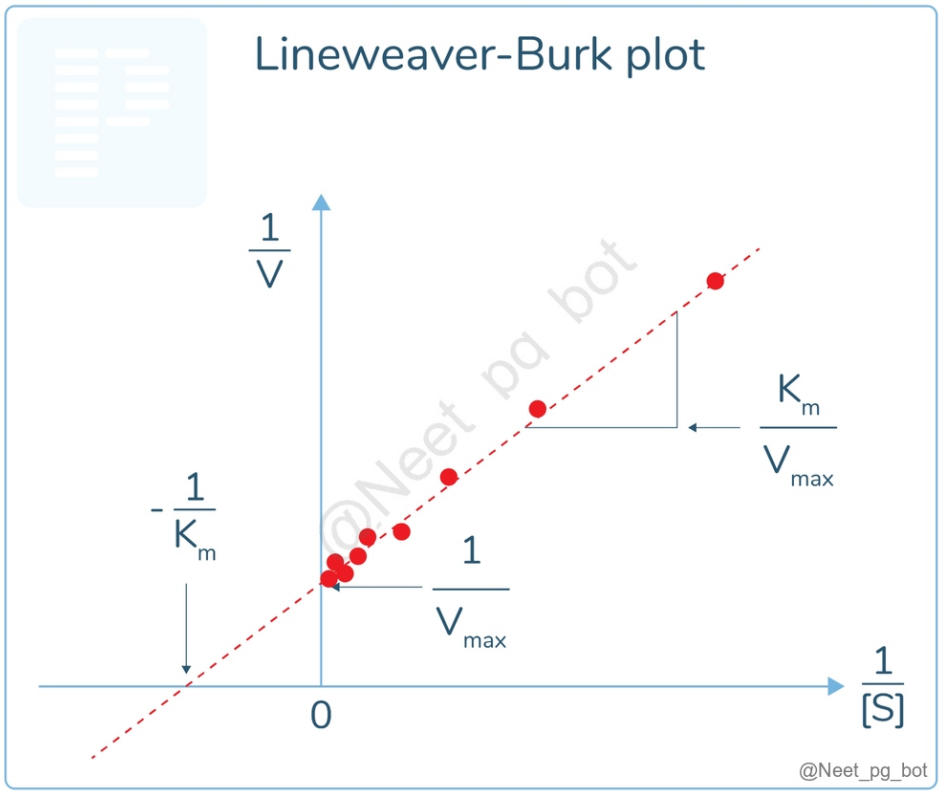
D. Km increases, Vmax stays the same

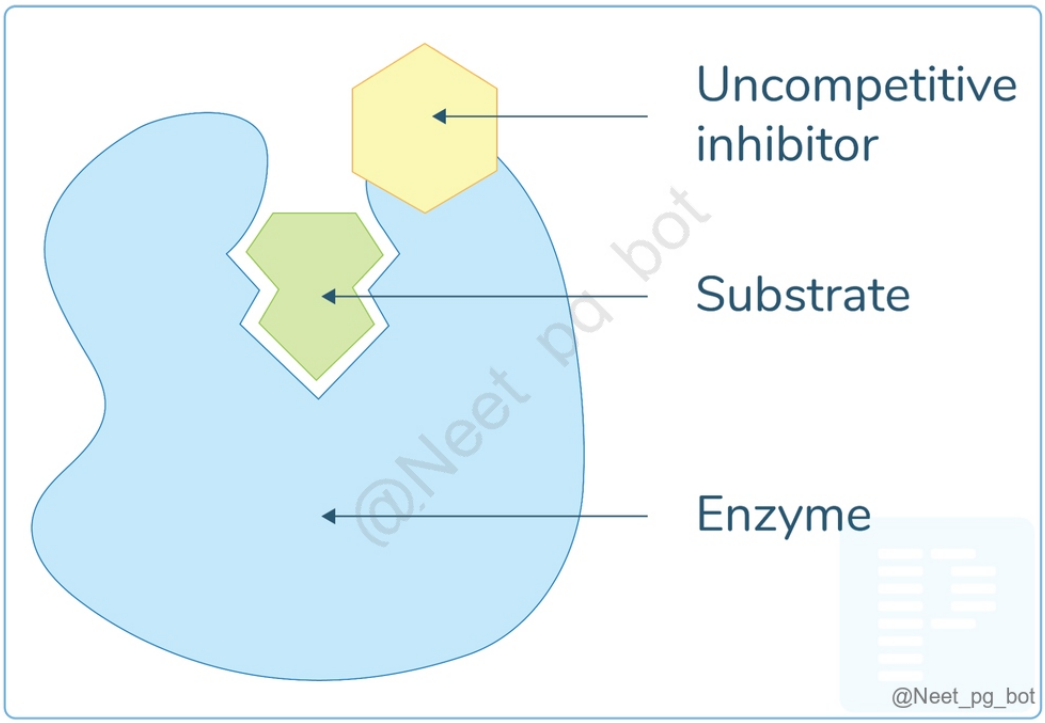
Correct Answer: B. Km decreases, Vmax decreases

■ *Listen to Audio 1*

Explanation: Uncompetitive Inhibition occurs when an inhibitor binds only to the enzyme-substrate complex, preventing it from converting the substrate into product. This type of inhibition results in: Decrease in Km : Since the inhibitor binds to the enzyme-substrate complex, it effectively lowers the concentration of the enzyme available for reaction, making the apparent affinity of the enzyme for the substrate appear higher Decrease in Vmax : The inhibitor reduces the maximum rate of reaction because it prevents the enzyme-substrate complex from proceeding to form the product, thus lowering the overall reaction rate at saturating substrate concentrations. Therefore, in Uncompetitive Inhibition, both Km and Vmax decrease. Examples of Uncompetitive inhibition: Uncompetitive inhibitors Clinical correlation Lithium and Inositol Monophosphatase Inhibits the phosphatidylinositol pathway involved in mood regulation, explaining lithium’s efficacy in bipolar disorder. Phenylalanine and Alkaline Phosphatase Binds to the enzyme–substrate complex, reducing activity—seen in metabolic disorders involving phenylalanine.


Lineweaver-Burk plots for inhibition
1/V
Inhibitor
No inhibitor
Slope = K_m/V_{max}
1/[S]
Uncompetitive inhibition
K_m reduced
V_{max} reduced
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Lineweaver-Burk plot
$\frac{1}{V}$
$-\frac{1}{K_m}$
$\frac{K_m}{V_{max}}$
$\frac{1}{V_{max}}$
0
$\frac{1}{[S]}$
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References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 62

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1185393/

↑ *Back to this question in Part A*

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Q2: A patient presents with hepatomegaly and joint pain. Investigations suggest abnormal intracellular accumulation of macromolecules such as lipids or glycoconjugates. Which cellular organelle is primarily responsible for this accumulation? (Q882197)

A. Golgi apparatu

B. Lysosome

C. Mitochondria

D. Peroxisome

Correct Answer: B. Lysosome

Explanation: Lysosomal storage disorders result from a deficiency of specific lysosomal enzymes , leading to failure of degradation of macromolecules such as lipids, glycogen, and glycosaminoglycans. These undegraded substrates accumulate within lysosomes , causing organomegaly (especially hepatosplenomegaly) , skeletal abnormalities, and joint involvement. Hence, the lysosome is the primary organelle responsible for this abnormal accumulation. Golgi apparatus (Option A): Involved in protein modification and trafficking, not intracellular degradation. Mitochondria (Option C): Responsible for ATP production and apoptosis, not macromolecule breakdown. Peroxisome (Option D): Involved in very long-chain fatty acid oxidation and detoxification, not glycoconjugate degradation.

References:

pmc.ncbi.nlm.nih.gov/articles/PMC3514785/

Robbin's and Cotran's Pathologic Basis of Disease, 10th Edition, Page 154-160

↑ *Back to this question in Part A*

@Neet_pg_bot

Q3: Which of the following statements regarding creatine kinase (CK) is false? (Q843568)

A. Total CK is elevated in muscular dystrophies

B. CK levels start to rise within 3–6 hours after myocardial infarction

C. CK levels are increased in hemolysis and congestive heart failure

D. CK-MB is used as a marker for myocardial infarction

Correct Answer: C. CK levels are increased in hemolysis and congestive heart failure

■ *Listen to Audio 1*

Explanation: Creatine kinase (CK) is an enzyme that catalyzes the reversible conversion of creatine ↔ phosphocreatine , important in muscle energy metabolism. It is mainly found in skeletal muscle (CK-MM) , cardiac muscle (CK-MB) , and brain (CK-BB) . CK is not significantly elevated in hemolysis or congestive heart failure because red blood cells lack CK and mild cardiac strain in CHF doesn’t cause measurable CK elevation. Option: A) CK is markedly elevated in muscular dystrophies (due to muscle fiber breakdown). Option: B) In MI, CK (especially CK-MB) rises 3–6 hours after infarction, peaks in 12–24 hours, and normalizes in 2–3 days. Option: D) CK-MB is the cardiac-specific isoenzyme , used for diagnosis and monitoring of myocardial infarction .

References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page302-303

↑ *Back to this question in Part A*

Q4: Which of the following is used to study the kinetics of oligomeric enzymes? (Q843567)

A. Lineweaver Burke equation

B. Michaelis Menten equation

C. Hill equation

D. Dixon equation

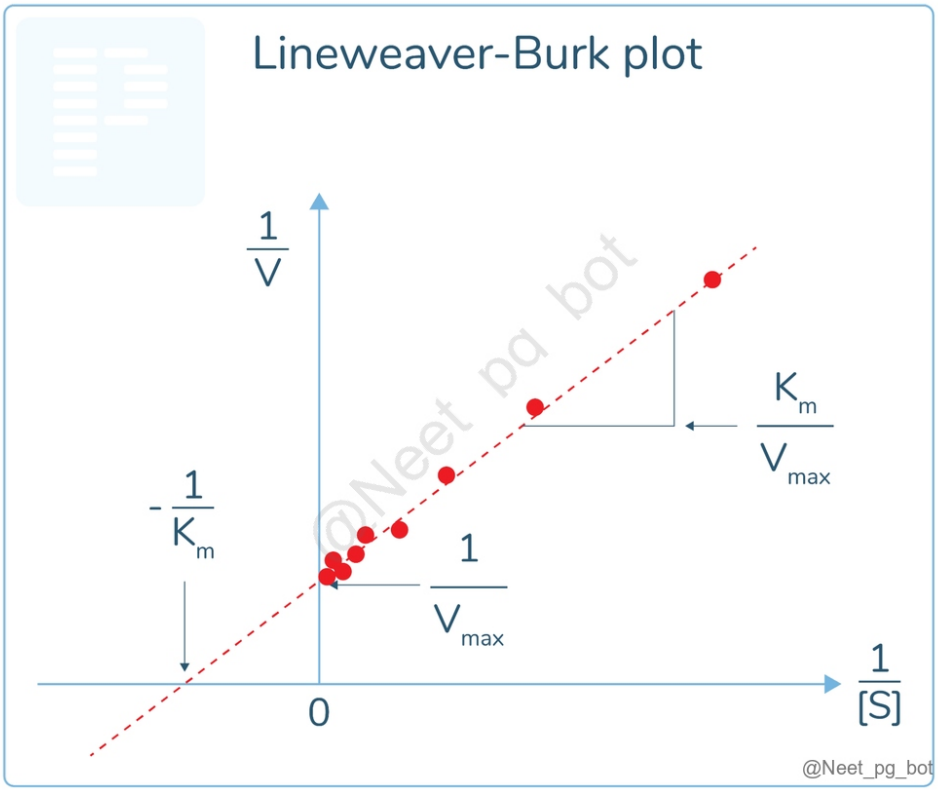
Correct Answer: C. Hill equation

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Explanation: Hill equation relates reaction velocity to substrate concentration in oligomeric enzymes Oligomeric enzymes often exhibit cooperative binding behavior due to their multiple substrate-binding sites They show sigmoidal kinetics Lineweaver-Burk Equation: Michaelis-Menten equation in a linear form (Option A) Michaelis-Menten Equation (Option B): Relates reaction rate to substrate concentration, might not accurately describe the kinetics of oligomeric enzymes. Dixon Equation (Option D): A method for determining the inhibition constant of an enzyme inhibitor by plotting the inverse of the reaction rate against the inhibitor concentration at different substrate concentrations

$$\frac{1}{V} = \frac{K_m + [S]}{V_{max} + [S]} = \frac{K_m}{V_{max}} \frac{1}{[S]} + \frac{1}{V_{max}}$$

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References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 79

↑ *Back to this question in Part A*

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Q5: Which among the following statements regarding the induction and repression of enzymes is NOT true? (Q843566)

A. Repression acts at the gene level to decrease the amount of enzyme

B. Induction relieves repression on the operator site

C. Glucokinase is induced by insulin

D. ALA(Aminolevulinic acid) synthase is induced by heme

Correct Answer: D. ALA(Aminolevulinic acid) synthase is induced by heme

■ *Listen to Audio 1*

Explanation: ALA (Aminolevulinic acid) synthase is induced by barbiturates and repressed by heme Induction and repression of enzymes increase and decrease enzyme biosynthesis respectively at the gene level Mechanism Process Examples Induction Relieves repression on the operator site, increasing enzyme biosynthesis (Option B) Lactose-utilizing enzymes in bacteria induced by lactose in the absence of glucose Tryptophan pyrrolase and transaminases induced by glucocorticoids Glucokinase induced by insulin (Option C) ALA (Aminolevulinic acid) synthase induced by barbiturates Repression Acts at the gene level, reducing the number of enzyme molecules (Option A) ALA (Aminolevulinic acid) synthase is repressed by heme

References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 64

↑ *Back to this question in Part A*

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Q6: All of the following enzymes are proteins except? (Q843565)

A. Aminoacyl tRNA synthetase

B. Peptidyl transferase

C. RNA polymerase

D. Acetyl-CoA synthetase

Correct Answer: B. Peptidyl transferase

■ *Listen to Audio 1*

Explanation: Peptidyl transferase is an RNA enzyme/Ribozyme while others are proteins Ribozymes/RNA enzymes Definition Enzymes made up of RNA Spliceosomes Ribozymes + protein components (stabilize the structure of ribozymes) Examples Peptidyl transferase - Catalyzes peptide bond formation during translation RNase P - Cleaves precursor tRNA to produce mature tRNA ends Group I introns - Self-splicing introns that remove themselves from RNA Group II introns - Self-splicing introns involved in RNA processing Hammerhead ribozyme - Self-cleaving RNA found in plant viroids Hairpin ribozyme - Catalyzes RNA cleavage reactions HDV ribozyme - Found in Hepatitis delta virus Spliceosome catalytic RNA - RNA component responsible for splicing activity Ribosomal RNA Acting as Ribozyme: Organism rRNA acting as Peptidyl Transferase Prokaryotes 23S rRNA of the 50S ribosomal subunit Eukaryotes 28S rRNA of the 60S ribosomal subunit

References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 592

↑ *Back to this question in Part A*

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Q7: In a Lineweaver-Burk plot for an enzyme experiencing non-competitive inhibition, which of the following statements is correct? (Q843312)

A. The slope of the Lineweaver-Burk plot increases, but the y-intercept remains the same.

B. The y-intercept increases while Km remains constant and the slope increases.

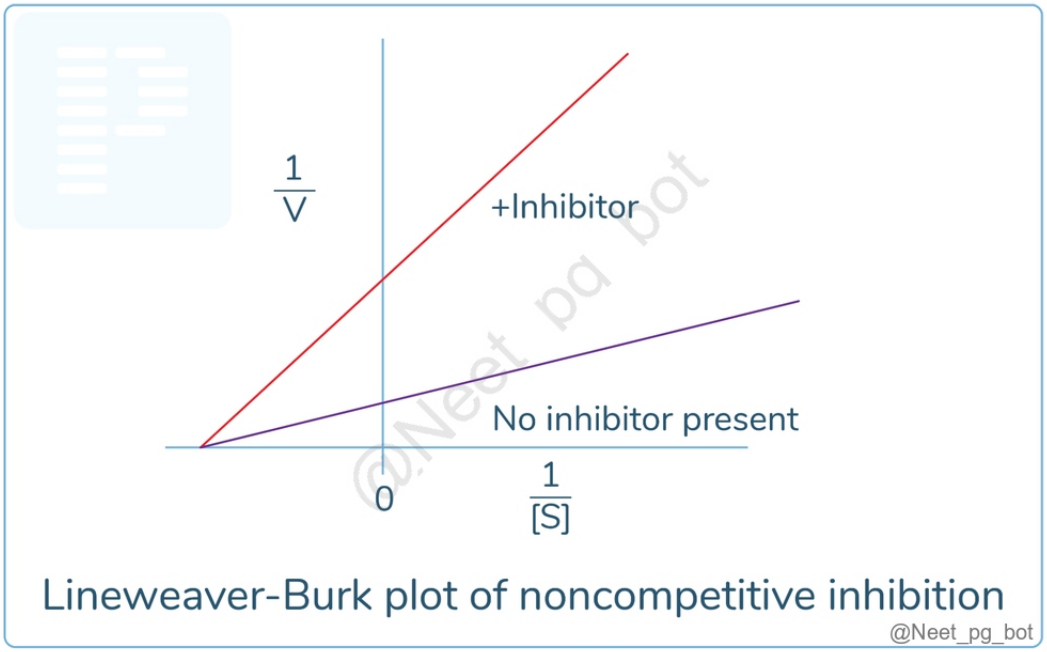
C. Both the slope and the y-intercept increase.

D. The slope of the Lineweaver-Burk plot decreases while the y-intercept increases.

Correct Answer: B. The y-intercept increases while Km remains constant and the slope increases.

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Explanation: Non-competitive inhibition occurs when an inhibitor binds to an enzyme at a site other than the active site, which is called an allosteric site. This binding changes the enzyme's shape and reduces its activity, regardless of whether the substrate is present. Effect on the Lineweaver-Burk Plot: The lineweaver burk plot is graphical representation of enzyme kinetics representing Vmax and Km. Effect on Vmax: The y-intercept of the Lineweaver-Burk plot, which represents 1/Vmax increases as Vmax decreases. Effect on Km: The slope of the plot, which is Km/Vmax, remains constant because Km does not change. The y-intercept increases while the Km remains constant for non competitive inhibition. Examples of non-competitive inhibitors: Cyanide acts as a non-competitive inhibitor by binding to a site on cytochrome c oxidase that is not the active site, thereby disrupting its function. Fluoride functions as a non-competitive inhibitor by chelating essential metal ions (magnesium or manganese) required for enolase activity, thus inhibiting the enzyme.



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 80 https://www.ncbi.nlm.nih.gov/books/NBK545242/

↑ *Back to this question in Part A*

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Q8: Which of the following statements are true regarding enzymes? a) Enzymes are proteins that can accelerate reactions by factors of 10^6 to 10^9 or more. b) Enzymes act on a wide range of substrates. c) Enzymes stabilize the ground state. d) Enzymes can catalyze both forward and reverse reactions. their e) Enzymes are regulated by pH, temperature, inhibitors, and activators. f) Enzyme are water insoluble (Q843236)

A. a, d, e, f ,c

B. a, b, d, e

C. a, d, e

D. a, e, f , c

Correct Answer: C. a, d, e

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Explanation: Properties of Enzyme Nature of Enzymes Enzymes are proteins, heat-sensitive, water-soluble. (Statement f ruled out) Efficiency Enzymes can accelerate reactions by factors 10 6 to 10 9 or more. (Statement a) Specificity Enzymes act on specific substrates and can distinguish stereoisomers. (Statement b ruled out) Cofactors/Coenzymes Some enzymes need non-protein molecules (cofactors or coenzymes) to be active. Substrate Binding Enzymes bind substrates at multiple points, creating chiral products from non-chiral substrates. Transition State Enzymes stabilize the transition state, lowering activation energy. (Statement c ruled out) Reversibility Enzymes can catalyze both forward and reverse reactions. (Statement d) Regulation Activity is regulated by pH, temperature, inhibitors, and activators. (Statement e)

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 59,60

↑ *Back to this question in Part A*

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Q9: Which of the following examples best illustrates competitive inhibition? (Q843310)

A. Methotrexate and Dihydrofolate Reductase

B. Cyanide and Cytochrome c Oxidase

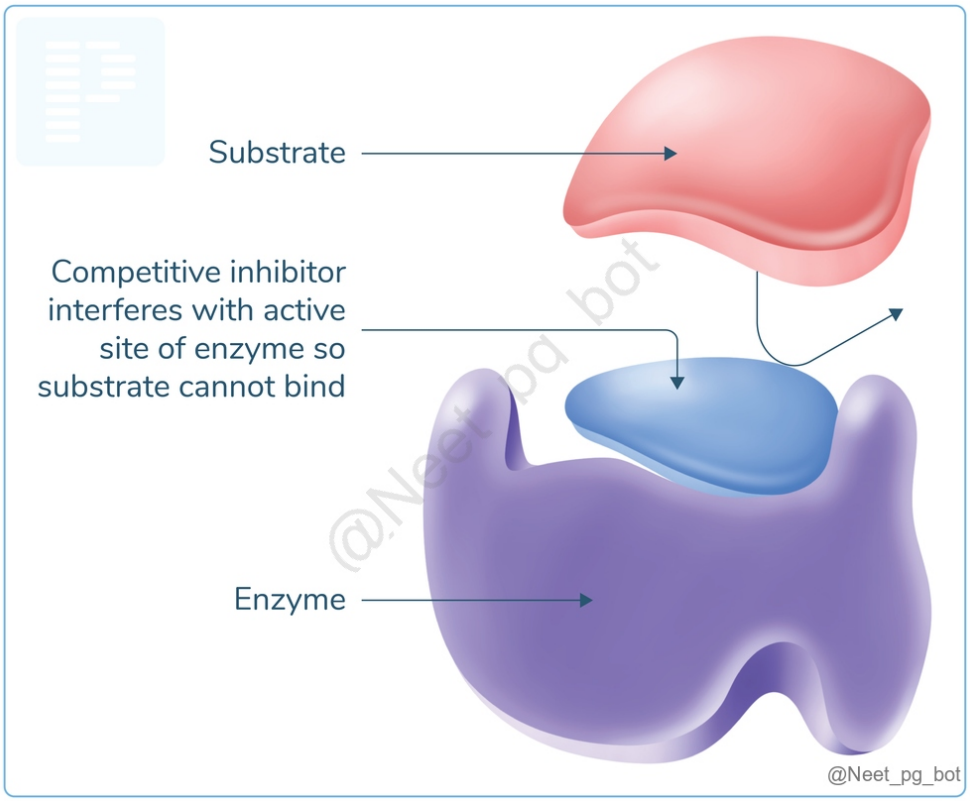
C. Alanine and Pyruvate Kinase

D. Citrate and Phosphofructokinase-1(PFK-1)

Correct Answer: A. Methotrexate and Dihydrofolate Reductase

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Explanation: Competitive inhibition occurs when an inhibitor molecule resembles the substrate and competes with the substrate for binding to the active site of the enzyme.It is usually reversible. Competitive inhibitors clinical correlation Methotrexate and Dihydrofolate Reductase Methotrexate acts as a competitive inhibitor by mimicking dihydrofolate, the natural substrate of DHFR, and binding to its active site, thereby preventing dihydrofolate from binding and inhibiting the enzyme's activity. Sulfa Drugs and PABA Sulfa drugs, such as sulfanilamide, competitively inhibit the enzyme dihydropteroate synthase by mimicking para-aminobenzoic acid (PABA), a substrate involved in folic acid synthesis in bacteria. Ethanol and Alcohol Dehydrogenase Ethanol can act as a competitive inhibitor of methanol metabolism by competing for the active site of alcohol dehydrogenase. This is used in cases of methanol poisoning. Captopril and Angiotensin-Converting Enzyme (ACE) Captopril competes with the natural substrate of ACE, thereby inhibiting the production of angiotensin II, a peptide that increases blood pressure. Cyanide and Cytochrome c Oxidase (Option B): Cyanide and Cytochrome C Oxidase describe non-competitive inhibitions. Alanine and Pyruvate Kinase (Option C): Alanine and Pyruvate Kinase describe non-competitive inhibitions. Citrate and Phosphofructokinase-1 (PFK-1) (Option D): Citrate and Phosphofructokinase-1(PFK-1) describes Allosteric Inhibition.



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 79, 80 Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 59, 60

↑ *Back to this question in Part A*

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Q10: Given the following enzyme kinetics parameters: Maximum velocity (Vmax): 100 µmol/min Michaelis constant (Km): 10 µM Initial velocity (Vo): 50 µmol/min What is the substrate concentration [S] of the given enzyme? (Q843309)

A. 5 µM

B. 10 µM

C. 20 µM

D. 25 µM

Correct Answer: B. 10 µM

■ *Listen to Audio 1*

Explanation: The substrate concentration [S] represents the amount of substrate available for the enzyme to act upon in a given reaction. It is typically measured in units of concentration, such as micromoles per liter (µM) or millimoles per liter (mM). To find the substrate concentration [S], you can use the Michaelis-Menten equation: Vo = Vmax[S] / Km + [S] Given: (Vmax): 100 µmol/min (Km): 10 µM (Vo): 50 µmol/min Substitute the given values into the equation: 50 = 100[S] / 10+ [S] 50 (10 + [S]) = 100 [S] Simplify and solve for [S]: 500 + 50[S] = 100[S] 500 = 50[S] [S] = 10 µM Therefore, the substrate concentration [S] is 10 µM.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 78

↑ *Back to this question in Part A*

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Q11: In enzyme kinetics, a double reciprocal plot (Lineweaver-Burk plot) is used to determine kinetic parameters. What do the x-intercept and y-intercept of a double reciprocal plot represent? (Q843308)

A. x-intercept: -1/Km; y-intercept: 1/Vmax

B. x-intercept: 1/Km; y-intercept: 1/Vmax

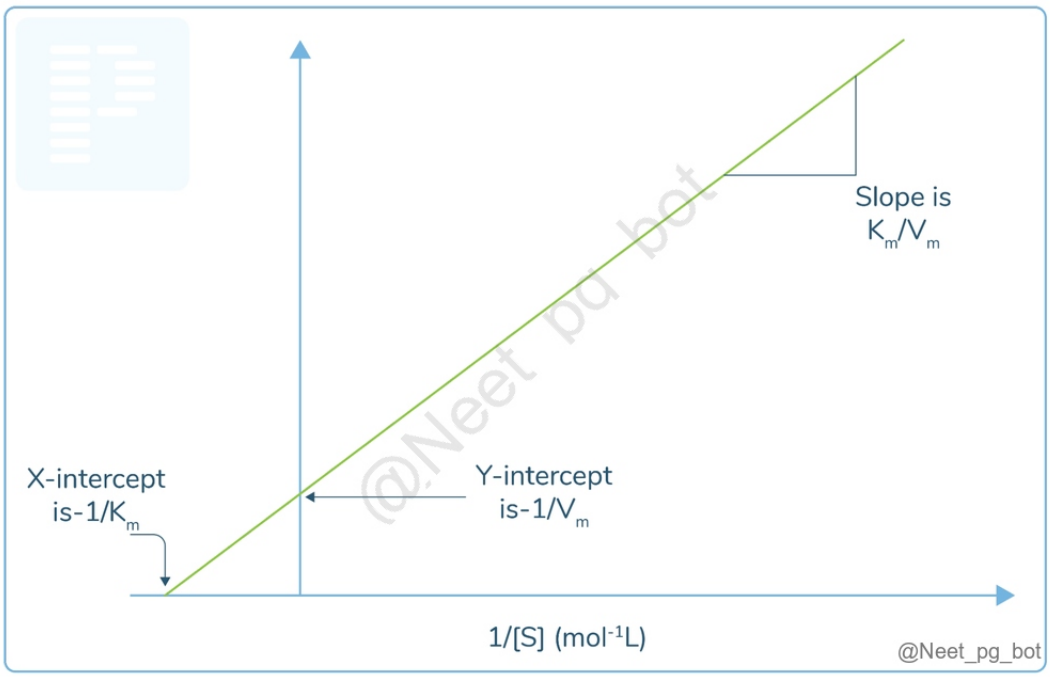
C. x-intercept:1/Vmax; y-intercept: -1/Km

D. x-intercept: -1/Km; y-intercept: -1/Vmax

Correct Answer: A. x-intercept: -1/Km; y-intercept: 1/Vmax

■ *Listen to Audio 1*

Explanation: Double reciprocal plots typically are used both to distinguish between competitive and non-competitive inhibitors and to simplify evaluation of inhibition constants. The Lineweaver-Burk plot is a double reciprocal plot used to linearize the Michaelis-Menten equation. The Michaelis-Menten equation is: V = Vmax[S]/Km + [S] Taking the reciprocal of both sides, we get the Lineweaver-Burk equation: 1/V = Km/Vmax[S] + 1/Vmax In this linear form, plotting 1/V(y-axis) against 1/[S] (x-axis) yields a straight line. The x-intercept of this plot is -1/Km and the y-intercept is 1/Vmax.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 80

↑ *Back to this question in Part A*

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Q12: Which of the following statements regarding Michaelis Menten equation is true? (Q843282)

A. Used to calculate the rate of reaction

B. Substrate concentration is required to calculate the rate using Michaelis Menten equation

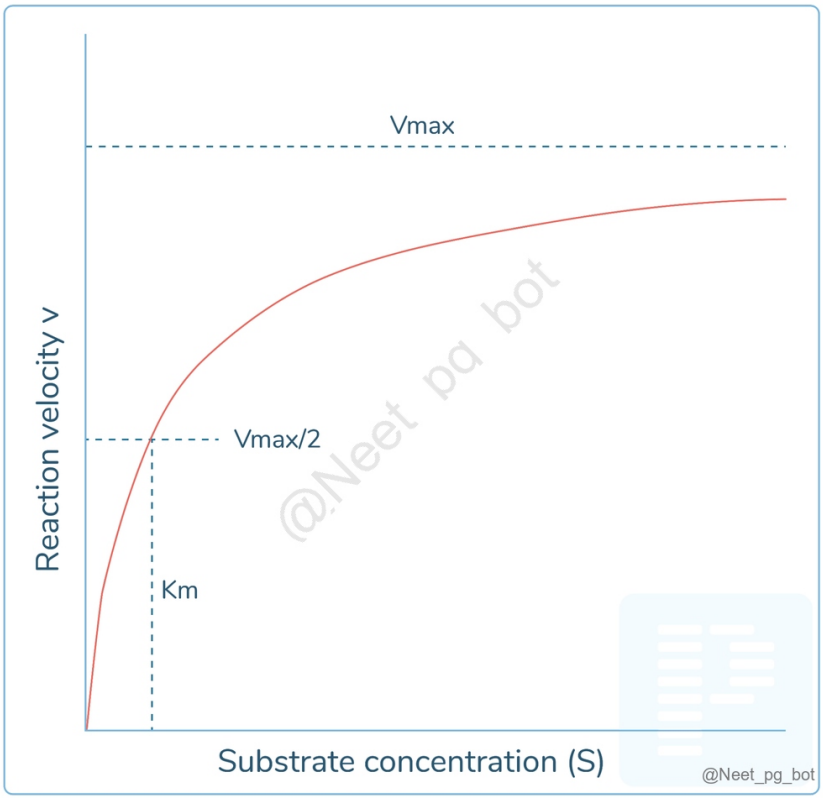
C. Michaelis constant is the substrate concentration at which the velocity of the reaction is half the maximal velocity

D. All are true

Correct Answer: D. All are true

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Explanation: The Michaelis–Menten equation describes the relationship between substrate concentration and the rate of enzyme-catalyzed reactions . Michalis-Menten equation: V = Vmax x S / (Km + S) Where, V = Velocity of reaction Vmax = Maximum velocity of the reaction S = Substrate concentration Km (Michaelis constant) is the substrate concentration at which the reaction velocity is half of the maximal velocity (Vmax/2). Factors Affecting Km: Enzyme structure: Changes affinity of enzyme for substrate pH: Alters ionization of active site Temperature: Extreme temperatures may alter enzyme structure Enzyme inhibitors: Some inhibitors modify Km Substrate type: Different substrates may have different Km values



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 76-77

↑ *Back to this question in Part A*

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Q13: What is the correct equation to calculate the catalytic efficiency of an enzyme? (Q843278)

A. Kcat/Km

B. Km/Kcat

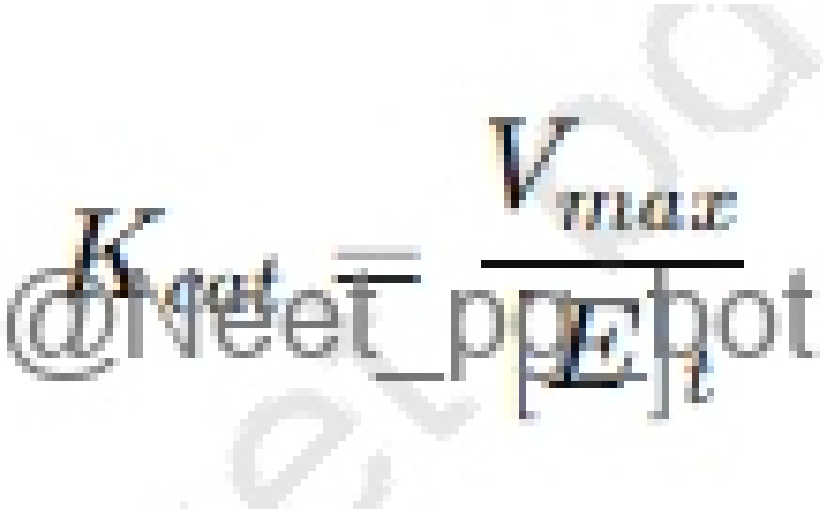
C. Vmax/St

D. Vmax/Km

Correct Answer: A. Kcat/Km

■ *Listen to Audio 1*

Explanation: The catalytic efficiency of an enzyme describes how efficiently an enzyme converts substrate into product, especially at low substrate concentrations. It is the ratio of Kcat to Km: Catalytic Efficiency = Kcat / Km Where, Catalytic speed (Kcat) Substrate affinity (Km) Significance of Catalytic Efficiency Feature Description High Kcat/Km Highly efficient enzyme Low Kcat/Km Poor catalytic performance Units M■¹ s■¹ Upper limit Diffusion-controlled enzymes approach 10■–10■ M■¹ s■¹ Catalytic Constant (Kcat) Kcat is called the turnover number. It represents the maximum number of substrate molecules converted into product per enzyme molecule per second when the enzyme is fully saturated with substrate. Where, Vmax is the maximum reaction velocity [E]t is the total enzyme concentration or the number of active sites Km/Kcat (Option B): Inverse of catalytic efficiency; not used to measure enzyme efficiency. Vmax/St (Option C): This actually represents Kcat , not catalytic efficiency. ■■■■■■■Vmax/Km (Option D): Does not account for enzyme concentration and therefore does not accurately measure the enzyme.



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 77-78

↑ *Back to this question in Part A*

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Q14: Which of the following statements is false Increase in enzyme concentration increases the rate of reaction Increase in substrate concentration increases the rate of reaction Increase in product concentration increases the rate of reaction (Q843277)

A. 1, 3

B. 3

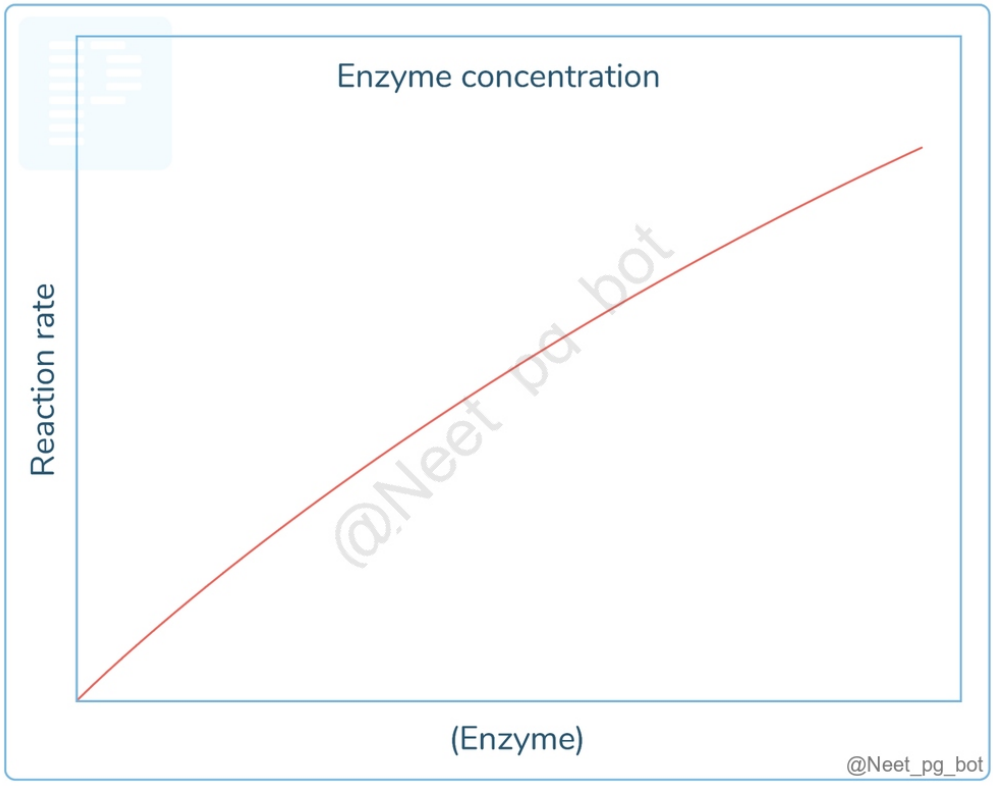
C. 1, 2, 3

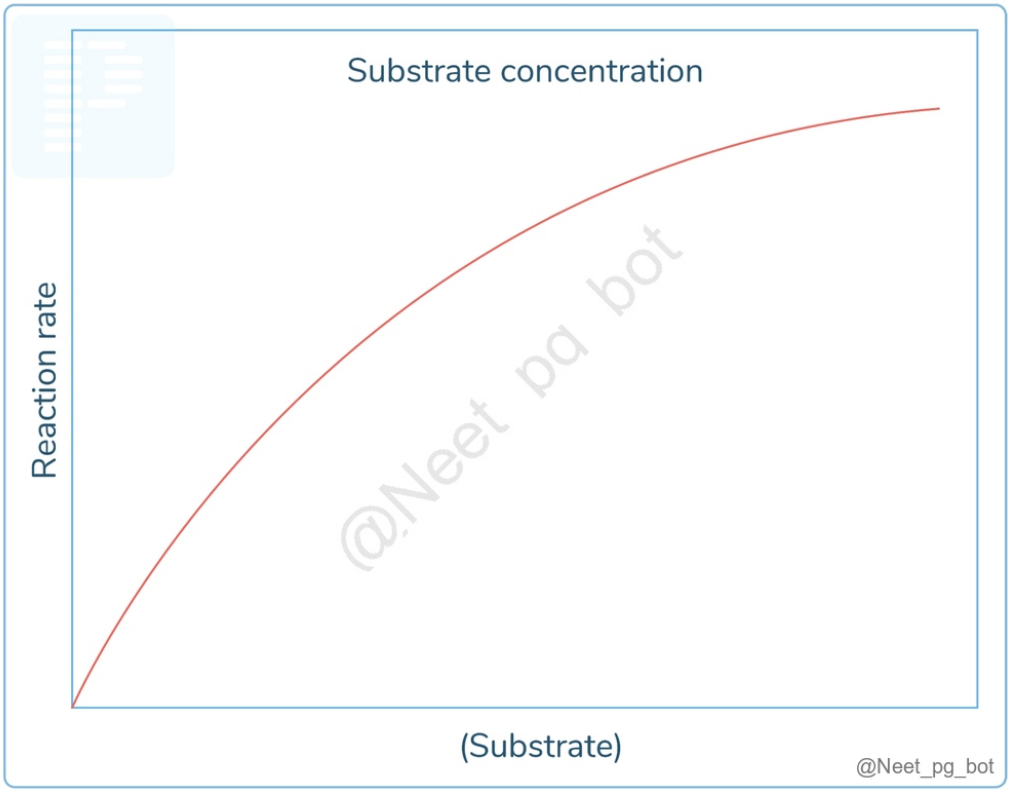
D. None

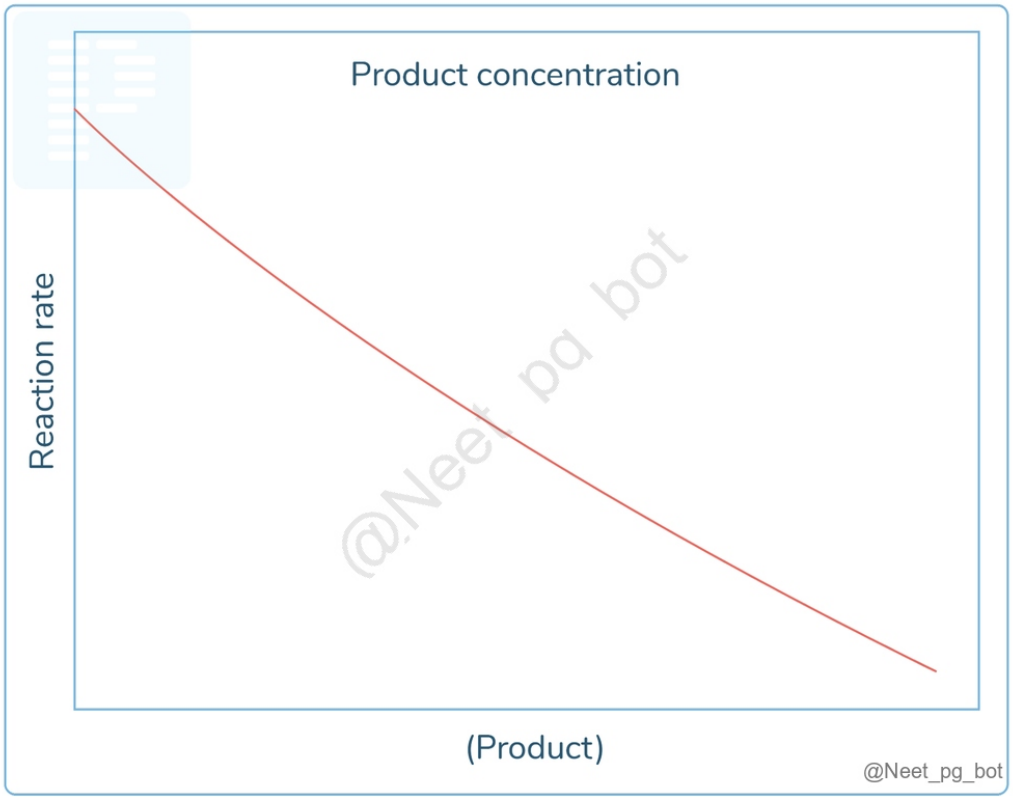
Correct Answer: B. 3

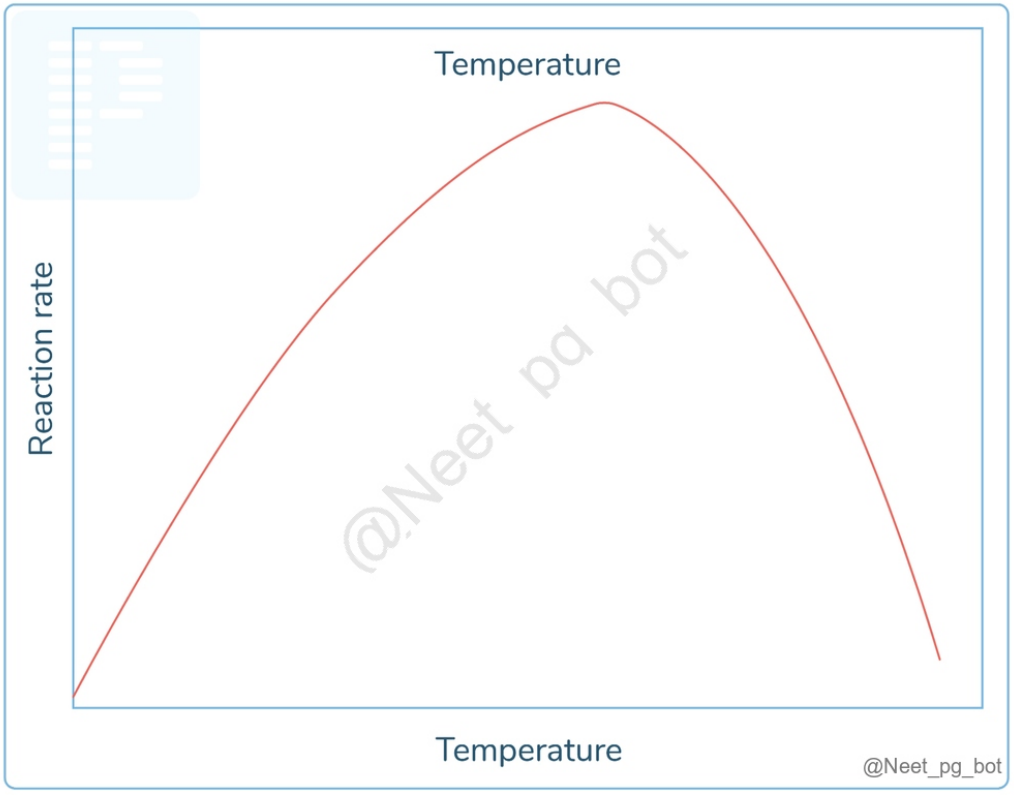
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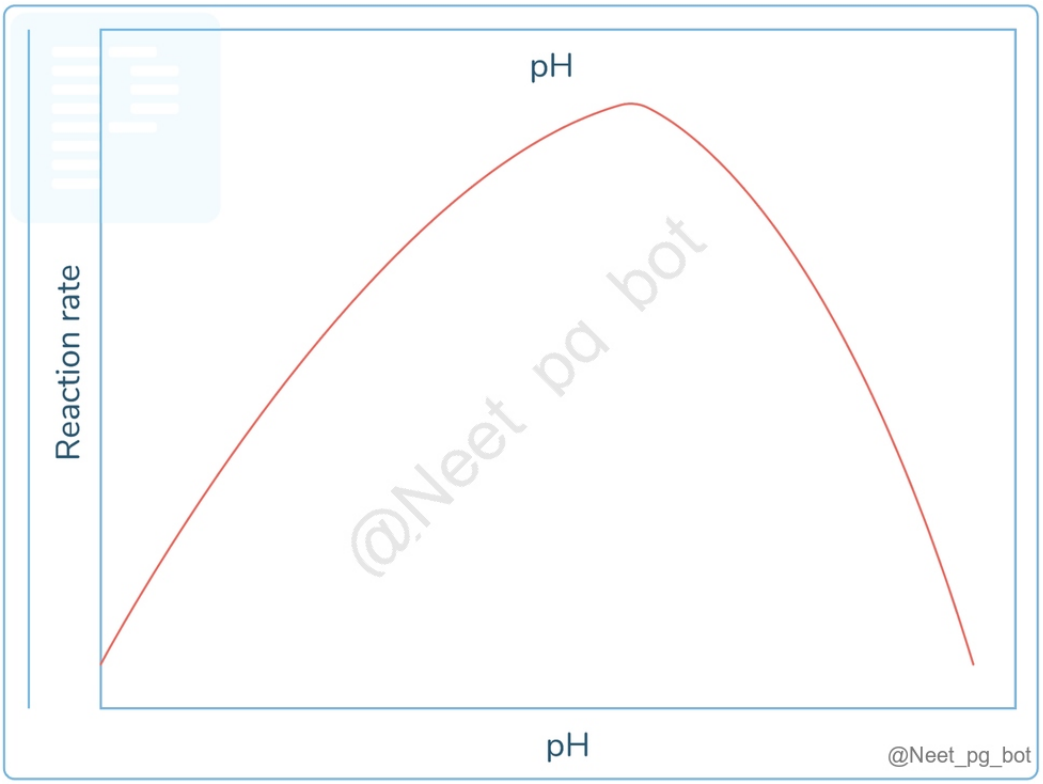
Explanation: Increased product concentration slows, stops, or reverses the reaction Factor Effect on Reaction Rate Enzyme Concentration Rate of reaction increases with an increase in enzyme concentration Substrate Concentration Initial phases: Velocity increases with increasing substrate concentration Vmax: Maximum velocity attainable at substrate saturation level Once Vmax is attained, changes in substrate concentration will not affect the rate of reaction Product Concentration Increased product concentration slows, stops, or reverses the reaction Temperature Increasing temperature increases reaction velocity Velocity reaches a maximum at an optimum temperature and then falls with further increase pH Each enzyme has an optimum pH pH value is nearer to optimum→reaction rate increases


Enzyme concentration
Reaction rate
(Enzyme)
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Substrate concentration
Reaction rate
(Substrate)
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Product concentration
Reaction rate
(Product)
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Temperature
Reaction rate
Temperature
@Neet_pg_bot



References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page56-58

↑ *Back to this question in Part A*

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Q15: Which of the following statements is/are true regarding the mechanisms of action of enzymes? Enzymes that act by altering the strain of substrates catalyze lytic reactions Enzymes forming covalent bonds are seen commonly in group transfer reactions When the functional groups of aminoacyl side chains of enzymes act as an acid or base, the rate of reaction increases (Q843275)

A. 1, 2

B. 2, 3

C. 1, 3

D. 1, 2, 3

Correct Answer: D. 1, 2, 3

■ *Listen to Audio 1*

Explanation: Different enzymes have different mechanisms to catalyze a reaction Mechanism Description Catalysis by Proximity Substrates come in bond-forming proximity to enzymes→Enzymes bind substrate molecules at their active site→region of high local substrate concentration created→substates are ideally positioned to chemically interact→reaction is catalyzed Acid-Base Catalysis Ionizable functional groups of aminoacyl side chains and prosthetic groups of enzymes act as acids or bases causing catalysis (Statement 3) Catalysis by Strain Enzymes bind substrates in a conformation that weakens the bond targeted for cleavage→ catalyses lytic reactions (Statement 1) Covalent Catalysis Formation of a covalent bond between the enzyme and one or more substrates catalyzes the reaction Common among enzymes that catalyze group transfer reactions (Statement 2)

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 62

↑ *Back to this question in Part A*

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Q16: A 35-year-old female with chronic diarrhea, weight loss, and fatty stools complains that her symptoms worsen after a high-fat meal. Reduced activity of pancreatic lipase is suspected. Which of the following ions is most likely to be low in this patient? (Q843272)

A. Calcium

B. Copper

C. Iron

D. Zinc

Correct Answer: A. Calcium

■ *Listen to Audio 1*

Explanation: Pancreatic lipase is an ion-activated enzyme. Its activity is enhanced by adding calcium ions, which is its cofactor. A cofactor is a collective term that includes coenzymes and metal ions Enzymes that require certain metal ions for their activity are called metalloenzymes Metal-ion (cofactor) Enzymes Calcium (Option A) Lecithinase, lipase Copper (Option B) Tyrosinase, cytochrome oxidase, superoxide dismutase Iron (Option C) Cytochrome oxidase, catalase, peroxidase, xanthine oxidase Zinc (Option D) Carbonic anhydrase, carboxypeptidase, alcohol dehydrogenase Magnesium Hexokinase, enolase, glucose-6-phosphatase Manganese Phosphoglucomutase, glycosyl transferases Molybdenum Xanthine oxidase Cobalt (Co^{2}■) Component of Vitamin B12-dependent enzymes (e.g., methylmalonyl-CoA mutase)

References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 50, 51

↑ *Back to this question in Part A*

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Q17: A patient presents with symptoms of fatigue and muscle weakness. Laboratory tests indicate a deficiency in riboflavin. Which coenzyme's function is most likely affected? (Q843240)

A. NAD

B. FAD

C. Coenzyme A

D. Folic acid

Correct Answer: B. FAD

■ *Listen to Audio 1*

Explanation: FAD is derived from riboflavin and is essential for redox reactions in the electron transport chain. Coenzyme Type Source (Vitamin) Function Example Reaction NAD/NADP (Option A) Nicotinamide (B3) Redox reactions Cellular respiration FAD / FM (Option B) Riboflavin (B2) Redox reactions Electron transport chain Coenzyme A (Option C) Pantothenic acid (B5) Acyl group transfer Fatty acid metabolism Folic acid/ Cobamide (Option D) Folate (B9), B12 One-carbon metabolism DNA synthesis

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 61

↑ *Back to this question in Part A*

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Q18: Which of the following is not a function of prosthetic groups? (Q843239)

A. Substrate binding and orientation

B. Formation of covalent bonds

C. Electron transfer

D. Protein degradation

Correct Answer: D. Protein degradation

■ *Listen to Audio 1*

Explanation: Prosthetic groups Definition Non-protein molecules tightly bound to proteins via covalent or non-covalent bonds. Essential for enzyme catalytic activity. Examples Pyridoxal Phosphate (PLP): Amino acid metabolism. Flavin Mononucleotide (FMN) and Flavin Adenine Dinucleotide (FAD): Redox reactions. Thiamin Pyrophosphate (TPP): Decarboxylation reactions. Functions Substrate Binding and Orientation: Proper orientation of substrates for catalytic reaction. (Option A) Formation of Covalent Bonds: Covalent bonds with reaction intermediates. (Option B) Electron Transfer: Facilitates electron transfer in redox reactions. (Option C) Activation of Substrates: Acts as Lewis acids or bases to enhance substrate reactivity. Characteristics Stable Incorporation: Tightly bound to enzymes. Catalytic Role: Extend enzyme activity beyond amino acids. Function: Participate in redox, group transfer, and covalent reactions.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 60,61

↑ *Back to this question in Part A*

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Q19: Match the enzyme class with its corresponding example: Enzyme Class Example A) Oxidoreductases 1) Hexokinase B) Transferases 2) Lactate dehydrogenase C) Hydrolases 3) DNA ligase D) Lyases 4) Trypsin E) Isomerases 5) Aldolase F) Ligases 6) Triosephosphate isomerase (Q843238)

A. A-2, B-1, C-4, D-5, E-6, F-3

B. A-1, B-2, C-4, D-5, E-3, F-6

C. A-2, B-4, C-5, D-1, E-6, F-3

D. A-3, B-2, C-4, D-5, E-6, F-1

Correct Answer: A. A-2, B-1, C-4, D-5, E-6, F-3

■ *Listen to Audio 1*

Explanation: Class Function Examples Oxidoreductases Catalyze oxidation–reduction reactions Lactate dehydrogenase, glucose-6-phosphate dehydrogenase Transferases Transfer functional groups from one molecule to another Hexokinase, aminotransferases Hydrolases Catalyze hydrolytic cleavage of bonds using water Trypsin, acetylcholinesterase Lyases Cleave bonds without hydrolysis or oxidation; often form double bonds Aldolase, decarboxylases Isomerases Catalyze intramolecular rearrangement of atoms Triosephosphate isomerase, racemases Ligases (Synthetases) Join two molecules using ATP energy DNA ligase, acetyl-CoA synthetase Translocases (recently added 7th class) Catalyze movement of ions or molecules across membranes Na■/K■-ATPase, proton pumps

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 60

↑ *Back to this question in Part A*

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Q20: What is the role of activation energy in a chemical reaction, and how do enzymes influence it? (Q843237)

A. Activation energy converts molecules from transition to ground state; enzymes increase it.

B. Activation energy converts molecules from ground state to transition state; enzymes lower it.

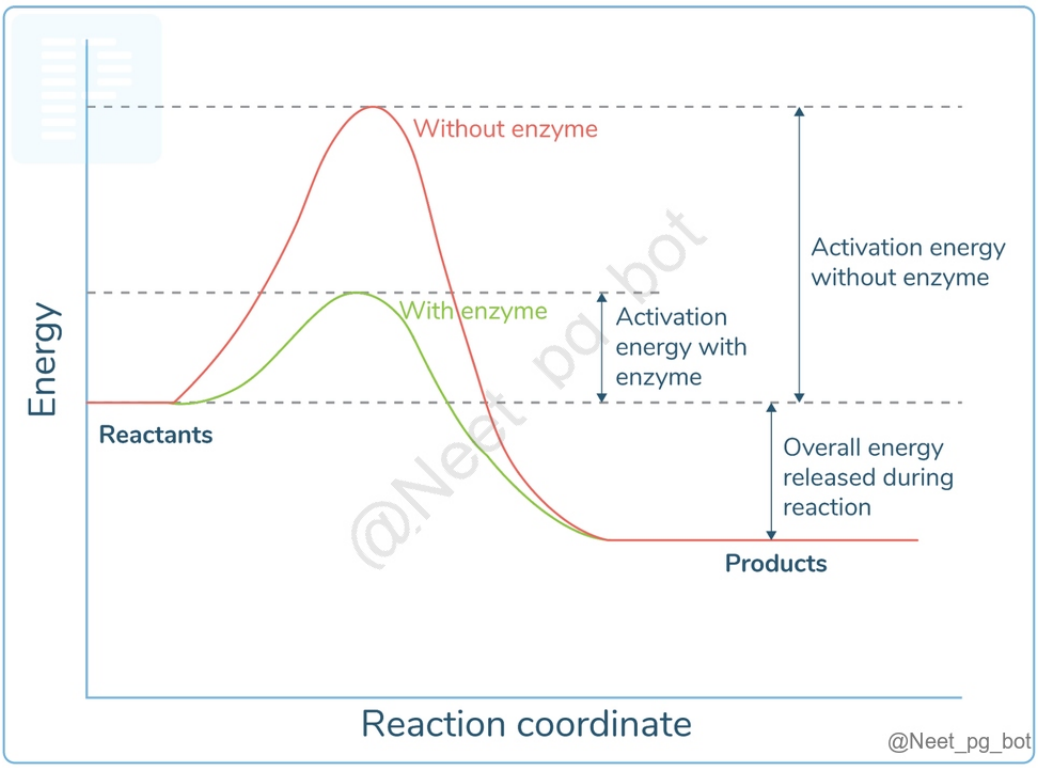
C. Activation energy is released during product formation; enzymes do not affect it.

D. Activation energy breaks bonds in products; enzymes increase it.

Correct Answer: B. Activation energy converts molecules from ground state to transition state; enzymes lower it.

■ *Listen to Audio 1*

Explanation: Activation energy Definition Energy needed to convert reactants to the unstable transition state with partially formed/broken bonds; a positive energy barrier is required for reaction progress. Role of Enzymes Enzymes lower activation energy, making it easier for reactions to occur. Reaction Rate Factors Reaction rate increases as activation energy decreases; temperature increases kinetic energy, leading to more frequent and energetic collisions. Visualization Transition state visualized as neither free substrate nor product, with partial bonds forming and rupturing. Substrate Binding Enzymes optimize weak interactions with substrates, providing the driving force for catalysis. Influencing Factors Enzyme activity is affected by pH, temperature, and presence of inhibitors or activators. Example Acid hydrolysis of sucrose: Activation energy with enzyme (sucrase) is 9,000 cal/mol while without enzyme is around 26,000 cal/mol. Activation energy converts molecules from transition to ground state; enzymes increase it (Option A): Incorrect—activation energy is needed to reach the transition state and enzymes lower it. Activation energy is released during product formation; enzymes do not affect it (Option C): Incorrect—activation energy is absorbed to start a reaction and enzymes do lower it. Activation energy breaks bonds in products; enzymes increase it (Option D): Incorrect—activation energy breaks bonds in reactants and enzymes lower , not raise, it.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 72,73

↑ *Back to this question in Part A*

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Q21: Which of the following statements about suicide inhibition is incorrect? (Q843320)

A. The inhibitor covalently binds to the enzyme after being converted to a reactive intermediate.

B. Suicide inhibition is a reversible form of enzyme inhibition.

C. Suicide inhibitors resemble the enzyme's natural substrate.

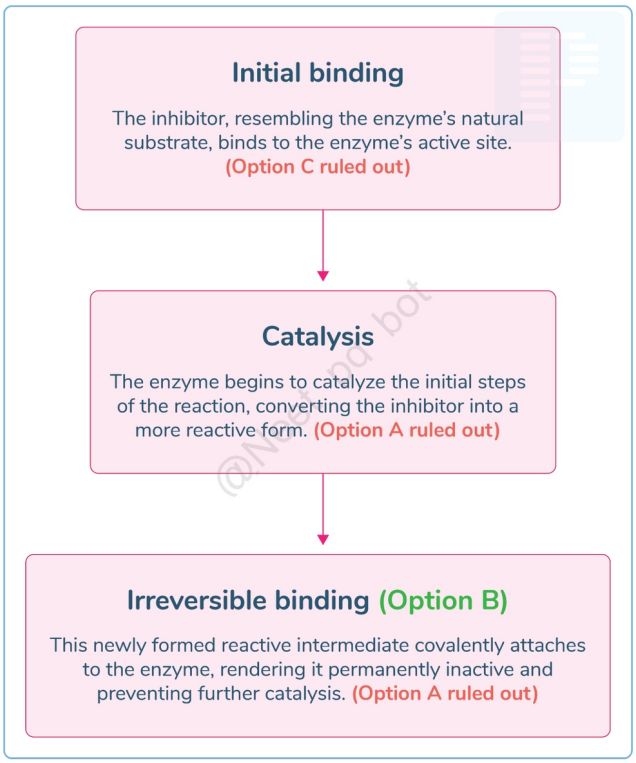
D. Allopurinol acts as a suicide inhibitor of xanthine oxidase to manage gout.

Correct Answer: B. Suicide inhibition is a reversible form of enzyme inhibition.

■ *Listen to Audio 1*

Explanation: Suicide inhibition, is a special type of irreversible enzyme inhibition. Process of Suicide Inhibition: Examples of Suicide Inhibition: Enzyme Inhibitor Mechanism Clinical Application Ornithine Decarboxylase

(ODC) Difluoromethylornithine (DFMO) DFMO binds to ODC, is converted to a reactive form, and irreversibly inactivates the enzyme. Treats trypanosomiasis by stopping polyamine synthesis in Trypanosoma parasites. Xanthine Oxidase (Option D) Allopurinol Allopurinol is oxidized to alloxanthine, which forms an irreversible complex with xanthine oxidase. Manages gout by reducing uric acid levels. Cyclo-oxygenase (COX) Aspirin Aspirin acetylates a serine residue in COX, irreversibly inhibiting the enzyme. Reduces inflammation and pain, and prevents heart attacks and strokes.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 81,82.

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Pagee 62.

↑ *Back to this question in Part A*

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Q22: Which of the following biochemical reactions is not regulated by allosteric regulation? (Q843321)

A. Phosphofructokinase-1 in glycolysis

B. Aspartate transcarbamoylase in pyrimidine biosynthesis

C. Glycogen phosphorylase in glycogenolysis

D. Glucokinase in glycolysis

Correct Answer: D. Glucokinase in glycolysis

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Explanation: Glucokinase catalyzes the first step of glycolysis, converting glucose to glucose-6-phosphate. Unlike some other enzymes, glucokinase is primarily regulated by product inhibition (glucose-6-phosphate) rather than allosteric regulation. Allosteric regulation is a mechanism where an enzyme's activity is modified by the binding of an effector molecule at a site other than the active site, known as the allosteric site. Allosteric Site: A specific location on an enzyme distinct from the active site where effector molecules bind, inducing conformational changes that influence enzyme activity. Effectors: Molecules that bind to the allosteric site and modify enzyme activity. Conformational Changes : Binding of an effector to the allosteric site causes a conformational change in the enzyme, which can alter the enzyme's affinity for its substrate and modify the enzyme's catalytic efficiency. Examples of Allosteric Regulation Enzyme Allosteric Inhibitor Allosteric Activator Notes Phosphofructokinase (Option A) ATP, citrate AMP, F-2,6-P Key regulatory enzyme in glycolysis Aspartate transcarbamoylase (Option B) CTP ATP Important in pyrimidine synthesis Glycogen phosphorylase (Option C) ATP, glucose-6-P AMP Regulates glycogen breakdown Pyruvate kinase ATP, alanine F-1,6-BP Regulates glycolysis

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 88-91.

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 62,63.

↑ *Back to this question in Part A*

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Q23: Which of the following shapes of the Michaelis-Menten plot is observed for allosteric regulation? (Q843322)

A. Hyperbolic curve

B. Sigmoidal curve

C. Linear increase

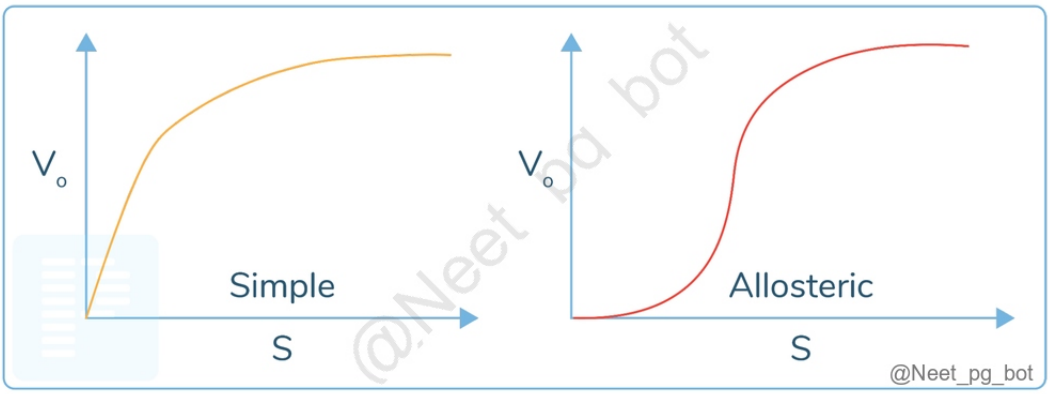
D. Parabolic curve

Correct Answer: B. Sigmoidal curve

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Explanation: A sigmoidal curve, typical of allosteric enzymes, shows cooperative binding. Initially, reaction velocity rises slowly due to low substrate affinity. As more substrates bind, conformational changes enhance

enzyme affinity, causing a sharp increase in velocity before saturation. In enzyme kinetics, a hyperbolic curve in Michaelis-Menten plots represents enzymes with simple kinetics, where reaction velocity increases rapidly at low substrate concentrations and levels off as the enzyme becomes saturated.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 88-91.

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 62,63.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q24: Match the following regulation mechanisms with their respective enzymatic reactions: Regulation mechanisms Enzymatic reactions 1. Compartmentation a. Phosphorylation of glycogen phosphorylase 2. Feedback inhibition b. Negative regulation of phosphofructokinase by ATP 3. Covalent modification c. Segregation of glycolytic enzymes in the cytosol 4. Proteolytic activation d. Activation of pepsinogen to pepsin (Q843323)

A. 1 - c, 2 - d, 3 - a, 4 - b

B. 1 - b, 2 - c, 3 - d, 4 - a

C. 1 - c, 2 - b, 3 - a, 4 - d

D. 1 - d, 2 - a, 3 - b, 4 - c

Correct Answer: C. 1 - c, 2 - b, 3 - a, 4 - d

■ *Listen to Audio 1*

Explanation: Regulatory Mechanism Description Examples Compartmentation Separating metabolic processes into different cellular compartments to regulate their activity. Segregation of glycolytic enzymes in the cytosol. Separation of fatty acid oxidation in the mitochondria from fatty acid synthesis in the cytosol. Feedback Inhibition A molecule decreases the activity of an enzyme. Negative regulation of phosphofructokinase by ATP. Inhibition of acetyl-CoA carboxylase by palmitoyl-CoA in fatty acid synthesis. Covalent Modification Addition or removal of a covalent bond of a chemical group to alter an enzyme's activity. Phosphorylation of glycogen phosphorylase. Acetylation of histones affecting gene expression. Proteolytic Activation Converting an inactive enzyme precursor into its active form through proteolytic cleavage. Activation of pepsinogen to pepsin. Activation of trypsinogen to trypsin.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 86-92

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 63, 64, 65, 196, 197

↑ *Back to this question in Part A*

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Q25: Which of the following statements about isoenzymes is incorrect? (Q843324)

A. Isoenzymes can be distinguished by their different amino acid sequences or structural properties

B. Allozymes are produced by different genes and can have distinct Km values or substrate affinities

C. Isoforms result from post-translational modifications, such as changes in sialic acid content in alkaline phosphatase (ALP)

D. Electrophoresis is a technique used to separate isoenzymes based on their mobility.

Correct Answer: B. Allozymes are produced by different genes and can have distinct Km values or substrate affinities

■ *Listen to Audio 1*

Explanation: Allozymes are variants of the same gene, not produced by different genes. They do exhibit differences in Km values or substrate affinities. Isoenzymes Isoenzymes or isozymes, are distinct forms of the same enzyme that catalyze the same chemical reaction but differ in their amino acid sequences, structural properties, or regulatory mechanisms (Option A) These variations allow isoenzymes to be tissue-specific or associated with different developmental stages, reflecting their adaptation to specific physiological needs or conditions while performing the same fundamental biochemical function. Variants: True Isoenzymes: Produced by different genes, e.g., salivary vs. pancreatic amylase. Allozymes: Variants of the same gene, e.g., glucose-6-phosphate dehydrogenase Isoforms: Result from post-translational modifications, e.g., sialic acid content in ALP (Option C) Identification Techniques: Electrophoresis: Isoenzymes can be separated based on mobility (Option D) Heat Stability: Some isoenzymes are more heat-sensitive. Inhibitors: Sensitivity to specific inhibitors can help differentiate isoenzymes. Km Value/Substrate Specificity: Different isoenzymes may have

distinct Km values or substrate affinities. Cofactor Requirements: Isoenzymes might differ in their cofactor needs. Tissue Localization: Isoenzymes are often found in specific tissues. Examples: Lactate Dehydrogenase (LDH): 5 isoforms (LDH-1 to LDH-5) Creatine Kinase (CK): 3 isoforms (CK-MM, CK-BB, CK-MB) Alkaline Phosphatase (ALP): 3 isoforms (Bone, Liver, Placental)

References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 66, 67, 302-308

↑ *Back to this question in Part A*

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Q26: A 45-year-old male presents with severe pain and swelling in his right calf following an intense workout session two days ago. Laboratory tests, including measurements of lactate dehydrogenase (LDH) isoenzymes, are ordered. Which LDH isoenzyme is most indicative of the patient's condition? (Q843325)

A. LDH-1

B. LDH-2

C. LDH-3

D. LDH-5

Correct Answer: D. LDH-5

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Explanation: Lactate dehydrogenase (LDH) is an enzyme involved in the interconversion of lactate and pyruvate during anaerobic glycolysis . LDH exists as five isoenzymes , each composed of four subunits of two types: H (Heart) subunit M (Muscle) subunit Different combinations of these subunits produce five isoenzymes with different tissue distributions : Isoenzyme Subunit Composition Major Tissue Distribution Clinical Significance LDH-1 (Option A) H■ Heart, RBCs Increased in myocardial infarction , hemolysis LDH-2 (Option B) H■M■ Reticuloendothelial system, RBCs Normally highest LDH fraction in serum LDH-3 (Option C) H■M■ Lungs Increased in pulmonary diseases LDH-4 H■M■ Kidney, placenta, pancreas Increased in renal disease LDH-5 M■ Liver, skeletal muscle Increased in muscle injury, rhabdomyolysis, liver disease Since the patient has skeletal muscle injury after intense exercise , the isoenzyme most elevated is LDH-5 , which predominates in skeletal muscle and liver .

References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Pages 119, 302-308

↑ *Back to this question in Part A*

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• [05_Enzymes] 019 Previous Year Questions

« Back to Folder Directory

[05_Enzymes] 019 Previous Year Questions — Quick Jump

Q1 (p. 948)	*Q2 (p. 948)*	*Q3 (p. 949)*	*Q4 (p. 950)*	*Q5 (p. 951)*	*Q6 (p. 952)*
Q7 (p. 955)	*Q8 (p. 957)*	*Q9 (p. 958)*	*Q10 (p. 958)*	*Q11 (p. 959)*	*Q12 (p. 960)*
Q13 (p. 961)	*Q14 (p. 962)*	*Q15 (p. 963)*	*Q16 (p. 963)*	*Q17 (p. 964)*	*Q18 (p. 965)*
Q19 (p. 966)	*Q20 (p. 966)*	*Q21 (p. 967)*			

Q1: Deficiency of copper which is necessary for lysyl oxidase is seen in (Q853010)

A. Scurvy

B. Scoliosis

C. Ehler Danlos Syndrome

D. Menke's disease

Correct Answer: D. Menke's disease

Explanation: Menke’s disease is a congenital disorder of copper transport caused by ATP7A gene mutation, leading to severe copper deficiency. Copper is essential for lysyl oxidase, an enzyme required for collagen cross-linking. Deficiency results in weak connective tissues, kinky hair, growth failure, and vascular fragility. Menke’s disease: Genetic Basis X-linked defect (affects only male children) Cause Mutation in the ATP7A gene (absence of intracellular copper binding ATPase) Presentation Dietary copper is absorbed from the GI tract but cannot be transported to blood Copper cannot be mobilized from intestinal and other tissue cells Copper accumulates within cells due to the inability to export it Defective cross-linking of connective tissue, affecting vascular and connective tissues Typically fatal in infancy due to severe connective tissue and vascular defects Scurvy (Option A): Caused by vitamin C deficiency, affecting proline and lysine hydroxylation—not lysyl oxidase. Scoliosis (Option B): A spinal deformity not related to copper deficiency or lysyl oxidase. Ehlers-Danlos Syndrome (Option C): A collagen synthesis/connective tissue disorder but not due to copper deficiency; usually enzyme or collagen gene defects.

References:

Vasudevan’s Textbook of Biochemistry for Medical Students, 7th edition, Pages 521

↑ *Back to this question in Part A*

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Q2: Deficiency of which enzyme leads to increased 2,3-BPG levels and low glucose values in vitro? (Q879288)

A. Pyruvate kinase

B. Phosphoglycerate kinase

C. G6PD

D. Hexokinase

Correct Answer: B. Phosphoglycerate kinase

Explanation: Phosphoglycerate kinase (PGK) deficiency blocks the glycolytic step converting 1,3-BPG to 3-phosphoglycerate , causing accumulation of 1,3-BPG , which is then converted to 2,3-BPG , increasing its levels. Because glycolysis is slowed, RBCs consume less glucose , causing low glucose values in vitro (artificial hypoglycemia in the sample). Incorrect Options: Pyruvate kinase (Option A): Its deficiency causes hemolytic anaemia but does not increase 2,3-BPG due toan upstream block. G6PD (Option C): Leads to oxidative hemolysis; no increase in 2,3-BPG. Hexokinase (Option D): Early glycolytic block reduces 2,3-BPG because upstream intermediates decrease.

References:

Phosphoglycerate Kinase Deficiency - Symptoms, Causes, Treatment | NORD

↑ *Back to this question in Part A*

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Q3: A 13-year-old presents with acute flaccid paralysis. Cerebrospinal fluid is collected, and an RT-PCR test is ordered to identify the causative virus. Which viral enzyme is responsible for replicating the genome of this RNA virus? (Q859716)

A. DNA-dependent RNA polymerase

B. RNA-dependent DNA polymerase

C. DNA-dependent DNA polymerase

D. RNA-dependent RNA polymerase

Correct Answer: D. RNA-dependent RNA polymerase

Explanation: The virus causing acute flaccid paralysis is most likely Poliovirus , a positive-sense single-stranded RNA virus (Picornavirus family). To replicate its RNA genome, the virus uses the enzyme RNA-dependent RNA polymerase , which synthesizes a complementary negative-sense RNA strand that serves as a template to

synthesize the new RNA strands required for replication. In laboratory diagnosis, RT-PCR employs reverse transcriptase (an RNA-dependent DNA polymerase) to convert viral RNA into complementary DNA (cDNA), enabling amplification and detection. DNA-dependent RNA polymerase (Option A) is found in cells and some DNA viruses; it synthesizes RNA from a DNA template, not RNA from RNA. RNA-dependent DNA polymerase (Option B): Also called reverse transcriptase, it converts RNA into DNA, used in RT-PCR, but not for viral RNA genome replication in poliovirus. DNA-dependent DNA polymerase (Option C) replicates DNA from a DNA template and is irrelevant for RNA viruses like poliovirus.

References:

The Basics of Reverse Transcription PCR (RT-PCR)

BCMJ_56_Vol10_wrap4.indd

Poliovirus RNA-dependent RNA polymerase

↑ *Back to this question in Part A*

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Q4: Which of the following is not a copper-containing enzyme? (Q859570)

A. Mitochondrial superoxide dismutase

B. Cytosolic superoxide dismutase

C. Tyrosinase

D. Lysyl oxidase

Correct Answer: A. Mitochondrial superoxide dismutase

Explanation: Mitochondrial superoxide dismutase (MnSOD or SOD2) is not a copper-containing enzyme . Instead, it contains manganese (Mn) as its cofactor, not copper. It is one of the three isoforms of superoxide dismutase (SOD) that help protect cells from oxidative stress by converting superoxide radicals ($O_2^{\bullet -}$) into hydrogen peroxide (H_2O_2) and molecular oxygen (O_2). The copper-containing enzymes rely on copper to carry out various essential functions in the body. Ceruloplasmin Cytochrome oxidase Cytochrome c Tyrosinase (Option C ruled out) Lysyl oxidase (Option D ruled out) ALA synthase Monoamine oxidase Cytosolic Superoxide dismutase (Option B ruled out) Phenol oxidase

References:

Vasudevan 7th edition Pg no. 520-523

Superoxide Dismutases: Role in Redox Signaling, Vascular Function, and Diseases - PMC

↑ *Back to this question in Part A*

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Q5: Which enzymes show deficiency in Von Gierke's disease? (Q859425)

A. Liver glycogen phosphorylase

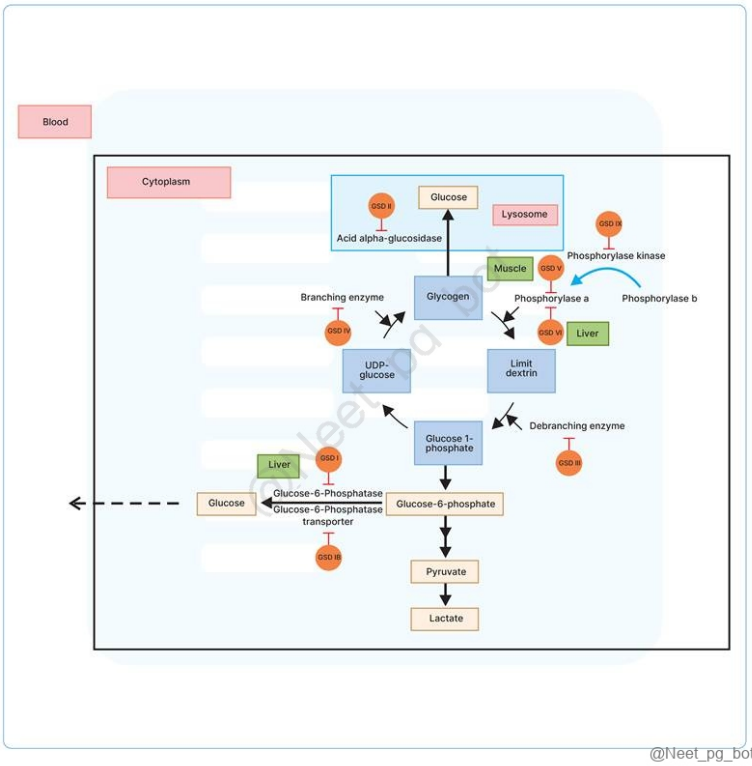
B. Muscle glycogen phosphorylase

C. Glycogen debranching enzyme

D. Glucose-6-phosphatase

Correct Answer: D. Glucose-6-phosphatase

Explanation: Von Gierke's disease is characterized by a deficiency of glucose-6-phosphatase, which impairs the liver’s ability to convert glycogen to glucose. This results in severe hypoglycemia, hepatomegaly, and elevated levels of lactate, triglycerides, and uric acid. Von Gierke's Disease (Glycogen Storage Disease Type I) Cause Deficiency of glucose-6-phosphatase enzyme Pathophysiology Inability to convert glucose-6-phosphate to glucose; leads to accumulation of glycogen, lactate, and triglycerides Symptoms Severe hypoglycemia Hepatomegaly and Renomegaly Growth retardation Lactic acidosis Hyperuricemia and Hyperlipidemia Diagnosis Biochemical tests (hypoglycemia, lactic acidosis, hyperuricemia, hyperlipidemia) Genetic testing Management Frequent feedings with slow-release carbohydrates Blood glucose monitoring Medications for associated conditions Liver transplantation (in severe cases) Glycogen Storage Disease Enzyme Deficiency Type 0 Glycogen synthase deficiency Type I (von Gierke's Disease) Glucose-6-phosphatase Type II (Pompe's Disease) Acid α-glucosidase (acid maltase) Type III (Cori's Disease) Debranching enzyme (amylo-1,6-glucosidase) (Option C ruled out) Type IV (Andersen's Disease) Branching enzyme Type V (McArdle's Disease) Muscle glycogen phosphorylase (myophosphorylase) (Option B ruled out) Type VI (Hers' Disease) Liver glycogen phosphorylase (Option A ruled out) Type VII (Tarui's Disease) Phosphofructokinase (muscle type) {{caption_text}}



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References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page no: 123, 128, 129

Harper's Illustrated Biochemistry (23rd Edition), Page 174.

↑ *Back to this question in Part A*

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Q6: What is the likely diagnosis for a 36-year-old man who has been experiencing bluish-black discoloration of the sclera and pinna for the past 6 months, along with a history of black urine upon standing since childhood? (Q859421)

A. Phenylketonuria

B. Alkaptonuria

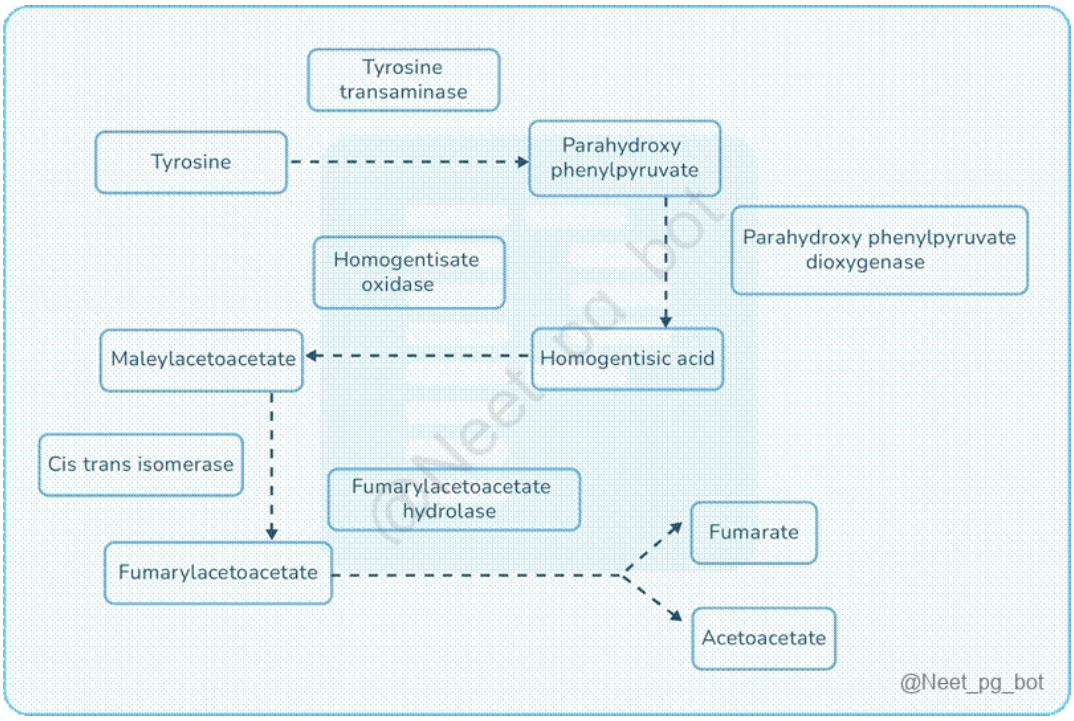
C. Tyrosinemia

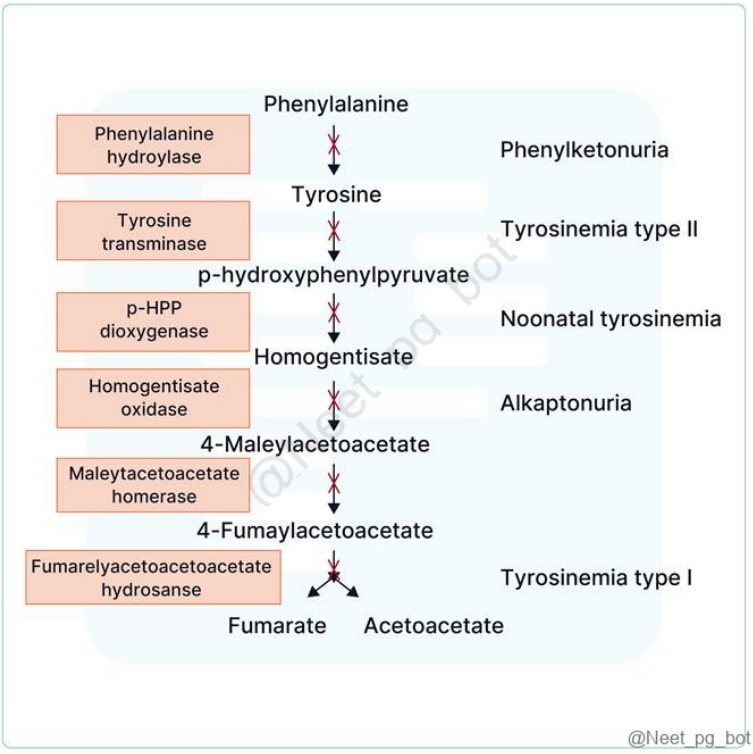
D. Maple syrup urine disease

Correct Answer: B. Alkaptonuria

Explanation: The clinical scenario described, including darkening of urine upon standing, bluish-black discoloration of the sclera and ear cartilage, and joint pain, is characteristic of Alkaptonuria , which is caused by a deficiency in the enzyme homogentisate oxidase. Garrod's tetrad in alkaptonuria 1. Homogentisic Aciduria Presence of homogentisic acid in the urine, which turns dark upon standing or when exposed to air. 2. Ochronosis {{caption_text}} Deposition of dark pigment in connective tissues, particularly noticeable in the ear cartilage, sclera, and other connective tissues, leading to a bluish-black discoloration.The image shows Osler’s sign. 3. Arthropathy Degenerative joint disease, especially affecting the spine and large joints, leading to chronic pain and stiffness. 4. Cardiac Involvement Includes heart valve calcification and atherosclerosis due to the deposition of pigment in cardiac tissues. {{caption_text}} {{caption_text}} Phenylketonuria (Option A): PKU causes mental retardation, hypopigmentation, and a mousy odor due to phenylalanine accumulation. However, it does not cause bluish-black discoloration of the sclera or black urine upon standing. Tyrosinemia (Option C): This disorder involves the accumulation of tyrosine or its metabolites, causing liver and kidney damage, but does not lead to bluish-black discoloration of the sclera or pinna or black urine upon standing. Maple Syrup Urine Disease (Option D): This is a metabolic disorder caused by the inability to break down branched-chain amino acids, leading to a sweet, maple syrup odor in the urine. It does not cause black urine or scleral discoloration.







References:

Harper’s Illustrated Biochemistry, 32nd Edition Pg. 296, 573, 574

Phenylketonuria - StatPearls - NCBI Bookshelf

Tyrosinemia - MSD Manual Consumer Version

↑ *Back to this question in Part A*

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Q7: Which of the following is true regarding competitive inhibition? (Q854885)

A. The inhibitor binds to an allosteric site on the enzyme.

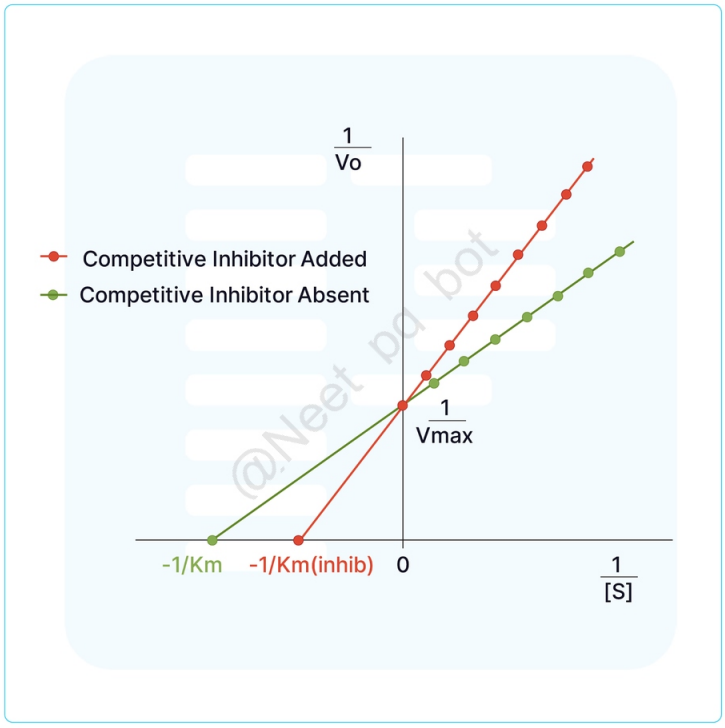
B. Competitive inhibition decreases the apparent Vmax (maximum reaction rate) of the enzyme.

C. Increasing substrate concentration can overcome competitive inhibition.

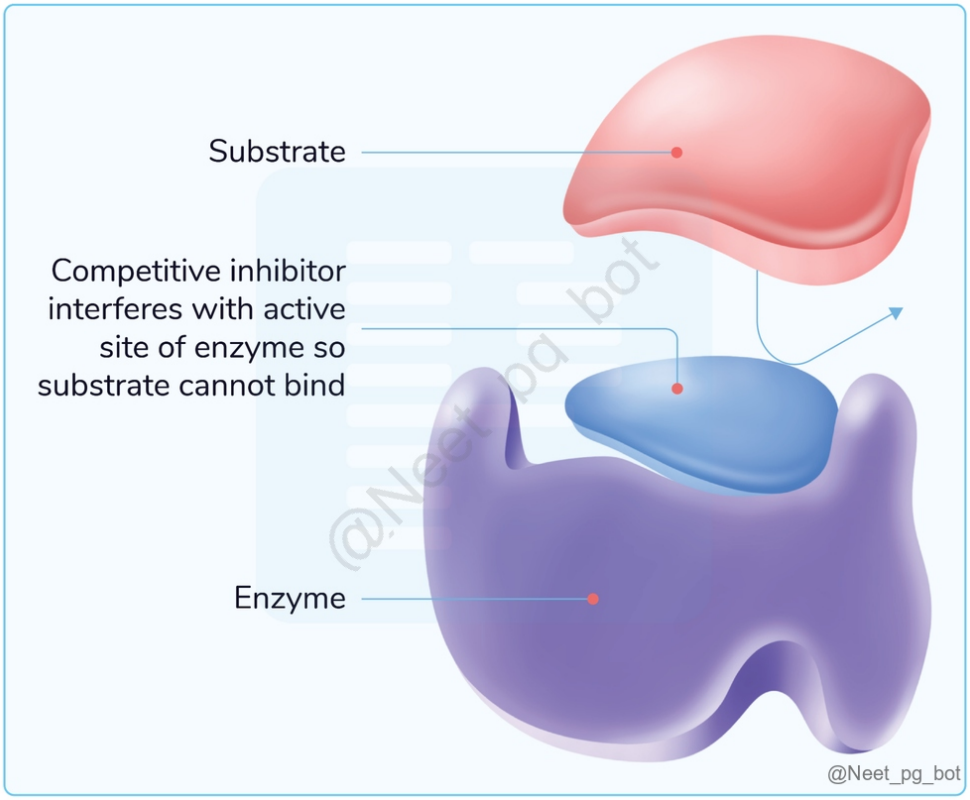
D. Competitive inhibition decreases the Km for the substrate

Correct Answer: C. Increasing substrate concentration can overcome competitive inhibition.

Explanation: Increasing the concentration of substrate can overcome the competitive inhibition. It is more likely to outcompete the inhibitor for binding to the active site, allowing enzyme to function at its maximum rate. Competitive inhibition is a type of enzyme inhibition where a molecule (the inhibitor) competes with the substrate for binding to the active site of the enzyme. Mechanism : The inhibitor resembles the substrate and binds to the enzyme’s active site. This prevents the substrate from binding, reducing the rate of the enzyme-catalyzed reaction. Effect on Enzyme Kinetics: Km (Michaelis constant): Increases because a higher substrate concentration is needed to overcome the competition. Vmax (Maximum reaction velocity): Unchanged because at sufficiently high substrate concentrations, the inhibitor is outcompeted. Reversibility : Competitive inhibition is reversible by increasing the concentration of the substrate. Examples : Competitive inhibitors Clinical Correlation Methotrexate and Dihydrofolate Reductase Methotrexate acts as a competitive inhibitor by mimicking dihydrofolate, the natural substrate of DHFR, and binding to its active site, thereby preventing dihydrofolate from binding and inhibiting the enzyme's activity. Sulfa Drugs and PABA Sulfa drugs, such as sulfanilamide, competitively inhibit the enzyme dihydropteroate synthase by mimicking para-aminobenzoic acid (PABA), a substrate involved in folic acid synthesis in bacteria. Ethanol and Alcohol Dehydrogenase Ethanol can act as a competitive inhibitor of methanol metabolism by competing for the active site of alcohol dehydrogenase. This is used in cases of methanol poisoning. Captopril and Angiotensin-Converting Enzyme (ACE) Captopril competes with the natural substrate of ACE, thereby inhibiting the production of angiotensin II, a peptide that increases blood pressure.



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References:

Harper biochemistry ed 32,Pg 79 & 80

Vasudevan ed 7,pg 59&60

↑ *Back to this question in Part A*

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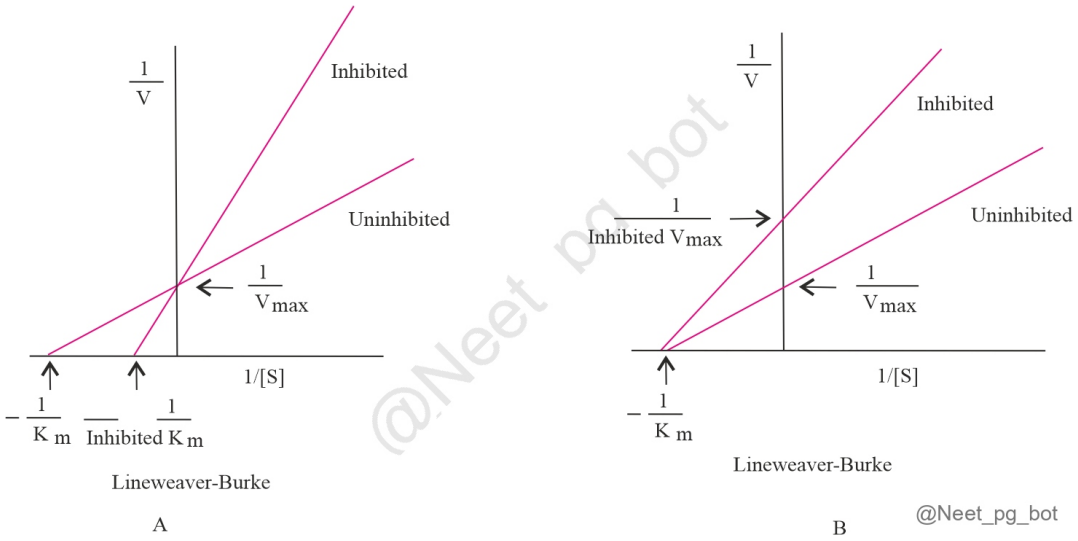
Q8: In the given enzyme kinetics graphs, identify the types of inhibitors labeled as A and B. (Q839952)

A. Competitive and Non-Competitive

B. Uncompetitive and Non-competitive

C. Non-Competitive and Uncompetitive

D. Non-competitive and competitive



Correct Answer: A. Competitive and Non-Competitive

Explanation: Correct Option A - Competitive and Non-Competitive: Competitive Inhibition (Inhibitor A): In a Lineweaver-Burk plot, competitive inhibition is characterized by lines that intersect on the y-axis. This is because the presence of a competitive inhibitor increases the apparent Km (Michaelis constant), but the Vmax■ (maximum velocity) remains unchanged. The inhibitor competes with the substrate for the active site of the enzyme. Non-Competitive Inhibition (Inhibitor B): In a Lineweaver-Burk plot, non-competitive inhibition is shown by lines that intersect on the x-axis. This occurs because non-competitive inhibitors decrease the Vmax■ but do not affect the Km■. The inhibitor binds to an allosteric site (not the active site), affecting the enzyme's function regardless of the substrate concentration. Incorrect Options: Option B - Uncompetitive and Non-Competitive: Uncompetitive inhibition shows parallel lines in a Lineweaver-Burk plot, as both Vmax■ and Km ■ are reduced proportionally. This does not match the provided data for inhibitor A. Option C - Non-Competitive and Un-competitive: This option incorrectly identifies the inhibitors in reverse order. The data provided for inhibitor A matches competitive inhibition, not uncompetitive. Option D - Non-competitive and competitive: Like option C, this option reverses the

identification of inhibitors A and B.

↑ *Back to this question in Part A*

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Q9: What is the most likely diagnosis of a hypertensive patient presenting with Tanner stage 4 development and hyperpigmentation? (Q839950)

A. 21 beta hydroxylase deficiency

B. 11 Beta hydroxylase deficiency

C. 17 beta hydroxylase deficiency

D. 17 alpha hydroxylase deficiency

Correct Answer: B. 11 Beta hydroxylase deficiency

Explanation: Correct Option B - 11 Beta hydroxylase deficiency: This enzyme deficiency leads to an accumulation of 11-deoxycorticosterone (DOC), which has mineralocorticoid activity, causing hypertension. Patients with 11-beta hydroxylase deficiency typically present with signs of virilization (such as Tanner stage 4) and hyperpigmentation due to increased ACTH stimulation. Incorrect Options: Option A - 21 beta hydroxylase deficiency: This deficiency typically causes virilization and hyperpigmentation but is associated with hypotension, not hypertension. Option C - 17 beta hydroxylase deficiency: This enzyme deficiency usually presents with hypertension and ambiguous genitalia in males or lack of secondary sexual characteristics in females, but not virilization (Tanner stage 4). Option D - 17 alpha hydroxylase deficiency: This condition leads to hypertension and sexual infantilism, but not virilization or hyperpigmentation.

References:

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↑ *Back to this question in Part A*

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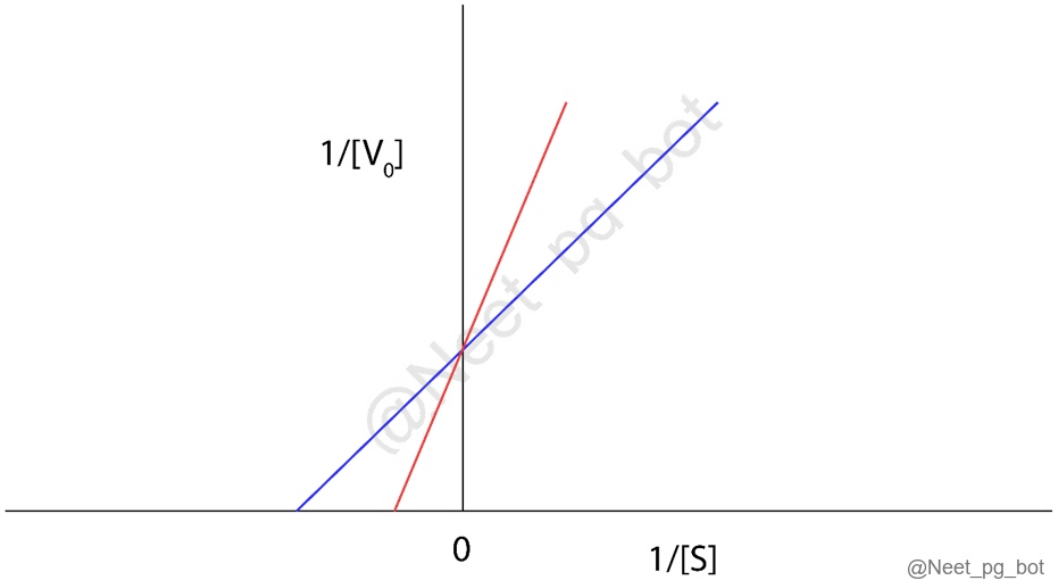
Q10: In the given graph, identify the type of inhibition: (Q844275)

A. Competitive

B. Non-competitive

C. Mixed

D. None of the above



Correct Answer: A. Competitive

Explanation: The Lineweaver–Burk plot shows both lines intersecting at the same point on the Y-axis (same 1/Vmax). This means Vmax is unchanged. The slopes are different and the X-intercepts shift → Km increases in the presence of inhibitor. These features are classic for competitive inhibition. In competitive inhibition, the inhibitor competes with substrate for the active site. Increasing substrate concentration can overcome inhibition. Therefore, the graphical pattern fits competitive inhibition exactly. Incorrect Options: Option B) Non-competitive inhibition : Would show same X-intercept (Km unchanged) but different Y-intercepts (Vmax decreases). In the graph, the Y-intercept is the same → so this is NOT non-competitive. Option C) Mixed inhibition : Both Km and Vmax change, so neither X-intercept nor Y-intercept remain the same. Graph would show lines intersecting off both axes, not at Y-axis. Not matching the given graph. Option D) None of the above : Not correct because the graph clearly matches a known inhibition type (competitive).

References:

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↑ *Back to this question in Part A*

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Q11: A patient presents with severe abdominal pain and elevated lipase levels. Imaging reveals a stone in the common bile duct (CBD). Which enzyme is likely to be elevated in this condition? (Q844268)

A. Gamma-glutamyl transferase (GGT)

B. Lactate Dehydrogenase (LDH)

C. Alanine Aminotransferase (ALT)

D. Aspartate Aminotransferase (AST)

Correct Answer: A. Gamma-glutamyl transferase (GGT)

Explanation: Correct Option A - Gamma-glutamyl transferase (GGT): Gamma-glutamyl transferase levels are elevated in bile duct obstruction, such as a stone in the common bile duct, along with alkaline phosphatase (ALP). Incorrect Options: Options B, C, and D are not elevated in a patient with common bile duct stones.

↑ *Back to this question in Part A*

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Q12: A 13-year-old boy presents with jaundice, fatigue, muscle stiffness, tremors, behavioral changes, hepatosplenomegaly, and Kayser-Fleischer rings. Which test is definitive for the diagnosis? (Q844273)

A. Urinary copper

B. Serum ceruloplasmin

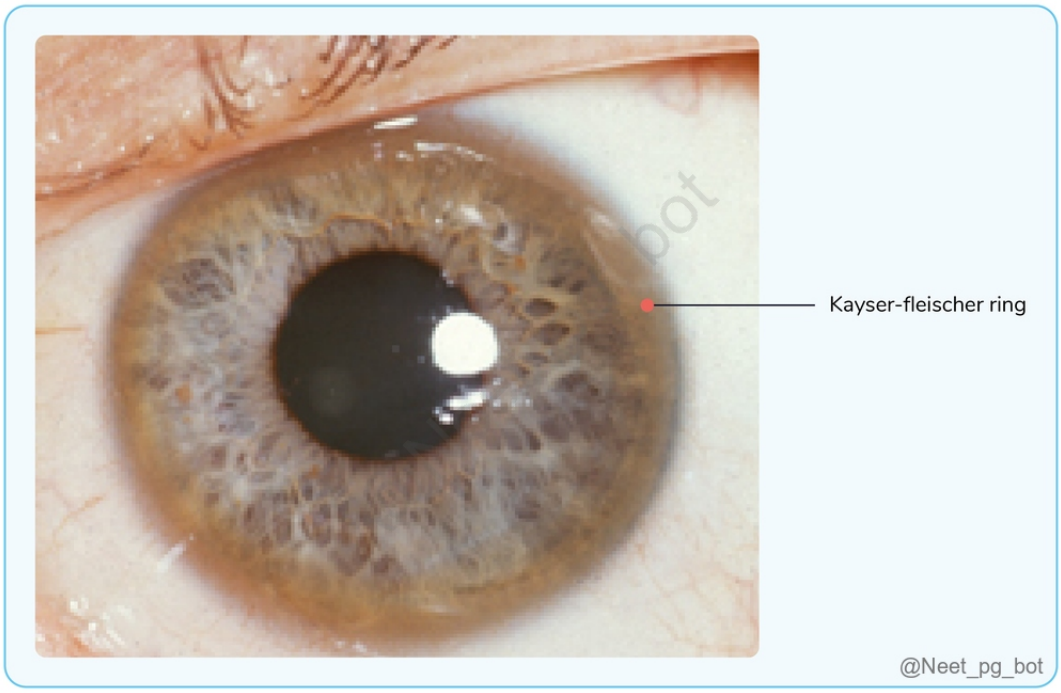
C. Hepatic parenchymal copper concentration

D. Genetic testing for ATP7B mutation

Correct Answer: C. Hepatic parenchymal copper concentration

Explanation: The clinical presentation of jaundice, fatigue, muscle stiffness, tremors, behavioral changes, hepatosplenomegaly, and Kayser-Fleischer rings(most specific) suggests Wilson's disease and measuring the Hepatic parenchymal copper concentration is the most definitive test for diagnosis of Wilson's disease . Wilson Disease : Aspect Details Definition Autosomal recessive disorder of copper metabolism leading to copper accumulation in tissues. Gene Affected ATP7B gene mutation on chromosome 13. (Option D) - Helpful, but ATP7B has many mutations; testing can miss variants, and results take time. Pathophysiology Defective copper transport leads to: Decreased copper incorporation into ceruloplasmin. Impaired biliary excretion of copper. Copper accumulates in the liver, spills into the bloodstream, and deposits in various tissues. Clinical Features Hepatic Acute hepatitis Chronic liver disease (cirrhosis) Fulminant hepatic failure Neurological Dysarthria Movement disorders (tremors, dystonia, chorea) Rigidity, bradykinesia Psychiatric Depression, irritability, or psychosis Cognitive decline Ophthalmological Kayser-Fleischer rings (golden-brown ring at the corneal margin due to copper deposition) in slit lamp examination. Sunflower cataracts (less common) Diagnosis Biochemical Gold standard: Elevated hepatic copper content on liver biopsy (>250 µg/g dry weight) (Option C) Low serum ceruloplasmin (<20 mg/dL) (Option B) Elevated 24-hour urinary copper excretion (>100 µg/day) (Option A)

Imaging Brain MRI: Hyperintensities in basal ganglia (e.g., "face of the giant panda" sign in the midbrain) Treatment Copper Chelation D-Penicillamine (binds and increases urinary copper excretion) Copper Absorption Blockers Zinc salts : Compete with copper absorption in the gut Dietary Low-copper diet (avoid shellfish, nuts, chocolate, liver) Liver Transplant For severe liver failure or unresponsive cases



References:

Vasudevan Textbook of Biochemistry, 7th Edition, Pages 384-385

↑ *Back to this question in Part A*

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Q13: Which of the following enzymes is dependent on Vitamin C for its activity? (Q852557)

A. Lysyl dehydrogenase

B. Lysyl oxidase

C. Prolyl hydroxylase

D. Hydroxyprolyl kinase

Correct Answer: C. Prolyl hydroxylase

Explanation: Role of Prolyl Hydroxylase and Vitamin C: Prolyl hydroxylase catalyzes the hydroxylation of proline residues in collagen. This process is essential for collagen stability via hydrogen bonding. Vitamin C (ascorbic acid) is a cofactor for prolyl hydroxylase, aiding in the regeneration of the enzyme's Fe^{2}■ active site . Vitamin C Deficiency and Scurvy: Deficiency leads to impaired collagen synthesis , causing fragile blood vessels, poor wound healing, and gingival bleeding (Scurvy). Vitamin C-Dependent Enzymes in Collagen Synthesis Enzyme Function Vitamin C Dependency Prolyl hydroxylase Hydroxylates proline residues in collagen for stability. Yes Lysyl hydroxylase Hydroxylates lysine residues in collagen for cross-linking. Yes Lysyl oxidase Cross-links collagen via aldehyde formation. No (Requires copper) Hydroxyprolyl kinase Phosphorylates hydroxyproline. No Lysyl dehydrogenase (Option A): Incorrect because this enzyme is not involved in collagen synthesis and does not require Vitamin C. Lysyl oxidase (Option B): Incorrect because it requires copper (Cu^{2}■), not Vitamin C , for collagen cross-linking. Hydroxyprolyl kinase (Option D): Incorrect because it phosphorylates hydroxyproline and is not dependent on Vitamin C.

References:

https://pmc.ncbi.nlm.nih.gov/articles/PMC5045062/

https://pmc.ncbi.nlm.nih.gov/articles/PMC2692800/

↑ *Back to this question in Part A*

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Q14: Which of the following acts as a major intracellular antioxidant and helps in detoxifying reactive oxygen species? (Q866659)

A. Glutathione

B. Catalase

C. Superoxide dismutase

D. Peroxidase

Correct Answer: A. Glutathione

Explanation: Glutathione is the primary intracellular antioxidant that neutralizes reactive oxygen species by donating electrons, detoxifying them, regenerating other antioxidants, and maintaining cellular redox balance. Major intracellular antioxidant composed of glutamate, cysteine, and glycine. (Option A) Function: Neutralizes reactive oxygen species (ROS) by donating electrons, converting ROS to less harmful compounds. (Option A) Regeneration: Helps regenerate other antioxidants like vitamins C and E. Role: Maintains cellular redox balance and protects against oxidative stress. Other antioxidants: Superoxide dismutase (SOD): Converts superoxide radicals to hydrogen peroxide. (Option C ruled out) Catalase: Converts hydrogen peroxide to water and oxygen. (Option B ruled out) Peroxidase: Breaks down peroxides using reducing agents like glutathione. (Option D ruled out).

↑ *Back to this question in Part A*

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Q15: A 2-month-old infant is brought to the pediatrician due to feeding difficulties, vomiting, and failure to thrive. The child's history reveals that the infant wasborn full-term and had a normal birth weight. On physical examination, the infant appears jaundiced, and the liver is palpable below the costal margin. Oil drop Cataracts are also noted on eye examination. Laboratory tests show elevated levels of galactose in the blood and urine. Based on these clinical findings, which enzyme deficiency is most likely responsible for this presentation? (Q820344)

A. G6PD (Glucose-6-phosphate dehydrogenase)

B. GALT (Galactose-1-phosphate uridylyltransferase)

C. ALDH (Aldehyde dehydrogenase)

D. PKU (Phenylalanine hydroxylase)

Correct Answer: B. GALT (Galactose-1-phosphate uridylyltransferase)

Explanation: Correct Option B - GALT (Galactose-1-phosphate uridylyltransferase) deficiency: GALT deficiency, also known as galactosemia, is a genetic disorder that impairs the ability to metabolize galactose properly. Galactose is a sugar found in milk and other dairy products. In individuals with GALT deficiency, galactose-1-phosphate accumulates, leading to various symptoms, including feeding difficulties, vomiting, failure to thrive, jaundice, and liver enlargement. Cataracts are also a characteristic feature of galactosemia. This condition requires the removal of galactose from the diet to prevent complications. Incorrect Options: Option A - G6PD (Glucose-6-phosphate dehydrogenase) deficiency: G6PD deficiency is a genetic disorder that leads to a deficiency of the enzyme glucose-6-phosphate dehydrogenase, which is important for protecting red blood cells from oxidative damage. Option C - ALDH (Aldehyde dehydrogenase) deficiency: ALDH deficiency is associated with a condition called alcohol-induced flushing, where individuals who carry certain genetic variants of the aldehyde dehydrogenase enzyme experience facial flushing and other symptoms when they consume alcohol. Option D - PKU (Phenylalanine hydroxylase) deficiency: PKU is a genetic disorder characterized by the deficiency of the enzyme phenylalanine hydroxylase, which is responsible for converting phenylalanine to tyrosine.

References:

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↑ *Back to this question in Part A*

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Q16: Hormone-sensitive lipase is activated by all of the following EXCEPT? (Q820391)

A. Glucagon

B. Epinephrine

C. ACTH

D. Insulin

Correct Answer: D. Insulin

Explanation: Correct Option D - Insulin: Insulin is a hormone produced by the pancreas that plays a crucial role in glucose metabolism. It promotes glucose uptake into cells and storage as glycogen in the liver and muscle tissues. However, insulin inhibits the activation of hormone-sensitive lipase. When insulin levels are high (e.g., after a meal), lipolysis (breakdown of fats) is suppressed, and the body primarily uses glucose as an energy source. Incorrect Options: Option A - Glucagon: It also stimulates the activation of hormone-sensitive lipase, which leads to the breakdown of stored triglycerides (lipids/fats) into fatty acids in adipose tissue. Option B - Epinephrine: It activates hormone-sensitive lipase in adipose tissue, causing the breakdown of triglycerides into fatty acids and glycerol. Option C - ACTH (Adrenocorticotropic Hormone): While cortisol plays a role in metabolism, it does not directly activate hormone-sensitive lipase. Instead, cortisol can indirectly influence lipid metabolism by affecting other regulatory pathways.

↑ *Back to this question in Part A*

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Q17: A 62-year-old male with CAD comes to the physician for a follow-up examination. His blood pressure is 130/80 mm Hg, height of 182 cm, weight of 85 kg, and a BMI of 25 kg/m². Serum studies shows a glucose concentration of 95 mg/dL and homocysteine concentration of 19.3 μmol/L (N=5–15). Serum lipid concentrations are within the normal range. which of the following enzyme deficiencies is most likely to be found in this patient? (Q820409)

A. Homogentisate oxidase

B. Cystathionine beta synthase

C. Cystathionine gamma synthase

D. HGPRTase

Correct Answer: B. Cystathionine beta synthase

Explanation: Correct Option B -Cystathionine beta synthase: Cystathionine beta synthase deficiency is associated with homocystinuria, a genetic disorder characterized by elevated levels of homocysteine in the blood. The clinical manifestations of homocystinuria can include cardiovascular issues (such as early-onset atherosclerosis), skeletal abnormalities, intellectual disabilities, and other systemic complications. The patient in the question has an elevated homocysteine concentration, which is characteristic of homocystinuria. Incorrect Options: Option A - Homogentisate oxidase: Homogentisate oxidase deficiency leads to alkaptonuria, a condition characterized by the accumulation of homogentisic acid, causing darkening of urine and connective tissue. This condition is not consistent with the patient's presentation and lab values. Option C - Cystathionine gamma synthase: Cystathionine gamma synthase deficiency is not a well-known enzyme deficiency associated with a specific clinical disorder. Cystathionine gamma synthase is involved in the transsulfuration pathway, converting cystathionine to cysteine, but its deficiency doesn't typically lead to a recognized metabolic disorder. Option D - HGPRTase (Hypoxanthine-guanine phosphoribosyltransferase): HGPRTase deficiency is seen in Lesch-Nyhan syndrome, which is characterized by neurological and behavioral abnormalities, along with uric acid overproduction leading to gout. The patient's presentation and lab values do not match this disorder.

↑ *Back to this question in Part A*

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Q18: A 35-year-old battery factory worker presents with abdominal colic, a blue line on the gums, and anemia. Laboratory findings suggest lead poisoning. Which of the following enzymes is the most sensitive and primarily inhibited by lead in this condition? (Q830705)

A. ALA synthase

B. ALA dehydratase

C. Heme synthase

D. Coproporphyrinogen oxidase

Correct Answer: B. ALA dehydratase

Explanation: Correct Option B - ALA dehydratase: Lead is a heavy metal that interferes with heme biosynthesis by sulfhydryl (–SH) group inhibition of multiple enzymes. While it affects three steps, ALA Dehydratase is the most clinically significant and sensitive marker. It catalyzes the conversion of delta-Aminolevulinic acid (ALA) to Porphobilinogen. Lead highly stabilizes the inactive form of this enzyme. Its inhibition leads to an accumulation of ALA in the blood and urine, which is a hallmark of lead poisoning. ALA is neurotoxic, contributing to the "lead palsy" and abdominal symptoms. Option A: ALA Synthase: This is the rate-limiting enzyme. Lead does not inhibit it; in fact, because heme levels fall (negative feedback), ALA Synthase activity may actually increase, further worsening the accumulation of toxic ALA. Option C: Heme Synthase (Ferrochelatase): Lead directly inhibits this enzyme, which prevents the insertion of iron (Fe 2+) into Protoporphyrin IX. This leads to the accumulation of Erythrocyte Protoporphyrin (EP), which often binds with zinc to form Zinc Protoporphyrin (ZPP)—a key screening marker for lead toxicity. Option D: Coproporphyrinogen Oxidase: Lead inhibits this enzyme as well,

leading to increased excretion of Coproporphyrin III in the urine.

↑ *Back to this question in Part A*

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Q19: Which of the statements below accurately describes the characteristics of the Michaelis-Menten graph? (Q793383)

A. Km is directly proportional to substrate concentration

B. Km will vary with the enzyme concentration

C. Km is indirectly proportional to substrate concentration

D. Km has no relation to substrate concentration

Correct Answer: D. Km has no relation to substrate concentration

Explanation: Km has no relation to substrate concentration The Michaelis-Menten equation describes how the rate of an enzyme-catalyzed reaction changes with substrate concentration: v=Km■+[S]Vmax■[S]■ Where: v = rate of reaction Vmax■ = maximum rate [S] = substrate concentration Km■ = Michaelis constant — the substrate concentration at which the reaction rate is half of Vmax■ Km is a constant for a given enzyme-substrate pair and does not depend on enzyme concentration . Km is not directly or inversely proportional to substrate concentration (Option A and C): It represents the substrate concentration needed to reach half-maximal enzyme activity, but it is a fixed value, not a variable with [S] .. Km reflects the enzyme's affinity for the substrate : Low Km = high affinity (enzyme reaches half Vmax at low [S]) High Km = low affinity Km does NOT vary with enzyme concentration (Option B) .

References:

Biochemistry, 4 th edition, U. Satyanarayan & U. Chakrapani

↑ *Back to this question in Part A*

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Q20: By the action of the enzyme aromatase, testosterone is transformed into which of the subsequent hormones? (Q793389)

A. 5-hydroxy testosterone

B. Estradiol

C. Cortisol

D. Cortisone

Correct Answer: B. Estradiol

Explanation: Correct Option B - Estradiol: Aromatase enzyme is used in the process of androgen (such as testosterone) to estrogen (such as 17β-estradiol) conversion. Aromatase enzyme is used as a therapeutic target for the treatment process of endocrine-responsive breast cancer. Aromatase also converts androstenedione to estrone. Incorrect Options: Option A - 5-hydroxy testosterone is a metabolite of testosterone, but it is not converted to this hormone by the aromatase enzyme. 5-hydroxy testosterone is produced by the action of the enzyme 5-alpha reductase on testosterone. Option C - Cortisol is a steroid hormone that is produced in the adrenal gland, not in the testis where testosterone is produced. Cortisol is synthesized from cholesterol through a series of enzymatic reactions, but aromatase is not involved in its production. Option D - Cortisone is a steroid hormone that is closely related to cortisol, but it is not produced directly from testosterone. Cortisone is a metabolite of cortisol, and its production involves a series of enzymatic reactions that are different from those involved in the conversion of testosterone to estradiol.

↑ *Back to this question in Part A*

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Q21: What is the substance that is most likely to be secreted by the hypothalamus? (Q793467)

A. Hexokinase I

B. Hexokinase II

C. Hexokinase III

D. Hexokinase IV

Correct Answer: D. Hexokinase IV

Explanation: Correct Option D - Hexokinase IV: A hexose phosphate is produced from hexose which is a six-carbon sugar with the help of a hexokinase enzyme. Glucose is present as the most important substrate for the hexokinases that further produce glucose-6-phosphate as the most important product. Hexokinases I, II, and III are isozymes that have a “low-Km” value; hence, it has a high-affinity process for glucose, even at the low concentrations of it. The hypothalamus releases Hexokinase IV likely. HK IV or Glucokinase is also expressed in the pancreas and liver. Incorrect Choices: Option A - Hexokinase I: HK1 is the most expressed isoform of hexokinase among the four hexokinases ubiquitously. It is expressed in many tissues majorly in the kidney, brain, and red blood cells (RBCs). Option B - Hexokinase II enzyme is encoded by the HK2 gene present on chromosome 2 in humans. It is the most active isozyme of the hexokinase family. It is expressed in the insulin-sensitive tissues. Option C - Hexokinase III is predominantly produced from myeloid cells. HKI and II are able to bind to mitochondria and protect cells against cell death. HKIII is not able to bind mitochondria. Very little

is known about its cytoprotective effect.

References:

Biochemistry, 4 th edition, U. Satyanarayan & U. Chakrapani

↑ *Back to this question in Part A*

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• [06_Free Radicals_ Antioxidants _ Trace Elements] 020 Previous Year Questions

« Back to Folder Directory

[06_Free Radicals_ Antioxidants _ Trace Elements] 020 Previous Year Questions — Quick Jump

Q1 (p. 969)	*Q2 (p. 970)*	*Q3 (p. 970)*	*Q4 (p. 972)*	*Q5 (p. 973)*	*Q6 (p. 974)*
Q7 (p. 975)	*Q8 (p. 976)*	*Q9 (p. 977)*	*Q10 (p. 978)*	*Q11 (p. 979)*	*Q12 (p. 980)*
Q13 (p. 981)	*Q14 (p. 982)*	*Q15 (p. 984)*	*Q16 (p. 985)*	*Q17 (p. 986)*	*Q18 (p. 987)*
Q19 (p. 988)	*Q20 (p. 989)*	*Q21 (p. 990)*	*Q22 (p. 991)*	*Q23 (p. 991)*	*Q24 (p. 992)*
Q25 (p. 993)	*Q26 (p. 993)*	*Q27 (p. 994)*	*Q28 (p. 995)*	*Q29 (p. 996)*	*Q30 (p. 996)*
Q31 (p. 997)	*Q32 (p. 998)*	*Q33 (p. 998)*	*Q34 (p. 999)*	*Q35 (p. 1000)*	*Q36 (p. 1001)*
Q37 (p. 1002)	*Q38 (p. 1002)*	*Q39 (p. 1003)*	*Q40 (p. 1004)*	*Q41 (p. 1005)*	*Q42 (p. 1007)*
Q43 (p. 1008)	*Q44 (p. 1009)*	*Q45 (p. 1011)*	*Q46 (p. 1012)*	*Q47 (p. 1013)*	

Q1: Which enzyme deficiency causesVon Gierke's disease? (Q857492)

A. Liver phosphorylase

B. Lysosomal glucosidase

C. Hepatic glycogen synthase

D. Glucose 6-phosphatase

Correct Answer: D. Glucose 6-phosphatase

Explanation: Von Gierke’s disease (Glycogen Storage Disease Type I) is caused by a deficiency of glucose-6-phosphatase, an enzyme responsible for the final step of glycogenolysis and gluconeogenesis. Pathophysiology: Impaired conversion of glucose-6-phosphate to free glucose. Leads to glycogen accumulation in the liver and kidneys, causing hepatomegaly and severe hypoglycemia. Key symptoms: Severe fasting hypoglycemia Hepatomegaly Lactic acidosis Hyperuricemia and hyperlipidemia Liver phosphorylase (Option A): Deficiency causes Hers disease (GSD VI). Lysosomal glucosidase (Option B): Deficiency causes Pompe disease (GSD II). Hepatic glycogen synthase (Option C): Deficiency causes GSD 0 (reduced glycogen storage). Glycogen storage disease: Type Name Enzyme defect Type 1 Von Gierke's Disease Glucose-6-phophatase Type 2 Pompe's Disease Acid maltase/ alpha glucosidase Type 3 Cori's Disease Debranching enzyme Type 4 Anderson's Disease Branching enzyme Type 5 McArdle's Disease Muscle Phosphorylase Type 6 Hers Disease Liver phosphorylase Type 7 Tarui's Disease Phosphofructokinase- I

References:

Von gierke disease

↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: You are conducting an experiment on mitochondrial respiration. You add malate/pyruvate and respiration is normal. You add succinate and respiration is normal. When you add another substance in the presence of pyruvate/succinate and malate, respiration is blocked. Which of the following substances is most likely added? (Q855978)

A. Rotenone

B. Antimycin A

C. Oligomycin

D. 2,4-dinitrophenol

Correct Answer: B. Antimycin A

Explanation: In this experiment, respiration proceeds normally when malate/pyruvate (Complex I substrates) and succinate (Complex II substrate) are added. This means both Complex I and II are functioning properly. When respiration stops completely after adding another substance, it indicates the block occurs after Coenzyme Q , where both pathways converge — this points to Complex III inhibition. Antimycin A acts at Complex III (cytochrome bc■ complex) , blocking electron transfer from reduced Coenzyme Q to cytochrome c, which halts the entire electron transport chain and stops oxygen consumption. By contrast: Rotenone (Option A) inhibits Complex I , but respiration would still occur with succinate. Oligomycin (Option C) inhibits ATP synthase (Complex V) , preventing ATP synthesis but not respiration; electrons continue to flow. 2,4-Dinitrophenol (DNP) (Option D) uncouples oxidative phosphorylation, increasing oxygen consumption rather than blocking it.

References:

Harper’s Illustrated Biochemistry, 32nd edition, pg: 126

↑ *Back to this question in Part A*

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Q3: In this scenario, what is the origin of the blood glucose measured at 7 am the following morning for an individual who had dinner at 8 pm the previous night? (Q855985)

A. Hepatic glycogenolysis

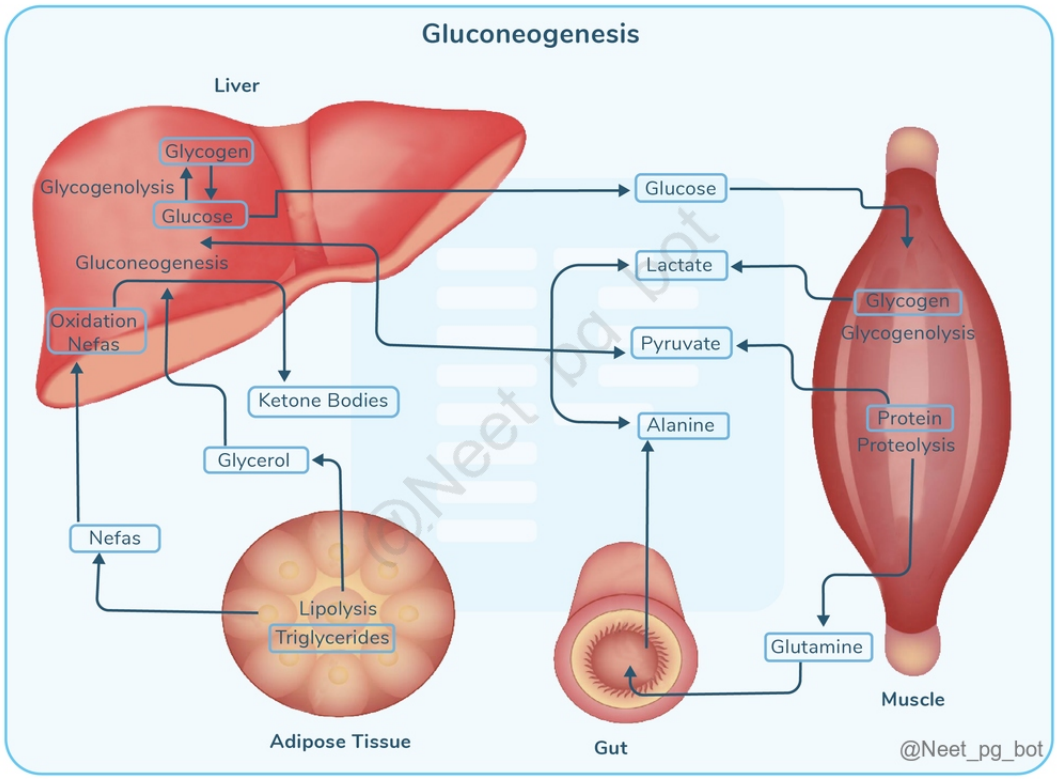
B. Hepatic gluconeogenesis

C. Muscle glycogenolysis

D. Dietary glucose

Correct Answer: A. Hepatic glycogenolysis

Explanation: During fasting: After 4-16 hrs, the primary source of glucose is glycogenolysis After 16-48 hrs, the primary source of glucose is gluconeogenesis (Option B) Gluconeogenesis: It is a process in which glucose is synthesized by non-carbohydrate sources such as amino acids, lactate, glycerol, and propionyl CoA. The major site of gluconeogenesis the liver > kidney. Regulation of gluconeogenesis: Glucagon activates fructose 1,6-bisphosphatase and inhibits PFK-1, favoring gluconeogenesis and inhibiting glycolysis The availability of substrates such as alanine, lactate, and glycerol favors gluconeogenesis Acetyl CoA is an allosteric activator of 1st regulatory step of gluconeogenesis (pyruvate carboxylase) Muscle glycogenolysis (Option C) is used during exercise and muscle activity. Dietary glucose (Option D) is used in the first 4 hrs after meals.



References:

Harper’s Illustrated Biochemistry, Ed 32, Pg 180-189

Lippincott Illustrated Reviews: Biochemistry 7e, pg no: 376-78

↑ *Back to this question in Part A*

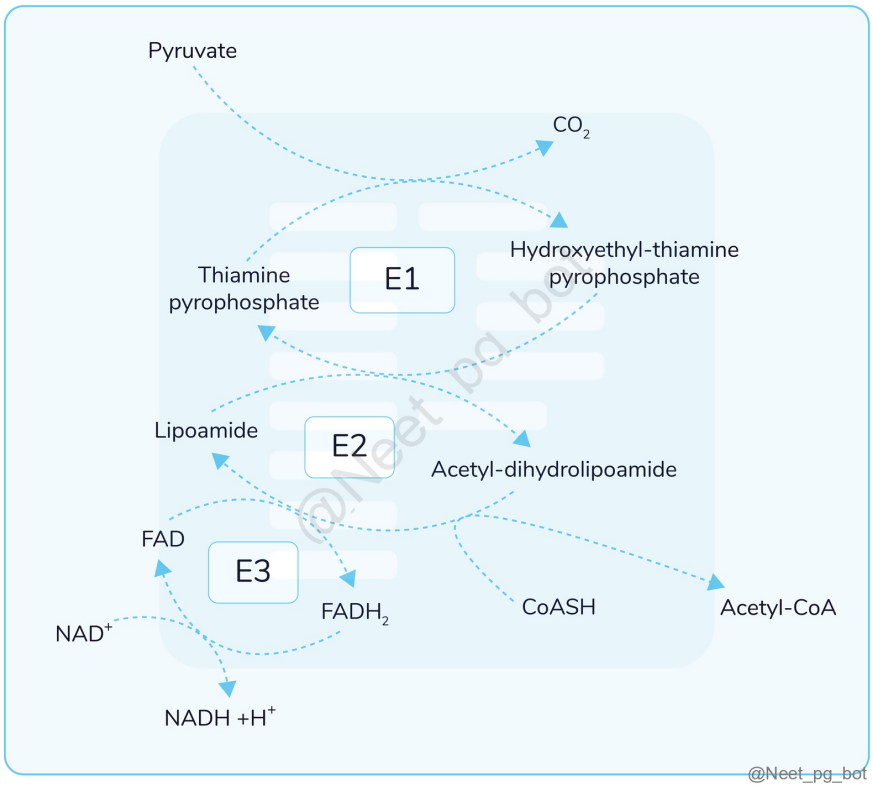
Q4: In the diagram provided, which enzymes are identified as E1 and E2, responsible for the conversion of pyruvate to acetyl CoA? (Q856000)

A. Pyruvate dehydrogenase and dihydrolipoyl transacetylase

B. Pyruvate carboxylase and dihydrolipoyl transacetylase

C. Pyruvate dehydrogenase and dihydrolipoyl dehydrogenase

D. Pyruvate carboxylase and dihydrolipoyl dehydrogenase



Correct Answer: A. Pyruvate dehydrogenase and dihydrolipoyl transacetylase

Explanation: The pyruvate dehydrogenase complex is responsible for the irreversible conversion of pyruvate to acetyl-CoA, a crucial step in aerobic glucose metabolism. A deficiency in this complex leads to the accumulation of pyruvate, which is then shunted towards lactate production, resulting in lactic acidosis. Enzyme Label Full Name Cofactor Function E1 Pyruvate dehydrogenase (decarboxylase) Thiamine pyrophosphate (TPP) Catalyzes oxidative decarboxylation of pyruvate to form hydroxyethyl–TPP. E2 Dihydrolipoyl transacetylase Lipoic acid Transfers the acetyl group from hydroxyethyl–TPP to CoA, forming acetyl-CoA . E3 Dihydrolipoyl dehydrogenase FAD and NAD■ Regenerates oxidized lipoamide by transferring electrons to NAD■ → NADH + H■.

References:

Harper's Illustrated Biochemistry, Ed 32, Pg 168-170.

↑ *Back to this question in Part A*

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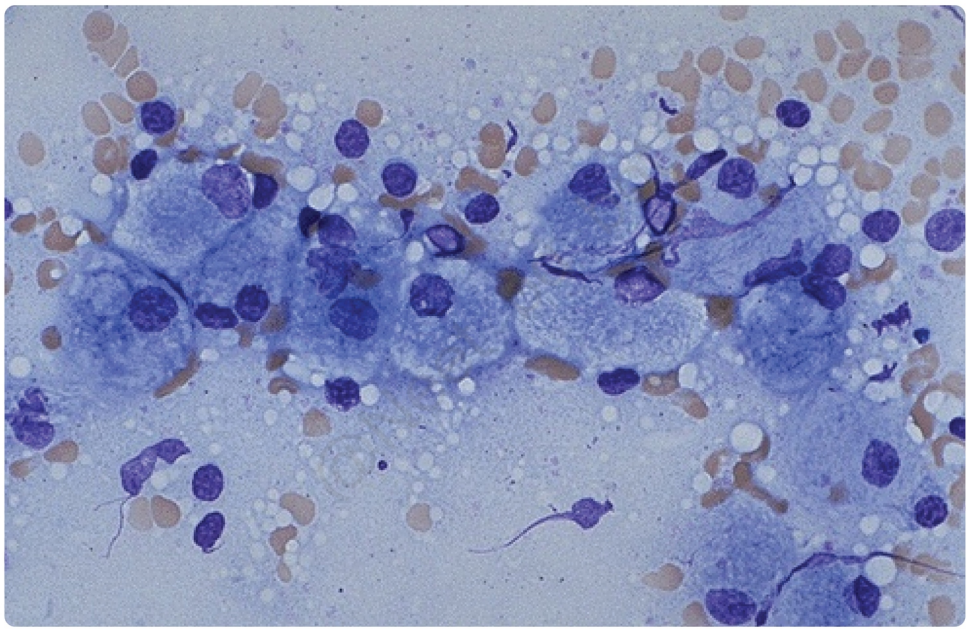
Q5: A 5 year old boy presented with easy fatigability, irritability and inability to concentrate. Labs revealed the following findings a and b (light and oil immersion respectively) on the bone marrow aspiration. Which of the following is the most likely enzyme deficient in this condition? (Q856005)

A. Hexosaminidase

B. Glucocerebrosidase

C. Sphingomyelinase

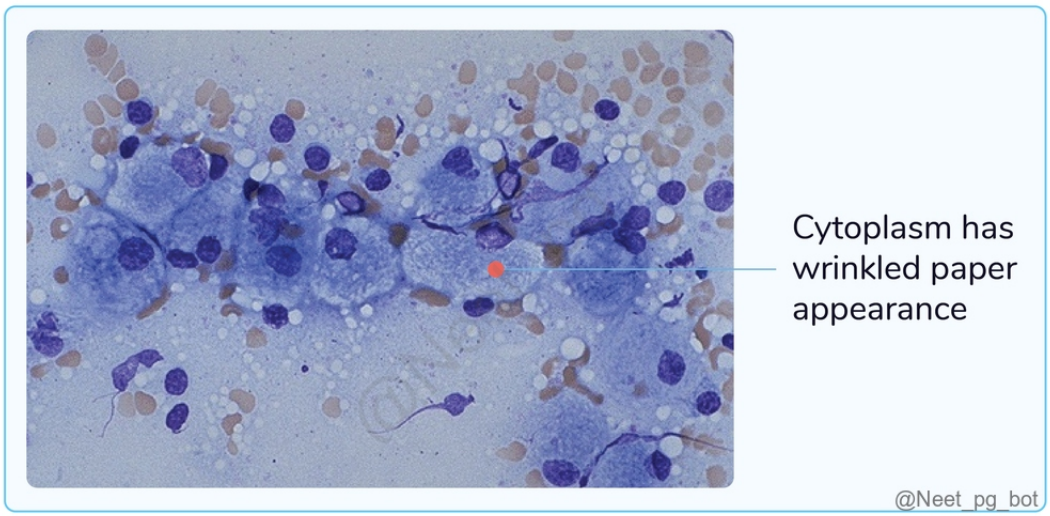
D. N-acetyglucosaminidase



Correct Answer: B. Glucocerebrosidase

Explanation: The Clinical scenario of easy fatiguability, irritability and inability to concentrate along with the microscopic findings of crumpled tissue appearance of macrophages. Gaucher's disease is characterized by the accumulation of glucosylceramide in RBCs and platelet membranes due to a deficiency in the enzyme

Glucocerebrosidase , leading to anemia, hepatosplenomegaly, and thrombocytopenia. Bone marrow biopsy shows macrophages with a characteristic "wrinkled paper" appearance. Gaucher's disease: Lysosomal storage disease Defective enzyme: Glucocerebrosidase Defect in metabolism: Accumulation of cerebroside (Glucosylceramide) in RBCs and platelet membranes. (Option B) Pathophysiology: Abnormal RBCs and platelets are engulfed by macrophages leading to accumulation inside macrophages causing wrinkled paper appearance. {{caption_text}} Disease Enzyme deficiency Substance accumulation Features Tay-Sachs disease Hexosaminidase A (Option A ruled out) Ganglioside Mental retardation Blindness Muscular weakness Cherry red spots in the macula Niemann-Pick disease Sphingomyelinase (Option C ruled out) Sphingomyelin Enlarged liver and spleen Mental retardation Fatal in early life Sanfilippo Syndrome N-acetyglucosaminidase (Option D ruled out)



References:

https://www.ncbi.nlm.nih.gov/books/NBK448080/

Harper’s illustrated Biochemistry 32e pg 245,

Vasudevan textbook of Biochemistry 7e pg 194

Image: https://www.tau.ac.il/medicine/tau-only/webpath/hemehtml/heme057.htm

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: Which enzyme is shared by both glycolysis and gluconeogenesis? (Q856507)

A. Pyruvate kinase

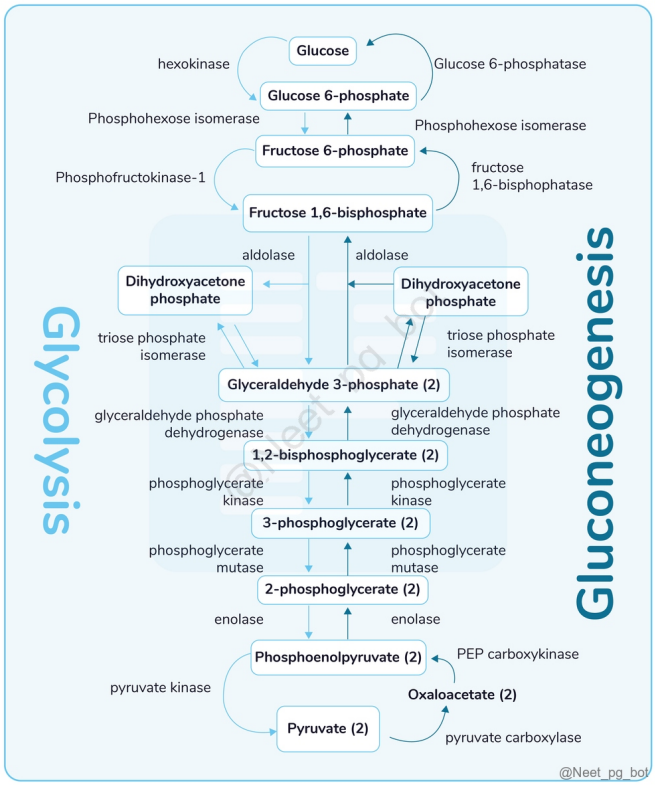
B. Phosphofructokinase

C. Phosphoenolpyruvate carboxy kinase

D. Phosphoglycerate kinase

Correct Answer: D. Phosphoglycerate kinase

Explanation: Phosphoglycerate kinase is involved in a reversible reaction in both glycolysis and gluconeogenesis: Glycolysis : Converts 1,3-bisphosphoglycerate to 3-phosphoglycerate, generating ATP. Gluconeogenesis : Reverses the above reaction, utilizing ATP to convert 3-phosphoglycerate to 1,3-bisphosphoglycerate. The other enzymes listed are specific to one pathway: Pyruvate kinase (Option A): Specific to glycolysis (irreversible). Phosphofructokinase (Option B): Specific to glycolysis (irreversible). Phosphoenolpyruvate carboxykinase (Option C): Specific to gluconeogenesis.



References:

PGK

↑ *Back to this question in Part A*

@Neet_pg_bot

Q7: Ketosis is common in type 1 DM due to: - Decreased beta-oxidation of fatty acids Increased mobilization of fat from the adipose cell Decreased acetyl CoA in TCA cycle Increase beta-hydroxy butyryl synthase (Q856515)

A. 2,3 and 4

B. 1,2,3 and 4

C. 4 only

D. 1 and 2 only

Correct Answer: A. 2,3 and 4

Explanation: Ketosis in Type 1 Diabetes Mellitus (T1DM) occurs due to the following mechanisms: Increased mobilization of fat from adipose tissue (statement 2): Lack of insulin leads to unregulated lipolysis, releasing free fatty acids (FFAs) into the bloodstream. Decreased acetyl-CoA utilization in the TCA cycle (statement 3): Due to the depletion of oxaloacetate (used for gluconeogenesis in a hyperglycemic state), acetyl-CoA from beta-oxidation cannot enter the TCA cycle efficiently and is diverted toward ketone body synthesis. Increased activity of beta-hydroxybutyryl-CoA synthase (statement 4): This enzyme catalyzes the synthesis of ketone bodies (e.g., beta-hydroxybutyrate), contributing to ketosis. Decreased beta-oxidation of fatty acids (Statement 1) because beta-oxidation is actually increased in T1DM due to elevated fatty acid mobilization and lack of insulin-mediated inhibition.

References:

DKA

↑ *Back to this question in Part A*

@Neet_pg_bot

Q8: Which of the following poisoning agent does not inhibit cytochrome oxidase? (Q856558)

A. Hydrogen sulfide

B. Carbon monoxide

C. Methane

D. Cyanide

Correct Answer: C. Methane

Explanation: Cytochrome c oxidase is complex 4 in the electron transport chain. It is inhibited by the following: Carbon monoxide Cyanide Hydrogen sulfide Thus, cytochrome oxidase is not inhibited by methane.

References:

Harper’s Illustrated Biochemistry, 32nd edition, pg: 126

↑ *Back to this question in Part A*

@Neet_pg_bot

Q9: Which one of the options provided does not serve as a substrate for the synthesis of glucose? (Q856613)

A. Acetyl coenzyme A

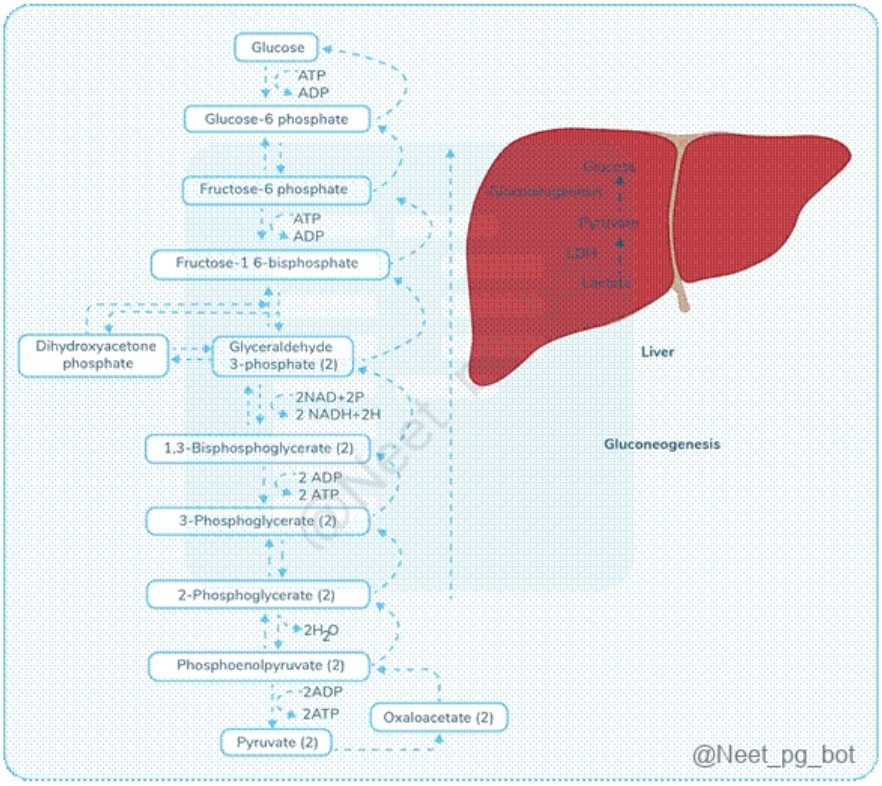
B. Glycerol

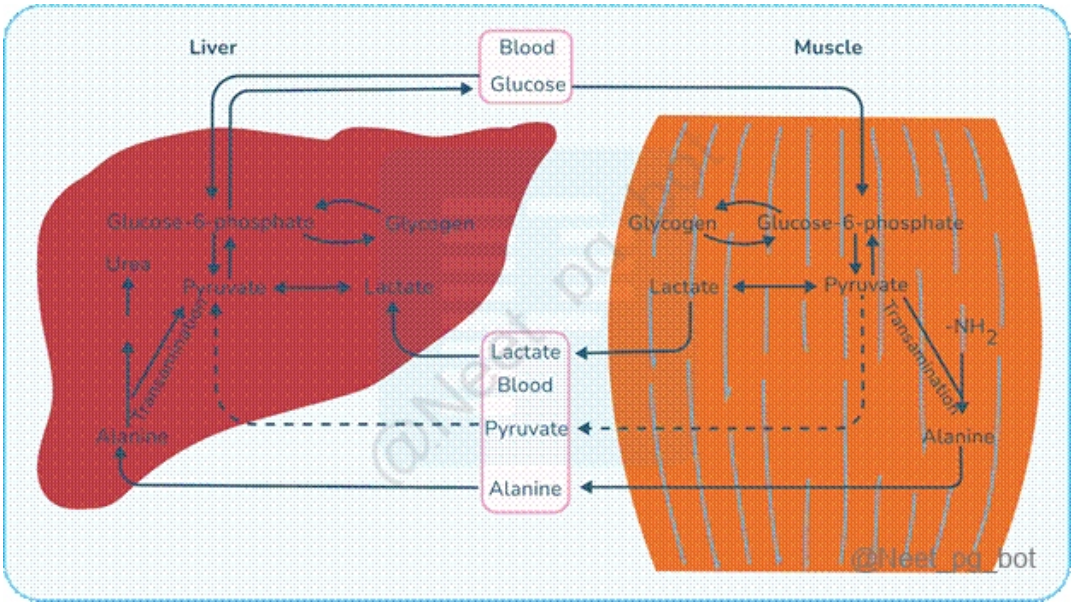
C. Alanine

D. Lactate

Correct Answer: A. Acetyl coenzyme A

Explanation: Acetyl-CoA is NOT a substrate for gluconeogenesis. Although acetyl-CoA is an important metabolic intermediate, it cannot be converted to glucose because the reaction catalysed by pyruvate dehydrogenase is irreversible. Substrates for gluconeogenesis: Lactate (Option D): Produced during anaerobic glycolysis in skeletal muscles and can be converted back to glucose in the liver through the Cori cycle. Glycerol (Option B) is released from the breakdown of triglycerides in adipose tissue and can enter the gluconeogenesis pathway. Alanine (Option C) and Glutamine: Released from the breakdown of proteins, primarily in muscle tissue. Alanine is converted to pyruvate, which enters the gluconeogenesis pathway as part of the glucose-alanine (Cahill) cycle. Propionate : Obtained from the metabolism of odd-chain fatty acids. Cori’s cycle/ Glucose Lactate cycle and Cahill cycle/Glucose Alanine cycle





References:

Harper's Biochemistry, 32nd edition, Pg 142, 186, 187

↑ *Back to this question in Part A*

@Neet_pg_bot

Q10: Glucose is stored in the form of glycogen because (Q856876)

A. It is compact

B. It has multiple reducing ends

C. It can be stored at multiple organs

D. It can provide glucose for about 1 week when needed

Correct Answer: A. It is compact

Explanation: Glycogen is a polysaccharide composed of many glucose molecules linked together through alpha-1,4-glycosidic bonds and branched with alpha-1,6-glycosidic bonds. By storing glucose as glycogen, the body can efficiently pack a large amount of glucose molecules into a compact and readily available form. This compact storage allows for efficient storage of glucose within cells, especially in tissues such as the liver and muscles, where glycogen serves as a reserve energy source. It has multiple reducing ends (Option B): While it is true that glycogen has multiple reducing ends due to its branched structure, this is not the primary reason why glucose is stored as glycogen. It can be stored at multiple organs (Option C): Although glycogen can be stored in multiple organs, such as the liver and muscles, this is not the main reason why glucose is stored as glycogen. It can provide glucose for about 1 week when needed (Option D): The storage capacity of glycogen varies depending on the individual and their glycogen reserves.

References:

Glycogen

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: Which one of the following choices does not represent a function of glucagon? (Q856992)

A. Glycolysis

B. Gluconeogenesis

C. Glycogenolysis

D. Ketogenesis

Correct Answer: A. Glycolysis

Explanation: Glucagon is a hormone produced by the alpha cells of the pancreas that primarily increases blood glucose levels, especially during fasting or low glucose states. It achieves this by stimulating processes that

increase glucose availability and energy production. Glycolysis is the metabolic pathway that breaks down glucose to produce energy (ATP). Glucagon inhibits glycolysis in the liver to conserve glucose and promote the release of glucose into the bloodstream. Instead of promoting glucose breakdown, glucagon promotes glucose production and release. Gluconeogenesis (Option B): Stimulates the production of glucose from non-carbohydrate sources (amino acids, lactate) in the liver. Glycogenolysis (Option C): Stimulates the breakdown of glycogen into glucose in the liver to release glucose into the bloodstream. Ketogenesis (Option D): Stimulates the production of ketone bodies in the liver from fatty acids during periods of prolonged fasting or carbohydrate restriction.

References:

Glucagon

↑ *Back to this question in Part A*

@Neet_pg_bot

Q12: Which enzyme is responsible for the deficiency causing classical galactosemia? (Q856998)

A. Aldose reductase

B. Galactokinase

C. Galactose-1-phosphate uridyl transferase

D. UDP galactose-4-epimerase

Correct Answer: C. Galactose-1-phosphate uridyl transferase

Explanation: Classical galactosemia is a genetic metabolic disorder caused by a deficiency of the enzyme galactose-1-phosphate uridyl transferase (GALT). This enzyme is essential for the proper metabolism of galactose, a sugar found in milk and other dairy products. Role of GALT in Galactose Metabolism: GALT converts galactose-1-phosphate to UDP-galactose, which is required for various biochemical processes, including the synthesis of glycoproteins and glycolipids. A deficiency in GALT leads to the accumulation of galactose-1-phosphate and galactose in tissues, which can cause toxicity and damage to the liver, kidneys, and brain, leading to the symptoms of classical galactosemia. Clinical Features of Classical Galactosemia: Failure to thrive and jaundice in the neonatal period after feeding with milk. Liver damage, cataracts, intellectual disability, and sepsis from E. coli infections (due to impaired immune function). Diagnosis: Elevated galactose-1-phosphate in red blood cells. Genetic testing for mutations in the GALT gene. Treatment: Lactose-free diet (avoiding galactose) to prevent the accumulation of galactose and its metabolites. Aldose reductase (Option A): Involved in the reduction of galactose to galactitol, which is implicated in galactose toxicity but is not the enzyme deficient in classical galactosemia. Galactokinase (Option B): Deficiency causes galactokinase deficiency or non-classical galactosemia, which leads to cataract formation but does not cause the systemic effects seen in classical galactosemia (liver damage, intellectual disability). UDP-galactose-4-epimerase (Option D): Deficiency causes epimerase-deficient galactosemia, a less common form of galactosemia with milder symptoms than classical galactosemia.

References:

Galactosemia

↑ *Back to this question in Part A*

@Neet_pg_bot

Q13: A patient with thiamine deficiency develops lactic acidosis. Which of the following enzymes is affected? (Q855957)

A. Pyruvate carboxylase

B. Pyruvate dehydrogenase

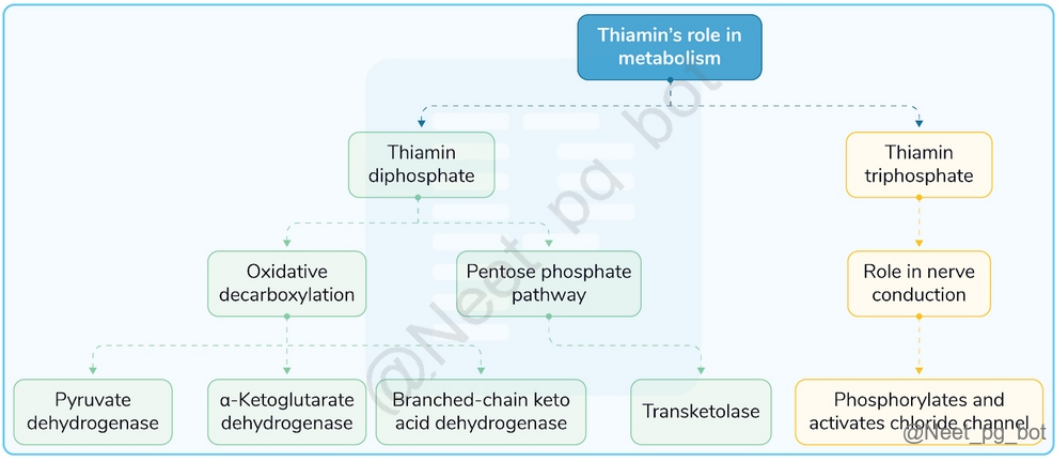
C. Phosphoenolpyruvate carboxykinase

D. Transketolase

Correct Answer: B. Pyruvate dehydrogenase

Explanation: Thiamine (vitamin B1) is an essential coenzyme involved in several metabolic pathways, including the conversion of pyruvate to acetyl-CoA through the pyruvate dehydrogenase complex (PDC). When deficient, the activity of the pyruvate dehydrogenase complex is impaired. As a result, there is a buildup of pyruvate. This pyruvate is then diverted to other metabolic pathways, like the conversion to lactic acid (lactate), resulting in lactic acidosis. Thiamine deficiency : The Aleurone layer of cereals (food grains) is a rich source of thiamine. Therefore, whole wheat flour and unpolished hand-pound rice have better nutritive value than completely polished refined foods. When the grains are polished, the aleurone layer is usually removed. Biochemical parameters: Thiamine deficiency affects the enzyme transketolase, which is involved in the pentose phosphate pathway. Measuring transketolase activity in red blood cells is a specific test for thiamine deficiency and can confirm the diagnosis. Erythrocyte transketolase activity is reduced; this is the earliest manifestation seen even before clinical disturbances. Conditions Biochemical Role of Thiamine Deficiency Manifestations Dry Beriberi Essential for carbohydrate metabolism and nerve function Peripheral neuritis, sensory disturbances, difficulty walking, and paralysis due to CNS manifestations. Wet Beriberi Thiamine is necessary for the PDH complex and low activity causes Lactic acidosis. Lactic acidosis causes vasodilatation and hence cardiovascular manifestations. Cardiovascular manifestations such as oedema, palpitations, breathlessness, and heart failure. Maple Syrup Urine Disease Cofactor for decarboxylation of branched-chain keto acids Accumulation of branched-chain keto acids leads to convulsions, severe mental retardation, vomiting, acidosis, coma, and death. Lactic Acidosis Cofactor for pyruvate dehydrogenase, converting pyruvate to acetyl-CoA Impaired conversion leads to increased lactate and pyruvate levels, causing lactic acidosis. Pyruvate carboxylase (Option A) is not directly involved in the conversion of pyruvate to acetyl-CoA. Pyruvate carboxylase is responsible for the conversion of pyruvate to oxaloacetate in an anaplerotic reaction that replenishes TCA cycle intermediates. It is not directly affected by thiamine deficiency in the context of lactic acidosis. Phosphoenolpyruvate carboxykinase (Option C) is responsible for the conversion of oxaloacetate to phosphoenolpyruvate (PEP), an important step in gluconeogenesis. It is not directly involved in the conversion of pyruvate to acetyl-CoA and is not typically affected by thiamine deficiency. Transketolase (Option D) is involved in the non-oxidative phase of the pentose phosphate pathway (PPP), which generates

ribose-5-phosphate and other sugars. Transketolase requires thiamine pyrophosphate as a cofactor.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 541, 542

↑ *Back to this question in Part A*

@Neet_pg_bot

Q14: A 2-month-old infant is brought to the pediatrician due to feeding difficulties, vomiting, and failure to thrive. The child's history reveals that the infant was born full-term and had a normal birth weight. On physical examination, the infant appears jaundiced, and the liver is palpable below the costal margin. Oil drop Cataracts are also noted on eye examination. Laboratory tests show elevated levels of galactose in the blood and urine. Based on these clinical findings, which enzyme deficiency is most likely responsible for this presentation? (Q857857)

A. G6PD (Glucose-6-phosphate dehydrogenase)

B. GALT (Galactose-1-phosphate uridylyltransferase)

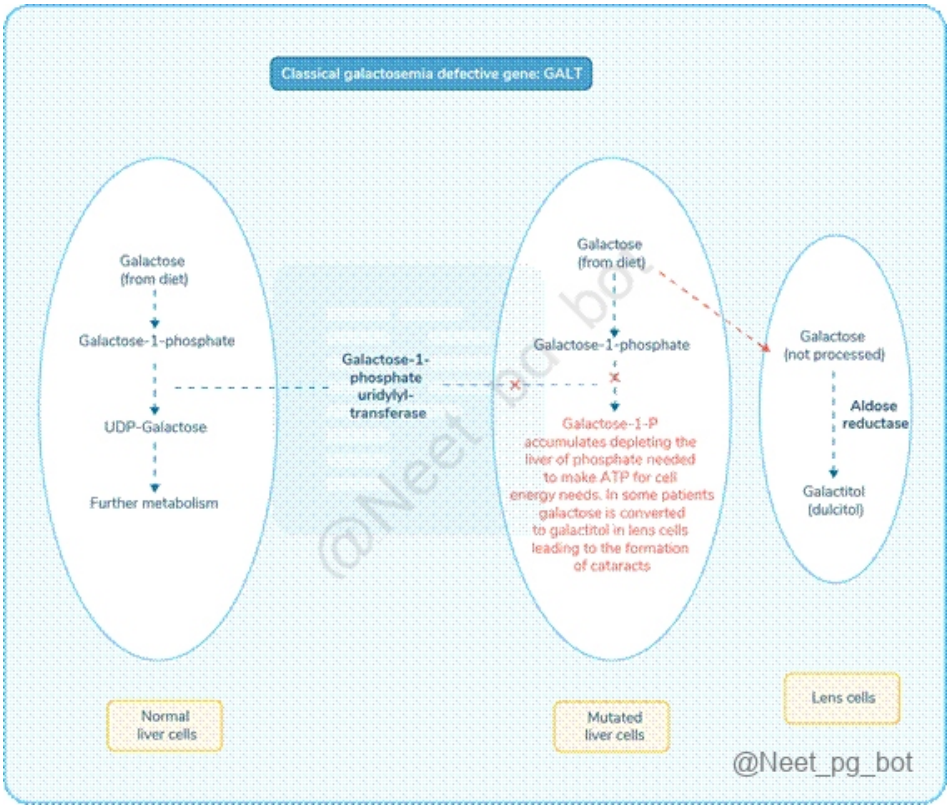
C. ALDH (Aldehyde dehydrogenase)

D. PKU (Phenylalanine hydroxylase)

Correct Answer: B. GALT (Galactose-1-phosphate uridylyltransferase)

Explanation: GALT deficiency, also known as galactosemia, is a genetic disorder that impairs the ability to metabolize galactose properly. Galactose is a sugar found in milk and other dairy products. Galactose-1-phosphate Uridyltransferase (GALT) Deficiency Severely deficient in individuals with Classic Galactosemia in which

galactose-1-phosphate and, therefore, galactose accumulate. (Option A) It Is an inborn error of metabolism Accumulation Accumulated galactose is shunted into side pathways like galactitol production. Aldose reductase catalyzes it. Galactose-1 phosphate accumulates in the liver, inhibiting galactokinase and glycogen phosphorylase, leading to hypoglycemia. Bilirubin Reduced bilirubin uptake and conjugation, increasing unconjugated bilirubin in blood Symptoms Hypoglycemia, Hypophosphatemia, Hyperuricemia, Lactic acidosis Hepatomegaly, Jaundice, Severe mental retardation Galactosuria (galactose excreted in urine) Oil drop Cataracts from dulcitol accumulation in the lens Tubular damage and generalized aminoaciduria Diagnosis Clinical manifestations (e.g., congenital cataract), presence of galactose in urine and elevated blood galactose levels Prenatal diagnosis via amniocentesis Detection of heterozygous parents by elevated galactose levels after a galactose load Complications in untreated cases E. coli sepsis, Fanconi syndrome Feeding problems, Failure to thrive, Hepatocellular damage, Bleeding Treatment Withdrawal of galactose and lactose from the diet. GALT deficiency is a part of the newborn screening panel G6PD (Glucose-6-phosphate dehydrogenase) deficiency (Option A) is a genetic disorder that leads to a deficiency of the enzyme glucose-6-phosphate dehydrogenase, which is important for protecting red blood cells from oxidative damage. ALDH (Aldehyde dehydrogenase) deficiency (Option C) is associated with a condition called alcohol-induced flushing, where individuals who carry certain genetic variants of the aldehyde dehydrogenase enzyme experience facial flushing and other symptoms when they consume alcohol. PKU (Phenylalanine hydroxylase) deficiency (Option D) is a genetic disorder characterized by the deficiency of the enzyme phenylalanine hydroxylase, which is responsible for converting phenylalanine to tyrosine.



References:

Lippincott Edition 7 page no:430, Chapter 12. II. FRUCTOSE METABOLISM

Vasudevan Edition 7 page no:139

↑ *Back to this question in Part A*

Q15: A newborn infant is brought to the clinic with jaundice and a white, punctate, and scattered opacity in the central lens. The condition is consistent with Oil drop cataract. Which of the following metabolic disorders is most likely responsible for this presentation? (Q857861)

A. Phenylketonuria (PKU)

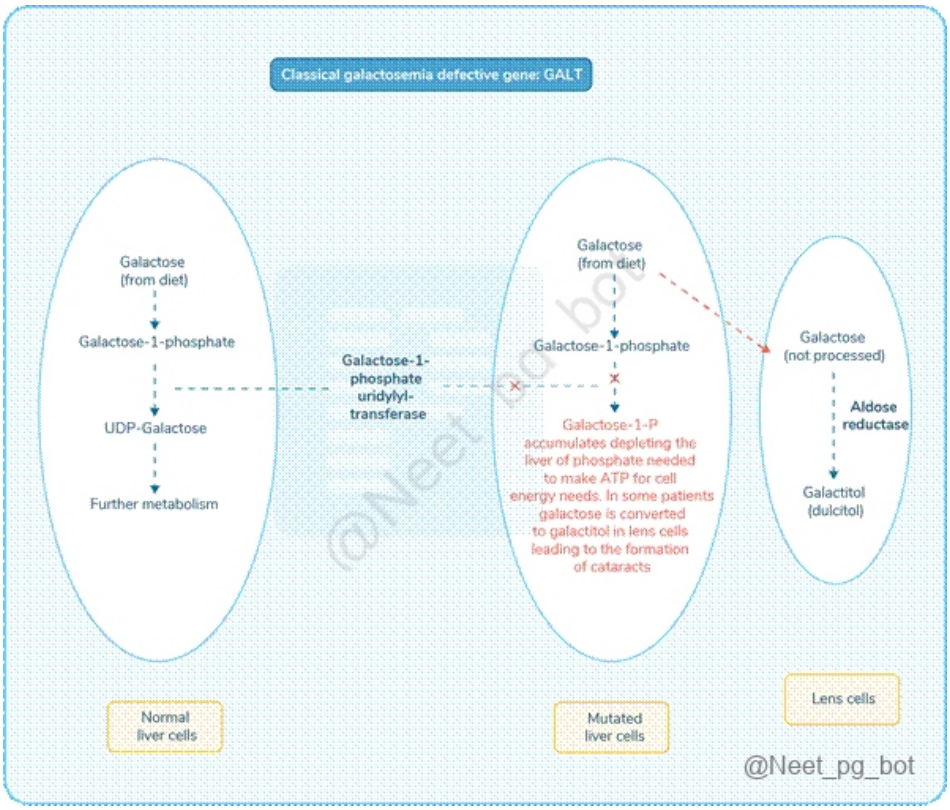
B. Maple syrup urine disease

C. Galactosemia

D. Wilson's disease

Correct Answer: C. Galactosemia

Explanation: The most likely metabolic disorder responsible for the presentation of oil drop cataract in a newborn infant with jaundice and lens opacities is Galactosemia. GALT deficiency, also known as galactosemia, is a genetic disorder that impairs the ability to metabolize galactose properly. Galactose is a sugar found in milk and other dairy products. Galactose-1-phosphate Uridyltransferase (GALT) Deficiency Severely deficient in individuals with Classic Galactosemia in which galactose-1-phosphate and, therefore, galactose accumulate. (Option A) It Is an inborn error of metabolism Accumulation Accumulated galactose is shunted into side pathways like galactitol production. Aldose reductase catalyzes it. Galactose-1 phosphate accumulates in the liver, inhibiting galactokinase and glycogen phosphorylase, leading to hypoglycemia. Bilirubin Reduced bilirubin uptake and conjugation, increasing unconjugated bilirubin in blood Symptoms Hypoglycemia, Hypophosphatemia, Hyperuricemia, Lactic acidosis Hepatomegaly, Jaundice, Severe mental retardation Galactosuria (galactose excreted in urine) Oil drop Cataracts from dulcitol accumulation in the lens, reversible cataract Tubular damage and generalized aminoaciduria Diagnosis Clinical manifestations (e.g., congenital cataract), presence of galactose in urine and elevated blood galactose levels Prenatal diagnosis via amniocentesis Detection of heterozygous parents by elevated galactose levels after a galactose load Complications in untreated cases E. coli sepsis, Fanconi syndrome Feeding problems, Failure to thrive, Hepatocellular damage, Bleeding Treatment Withdrawal of galactose and lactose from the diet. GALT deficiency is a part of the newborn screening panel Phenylketonuria (PKU) (Option A) is a metabolic disorder caused by a deficiency of the enzyme phenylalanine hydroxylase. This leads to the buildup of phenylalanine in the body. It can lead to intellectual disability, developmental delays, and other neurological problems if left untreated. Maple syrup urine disease (Option B) is a metabolic disorder that affects the breakdown of branched-chain amino acids (leucine, isoleucine, and valine). It can cause neurological symptoms and a characteristic sweet odor to the urine. Wilson's disease (Option D) is a disorder of copper metabolism, leading to copper accumulation primarily in the liver and brain. It can cause various neurological and hepatic symptoms.



References:

Lippincott Edition 7 page no:430, Chapter 12. II. FRUCTOSE METABOLISM

Vasudevan Edition 7 page no:139

↑ *Back to this question in Part A*

@Neet_pg_bot

Q16: A patient with a history of strenuous exercise forgot to eat a meal and later consumed alcohol at a party. Which of the following metabolic processes is inhibited by alcohol consumption? (Q860060)

A. Gluconeogenesis

B. Glycogenolysis

C. HMP shunt

D. Ketone body formation

Correct Answer: A. Gluconeogenesis

Explanation: Alcohol consumption inhibits gluconeogenesis. Alcohol metabolism increases the NADH/NAD+ ratio in the liver, which inhibits key enzymes involved in gluconeogenesis, leading to hypoglycemia, especially in a fasting state after strenuous exercise. Alcohol produces a high NADH/NAD+ ratio in the cytoplasm, the high NADH level favours the conversion of pyruvate to lactate, leading to lactic acidosis. Deficiency of pyruvate leads to inadequate formation of oxaloacetate which blocks gluconeogenesis This results in low blood sugar levels. Symptoms of alcohol-induced hypoglycemia include confusion, irritability, and, in severe cases, loss of consciousness. Glycogenolysis (Option B): Glycogenolysis is generally stimulated by alcohol consumption due to the liver's attempt to maintain glucose levels. While alcohol inhibits gluconeogenesis, it does not directly inhibit glycogenolysis. HMP shunt (Option C): The hexose monophosphate (HMP) shunt is primarily involved in the production of NADPH and ribose-5-phosphate. Alcohol does not directly inhibit this pathway. Ketone body formation (Option D): Ketone body formation can actually be promoted in alcohol consumption as the body shifts to using fatty acids for energy when glucose is unavailable.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg.542

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg.140, 478

https://pubmed.ncbi.nlm.nih.gov/12444383/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: Which of the following is the rate-limiting enzyme of the hexose monophosphate (HMP) pathway? (Q860387)

A. Glucose-6-phosphatase

B. Glucose-6-phosphate dehydrogenase

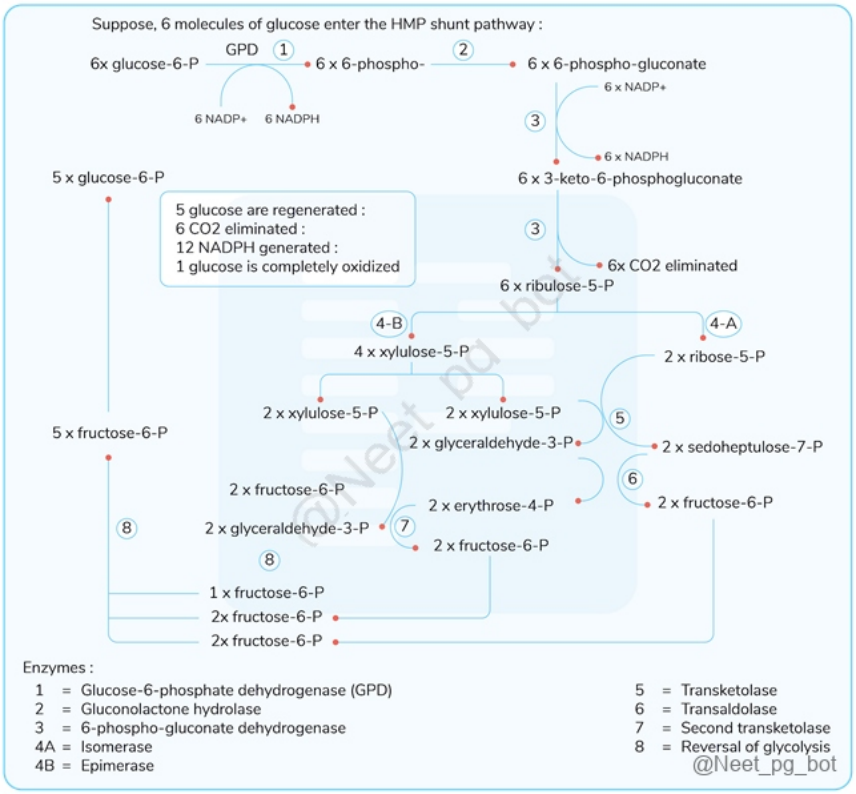
C. Phosphofructokinase-1

D. Glycogen phosphorylase

Correct Answer: B. Glucose-6-phosphate dehydrogenase

Explanation: Glucose-6-phosphate dehydrogenase is the rate-limiting step of the HMP pathway. It catalyzes the oxidation of glucose-6-phosphate to 6-phosphoglucono-δ-lactone, generating NADPH, which is critical for reductive biosynthesis and maintaining cellular redox balance. The pentose phosphate pathway (PPP) is essential in erythrocytes because it generates NADPH. NADPH is crucial for maintaining glutathione in its reduced form, which is necessary for the activity of glutathione peroxidase. This enzyme removes harmful hydrogen peroxide (H■O■), protecting red blood cells from oxidative damage and preventing hemolysis. Pentose Phosphate Pathway (PPP) The Pentose Phosphate Pathway (PPP), also known as the hexose monophosphate shunt, is an alternative metabolic pathway for glucose that doesn't generate ATP. Location: Cytosol It serves two primary functions: NADPH Production The PPP is a major source of NADPH, vital for biosynthesis (fatty acids and steroids).

NADPH Production: RBCs rely on glycolysis for ATP due to the lack of mitochondria and depend solely on the PPP for NADPH, as they lack mitochondrial dehydrogenases. Glutathione Maintenance: NADPH keeps glutathione reduced, enabling glutathione peroxidase to neutralize hydrogen peroxide and prevent oxidative damage. Impact of NADPH Deficiency : Low NADPH reduces glutathione, impairs glutathione peroxidase, increases oxidative damage , and leads to hemolysis . Ribose-5-Phosphate (R-5-P) Synthesis Ribose-5-phosphate is essential for nucleotide synthesis needed for RNA and DNA. Homeostasis between NADPH and R-5-P production. When NADPH demand exceeds R-5-P's need, the non-oxidative phase converts R-5-P to glycolytic intermediates (fructose-6-phosphate and glyceraldehyde-3-phosphate). If the demand for R-5-P is higher than for NADPH, the non-oxidative phase generates R-5-P from fructose-6-phosphate and glyceraldehyde-3-phosphate, bypassing the oxidative steps. Differences from Glycolysis Uses NADP+ instead of NAD+ and generates CO2, not ATP. Glucose-6-phosphatase (Option A): This enzyme catalyzes the conversion of glucose-6-phosphate to glucose during gluconeogenesis and glycogenolysis. It is not involved in the HMP pathway. Phosphofructokinase-1 (Option C): This enzyme is the rate-limiting enzyme of glycolysis , not the HMP pathway. Glycogen phosphorylase (Option D): This enzyme catalyzes the breakdown of glycogen to glucose-1-phosphate in glycogenolysis and is not related to the HMP pathway.



References:

Harper’s biochemistry 32ed Pg 191;194-196

↑ *Back to this question in Part A*

@Neet_pg_bot

Q18: What is the rate-limiting enzyme of cholesterol synthesis? (Q860403)

A. HMG-CoA reductase

B. 7-alpha hydroxylase

C. Carnitine acyltransferase 1

D. Carnitine acyltransferase 2

Correct Answer: A. HMG-CoA reductase

Explanation: The principal regulatory step in the cholesterol synthesis pathway: HMG-CoA is reduced to mevalonate It is catalyzed by HMG-CoA reductase Regulation of HMG-CoA reductase: Regulation Method Mechanism Sufficient Cholesterol Suppresses transcription of HMG CoA reductase gene Low Dietary Cholesterol Increases transcription of HMG CoA reductase gene AMP-activated Kinase Phosphorylates HMG CoA reductase inactivating it Protein Phosphatase 1 Dephosphorylates HMG CoA reductase activating it Insulin and Thyroxine Increase activity of HMG CoA reductase Cortisol and Glucagon Decrease activity of HMG CoA reductase Statins (e.g., Lovastatin) Competitive inhibition of HMG CoA reductase 7-alpha hydroxylase (Option B): This enzyme is the rate-limiting step in bile acid synthesis, not cholesterol synthesis. Carnitine acyltransferase 1 (Option C): This enzyme is the rate-limiting enzyme in fatty acid oxidation, facilitating the transport of long-chain fatty acids into the mitochondria. Carnitine acyltransferase 2 (Option D): This enzyme is involved in fatty acid oxidation but is not the rate-limiting step. It helps regenerate free carnitine in the mitochondrial matrix.

References:

Harper 32nd edition, page 260

Vasudevan 7th edition, pages 171,173

↑ *Back to this question in Part A*

@Neet_pg_bot

Q19: Which receptor plays a crucial role in glucose uptake by skeletal muscle? (Q860412)

A. GLUT 1

B. GLUT 2

C. GLUT 3

D. GLUT 4

Correct Answer: D. GLUT 4

Explanation: GLUT 4 is the insulin-dependent glucose transporter predominantly found in heart, skeletal muscle and adipose tissue . It translocates to the cell membrane in response to insulin, facilitating glucose uptake during periods of increased energy demand. GLUT proteins are distributed in various tissues according to their specific functions. Transporter Tissue Location Functions GLUT 1 (Option A ruled out) Brain, kidney, colon, placenta, erythrocytes Facilitates glucose uptake GLUT 2 (Option B ruled out) Liver, pancreatic β cells, small intestine, kidney Enables rapid glucose uptake or release GLUT 3 (Option C ruled out) Brain, kidney, placenta Assists in glucose uptake GLUT 4 (Option D) Heart and skeletal muscle, adipose tissue Mediates insulin-stimulated glucose uptake GLUT 5 Small intestine Facilitates fructose absorption SGLT 1 Small intestine and kidney Actively transports glucose against a concentration gradient GLUT 1-5 are facilitative bidirectional transporters, whereas SGLT 1 is a sodium-dependent unidirectional transporter.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Pg. 186,187.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q20: A child presents with fatigue and hepatomegaly. Liver enzymes (ALT, AST) are elevated. Ketosis was significant. Liver biopsy shows excess glycogen accumulation. After feeding, blood glucose levels rise, but there is no rise in glucose after overnight fasting. Which of the following enzyme deficiencies is most likely responsible for this presentation? (Q865410)

A. Glucose-6-phosphatase

B. Liver phosphorylase

C. Muscle phosphorylase

D. Phosphofructokinase

Correct Answer: A. Glucose-6-phosphatase

Explanation: The child's presentation—fatigue, hepatomegaly, elevated liver enzymes, marked ketosis, glycogen accumulation in the liver, fasting hypoglycemia with normal postprandial glucose—strongly points toward a Glucose-6-phosphatase deficiency Von Gierke's Disease (Glycogen Storage Disease Type I) Cause Deficiency of glucose-6-phosphatase enzyme Pathophysiology Inability to convert glucose-6-phosphate to glucose; leads to accumulation of glycogen, lactate, and triglycerides Symptoms Severe hypoglycemia Hepatomegaly and Renomegaly Growth retardation Lactic acidosis Hyperuricemia and Hyperlipidemia Diagnosis Biochemical tests (hypoglycemia, lactic acidosis, hyperuricemia, hyperlipidemia) Genetic testing Management Frequent feedings with slow-release carbohydrates Blood glucose monitoring Medications for associated conditions Liver transplantation (in severe cases) Liver phosphorylase (GSD VI – Hers disease) (Option B) causes mild hypoglycemia and hepatomegaly, but symptoms are less severe. Gluconeogenesis is intact, so fasting

hypoglycemia and ketosis are not as pronounced as in glucose-6-phosphatase deficiency. Muscle phosphorylase (GSD V – McArdle disease) (Option C) affects only skeletal muscle. Presents with exercise intolerance and muscle cramps, not fasting hypoglycemia or hepatomegaly. Phosphofructokinase (GSD VII – Tarui disease) (Option D) involves muscle and red blood cells. Causes muscle pain and hemolysis, but does not affect the liver or cause fasting hypoglycemia. Learning Objectives: Glycogen Storage Disease Enzyme Deficiency Type 0 Glycogen synthase deficiency Type I (von Gierke's Disease) Glucose-6-phosphatase Type II (Pompe's Disease) Acid α-glucosidase (acid maltase) Type III (Cori's Disease) Debranching enzyme (amylo-1,6-glucosidase) Type IV (Andersen's Disease) Branching enzyme Type V (McArdle's Disease) Muscle phosphorylase (myophosphorylase) Type VI (Hers' Disease) Liver phosphorylase Type VII (Tarui's Disease) Phosphofructokinase (muscle type)

References:

Harper’s Illustrated Biochemistry, 23rd Edition, Page 174.

Vasudevan Textbook of Biochemistry, 7th Edition, Page 128,129.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q21: Cancer cells take up excess glucose because? (Q865417)

A. Lactate is produced even in the presence of Oxygen

B. High NADH/NAD ratio

C. High GLUT2

D. Absence of Oxygen

Correct Answer: A. Lactate is produced even in the presence of Oxygen

Explanation: Cancer cells demonstrate the Warburg Effect by favoring the conversion of glucose to lactate even in the presence of adequate oxygen, a process known as aerobic glycolysis. The phenomenon in cancer cells where, even in the presence of oxygen, glucose is primarily converted to lactate through aerobic glycolysis. (Option A) Significance: This allows faster ATP production and provides intermediates for neoplastic cell growth and multiplication. It increases NADPH production, protecting neoplastic cells from oxidative stress. High NADH/NAD ratio (Option B): Although the NADH/NAD■ balance affects cellular metabolism, it is not the primary reason cancer cells take up excess glucose. The Warburg effect drives increased glucose uptake regardless of NADH levels. High GLUT2 (Option C): GLUT2 is a glucose transporter mainly found in liver, pancreatic beta cells, and kidney, not commonly involved in cancer cell glucose uptake. Cancer cells typically overexpress GLUT1 or other transporters to increase glucose intake. Absence of Oxygen (Option D): While low oxygen (hypoxia) can stimulate glucose uptake, many cancer cells continue to produce lactate and consume large amounts of glucose even when oxygen is plentiful, which is the hallmark of aerobic glycolysis, not simply hypoxia-induced metabolism.

References:

Vasudevan’s Textbook of Biochemistry, 7th Edition, Page 114.

Warburg Effect

↑ *Back to this question in Part A*

@Neet_pg_bot

Q22: After a high-carbohydrate meal, weight gain is explained by all except? (Q865420)

A. Malonyl CoA

B. Tricarboxylate transporter

C. Acetyl-CoA carboxylase

D. Mitochondrial carnitine

Correct Answer: D. Mitochondrial carnitine

Explanation: After a high-carbohydrate meal, excess glucose is converted into fatty acids and stored as fat, contributing to weight gain. This process involves: Malonyl CoA: An intermediate in fatty acid synthesis that inhibits fatty acid oxidation. Tricarboxylate transporter: Transports citrate from mitochondria to cytosol, providing acetyl-CoA for fatty acid synthesis. Acetyl-CoA carboxylase: The key enzyme that converts acetyl-CoA to malonyl-CoA, initiating fatty acid synthesis. Mitochondrial carnitine (Option D) transports fatty acids into mitochondria for β-oxidation (fat breakdown). After a high-carb meal, fat oxidation is inhibited, so mitochondrial carnitine doesn't contribute to weight gain.

References:

Mitochondrial Fatty Acid Oxidation in Obesity - PMC

↑ *Back to this question in Part A*

@Neet_pg_bot

Q23: When blood glucose concentration is suddenly increased to 2–3 times the normal level and maintained at that level, what will be the typical pattern of blood glucose change observed over time? (Q866337)

A. Rapid rise followed by sustained high glucose levels

B. Continuous increase in insulin secretion without plateau

C. Rise in glucose followed by a fall below baseline

D. Initial rise and then stabilization slightly above baseline

Correct Answer: C. Rise in glucose followed by a fall below baseline

Explanation: When plasma glucose suddenly increases and stays elevated, insulin secretion occurs in two phases : Phase 1: A rapid burst of preformed insulin causes a quick fall in glucose. Phase 2: A slower, sustained insulin release maintains glucose control. As a result, glucose levels initially rise , then overshoot downward , producing a temporary fall below baseline before normalizing — known as reactive hypoglycemia .

References:

Guyton and Hall Textbook of Medical Physiology, 15th Ed., Ch. 79 — “Insulin, Glucagon, and Diabetes Mellitus.” Harrison’s Principles of Internal Medicine, 22nd Ed., Ch. 417 — “Glucose Homeostasis and Insulin Secretion.”

↑ *Back to this question in Part A*

@Neet_pg_bot

Q24: A breastfed infant presents with lethargy, hepatomegaly, and cataracts. Which of the following enzyme deficiencies is most likely responsible for this presentation? (Q866651)

A. Galactokinase

B. Galactose-1-phosphate uridyltransferase (GALPUT)

C. Aldolase B

D. Fructokinase

Correct Answer: B. Galactose-1-phosphate uridyltransferase (GALPUT)

Explanation: The infant’s lethargy, hepatomegaly, and cataracts after breastfeeding indicate classic galactosemia caused by Galactose-1-phosphate uridyltransferase deficiency, leading to toxic buildup of galactose-1-phosphate and galactitol, resulting in liver damage, cataracts, and neurological issues. Classic Galactosemia Galactose-1-phosphate Uridyltransferase (GALT) Deficiency (Option B) Severely deficient in individuals with Classic Galactosemia in which galactose-1-phosphate and, therefore, galactose accumulate. It Is an inborn error of metabolism Accumulation Accumulated galactose is shunted into side pathways like galactitol production. Aldose reductase catalyzes it. Galactose-1 phosphate accumulates in the liver, inhibiting galactokinase and glycogen phosphorylase, leading to hypoglycemia. Bilirubin Reduced bilirubin uptake and conjugation, increasing unconjugated bilirubin in blood Symptoms Hypoglycemia, Hypophosphatemia, Hyperuricemia, Lactic acidosis

Hepatomegaly, Jaundice, Severe mental retardation Galactosuria (galactose excreted in urine) Cataracts from dulcitol accumulation in the lens Tubular damage and generalized aminoaciduria Diagnosis Clinical manifestations (e.g., congenital cataract), presence of galactose in urine and elevated blood galactose levels Prenatal diagnosis via amniocentesis Detection of heterozygous parents by elevated galactose levels after a galactose load Complications in untreated cases E. coli sepsis, Fanconi syndrome Feeding problems, Failure to thrive, Hepatocellular damage, Bleeding Treatment Withdrawal of galactose and lactose from the diet. GALT deficiency is a part of the newborn screening panel Incorrect Options: Galactokinase (Option A) causes isolated cataracts but milder disease without severe liver involvement. Aldolase B (Option C) causes hereditary fructose intolerance, presenting with hypoglycemia and liver problems after fructose ingestion. Fructokinase (Option D) causes essential/fructosuria, a benign condition without serious symptoms.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q25: Glycogen phosphorylase is activated by: (Q855065)

A. Calcium

B. Glucose-6-phosphate

C. Insulin

D. Protein phosphatase

Correct Answer: A. Calcium

Explanation: Glycogen phosphorylase is activated by calcium in tissues, particularly in muscle cells. Calcium and Muscle Contraction: During muscle contraction, calcium ions are released from the sarcoplasmic reticulum in response to a neural signal. Calcium then binds to calmodulin, a calcium-binding protein, forming the calcium-calmodulin complex. Activation of Phosphorylase Kinase: The calcium-calmodulin complex activates phosphorylase kinase, an enzyme that plays a key role in glycogen breakdown. Activation of Glycogen Phosphorylase: The phosphorylation of glycogen phosphorylase activates it, which in turn stimulates the breakdown of glycogen into glucose-1-phosphate, providing energy for muscle contraction. Cofactor of glycogen phosphorylase is pyridoxal phosphate (PLP) Glucose-6-phosphate (Option B): Inhibits glycogen breakdown; favors glycogen synthesis. Insulin (Option C): Promotes dephosphorylation → inactivates glycogen phosphorylase. Protein phosphatase (Option D): Dephosphorylates and inactivates glycogen phosphorylase.

References:

pubmed.ncbi.nlm.nih.gov/11341967/#:~:text=The%20presence%20of%20Mem-CC,-CC%20(0.3%20nM).

↑ *Back to this question in Part A*

@Neet_pg_bot

Q26: Match the following with the most appropriate feature. 1. Von Gierke's a. Branched structure in liver biopsy 2. Pompe's b. Glycogen deposition in lysosomes 3. McArdle's c. Hypoglycemia with lactic acidosis 4. Cori's d. Exercise intolerance (Q839906)

A. 1c, 2a,3d,4b

B. 1c, 2b,3d,4a

C. 1a, 2c,3d,4b

D. 1d, 2c,3b,4d

Correct Answer: B. 1c, 2b,3d,4a

Explanation: Von Gierke's Disease: Characterized by hypoglycemia, and lactic acidosis due to the deficiency of glucose-6-phosphatase. Pompe's Disease: Characterized by glycogen deposition in lysosomes due to acid alpha-glucosidase deficiency. McArdle's Disease: Causes exercise intolerance due to the lack of muscle glycogen phosphorylase. Cori's Disease: Characterized by the accumulation of branched glycogen due to deficiency of debranching enzyme. Type Name Enzyme defect 1 Von Gierke's Disease Glucose-6-phosphatase 2 Pompe's Disease Acid maltase/ alpha glucosidase 3 Cori's Disease Debranching enzyme 4 Anderson's Disease Branching enzyme 5 McArdle's Disease Muscle Phosphorylase 6 Hers Disease Hepatic phosphorylase 7 Tarui's Disease Phosphofructokinase-1

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q27: Which of the following deficiency is found in congenital lactic acidosis? (Q839919)

A. Transketolase

B. Branched chain ketoacid dehydrogenase

C. Isocitrate dehydrogenase

D. Pyruvate dehydrogenase

Correct Answer: D. Pyruvate dehydrogenase

Explanation: Pyruvate dehydrogenase: Pyruvate dehydrogenase (PDH) is a crucial enzyme that converts pyruvate into acetyl-CoA, linking glycolysis to the citric acid cycle. A deficiency in pyruvate dehydrogenase leads to congenital lactic acidosis. Without functional PDH, pyruvate accumulates and is shunted to lactate production, leading to elevated lactate levels in the blood. Transketolase (Option A): Transketolase is an enzyme of the pentose phosphate pathway, which helps in the production of NADPH and ribose-5-phosphate A deficiency in transketolase is not associated with congenital lactic acidosis. Branched chain Ketoacid dehydrogenase (Option B): This enzyme is involved in the catabolism of branched-chain amino acids (leucine, isoleucine, and valine). A deficiency in branched-chain ketoacid dehydrogenase leads to Maple Syrup Urine Disease (MSUD), not congenital lactic acidosis. MSUD is characterized by the accumulation of branched-chain amino acids and their corresponding keto acids. Isocitrate dehydrogenase (Option C): Isocitrate dehydrogenase is an enzyme in the citric acid cycle that helps in the conversion of isocitrate to α-ketoglutarate, producing NADPH or NADH in the process While important in the citric acid cycle, a deficiency in isocitrate dehydrogenase is not typically associated with congenital lactic acidosis

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q28: A patient’s X-ray shows cardiomegaly, and they present with a "floppy baby" syndrome, hypotonia, macroglossia, and hepatomegaly. What is the most likely diagnosis? (Q844238)

A. Pompe disease

B. Ebstein anomaly

C. Down syndrome

D. Cori's Disease

Correct Answer: A. Pompe disease

Explanation: Correct Option A - Pompe disease: Pompe disease (Glycogen storage disease type II) is caused by a deficiency of lysosomal acid α-1,4-glucosidase (acid maltase) , leading to accumulation of glycogen within lysosomes in cardiac and skeletal muscle. This results in: Cardiomegaly (hypertrophic cardiomyopathy) Severe hypotonia (“floppy baby” syndrome) Macroglossia Hepatomegaly Early death from heart failure or respiratory failure in infancy Incorrect Options: B) Ebstein anomaly: Congenital heart defect involving tricuspid valve displacement; presents with cyanosis and murmur, not hypotonia or macroglossia. C) Down syndrome : May cause hypotonia and macroglossia but not cardiomegaly or glycogen accumulation. D) Cori’s disease : (Glycogen storage disease type III) involves debranching enzyme deficiency, causing hepatomegaly and mild hypoglycemia, but no cardiomyopathy or severe hypotonia.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q29: A diabetic patient is advised to exercise. Which of the following glucose transporters is insulin-sensitive? (Q844249)

A. GLUT4

B. GLUT1

C. GLUT3

D. GLUT2

Correct Answer: A. GLUT4

Explanation: Correct Option A - GLUT4: This glucose transporter is insulin-sensitive and is primarily found in adipose tissue and muscle. During exercise, insulin facilitates glucose uptake via GLUT4, helping regulate blood glucose levels. Incorrect Options: Options B, C, and D are Insulin-independent transporters.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q30: A patient with high triglycerides esterified with long-chain fatty acids presents with fatigue, and muscle biopsy shows fat vacuoles. What is the most likely diagnosis? (Q844255)

A. Carnitine deficiency

B. Fatty Acid Synthase defect

C. Lipoprotein Lipase (LPL) deficiency

D. Medium-chain acyl-CoA dehydrogenase (MCAD) deficiency

Correct Answer: A. Carnitine deficiency

Explanation: Correct Option A - Carnitine deficiency: Carnitine deficiency impairs the transport of long-chain fatty acids into the mitochondrial matrix for β-oxidation . As a result: Fatty acids accumulate in muscle cytoplasm (fat vacuoles), ATP production decreases → fatigue, weakness , and exercise intolerance , Hypoketotic hypoglycemia may occur during fasting due to impaired fatty acid oxidation. The condition can be primary (defect in carnitine transporter) or secondary (from liver disease, malnutrition, or valproate use). Incorrect Options: B) Fatty Acid Synthase defect: Affects fatty acid synthesis, not oxidation; would not cause fat accumulation or myopathy. C) Lipoprotein Lipase (LPL) deficiency: Causes hypertriglyceridemia and pancreatitis, not muscle lipid vacuolation. D) Medium-chain acyl-CoA dehydrogenase (MCAD) deficiency: Causes hypoketotic hypoglycemia but does not affect long-chain fatty acid transport ; fat vacuoles are less prominent.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q31: A neonate presents with poor feeding and vomiting, cataract, and acidosis with a positive Benedict's test on urine and negative urinary glucose. Which enzyme is likely defective? (Q844276)

A. Galactose-1-phosphate uridyl transferase

B. Aldolase B

C. Fructokinase

D. Glucose-6-phosphatase

Correct Answer: A. Galactose-1-phosphate uridyl transferase

Explanation: Correct Option A - Galactose-1-phosphate uridyl transferase: This presentation is classic for galactosemia due to deficiency of galactose-1-phosphate uridyl transferase (GALT) — the most common and severe form. Pathophysiology: In normal metabolism: Galactose → Galactose-1-phosphate → Glucose-1-phosphate (via galactokinase and GALT) In GALT deficiency , galactose-1-phosphate accumulates in the liver, kidneys, and brain. Excess galactose is reduced to galactitol (dulcitol) in the lens → cataracts . Accumulation causes hepatic dysfunction , renal tubular damage , and metabolic acidosis . Urine Findings: Benedict’s test positive (detects reducing sugars like galactose, fructose, lactose — not just glucose) Urine glucose test negative (since glucose is absent) Incorrect Options: B) Aldolase B: Defective in hereditary fructose intolerance — presents after fructose ingestion (e.g., fruit, sucrose) with hypoglycemia , vomiting , and hepatomegaly , not cataracts. C) Fructokinase: Causes essential fructosuria , a benign condition with positive Benedict’s test but no symptoms . D) Glucose-6-phosphatase: Defective in Von Gierke’s disease (Glycogen storage disease type I) — presents with hypoglycemia , hepatomegaly , and lactic acidosis , not with cataracts or positive Benedict’s test.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q32: Which of the following is excreted in the urine of patients with pentosuria? (Q852461)

A. L-Xylulose

B. D-Ribose

C. D-Ribulose

D. D-Lactose

Correct Answer: A. L-Xylulose

Explanation: L-Xylulose is excreted in the urine of patients with essential pentosuria , a rare benign metabolic disorder caused by a deficiency of L-xylulose reductase . This leads to the accumulation and urinary excretion of L-xylulose , which can be mistaken for glucose in urine tests, but patients remain asymptomatic with no metabolic consequences. Essential Pentosuria Feature Essential Pentosuria Cause Deficiency of L-xylulose reductase Metabolic Effect Increased excretion of L-xylulose in urine Clinical Symptoms None (asymptomatic) Diagnosis Urine sugar analysis + Chromatography Treatment No treatment needed D-Ribose (Option B) is incorrect because it is a naturally occurring sugar involved in nucleotide metabolism and is not significantly excreted in pentosuria. D-Ribulose (Option C) is incorrect because it is an intermediate in the pentose phosphate pathway but is not associated with pentosuria. D-Lactose (Option D) is incorrect because lactose is a disaccharide and is not involved in pentose metabolism or pentosuria.

References:

Harper’s Biochemistry: Pg 194

Vasudevan’s Textbook of Biochemistry for Medical Students, 7th Edition, Page 131, 316, 317.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q33: A patient with a history of strenuous exercise skipped a meal and later consumed alcohol at a party. Alcohol inhibits which of the following biochemical processes,

potentially leading to hypoglycemia? (Q852549)

A. Gluconeogenesis

B. Glycogenolysis

C. Hexose Monophosphate (HMP) Shunt

D. Ketone Body Formation

Correct Answer: A. Gluconeogenesis

Explanation: Alcohol metabolism increases NADH/NAD+ ratio: Ethanol is metabolized in the liver by alcohol dehydrogenase and acetaldehyde dehydrogenase , leading to excessive NADH production. Inhibition of Gluconeogenesis: High NADH shifts metabolic balance , preventing conversion of lactate to pyruvate and malate to oxaloacetate , both of which are essential for gluconeogenesis. Hypoglycemia Risk: After exercise and fasting, glycogen stores are depleted. The body relies on gluconeogenesis for glucose , but alcohol blocks this pathway , leading to hypoglycemia after drinking. Alcohol-Induced Hypoglycemia Feature Effect of Alcohol Key Mechanism ↑ NADH inhibits gluconeogenesis Primary Effect ↓ Glucose production → Hypoglycemia Clinical Features Weakness, dizziness, confusion, seizures Risk Factors Fasting, exercise, malnutrition, chronic alcoholism Treatment IV dextrose, thiamine in alcoholics Glycogenolysis (Option B): Incorrect because alcohol does not directly inhibit glycogen breakdown. However, after prolonged fasting, glycogen stores are already depleted. Hexose Monophosphate (HMP) Shunt (Option C): Incorrect because this pathway primarily generates NADPH for anabolic processes and does not significantly contribute to blood glucose levels. Ketone Body Formation (Option D): Incorrect because alcohol metabolism can actually promote ketogenesis, especially in chronic alcoholism, rather than inhibiting it.

References:

Harper’s illustrated Biochemistry 32nd edition, pages 142, 162,163,171, 186, 187

↑ *Back to this question in Part A*

@Neet_pg_bot

Q34: During starvation, which of the following hormones is primarily produced to maintain blood glucose levels? (Q852554)

A. Glucagon

B. Insulin

C. Amylase

D. Somatostatin

Correct Answer: A. Glucagon

Explanation: Role of Glucagon in Starvation: During starvation, glucagon is the primary hormone that maintains blood glucose levels. It promotes gluconeogenesis (glucose synthesis) and glycogenolysis (breakdown of glycogen) in the liver. Increases lipolysis in adipose tissue, providing free fatty acids for energy. Phases of Starvation: Early fasting (first few hours): Blood glucose is maintained by glycogenolysis. Prolonged fasting (after 24 hours): Gluconeogenesis becomes the primary glucose source, with glucagon playing a key role. Later starvation (after 3-5 days): Ketogenesis increases as an alternative fuel. Hormonal Changes in Starvation Hormone Effect in Starvation Glucagon Increases to stimulate gluconeogenesis and glycogenolysis. Insulin Decreases to reduce glucose uptake by tissues. Cortisol Increases to promote protein catabolism for gluconeogenesis. Epinephrine Increases to enhance glycogen breakdown and lipolysis. Growth Hormone (GH) Increases to reduce glucose utilization and promote lipolysis. Insulin (Option B): Incorrect because insulin decreases during starvation to limit glucose uptake by tissues. Amylase (Option C): Incorrect because amylase is a digestive enzyme, not a hormone involved in blood glucose regulation. Somatostatin (Option D): Incorrect because somatostatin inhibits both insulin and glucagon but does not directly regulate blood glucose during starvation.

References:

Nelson Textbook of Paediatrics Edition 21, Page 778-781

↑ *Back to this question in Part A*

@Neet_pg_bot

Q35: Which of the following organs or tissues demonstrate insulin-dependent glucose uptake ? (Q854604)

A. RBC

B. Pancreas

C. Adipose tissue

D. Hepatocytes

Correct Answer: C. Adipose tissue

Explanation: Adipose tissue has GLUT 4 transporter which mediates insulin-stimulated glucose uptake. GLUT proteins are distributed in various tissues according to their specific functions. Glucose Transporters: Transporter Tissue Location Functions GLUT 1 Brain, kidney, colon, placenta, erythrocytes (Option A ruled out) Facilitates glucose uptake GLUT 2 Liver, pancreatic β cells, small intestine, kidney (Option B, D ruled out) Enables rapid glucose uptake or release GLUT 3 Brain, kidney, placenta Assists in glucose uptake GLUT 4 Heart and skeletal muscle, adipose tissue Mediates insulin-stimulated glucose uptake GLUT 5 Small intestine Facilitates fructose absorption SGLT 1 Small intestine and kidney Actively transports glucose against a concentration gradient GLUT 1-5 are facilitative bidirectional transporters, whereas SGLT 1 is a sodium-dependent unidirectional transporter.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 186,187.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q36: Glycogen phosphorylase is activated by? (Q854622)

A. Glucose 6 phosphatase

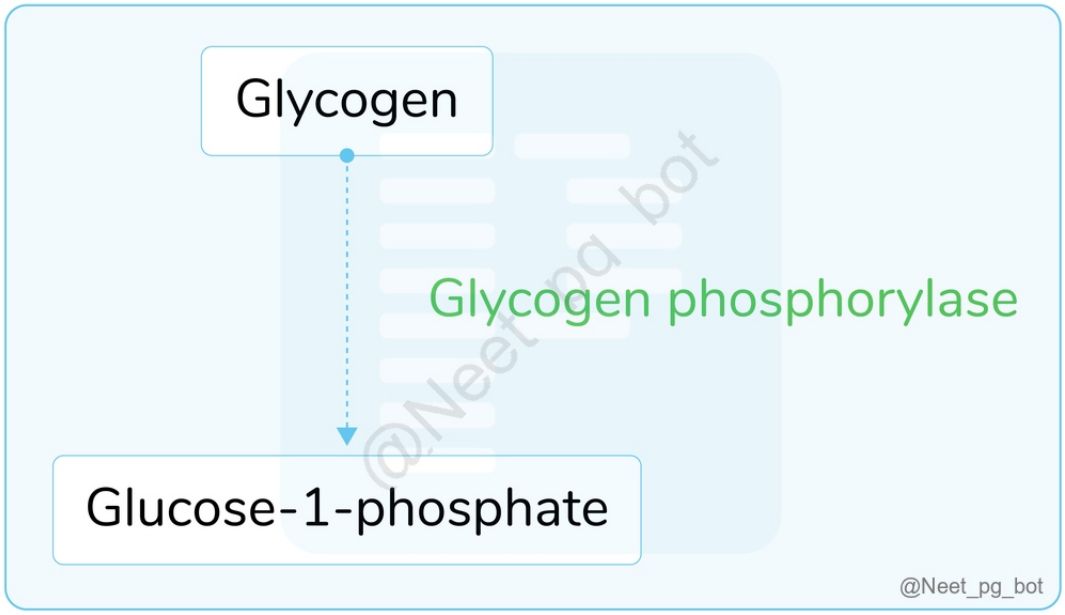
B. ATP

C. Calcium

D. Glucose

Correct Answer: C. Calcium

Explanation: Rate limiting step in glycogenolysis: Glycogen phosphorylase: Phosphorolytic cleavage of the 1→4 linkages of glycogen Different isoenzymes exist in liver, muscle, and brain, encoded by different genes Requires pyridoxal phosphate as coenzyme Activators: cAMP Glucagon - only in liver Epinephrine Calcium 5’AMP Inhibitors: Protein Phosphatase 1 (PP1) Insulin Glucose - only in liver Glucose 6 phosphate ATP Fructose 1 phosphate - only in liver



References:

Harper 32nd edition, page 173

↑ *Back to this question in Part A*

@Neet_pg_bot

Q37: A newborn infant is brought to the clinic with jaundice and a white, punctate, and scattered opacity in the central lens. The condition is consistent with Oil drop cataract. Which of the following metabolic disorders is most likely responsible for this presentation? (Q820353)

A. Phenylketonuria (PKU)

B. Maple syrup urine disease

C. Galactosemia

D. Wilson's disease

Correct Answer: C. Galactosemia

Explanation: Oil-drop cataract is a classical ocular finding in galactosemia. The disorder is caused by deficiency of galactose-1-phosphate uridyltransferase (GALT). This leads to accumulation of galactitol in the lens. Galactitol causes osmotic damage to lens fibers, producing cataracts. Affected newborns present with jaundice, vomiting, hepatomegaly, and failure to thrive. Early dietary restriction of galactose/lactose can prevent progression. Incorrect Options: Option A - Phenylketonuria (PKU) : Phenylketonuria is a metabolic disorder caused by a deficiency of the enzyme phenylalanine hydroxylase. This leads to the buildup of phenylalanine in the body. However, PKU is not associated with oil drop cataract; instead, it can lead to intellectual disability, developmental delays, and other neurological problems if left untreated. Option B - Maple syrup urine disease: This is a metabolic disorder that affects the breakdown of branched-chain amino acids (leucine, isoleucine, and valine). It can cause neurological symptoms and a characteristic sweet odor to the urine. It is not associated with oil drop cataract. Option D - Wilson's disease: Wilson's disease is a disorder of copper metabolism, leading to copper accumulation primarily in the liver and brain. It can cause various neurological and hepatic symptoms. It is not associated with oil drop cataract.

References:

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↑ *Back to this question in Part A*

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Q38: A young boy presents with muscle cramps and dark-colored urine after strenuous exercise. He reports that his symptoms improve after brief rest. Laboratory tests show normal blood glucose levels, no rise in serum lactate after exercise, and

elevated creatine kinase. Which of the following is the most likely diagnosis? (Q855193)

A. McArdle disease

B. Hers disease

C. Cori's disease

D. Andersen disease

Correct Answer: A. McArdle disease

Explanation: McArdle disease (Glycogen Storage Disease Type V) results from muscle glycogen phosphorylase deficiency , leading to an inability to break down glycogen in skeletal muscle . Characteristic features include: Exercise intolerance and cramps , Myoglobinuria (dark urine) after exertion , “Second wind” phenomenon , No rise in blood lactate after exercise , Normal liver function and blood glucose . Hers disease (Type VI) (Option B): Affects liver → hepatomegaly and fasting hypoglycemia. Cori disease (Type III) (Option C): Affects both liver and muscle → hypoglycemia, hepatomegaly, mild myopathy. Andersen disease (Type IV) (Option D): Affects liver → progressive cirrhosis and liver failure, not exercise-related. Glycogen Storage Disease Enzyme Deficiency Type 0 Glycogen synthase deficiency Type I (von Gierke's Disease) Glucose-6-phosphatase Type II (Pompe's Disease) Acid α-glucosidase (acid maltase) Type III (Cori's Disease) Debranching enzyme (amylo-1,6-glucosidase) Type IV (Andersen's Disease) Branching enzyme Type V (McArdle's Disease) Muscle phosphorylase (myophosphorylase) Type VI (Hers' Disease) Liver phosphorylase Type VII (Tarui's Disease) Phosphofructokinase (muscle type)

References:

Harper’s Illustrated Biochemistry (23rd Edition), Page 174.

Vasudevan Textbook of Biochemistry (7th Edition), Page 128,129.

Glycogen storage disease type III - NIH Genetic Testing Registry (GTR) - NCBI

Anderson-Fabry disease management:

Anderson's Disease

↑ *Back to this question in Part A*

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Q39: A 4-year-old child presents with easy fatiguability. The mother also complains that the child has increased hunger between meals, which is relieved after food. Liver examination revealed no glycogen. The enzyme most likely deficient is (Q855224)

A. Glycogen phosphorylase

B. Glycogen synthase

C. Debranching enzyme

D. Glucose 6 phosphatase

Correct Answer: B. Glycogen synthase

Explanation: The child has no hepatic glycogen , meaning the liver cannot store glucose. This points to a defect in glycogen synthesis , specifically glycogen synthase deficiency (GSD type 0) . These children develop fasting hypoglycemia with ketones and become hungry between meals, improving after eating. Glycogen storage disease type 0 (0a and 0b): Enzyme Deficiency: Glycogen synthase Affected Gene: GYS2 and GYS1 Clinical Features: Hypoglycemia, Hyperketonemia, early death Mode of Inheritance: Autosomal recessive Management: Supportive care: Frequent protein-rich meals and avoidance of fasting. Cornstarch may be given nighttime Glycogen Storage Disease Enzyme Deficiency Type 0 Glycogen synthase deficiency Type I (von Gierke's Disease) Glucose-6-phosphatase (Option D ruled out) Type II (Pompe's Disease) Acid α-glucosidase (acid maltase) Type III (Cori's Disease) Debranching enzyme (amylo-1,6-glucosidase) (Option C ruled out) Type IV (Andersen's Disease) Branching enzyme Type V (McArdle's Disease) Muscle phosphorylase (myophosphorylase) (Option A ruled out) Type VI (Hers' Disease) Liver phosphorylase Type VII (Tarui's Disease) Phosphofructokinase (muscle type)

References:

Harper’s Biochemistry 32e pg: 174

Vasudevan Textbook of Biochemistry (7th Edition), Page 129.

Management glycogen storage disease 0

↑ *Back to this question in Part A*

@Neet_pg_bot

Q40: Which of the following is seen in low insulin:glucagon ratio? (Q855240)

A. Activation of lipoprotein lipase

B. Activation of glycogen synthase

C. Activation of phosphofructokinase

D. Activation of hormone-sensitive lipase

Correct Answer: D. Activation of hormone-sensitive lipase

Explanation: Hormone-sensitive lipase (HSL) is activated in response to low insulin:glucagon ratio. HSL breaks down stored triglycerides in adipose tissue into free fatty acids and glycerol, providing energy during fasting. Insulin:Glucagon Bipolar Axis : Insulin : Anabolic hormone promoting glycogenesis, lipogenesis, and protein synthesis. Glucagon : Catabolic hormone stimulating glycogenolysis, gluconeogenesis, and lipolysis. IGR : High IGR → Anabolic state, reduced glucose production. Low IGR → Catabolic state, increased glucose production. Determinants of IGR : Influenced by insulin and glucagon secretion patterns, dietary composition, and metabolic status. Variability in systemic and portal concentrations complicates measurement accuracy. Activation of lipoprotein lipase (Option A): Lipoprotein lipase is activated by insulin, not glucagon. It facilitates triglyceride uptake from circulating lipoproteins into adipose tissue for storage, which occurs in the fed state. Activation of glycogen synthase (Option B): Glycogen synthase is activated by insulin, promoting glycogen synthesis in the liver and muscle during the fed state. Activation of phosphofructokinase (Option C): Phosphofructokinase (PFK) is a key enzyme in glycolysis. It is activated by insulin and suppressed during fasting (low insulin:glucagon ratio), as glycolysis is reduced in favor of gluconeogenesis.

References:

The Insulin:Glucagon Ratio and the Choice of Glucose-Lowering Drugs - PMC

Vasudevan Textbook of Biochemistry (7th Edition), Page 101, 102

↑ *Back to this question in Part A*

@Neet_pg_bot

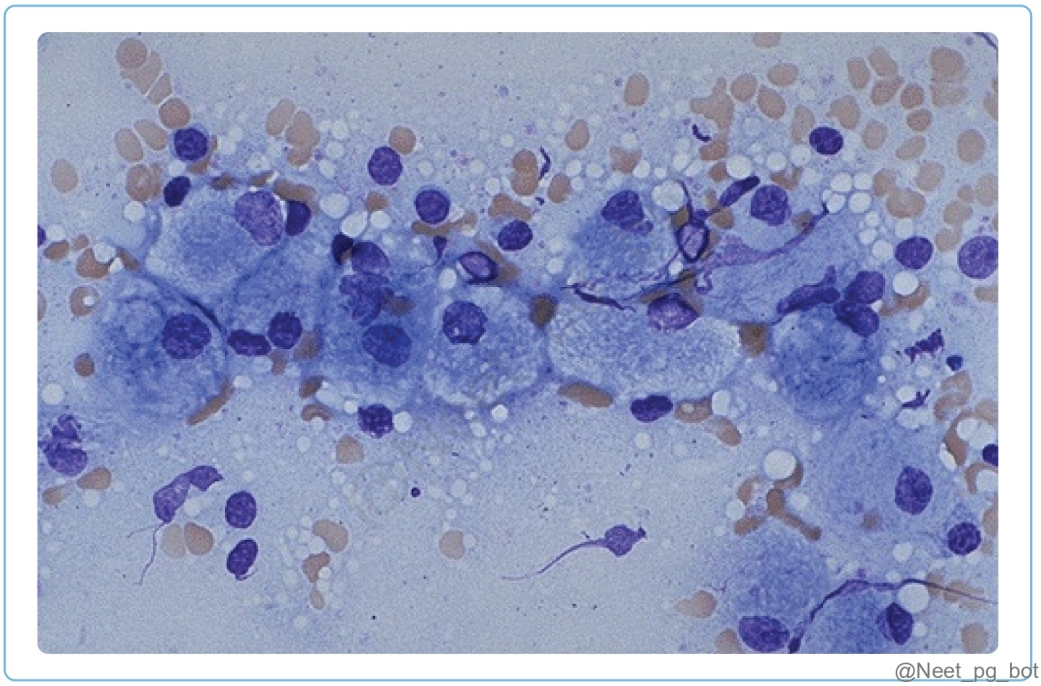
Q41: A child presents with bone pain and hepatosplenomegaly. A trephine biopsy and aspirate shows the following finding. Which of the following is the most likely enzyme deficient in this condition? (Q855278)

A. Hexosaminidase

B. Glucocerebrosidase

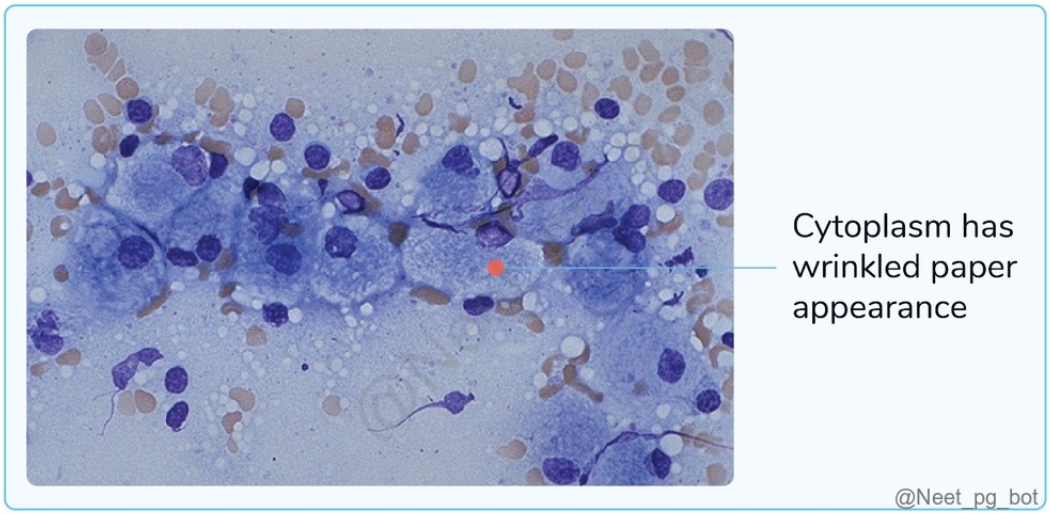
C. Sphingomyelinase

D. Alpha 1,4-glucosidase



Correct Answer: B. Glucocerebrosidase

Explanation: Gaucher's disease is characterized by the accumulation of glucosylceramide in RBCs and platelet membranes due to a deficiency in the enzyme beta-glucosidase (glucocerebrosidase) , leading to anemia, hepatosplenomegaly, and thrombocytopenia. Bone marrow biopsy shows macrophages with a characteristic "wrinkled paper" appearance. Gaucher's disease: Lysosomal storage disease Defective enzyme: Glucocerebrosidase (■-Glucosidase) Defect in metabolism: Accumulation of cerebroside (Glucosylceramide) in RBCs and platelet membranes. (Option B) Pathophysiology: Abnormal RBCs and platelets are engulfed by macrophages leading to accumulation inside macrophages causing wrinkled paper appearance. {{caption_text}} Disease Enzyme deficiency Features Tay-Sachs disease Hexosaminidase A (Option A ruled out) Mental retardation Blindness Muscular weakness Cherry red spots in the macula Fabry disease α-Galactosidase Skin rash Kidney failure (full symptoms only in males X-linked recessive) Krabbe disease β-Galactosidase Mental retardation Near absence of myelin Predominant neurological manifestation and cranial nerves causing deafness and blindness. Gaucher disease β-Glucosidase Enlarged liver and spleen Erosion of long bones Mental retardation in infants Anemia Thrombocytopenia Niemann-Pick disease Sphingomyelinase (Option C ruled out) Enlarged liver and spleen Mental retardation Fatal in early life Cherry red spot in macula Farber disease Ceramidase Hoarseness Dermatitis Skeletal deformation Mental retardation Fatal in early life Alpha 1,4-glucosidase (Option D): Deficiency of alpha 1,4-glucosidase (acid maltase) results in Pompe disease , which is associated with glycogen accumulation, hypotonia, cardiomegaly, and muscle weakness, rather than hepatosplenomegaly and bone pain.



References:

https://www.ncbi.nlm.nih.gov/books/NBK448080/

Harper's illustrated Biochemistry 32e pg 245,

Vasudevan textbook of Biochemistry 7e pg 194

Image: https://www.tau.ac.il/medicine/tau-only/webpath/hemehtml/heme057.htm

↑ *Back to this question in Part A*

@Neet_pg_bot

Q42: A child presents with developmental delay, bone pain, and inability to walk. On fundoscopy, a cherry-red spot is observed. There is no organomegaly. What is the most likely diagnosis? (Q855287)

A. Tay-Sachs disease

B. Niemann-Pick disease

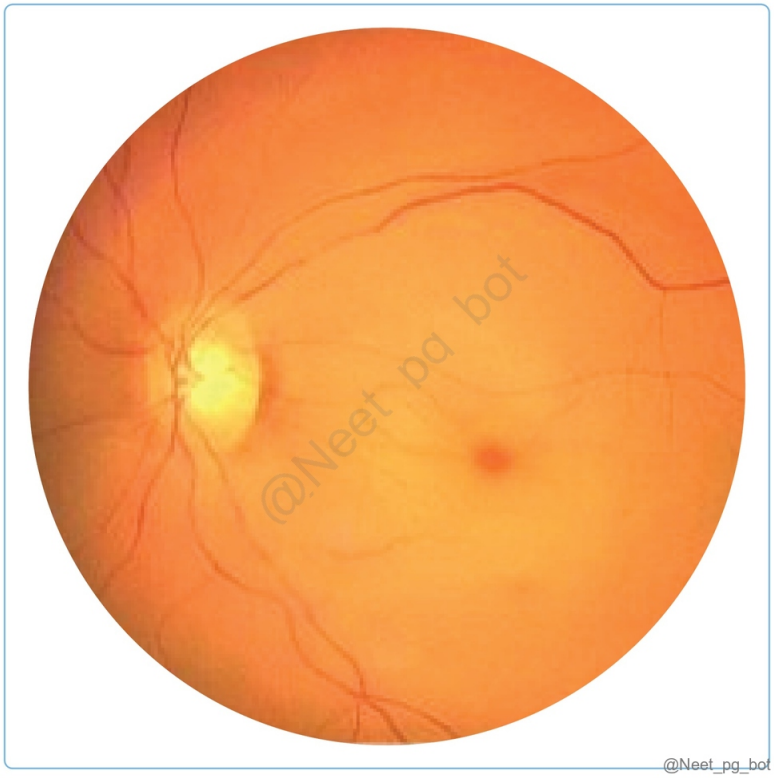
C. Hurler syndrome

D. Gaucher disease

Correct Answer: A. Tay-Sachs disease

Explanation: Tay–Sachs disease is a GM■ gangliosidosis caused by hexosaminidase A deficiency , leading to accumulation of GM■ gangliosides in neurons. Key features include: Neurodegeneration , developmental delay , and hypotonia , Cherry-red spot on macula (due to retinal ganglion cell storage material surrounding normal fovea)

No hepatosplenomegaly , distinguishing it from Niemann–Pick disease. {{caption_text}} Niemann–Pick disease (Option B): Also shows a cherry-red spot, but has massive hepatosplenomegaly due to sphingomyelinase deficiency. Hurler syndrome (Option C): A mucopolysaccharidosis presenting with coarse facial features, corneal clouding, and hepatosplenomegaly, not a cherry-red spot. Gaucher disease (Option D): Due to β-glucocerebrosidase deficiency, causing hepatosplenomegaly and bone pain, but no cherry-red spot. Disease Enzyme Deficiency Tay-Sachs disease Hexosaminidase A, S Fabry disease α-Galactosidase Metachromatic leukodystrophy Arylsulfatase A Krabbe disease β-Galactosidase Gaucher disease β-Glucosidase Niemann-Pick disease types A, B Sphingomyelinase Farber disease Ceramidase



References:

Harper’s illustrated Biochemistry 32e pg 245

https://www.ncbi.nlm.nih.gov/books/NBK564432/

↑ *Back to this question in Part A*

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Q43: Which enzyme deficiency could potentially be the cause of exercise intolerance and cramps in adolescent male patient? (Q855537)

A. Myophosphorylase

B. Hexokinase

C. Glucose-6-phosphatase

D. Hepatic glycogen phosphorylase

Correct Answer: A. Myophosphorylase

Explanation: McArdle disease, or myophosphorylase deficiency, affects glycogen utilization for energy. Type 5 Glycogen storage disorder. Myophosphorylase converts glycogen to glucose-1-phosphate in muscle tissue . Insufficient enzyme activity leads to exercise intolerance due impaired glycogen breakdown. Symptoms include muscle cramps, stiffness, and weakness during exertion, alleviated Hexokinase (Option B): Catalyzes glucose to glucose-6-phosphate conversion in glycolysis.Insufficiency affects glucose uptake, leading to exhaustion and weakness, not exercise intolerance or cramps. Glucose-6-phosphatase (Option C): Converts glucose-6-phosphate to glucose in gluconeogenesis.Deficiency causes hypoglycemia, not exercise intolerance. Hepatic glycogen phosphorylase (Option D): Breaks down liver glycogen. Deficiency affects liver, not causing exercise intolerance or cramps.

References:

McArdle disease

↑ *Back to this question in Part A*

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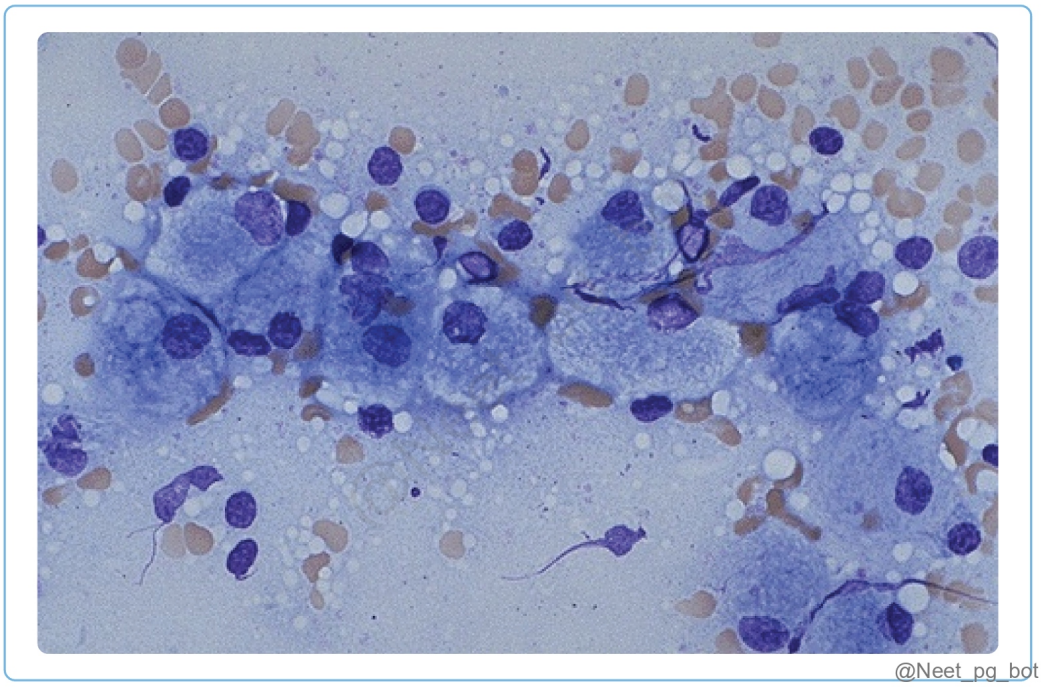
Q44: A baby is being evaluated for delayed developmental milestones. On examination, there is hepatosplenomegaly. Microscopic image of the bone marrow evaluation shown below. What is the diagnosis? (Q855544)

A. Gaucher disease

B. Niemann-pick disease

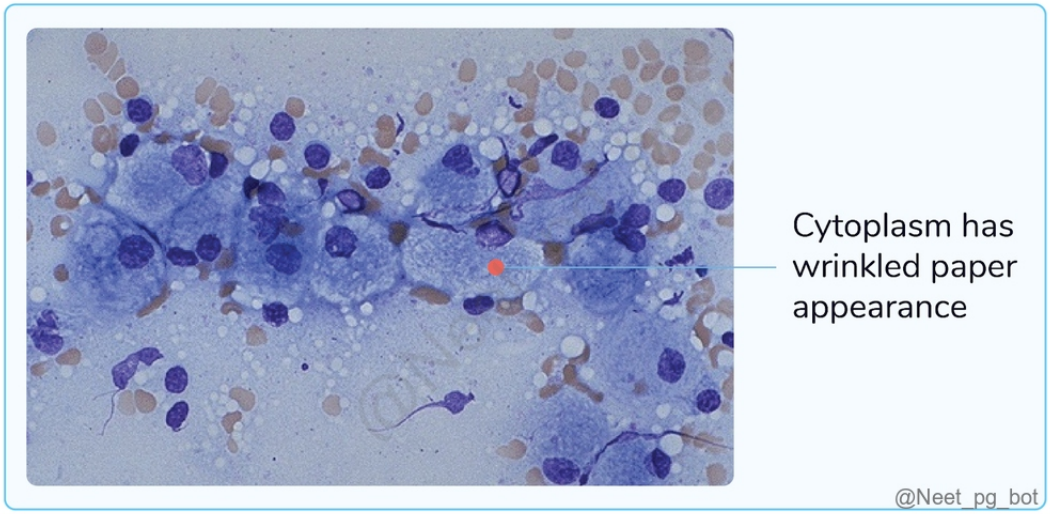
C. Hunter disease

D. Hurler disease



Correct Answer: A. Gaucher disease

Explanation: The given bone marrow image shows Gaucher cells, which have a large macrophage appearance with wrinkled or “crumpled tissue paper” cytoplasm. These cells result from accumulation of glucocerebroside due to β-glucocerebrosidase deficiency. Such cells are pathognomonic for Gaucher disease. Clinically, the child shows hepatosplenomegaly and developmental delay, fitting the disease profile. Bone involvement, anemia, and thrombocytopenia are also common. The combination of clinical features and the characteristic marrow cells strongly supports Gaucher disease. Hence, the diagnosis is Gaucher disease. {{caption_text}} Niemann-Pick disease (Option B): Foamy macrophages with lipid droplets (“soap-bubble” appearance), cherry-red macula. Hunter disease (Option C): Mucopolysaccharidosis with aggressive behavior; no corneal clouding. Hurler disease (Option D): Coarse facies, corneal clouding, but does not show Gaucher cells.



References:

pmc.ncbi.nlm.nih.gov/articles/PMC4017182/

↑ *Back to this question in Part A*

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Q45: Which of the following substances inhibits cytochrome complex IV, a component of the electron transport chain responsible for ATP synthesis? (Q855551)

A. Cyanide

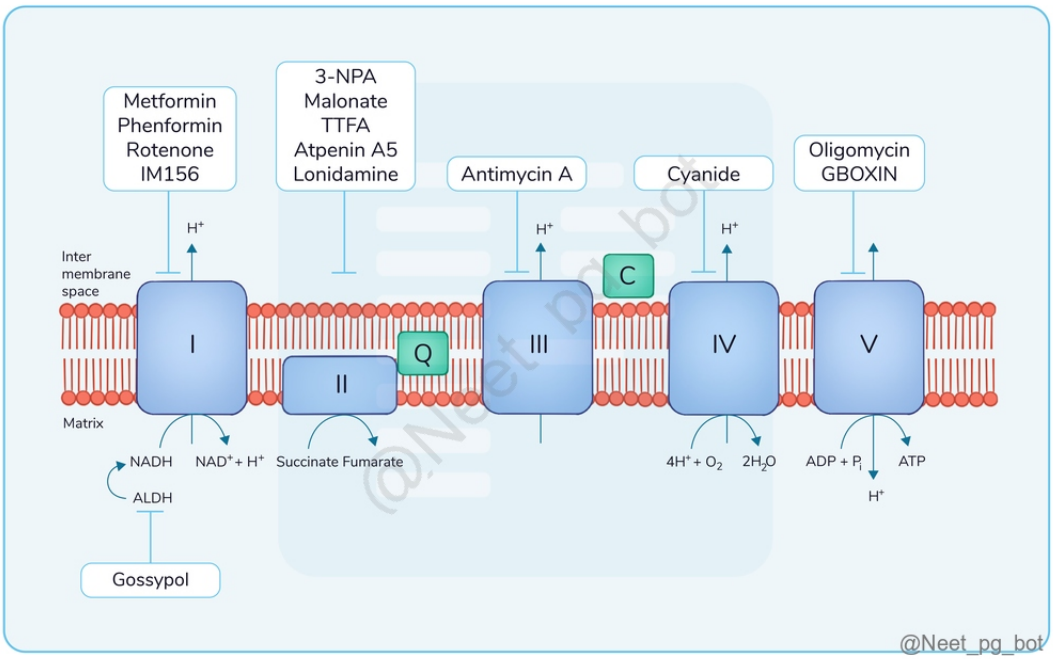
B. Carbon dioxide

C. Oligomycin

D. Ouabain

Correct Answer: A. Cyanide

Explanation: Cyanide: Powerful inhibitor of cytochrome complex IV is cyanide. It attaches to the enzyme's active site and stops the enzyme from transferring electrons to oxygen. Due to the blockage of the electron transport chain, reducing agents like NADH and FADH2 begin to accumulate. The last enzyme complex in the electron transport chain is cytochrome complex IV, also known as cytochrome c oxidase. When oxygen and protons are combined to form water, it receives electrons from cytochrome c. The ATP synthase uses the proton gradient created by this mechanism to manufacture ATP. inhibitor of Complex IV" data-author="" data-hash="11566" data-license="" data-source="" data-tags="May2025" src="" /> Carbon dioxide (Option B): Does not inhibit cytochrome complex IV.Bicarbonate ions, which are the predominant type of carbon dioxide carried in the blood, do not directly interact with the electron transport chain. Oligomycin (Option C): Cytochrome complex IV is not inhibited by oligomycin.It is an ATP synthase inhibitor that attaches to the Fo subunit of ATP synthase and stops protons from passing through the enzyme, hence preventing the production of ATP. Ouabain (Option D): Na/K ATPase pump, which keeps the electrochemical gradient across the plasma membrane of cell is inhibited by cardiac glycoside.



References:

ETC inhibitors

↑ *Back to this question in Part A*

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Q46: What are the main substrate and metabolic fuel used by RBCs during fasting? (Q855902)

A. Ketone bodies

B. Glucose

C. Free fatty acids

D. Amino acids

Correct Answer: B. Glucose

Explanation: RBCs cannot use fatty acid or ketone bodies as fuel during fasting as they do not have mitochondria where beta-oxidation of fatty acid occurs. Since ketone bodies and free fatty acids are utilized via beta-oxidation which occurs in mitochondria, RBCs cannot utilize them. RBC uses glucose as a substrate for energy production in the fed, fasting, and starvation state. Fuel in Fed, Fasting, and Starvation: Fed state Fasting Starvation Brain Glucose Glucose Ketone body Heart Fatty Acid (FA) FA Ketone body Liver Glucose FA Amino acid Muscle

Glucose FA FA and ketone body Adipose tissue Glucose FA FA RBC Glucose Glucose Glucose Main Fuel for Body Glucose Fat Amino acids Ketone body (Option A) are used as a substrate for energy production during starvation phase by vital organs brain and heart primarily. Muscles use FA and ketone bodies for energy during starvation to avoid using proteins as fuel to prevent muscle loss. Amino acid (Option C) is used by liver as a fuel in starvation state. It is the major source of fuel for the body in a starvation state. Free fatty acid (Option D) is used as a fuel in fasting state by Heart (uses fatty acid in both fed and fasting state) Liver Muscle Adipose tissue (uses fatty acid in both fasting and starvation state)

References:

Vasudevan, 7th Edition, Page 101

↑ *Back to this question in Part A*

@Neet_pg_bot

Q47: Which of the following diagnostic markers is /are incorrectly paired? 1. Beta glucocerebrosidase - Von Gierke disease 2. Lipase - Acute pancreatitis 3. Ceruloplasmin - Hepatolenticular degeneration 4. High LDH1: LDH2 ratio - Myocardial infarction (Q855916)

A. 1 only

B. 1 and 2

C. 1, 2 and 4

D. 2, 3 and 4

Correct Answer: A. 1 only

Explanation: Beta-glucocerebrosidase – Von Gierke disease: Incorrect Von Gierke disease (Glycogen Storage Disease type I) is due to glucose-6-phosphatase deficiency, not beta-glucocerebrosidase. Beta-glucocerebrosidase deficiency causes Gaucher disease, leading to glucocerebroside accumulation in macrophages ("Gaucher cells"). Lipase – Acute pancreatitis : Correct Serum lipase (and amylase) levels are the most specific markers of acute pancreatitis . Ceruloplasmin – Hepatolenticular degeneration: Correct Low ceruloplasmin levels are diagnostic of Wilson’s disease (hepatolenticular degeneration) , due to defective copper metabolism. High LDH1:LDH2 ratio – Myocardial infarction: Correct Normally LDH2 > LDH1, but in myocardial infarction , LDH1 > LDH2 (“flipped ratio”) due to cardiac tissue damage.

References:

Harper’s Illustrated Biochemistry (23rd Edition), Page 174.

Vasudevan Textbook of Biochemistry (7th Edition), Page 129.

↑ *Back to this question in Part A*

• [07_ Genetics] 021 Dna And Chromosome Biochemistry Structure Replication And Repair

« Back to Folder Directory

[07_ Genetics] 021 Dna And Chromosome Biochemistry Structure Replication And Repair — Quick Jump

Q1 (p. 1015)	*Q2 (p. 1015)*	*Q3 (p. 1016)*	*Q4 (p. 1017)*	*Q5 (p. 1018)*	*Q6 (p. 1019)*
Q7 (p. 1019)	*Q8 (p. 1021)*	*Q9 (p. 1021)*	*Q10 (p. 1022)*	*Q11 (p. 1023)*	*Q12 (p. 1024)*
Q13 (p. 1025)	*Q14 (p. 1026)*	*Q15 (p. 1026)*	*Q16 (p. 1027)*	*Q17 (p. 1028)*	*Q18 (p. 1029)*
Q19 (p. 1030)	*Q20 (p. 1031)*	*Q21 (p. 1032)*	*Q22 (p. 1033)*	*Q23 (p. 1035)*	*Q24 (p. 1036)*
Q25 (p. 1038)	*Q26 (p. 1039)*	*Q27 (p. 1041)*	*Q28 (p. 1041)*		

Q1: Steroid hormone receptors bind to specific sites on DNA by which of the following structures? (Q882301)

A. Zinc finger motif

B. RNA motif

C. Leucine zipper

D. Helix–turn–helix

Correct Answer: A. Zinc finger motif

Explanation: Correct answer A) Zinc finger motif Steroid hormone receptors are nuclear receptors that regulate gene transcription. They have a DNA-binding domain (DBD) that recognizes specific hormone response elements on DNA. This DBD contains zinc finger motifs, where zinc ions stabilize the finger-like loops. Each “finger” fits into the major groove of DNA, allowing specific sequence recognition. Incorrect Options: Option B) RNA motif – Binds RNA, not DNA; not involved in steroid receptor binding. Option C) Leucine zipper – DNA-binding motif in non-steroid transcription factors (e.g., AP-1); not used by steroid receptors. Option D) Helix–turn–helix – Found in prokaryotic transcription factors and homeobox proteins; not used by steroid hormone receptors.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: Which of the following is not a requirement for DNA replication in eukaryotes? (Q843167)

A. Origin of replication (ori)

B. DNA helicase

C. RNA primer

D. RNA polymerase

Correct Answer: D. RNA polymerase

■ *Listen to Audio 1*

Explanation: Origin of replication (ori) : Required to initiate replication. Eukaryotic DNA has multiple origins of replication to ensure efficient duplication. DNA helicase : Unwinds the DNA double helix to allow access for replication machinery. RNA primer : Required because DNA polymerase cannot initiate synthesis de novo ; RNA primase lays down short RNA primers for DNA polymerase to extend. RNA polymerase (Option D) is involved in transcription, not DNA replication . It synthesizes RNA from a DNA template but does not play a role in DNA synthesis. DNA Replication Requirements in Eukaryotes Component Function Origin of Replication (ori) (Option A ruled out) Specific DNA sequences where replication starts DNA Helicase (Option B ruled out) Unwinds the DNA double helix Single-Strand Binding Proteins (SSBPs) Stabilize unwound DNA strands Primase (RNA polymerase type) Synthesizes short RNA primers DNA Polymerase α, δ, and ε Synthesizes new DNA strands Sliding Clamp (PCNA) Enhances DNA polymerase processivity Topoisomerase Relieves supercoiling ahead of the replication fork DNA Ligase Seals Okazaki fragments on the lagging strand

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 372, 373, 374, 377, 378

↑ *Back to this question in Part A*

@Neet_pg_bot

Q3: A scientist observes the process of DNA replication in a human stem cell. He takes a sample of double-stranded DNA from the cell and studies it separately. He finds that the total amount of adenine in the sample is 24%. From the given information, what percentage of cytosine is present in the sample? (Q843584)

A. 52%

B. 24%

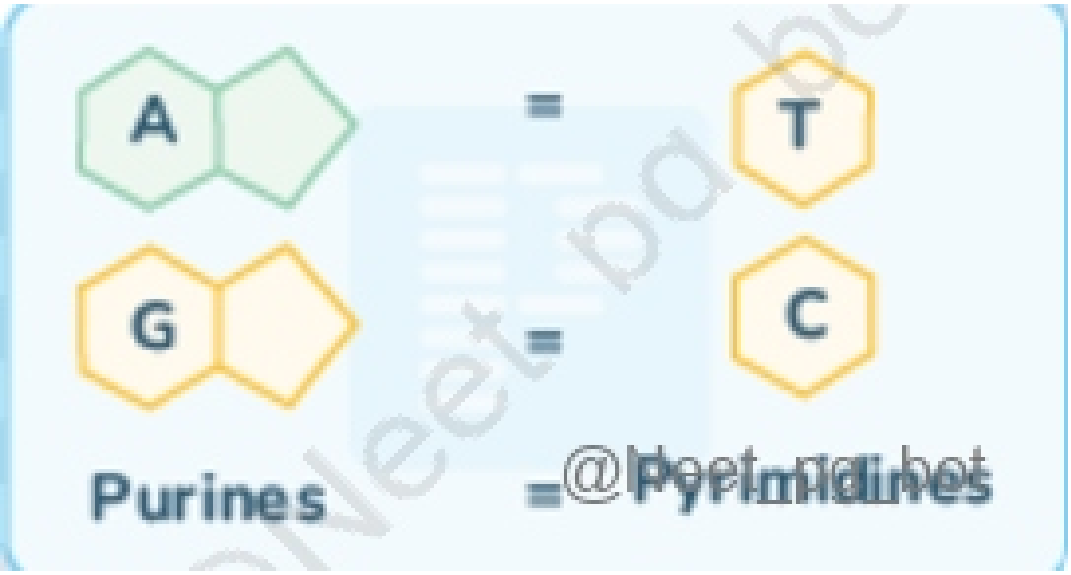
C. 30%

D. 26%

Correct Answer: D. 26%

■ *Listen to Audio 1*

Explanation: According to Chargaff’s Rule , DNA follows base-pairing where: Adenine (A) pairs with Thymine (T) Cytosine (C) pairs with Guanine (G) Given Adenine (A) = 24% , this means Thymine (T) = 24% as well. The total percentage of A + T = 24% + 24% = 48% . Since DNA is double-stranded , the remaining percentage must be from Cytosine (C) and Guanine (G) . C + G = 100% - 48% = 52% . As C and G are equal in amount , we divide: C = G = 52% ÷ 2 = 26% . It is not applicable to: Single-Stranded DNA viruses: (e.g., Parvovirus, phi X 174). All RNA molecules: (mRNA, tRNA, rRNA). In these single-stranded structures, complementary base pairing is not required for the primary sequence, so the molar ratios of A:T and G:C are independent. .



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 349

↑ *Back to this question in Part A*

@Neet_pg_bot

Q4: What is the wavelength at which nucleic acids show maximum absorbance of UV light? (Q843583)

A. 260 nm

B. 280 nm

C. 400 nm

D. 340 nm

Correct Answer: A. 260 nm

■ *Listen to Audio 1*

Explanation: Nucleic acids (DNA and RNA) absorb UV light maximally at 260 nm due to the presence of aromatic nitrogenous bases (adenine, guanine, cytosine, thymine, and uracil). The purines and pyrimidines contain conjugated double bonds that strongly absorb UV light at this wavelength. Protein contamination is assessed using the 260/280 nm ratio since proteins absorb maximally at 280 nm due to aromatic amino acids (tryptophan and tyrosine). UV Absorption of Biomolecules: Molecule Maximum Absorbance (nm) Reason DNA/RNA 260 nm Nitrogenous bases (purines & pyrimidines) Proteins 280 nm (Option B ruled out) Aromatic amino acids (tryptophan & tyrosine) NADH/NADPH 340 nm (Option D ruled out) Reduced nicotinamide ring Hemoglobin 400 nm (Option C ruled out) Heme group

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 331, 21, 318, 66

↑ *Back to this question in Part A*

@Neet_pg_bot

Q5: What sequence is repeated in human telomeres? (Q843398)

A. 5'-TTAAGG-3'

B. 5'-GGTACC-3'

C. 5'-TTAGGG-3'

D. 5'-AGCTAG-3'

Correct Answer: C. 5'-TTAGGG-3'

■ *Listen to Audio 1*

Explanation: Human telomeres consist of repetitive nucleotide sequences that protect chromosome ends from degradation. The repeated sequence in human telomeres is 5'-TTAGGG-3' , which is tandemly repeated thousands of times. Telomerase, a ribonucleoprotein enzyme, extends the telomeres using an RNA template to prevent progressive shortening. Telomere Structure and Function: Feature Description Sequence 5'-TTAGGG-3' repeat Function Protects chromosome ends from degradation and fusion Telomerase Activity Adds repeats using an RNA template Shortening Effect Telomeres shorten with each cell division due to incomplete lagging strand synthesis Associated Diseases Premature telomere shortening is linked to aging and diseases like dyskeratosis congenita and aplastic anemia

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 365, 701.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: Which of the following is not caused by a defective NER mechanism? (Q843184)

A. Xeroderma pigmentosum (XP)

B. Cockayne syndrome (CS)

C. Trichothiodystrophy (TTD)

D. Werner syndrome (WS)

Correct Answer: D. Werner syndrome (WS)

■ *Listen to Audio 1*

Explanation: Werner syndrome is caused by defective homologous repair and not a defect in the NER mechanism. Nucleotide Excision Repair (NER): Required when chemical damage to a segment occurs. Removes bulky DNA lesions, such as pyrimidine dimers and chemical adducts. Excises a variable number of nucleotides (typically 24-32 bases), followed by DNA synthesis to fill the gap. UV-Induced Damage: UV radiation causes the covalent joining of adjacent pyrimidines (usually thymine dimers). These bulky DNA lesions impede DNA replication and transcription. Recognition and Excision: In bacteria: UvrABC proteins (UvrABC excinuclease) recognize thymine dimers. The damaged strand is cleaved on both the 5′ and 3′ sides. A short oligonucleotide containing the dimer is excised. DNA polymerase I fills the gap, and DNA ligase seals it. Nucleotide Excision Repair in Humans: Repairs UV-induced damage and other bulky lesions throughout the cell cycle. Transcription-coupled repair (a type of NER) specifically targets lesions blocking RNA polymerase. Defects in NER cause: Xeroderma pigmentosum (XP) (Option A ruled out) Cockayne syndrome (CS) (Option B ruled out) Trichothiodystrophy (TTD) (Option C ruled out)

References:

1. Harper's Illustrated Biochemistry, 32nd Edition, Page 380 2. Textbook of Biochemistry for Medical Students, Vasudevan, 7th Edition, Page 583 3. Lippincott Illustrated Reviews: Biochemistry, 7th Edition, Page 1219-1222

↑ *Back to this question in Part A*

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Q7: What is the role of UvrABC exinuclease in nucleotide excision repair (NER)? (Q843182)

A. It fills in the gap with new nucleotides

B. It recognizes and excises damaged DNA segments.

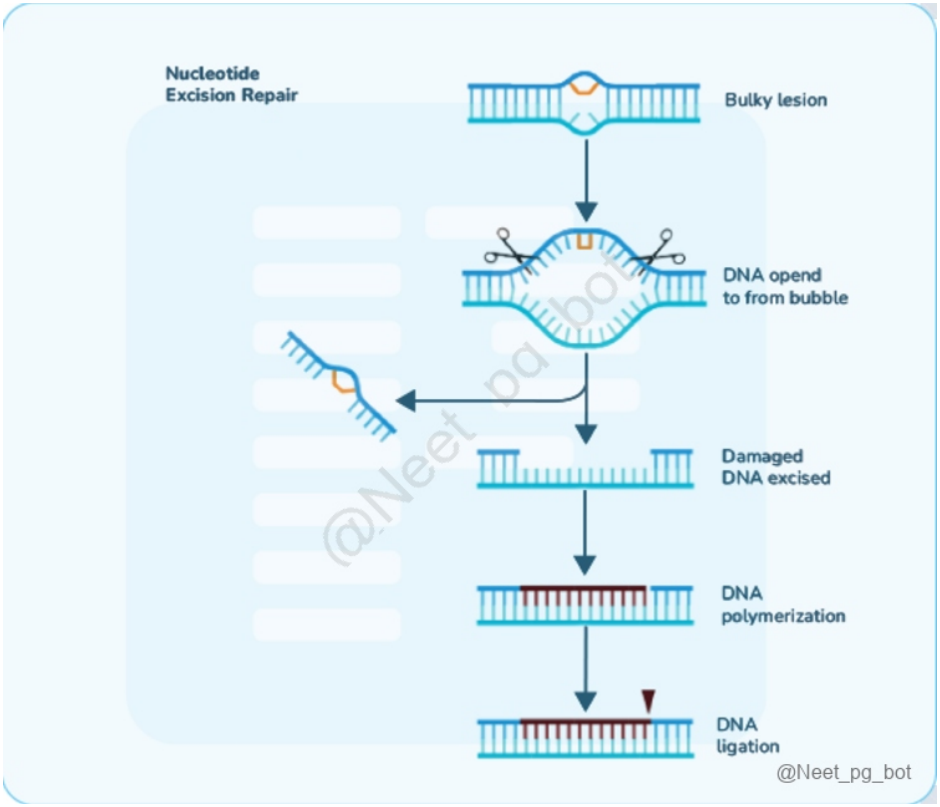
C. It seals the DNA strand after repair.

D. It unwinds the DNA double helix.

Correct Answer: B. It recognizes and excises damaged DNA segments.

■ *Listen to Audio 1*

Explanation: UvrABC excinuclease is responsible for recognizing bulky DNA lesions and excising the damaged DNA segments during nucleotide excision repair Nucleotide excision repair: Nucleotide excision repair is required when chemical damage to a segment occurs. 30 bases are removed and then the correct bases are added UV-Induced Damage: Exposure to UV radiation can cause the covalent joining of adjacent pyrimidines (usually thymine dimers), creating bulky DNA lesions that impede DNA replication. Recognition and Excision: In bacteria, UvrABC proteins (uvrABC excinuclease) recognize these dimers and cleave the damaged strand on both the 5′ and 3′ sides of the lesion. A short oligonucleotide containing the dimer is excised, leaving a gap in the DNA strand. The gap is subsequently filled in by DNA polymerase I and sealed by DNA ligase. Nucleotide Excision Repair in Humans: A related nucleotide excision repair (NER) pathway exists in humans, addressing UV-induced damage encountered during DNA synthesis. Transcription-coupled repair, a type of NER, specifically targets lesions that obstruct RNA synthesis. Clinical application: Pyrimidine dimers can form in human skin cells exposed to unfiltered sunlight. In xeroderma pigmentosum (XP), a genetic disorder, the inability to repair UV-induced DNA damage leads to the accumulation of mutations and a heightened risk of developing early and multiple skin cancers. XP results from defects in genes coding for proteins necessary for NER of UV damage It fills in the gap with new nucleotides (Option A): While filling in the gap is part of NER, it is the role of DNA polymerase I. It seals the DNA strand after repair (Option C): Sealing the DNA strand is the function of DNA ligase. It unwinds the DNA double helix (Option D): DNA helicase is responsible for unwinding the DNA double helix.



References:

1. Textbook of Biochemistry for Medical Students, Vasudevan, 7th Edition, Page 583 2. Lippincott Illustrated Reviews: Biochemistry, 7th Edition, Page 1219-1222

↑ *Back to this question in Part A*

@Neet_pg_bot

Q8: Mismatch repair defects are mostly associated with which of the following? (Q843180)

A. Hereditary Nonpolyposis Colon Cancer (HNPCC)

B. Familial Adenomatous Polyposis (FAP)

C. Li-Fraumeni syndrome

D. Retinoblastoma

Correct Answer: A. Hereditary Nonpolyposis Colon Cancer (HNPCC)

■ *Listen to Audio 1*

Explanation: Mismatch repair (MMR) defects lead to microsatellite instability (MSI) , which is a hallmark of Hereditary Nonpolyposis Colon Cancer (HNPCC), also known as Lynch syndrome . HNPCC is caused by mutations in MMR genes, including MLH1, MSH2, MSH6, and PMS2 . Individuals with HNPCC have a higher risk of colorectal cancer, endometrial cancer, and other malignancies . Mismatch Repair and Cancer Association Syndrome Genetic Defect Cancer Risk HNPCC (Lynch Syndrome) MMR gene mutations (MLH1, MSH2, MSH6, PMS2) Colorectal, endometrial, ovarian, gastric cancers FAP (Familial Adenomatous Polyposis) (Option B ruled out) APC gene mutation Multiple colorectal adenomas, colon cancer Li-Fraumeni Syndrome (Option C ruled out) TP53 mutation Breast, brain, sarcomas, leukemias Retinoblastoma (Option D ruled out) RB1 gene mutation Retinoblastoma, osteosarcoma

References:

1. Textbook of Biochemistry for Medical Students, Vasudevan, 7th Edition, Page 585 2. Lippincott Illustrated Reviews: Biochemistry, 7th Edition, Page 1217,1219 3. Harper's Illustrated Biochemistry, 32nd Edition, Page 715 4. www.researchgate.net/figure/ACMG-list-of-hereditary-cancer-syndromes-most-with-childhood-onset-for-reporting_tbl2_302988644

↑ *Back to this question in Part A*

@Neet_pg_bot

Q9: Why are methylases important in the mismatch repair mechanism in prokaryotes like E. coli? (Q843178)

A. They add methyl groups to the newly synthesized DNA strand, marking it for repair.

B. They methylate the parental DNA strand, allowing the Mut proteins to distinguish it from the daughter strand.

C. They remove mismatched bases from the DNA strand.

D. They act as exonucleases during the repair process.

Correct Answer: B. They methylate the parental DNA strand, allowing the Mut proteins to distinguish it from the daughter strand.

■ *Listen to Audio 1*

Explanation: DNA adenine methylase (DAM) methylates the parental strand, enabling Mut proteins to identify the parental strand as correct and target the unmethylated daughter strand for repair. Mismatch Repair: The defect occurs as a copying error, 1-5 bases unpaired Repair is done by strand cutting and exonuclease digestion Mismatched Strand Identification: MMR corrects errors that escape proofreading during DNA synthesis, significantly reducing the error rate. In prokaryotes like E. coli, Mut proteins identify and correct mismatches by distinguishing between the newly synthesized (unmethylated) and the parental (methylated) strands. GATC sequences in DNA are methylated on adenine by DNA adenine methylase (DAM), with the parental strand being methylated and the newly synthesized strand remaining unmethylated shortly after replication. The methylated parental strand is considered correct, guiding the repair of the unmethylated daughter strand. Repair Procedure: Once the mismatched strand is identified, an endonuclease nicks the strand, and an exonuclease removes the mismatched nucleotides along with additional nucleotides at the 5′ and 3′ ends. The gap is then filled using the sister strand as a template by DNA polymerase, typically DNA pol III in prokaryotes. DNA ligase seals the nick, completing the repair process. They add methyl groups to the newly synthesized DNA strand, marking it for repair (Option A) : Methylation occurs on the parental strand, not the newly synthesized strand, to mark it as the correct template They remove mismatched bases from the DNA strand (Option C): Methylases do not remove mismatched bases; they add methyl groups to the DNA. They act as exonucleases during the repair process (Option D): Exonucleases remove mismatched nucleotides, but methylases do not have this function.

References:

1. Lippincott Illustrated Reviews: Biochemistry, 7th Edition, Page 1217,1219 2. Textbook of Biochemistry for Medical Students, Vasudevan, 7th Edition, Page 583

↑ *Back to this question in Part A*

@Neet_pg_bot

Q10: Which of the following enzymes helps remove the defective base during base excision repair (BER)? (Q843175)

A. DNA polymerase I

B. DNA ligase

C. DNA glycosylase

D. AP endonuclease

Correct Answer: C. DNA glycosylase

■ *Listen to Audio 1*

Explanation: DNA glycosylase is the enzyme that recognizes and removes the defective base from the deoxyribose-phosphate backbone, initiating the base excision repair process. Base excision repair A defect occurs as a result of chemical damage to a single base The defective base is removed by N-glycosylase and a new base is added Steps: Abnormal Base Removal: BER targets abnormal bases, such as uracil, which can form from the deamination of cytosine or the incorporation of dUTP instead of dTTP during DNA synthesis. Specific DNA glycosylases recognize and cleave these abnormal bases from the deoxyribose-phosphate backbone, creating an apyrimidinic (AP) site if a pyrimidine is removed or an apurinic (AP) site if a purine is removed. AP Site Recognition and Repair: Specific AP endonucleases recognize the missing base and initiate repair by cutting just the 5′ side of the AP site. (Option D ruled out) A deoxyribose phosphate lyase removes the single, base-free sugar phosphate residue. DNA polymerase I fills in the gap with the correct nucleotide. (Option A ruled out) DNA ligase seals the nick, completing the repair process. (Option B ruled out)

References:

1. Lippincott Illustrated Reviews: Biochemistry, 7th Edition, Page 1224,1226 2. Textbook of Biochemistry for Medical Students, Vasudevan, 7th Edition, Page 583

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: A 6-year-old child presents with recurrent respiratory infections and poor growth despite a normal diet. Genetic testing reveals Cystic fibrosis. What type of mutation causes cystic fibrosis? (Q843174)

A. Phenotypic heterogeneity

B. Pleiotropy

C. Allelic heterogeneity

D. Heteroplasmy

Correct Answer: C. Allelic heterogeneity

■ *Listen to Audio 1*

Explanation: Cystic fibrosis (CF) is caused by mutations in the CFTR gene (located on chromosome 7q31.2), which encodes a chloride channel protein . Over 2,000 different mutations in the CFTR gene can cause CF — this

phenomenon is called allelic heterogeneity . The most common mutation is ΔF508 (deletion of phenylalanine at position 508), accounting for ~70% of CF cases. Other mutations in the same gene can produce variable phenotypic severity , but all affect CFTR function → defective chloride transport → thick secretions → chronic respiratory and pancreatic disease. Phenotypic heterogeneity (Option A): Refers to different mutations in the same gene causing different diseases (e.g., LMNA mutations causing progeria vs muscular dystrophy). CF always causes one disease, though with variable severity. Pleiotropy (Option B): Refers to one gene affecting multiple organ systems — CF is indeed pleiotropic (lungs, pancreas, GI, reproductive tract), but the question asks for the type of mutation , not its systemic effects. Heteroplasmy (Option D): Refers to the presence of both normal and mutant mitochondrial DNA in a cell — not applicable since CF is a nuclear autosomal recessive disorder .

References:

Sciencedirect.com/topics/biochemistry-genetics-and-molecular-biology/phenotypic-heterogeneity#

en.wikipedia.org/wiki/Phenotypic_heterogeneity

www.sciencedirect.com/topics/medicine-and-dentistry/pleiotropy

www.cancer.gov/publications/dictionaries/cancer-terms/def/allelic-heterogeneity

↑ *Back to this question in Part A*

@Neet_pg_bot

Q12: Which of the following statements accurately describes the role of DNA polymerase δ (Pol δ) in eukaryotic cells? (Q843172)

A. It synthesizes RNA primers necessary for DNA replication.

B. It is responsible for leading-strand DNA synthesis.

C. It requires a primer and provides proofreading during lagging-strand synthesis.

D. It directly repairs damaged DNA by filling in gaps.

Correct Answer: C. It requires a primer and provides proofreading during lagging-strand synthesis.

■ *Listen to Audio 1*

Explanation: DNA Polymerase δ (Pol δ) is the primary enzyme responsible for lagging-strand synthesis in eukaryotic cells. It requires an RNA primer synthesized by DNA polymerase α-primase complex to initiate replication. It has 3′ → 5′ exonuclease activity , enabling proofreading and ensuring high fidelity during DNA replication. Works in coordination with PCNA (proliferating cell nuclear antigen) for high processivity. Eukaryotic DNA Polymerases and Their Functions DNA Polymerase Function Pol α Synthesizes RNA primers and initiates DNA synthesis Pol δ Lagging-strand synthesis, proofreading, and high-fidelity replication Pol ε Leading-strand synthesis and proofreading Pol β Involved in base excision repair (BER) Pol γ Replicates mitochondrial DNA It synthesizes RNA primers necessary for DNA replication (Option A): Incorrect because RNA primers are synthesized by DNA polymerase α-primase , not Pol δ. It is responsible for leading-strand DNA

synthesis (Option B): Incorrect because leading-strand synthesis is performed by DNA polymerase ε . It directly repairs damaged DNA by filling in gaps (Option D): Incorrect because DNA repair is primarily performed by DNA polymerase β (base excision repair) and other polymerases in repair pathways.

References:

1. Harper's Illustrated Biochemistry, 32nd Edition, Page 374 2. www.ncbi.nlm.nih.gov/pmc/articles/PMC3694620/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q13: Which of the following statements about telomeres and telomerase is correct? (Q843170)

A. Telomeres prevent chromosome ends from degrading but do not shorten during cell division.

B. Telomerase adds repetitive DNA sequences to the 5′ end of chromosomes

C. Telomeres are composed of repetitive sequences that help prevent chromosome fusion.

D. The shortening of telomeres occurs due to the continuous synthesis of the lagging strand.

Correct Answer: C. Telomeres are composed of repetitive sequences that help prevent chromosome fusion.

■ *Listen to Audio 1*

Explanation: Telomeres are repetitive nucleotide sequences (TTAGGG in humans) at the ends of chromosomes that protect genetic information and prevent chromosome fusion. Telomerase is an RNA-dependent DNA polymerase that extends the 3′ end of chromosomes, not the 5′ end . Telomeres shorten with each cell division due to the end-replication problem in the lagging strand, but not solely because of its synthesis. While telomeres prevent chromosome degradation , they do shorten with every replication cycle in somatic cells due to incomplete lagging strand synthesis. Telomeres and Telomerase Feature Description Telomere Composition Repetitive TTAGGG sequences in humans Function Protect chromosome ends from degradation and fusion Shortening Mechanism Due to incomplete lagging strand synthesis in DNA replication Role of Telomerase Extends the 3′ end of DNA using an RNA template to compensate for shortening Cell Types with Active Telomerase Germ cells, stem cells, and cancer cells Telomeres prevent chromosome ends from degrading but do not shorten during cell division (Option A): Incorrect because telomeres do shorten with each cell cycle in somatic cells. Telomerase adds repetitive DNA sequences to the 5′ end of chromosomes (Option B): Incorrect because telomerase extends the 3′ end , not the 5′ end . The shortening of telomeres occurs due to the continuous synthesis of the lagging strand (Option D): Incorrect because telomeres shorten due to incomplete lagging strand synthesis, not its continuous synthesis.

References:

1. Harper’s Illustrated Biochemistry, 32nd Edition, Page 365 2. Textbook of Biochemistry for Medical Students, Vasudevan, 7th Edition, Page 585 3. www.ncbi.nlm.nih.gov/pmc/articles/PMC7139734/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q14: Which of the following statements about DNA polymerases in E. coli is true? (Q843168)

A. DNA Polymerase I is the primary enzyme for DNA synthesis at the replication fork.

B. DNA Polymerase II is primarily responsible for gap filling following DNA replication

C. DNA Polymerase III has the highest rate of chain elongation and is involved in both leading and lagging strand synthesis.

D. All three DNA polymerases perform identical functions in DNA synthesis.

Correct Answer: C. DNA Polymerase III has the highest rate of chain elongation and is involved in both leading and lagging strand synthesis.

■ *Listen to Audio 1*

Explanation: DNA Polymerase III is the main replicative enzyme in E. coli. DNA Polymerases in E. coli: DNA Polymerase I (pol I): (Option A ruled out) Involved in proofreading and DNA repair. Plays a role in gap-filling after DNA replication and recombination. DNA Polymerase II (pol II): (Option B ruled out) Primarily functions in proofreading and DNA repair. DNA Polymerase III (pol III): Main enzyme for DNA synthesis at the replication fork. Exhibits the highest chain elongation rate and processivity. Participates in both leading and lagging strand synthesis. High processivity due to association with β subunits of the sliding clamp, enabling it to add a large number of nucleotides (100 to >50,000) before detaching. All three DNA polymerases perform identical functions in DNA synthesis (Option D): They have distinct roles, with pol III being the main replicative polymerase while pol I and II focus on proofreading and repair.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 373, 374

↑ *Back to this question in Part A*

@Neet_pg_bot

Q15: Which of the following disorders is primarily caused by a defect in the RecQ DNA helicase (BLM protein), leading to a profound deficiency in homologous recombination (HR) and characterized by increased sister chromatid exchanges?

(Q842887)

A. Bloom syndrome

B. Ataxia telengectasia

C. LIG4 syndrome

D. Artemis-SCID

Correct Answer: A. Bloom syndrome

■ *Listen to Audio 1*

Explanation: Bloom syndrome is a rare autosomal recessive disorder caused by mutations in the BLM gene, which encodes a RecQ family DNA helicase involved in homologous recombination (HR)–mediated DNA repair. The BLM helicase normally unwinds DNA during replication and recombination to maintain genomic stability. Loss of BLM function causes: Defective homologous recombination repair, Chromosomal instability, Markedly increased sister chromatid exchanges (SCEs), and Predisposition to malignancies (especially leukemias, lymphomas, and GI cancers). Characteristic Features of Bloom Syndrome: Short stature and photosensitive facial rash (butterfly pattern) Immunodeficiency Genomic instability with ↑ SCE frequency (10× normal) High cancer susceptibility Ataxia-telangiectasia (Option B): Defect in the ATM gene, affecting DNA double-strand break signaling (NHEJ pathway). LIG4 syndrome (Option C): Mutation in DNA ligase IV, impairs NHEJ, leading to microcephaly and immunodeficiency. Artemis-SCID(Option D): Mutation in Artemis gene, causes defective V(D)J recombination (NHEJ defect), resulting in severe combined immunodeficiency.

References:

1. Harper's Illustrated Biochemistry, 32nd Edition, Page 380-382 2. Lippincott Illustrated Reviews: Biochemistry, 7th Edition, Page 1226

↑ *Back to this question in Part A*

@Neet_pg_bot

Q16: Which of the following is not a requirement for the activity of DNA polymerase during DNA replication? (Q843165)

A. A primer with a free 3′-OH group

B. A single-stranded DNA template

C. Free ribonucleoside triphosphates (rNTPs)

D. A divalent cation cofactor, typically Mg^{2}■

Correct Answer: C. Free ribonucleoside triphosphates (rNTPs)

■ *Listen to Audio 1*

Explanation: DNA polymerase requires deoxynucleoside triphosphates (dNTPs), not ribonucleoside triphosphates, which are used in RNA synthesis. DNA Polymerase requirements & function : Synthesis Direction : DNA polymerases synthesize DNA in the 5′ to 3′ direction, adding nucleotides to the 3′ end of a DNA strand. Primer Requirement : DNA polymerases require a short RNA primer, synthesized by primase, to initiate DNA synthesis. Primer Binding : The 3′-hydroxyl group of the RNA primer initiates the nucleophilic attack on the first deoxynucleoside triphosphate (dNTP). Template Requirement : A single-stranded DNA template is necessary to guide the order of dNTP addition. (Option B ruled out) dNTPs : The four dNTPs required for DNA synthesis are dATP, dGTP, dCTP, and dTTP. Cofactor : DNA polymerases require a divalent cation cofactor, typically Mg^{2}■. (Option D ruled out) Properties : Chain elongation, processivity, and proofreading ensure DNA replication and repair accuracy and efficiency. Replication Fork Dynamics: Leading Strand: Synthesized continuously 5'→3' toward the replication fork. Lagging Strand: Synthesized discontinuously away from the fork, creating Okazaki fragments . Enzyme Roles: Helicase: Unwinds DNA. Primase: Lays down RNA primers. DNA Polymerase: Extends strands. Ligase: Joins Okazaki fragments by forming phosphodiester bonds. A primer with a free 3′-OH group (Option A): DNA polymerase cannot initiate DNA synthesis de novo and requires a primer to provide a free 3′-OH group to which it can add nucleotides.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 372, 373, 374, 387

www.ncbi.nlm.nih.gov/pmc/articles/PMC3030377/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: A 45 y/o woman presents with visual disturbances, including gradual vision loss and difficulty seeing in low light, along with muscle weakness and fatigue. She has a history of early-onset cardiac conduction issues and worsening symptoms over the years. Her family history shows similar symptoms in her mother. What genetic condition best explains her symptoms? (Q843164)

A. Leber’s Hereditary Optic Neuropathy (LHON)

B. Myoclonic Epilepsy with Ragged Red Fibers (MERRF)

C. Mitochondrial Encephalopathy, Lactic Acidosis, and Stroke-like Episodes (MELAS)

D. Leigh’s Syndrome.

Correct Answer: A. Leber’s Hereditary Optic Neuropathy (LHON)

■ *Listen to Audio 1*

Explanation: Visual Disturbances : Gradual vision loss and difficulty seeing in low light are characteristic of LHON , a mitochondrial disorder affecting the optic nerve. Muscle Weakness and Fatigue : Mitochondrial dysfunction commonly leads to neuromuscular symptoms . Cardiac Conduction Issues : Some cases of LHON involve cardiac arrhythmias , particularly pre-excitation syndromes . Maternal Inheritance : Mitochondrial diseases, including LHON, follow maternal inheritance , consistent with the family history in her mother. Mitochondrial Syndromes and Their Features Syndrome Genetic Defect Key Clinical Features Leber's Hereditary Optic Neuropathy (LHON) mtDNA mutations in ND1, ND4, ND6 (Complex I subunits) Painless, progressive vision loss , optic atrophy, cardiac conduction defects , maternal inheritance Myoclonic Epilepsy with Ragged Red Fibers (MERRF) (Option B ruled out) mtDNA mutations in MT-TK (tRNA-Lys gene) Myoclonic epilepsy, muscle weakness , ataxia, hearing loss, ragged red fibers on muscle biopsy Mitochondrial Encephalopathy, Lactic Acidosis, and Stroke-like Episodes (MELAS) (Option C ruled out) mtDNA mutations in MT-TL1 (tRNA-Leu gene) Stroke-like episodes, lactic acidosis , seizures, progressive dementia, diabetes, muscle weakness Leigh's Syndrome (Option D ruled out) mtDNA or nuclear DNA mutations (Complex I or IV defects, NDUFS genes) Neurodegeneration in infancy , hypotonia, brainstem dysfunction, ataxia, respiratory failure Kearns-Sayre Syndrome (KSS) Large-scale mtDNA deletions Progressive external ophthalmoplegia, pigmentary retinopathy, cardiac conduction defects, muscle weakness

References:

Textbook of Biochemistry for Medical Students, Vasudevan, 7th Edition, Page 606

↑ *Back to this question in Part A*

@Neet_pg_bot

Q18: Which of the following is a characteristic of mitochondrial DNA (mtDNA)? (Q843162)

A. It is linear and single-stranded

B. It has very few non-coding regions and a higher mutation rate compared to nuclear DNA.

C. It uses the standard genetic code, including AGA and AGG as codons for arginine (Arg).

D. It is composed of linear strands and encodes for all 67 subunits of the respiratory chain.

Correct Answer: B. It has very few non-coding regions and a higher mutation rate compared to nuclear DNA.

■ *Listen to Audio 1*

Explanation: It has very few non-coding regions and a higher mutation rate compared to nuclear DNA. Explantion: Mitochondrial DNA is characteristic of having very few non-coding regions and a higher mutation rate of 5-10 times compared to nuclear DNA. Feature Description Structure Circular, double-stranded; composed of heavy (H) and light (L) chains. (Option A ruled out) Length 16,569 base pairs (bp). Protein-Encoding Subunits Most respiratory chain proteins are encoded by nuclear DNA and imported into mitochondria (Option D ruled out) Ribosomal RNAs Encodes 16S (large) and 12S (small) mitochondrial rRNAs. tRNAs Encodes 22 mitochondrial

tRNA molecules. Genetic Code Differences UGA typically a stop codon is read as tryptophan (Trp) AGA and AGG: normally arginine (Arg) codons are read as stop codons (Option C ruled out) Untranslated Sequences Very few non-coding regions. Mutation Rate 5-10 times higher than nuclear DNA. Evolutionary Studies Comparisons of mtDNA sequences provide insights into the evolutionary history of primates and other species.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 369

↑ *Back to this question in Part A*

@Neet_pg_bot

Q19: Which histone is least tightly bound to chromatin and can be easily removed with a salt solution? (Q843161)

A. H2A

B. H1

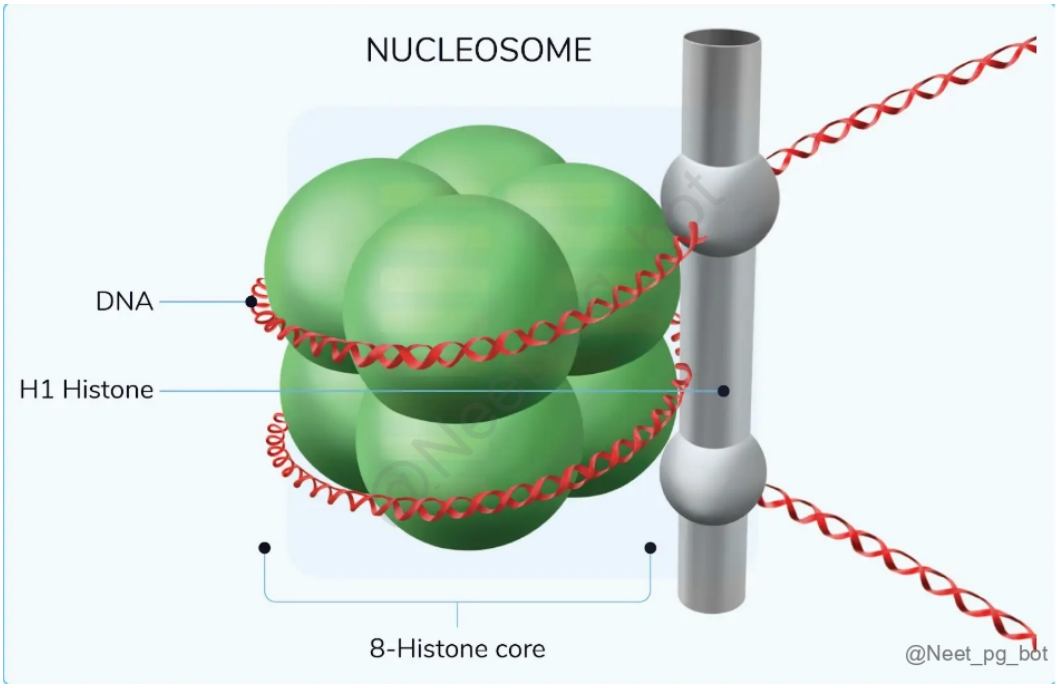
C. H3

D. H4

Correct Answer: B. H1

■ *Listen to Audio 1*

Explanation: H1 is a linker histone , not part of the core histone octamer. It binds to DNA at nucleosome entry and exit sites , helping in higher-order chromatin organization. Least tightly bound to chromatin and can be easily removed with a salt solution , increasing chromatin solubility. Histone Classification and Role Histone Type Function Binding to DNA H1 (Linker Histone) Stabilizes nucleosomes, promotes chromatin compaction Least tightly bound , removable with salt solutions H2A, H2B (Core Histones) (Option A ruled out) Form histone octamer, aid in nucleosome structure Tightly bound H3, H4 (Core Histones) (Options C & D ruled out) Critical for nucleosome stability and gene regulation Tightly bound



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 361, 363

↑ *Back to this question in Part A*

@Neet_pg_bot

Q20: Which of the following factors can cause the denaturation of DNA? a) Increasing temperature b) Increasing salt concentration c) Decreasing temperature d) DNA with a high A-T base composition e) DNA with a high G-C base composition f) Adding Urea g) Adding Formamide h) Decreasing salt concentration (Q843160)

A. b, c, e, g, h

B. c, d, e, h

C. a, d, f, g, h

D. a, b, e, g

Correct Answer: C. a, d, f, g, h

■ *Listen to Audio 1*

Explanation: Increasing temperature, Decreasing salt concentration, DNA with a high A-T base composition, and Adding Chaotropic agents like Urea and Formamide cause denaturation of DNA. Factors Influencing Denaturation

of DNA Increasing Temperature : Higher temperatures cause the double-stranded DNA to separate into single strands. Decreasing Solution Salt Concentrations : Lower salt concentrations reduce the stability of the double-stranded DNA, promoting denaturation. Adding Chaotropic Agents : Agents like urea and formamide form competing hydrogen bonds with nucleotide bases, destabilizing the hydrogen bonding between complementary bases and lowering the melting temperature (T m). Base Composition : DNA rich in G-C pairs melts at a higher temperature than DNA rich in A-T pairs due to stronger hydrogen bonding and base stacking interactions in G-C pairs. Denaturation: In Vivo Denaturation : In living cells, DNA denaturation and renaturation occur naturally during DNA replication, recombination, repair, and transcription. These processes are mediated by nucleic acid binding proteins, enzymes, and thermal or chemical energy from ATP hydrolysis. Experimental Denaturation : In the laboratory, a combination of increasing temperature, decreasing salt concentration, and adding chaotropic agents is often used to denature DNA, ensuring the strands separate efficiently without causing significant damage to the nucleotides.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 350, 351

↑ *Back to this question in Part A*

@Neet_pg_bot

Q21: Which of the following statements about the different types of DNA is TRUE? (Q843159)

A. B-DNA has a left-handed helix and a full turn with 12 bases.

B. A-DNA forms during dehydration and has grooves of the same dimensions.

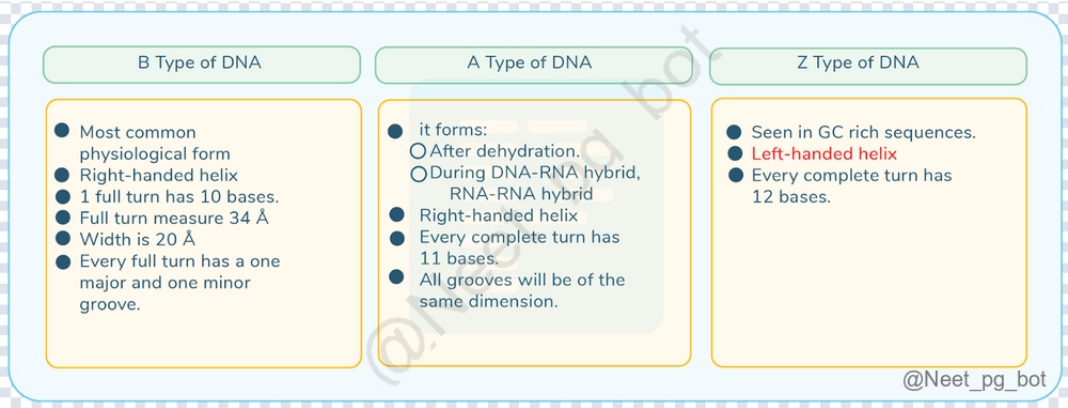
C. Z-DNA forms under normal physiological conditions and has a right-handed helix

D. A-DNA is the predominant form found in physiological conditions and has 11 bases per full turn.

Correct Answer: B. A-DNA forms during dehydration and has grooves of the same dimensions.

■ *Listen to Audio 1*

Explanation: A-DNA is a right-handed DNA helix that forms under dehydrated conditions. It has 11 base pairs per turn and a more compact structure than B-DNA. Unlike B-DNA, A-DNA has grooves of nearly equal dimensions (major and minor grooves are not as distinct).



References:

1. Harper’s Illustrated Biochemistry, 32nd Edition, Page 350 2. DNA: Alternative Conformations and Biology - Madame Curie Bioscience Database - NCBI Bookshelf (nih.gov) 3. B-DNA succumbs to form A-DNA in dehydrated conditions

↑ *Back to this question in Part A*

@Neet_pg_bot

Q22: In the analogy of the DNA double helix to a spiral staircase, what do the "handrails" and "steps" represent? (Q843157)

A. Handrails are nitrogenous bases; steps are phosphodiester bonds

B. Handrails are nitrogenous bases; steps are the sugar-phosphate backbone

C. Handrails are the sugar-phosphate backbone; steps are nitrogenous bases

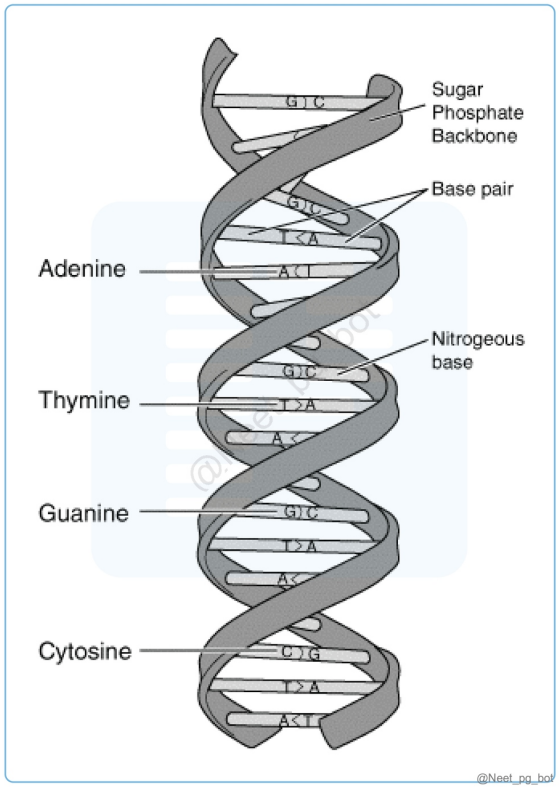
D. Handrails are hydrogen bonds; steps are deoxyribose sugars

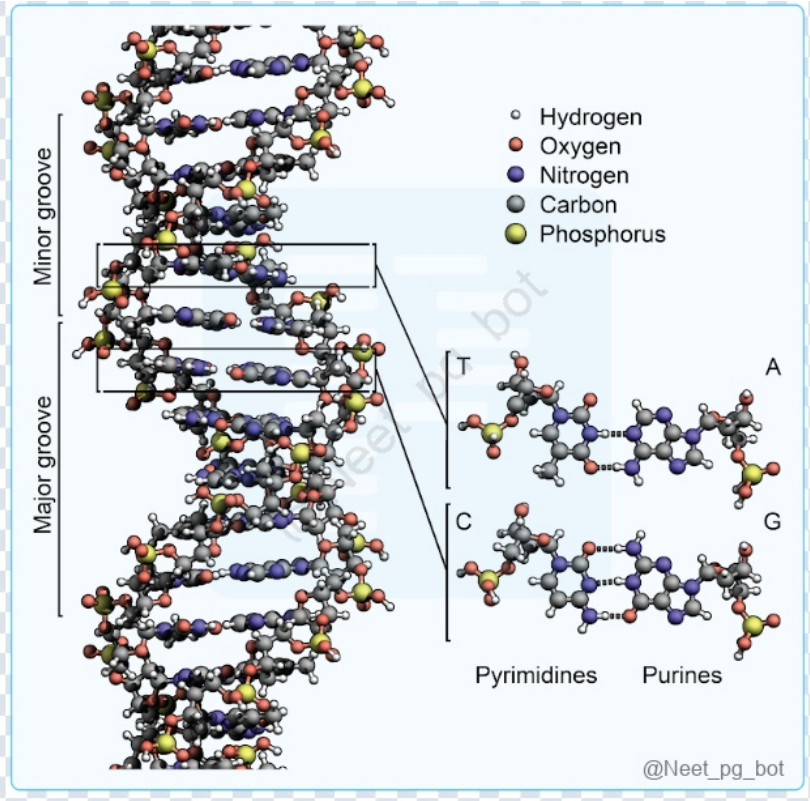
Correct Answer: C. Handrails are the sugar-phosphate backbone; steps are nitrogenous bases

■ *Listen to Audio 1*

Explanation: Handrails (Sugar-Phosphate Backbone): The sugar-phosphate backbone forms the structural framework of the DNA double helix. It consists of deoxyribose sugars connected by phosphodiester bonds. Steps (Nitrogenous Bases): The nitrogenous bases (adenine, thymine, cytosine, guanine) form the "steps" of the staircase. They pair via hydrogen bonds (A-T with two bonds, G-C with three bonds), connecting the two strands. Structural Features of the DNA Molecule: The DNA molecule consists of two antiparallel strands : one runs 5' to 3' , while the complementary strand runs 3' to 5' . It resembles a spiral staircase , where two polynucleotide chains twist together in a right-handed helix . The DNA helix has distinct major (2.2 nm) and minor (1.2 nm) grooves , providing protein-binding sites . The double helix is stabilized by base stacking , involving hydrophobic interactions and the specific arrangement of base ring systems . Each complete turn of the helix spans 3.4 nm and

includes ~10.5 base pairs . The helical diameter of B-DNA is 2 nm (20 Å) . Handrails are nitrogenous bases; steps are phosphodiester bonds (Option A): Incorrect because phosphodiester bonds form the backbone, not the steps. Handrails are nitrogenous bases; steps are the sugar-phosphate backbone (Option B): Incorrect because nitrogenous bases form the steps, not the handrails. Handrails are hydrogen bonds; steps are deoxyribose sugars (Option D): Incorrect because hydrogen bonds connect bases, and deoxyribose is part of the backbone, not the steps.





References:

1. Harper's Illustrated Biochemistry, 32nd Edition, Page 349, 335, 350 2. Textbook of Biochemistry- Vasudevan, 7th Edition, Page 575

↑ *Back to this question in Part A*

@Neet_pg_bot

Q23: In the structure of nucleotides, where does the esterification of the phosphate group typically occur? (Q843154)

A. On the 1' carbon of the pentose sugar

B. On the 2' hydroxyl group of the pentose sugar

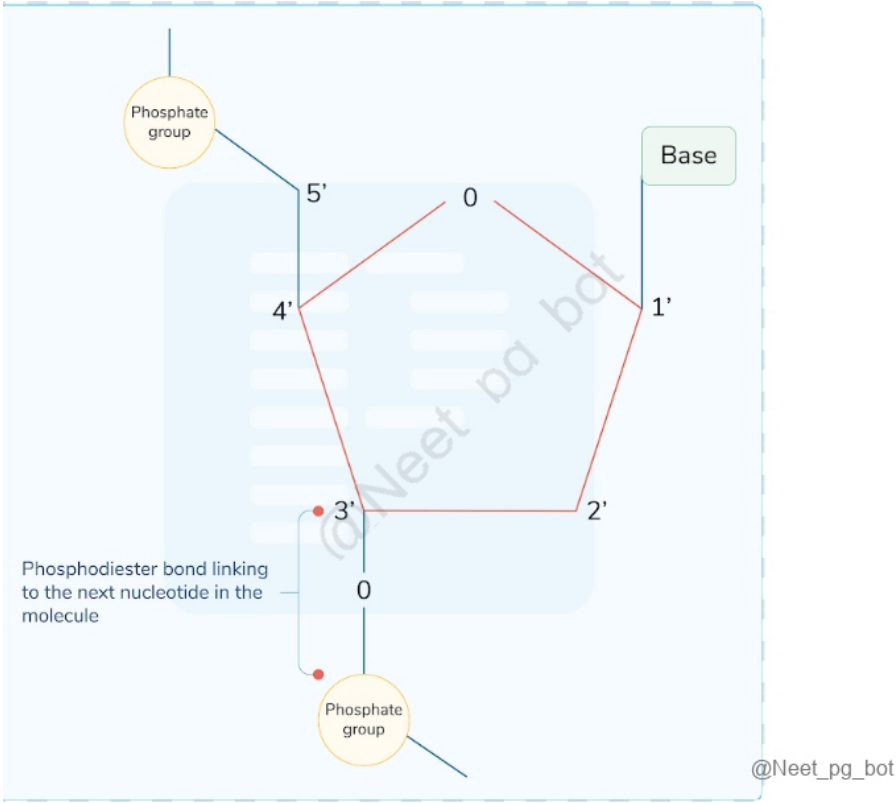
C. On the 5' or 3' hydroxyl group of the pentose sugar

D. On the N1 position of the nitrogenous base

Correct Answer: C. On the 5' or 3' hydroxyl group of the pentose sugar

■ *Listen to Audio 1*

Explanation: Phosphate groups in nucleotides are most commonly esterified to the 5' hydroxyl group of the pentose sugar, forming 5'-nucleotides. The 3' hydroxyl group can also participate in forming phosphodiester bonds, linking nucleotides together in nucleic acids. {{caption_text}} The 1' carbon of the pentose sugar (Option A): It is involved in forming the β-N-glycosidic bond with the nitrogenous base, not in esterification with the phosphate group. 2' hydroxyl group is present in ribose (in RNA) (Option B): it is not typically the site for phosphate esterification. The 2' position plays a role in distinguishing RNA from DNA, as deoxyribose lacks a hydroxyl group at this position. The N1 position (Option D): It is involved in attaching the base to the pentose sugar in pyrimidines, not in phosphate esterification. In purines, the corresponding attachment occurs at the N9 position.



References:

1. Harper’s Illustrated Biochemistry, 32nd Edition, Page 330, 348 2. Textbook of Biochemistry- Vasudevan, 7th Edition, Page 559, 561

↑ *Back to this question in Part A*

@Neet_pg_bot

Q24: What is the primary role of inosine monophosphate (IMP) in cellular metabolism? (Q843153)

A. Directly involved in energy transfer like ATP

B. Acts as a precursor for both adenosine monophosphate (AMP) and guanosine monophosphate (GMP)

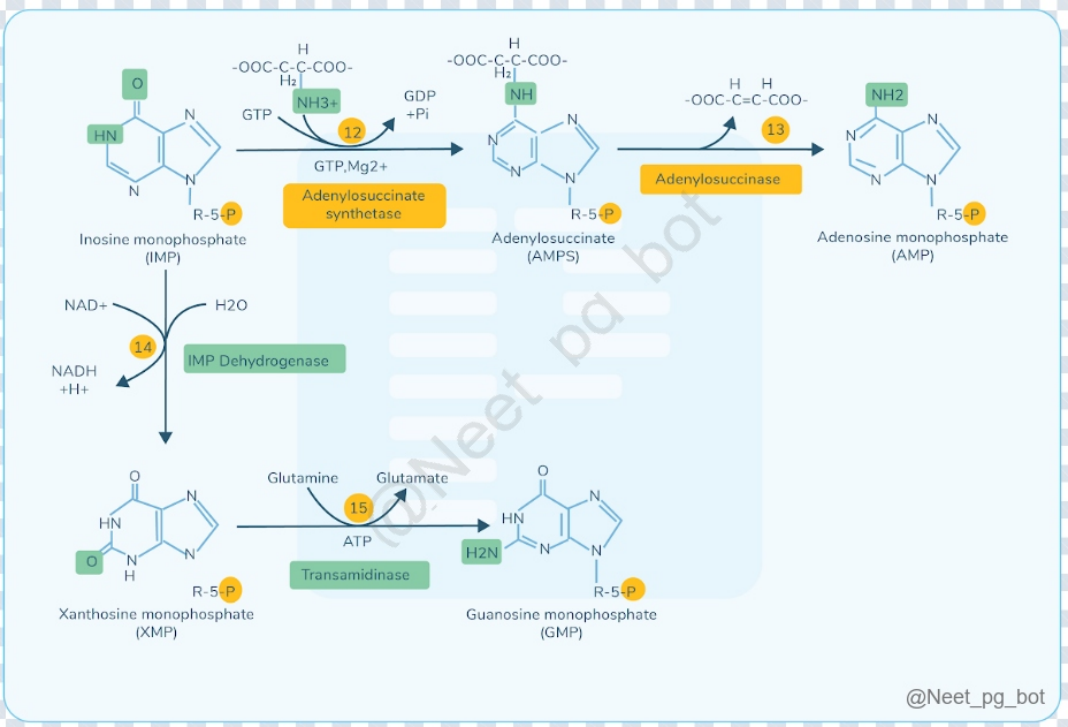
C. Inhibits the de novo synthesis of purine nucleotides

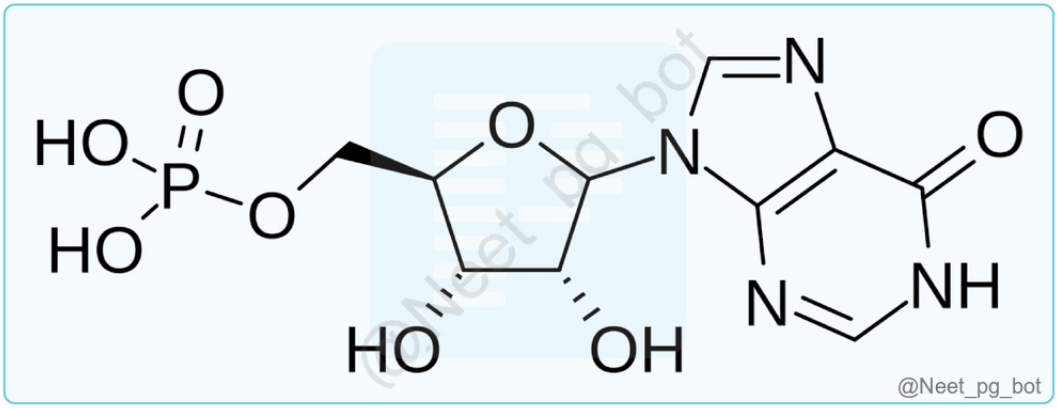
D. Serves as an end product of the salvage pathway for purine nucleotides

Correct Answer: B. Acts as a precursor for both adenosine monophosphate (AMP) and guanosine monophosphate (GMP)

■ *Listen to Audio 1*

Explanation: IMP is a central molecule in purine metabolism, serving as a precursor for AMP and GMP, which are crucial for various cellular processes, including nucleic acid synthesis and energy transfer. 1. De Novo Synthesis & Regulation: The Pathway: Purine synthesis begins with Phosphoribosyl pyrophosphate , which is converted to 5-phosphoribosylamine by the rate-limiting enzyme Amidophosphoribosyltransferase . Regulation (Feedback Inhibition): The synthesis is tightly regulated. IMP, AMP, and GMP all provide feedback inhibition of Amidophosphoribosyltransferase to prevent overproduction. Energy Cross-Regulation: To maintain a balanced ratio, the pathway to AMP requires GTP as an energy source, while the pathway to GMP requires ATP. This ensures that the cell produces one purine only when the other is available. (Option C ruled out) 2. Purine Catabolism: Purine nucleotides (AMP, GMP, IMP) are degraded by nucleotidases into their respective nucleosides. IMP's Catabolic Fate: IMP is converted to Inosine → Hypoxanthine . The Final Step: Hypoxanthine along with Guanine is converted to Xanthine and ultimately to Uric Acid by the enzyme Xanthine Oxidase. Conversion of IMP to AMP and GMP: Structure Of IMP: Directly involved in energy transfer like ATP (Option A) : While ATP is essential for energy transfer, IMP does not serve this function directly. IMP's primary role is as an intermediate in purine metabolism. Serves as an end product of the salvage pathway for purine nucleotides (Option D): Incorrect because IMP is an intermediate in the salvage pathway, not the final product.





References:

1. Harper’s Illustrated Biochemistry, 32nd Edition, Page 338, 340, 341 2. Textbook of Biochemistry- Vasudevan, 7th Edition, Page 564, 565

↑ *Back to this question in Part A*

@Neet_pg_bot

Q25: What type of bond forms between a nitrogenous base and a pentose sugar in a nucleoside? (Q843151)

A. α-N-glycosidic bond

B. β-N-glycosidic bond

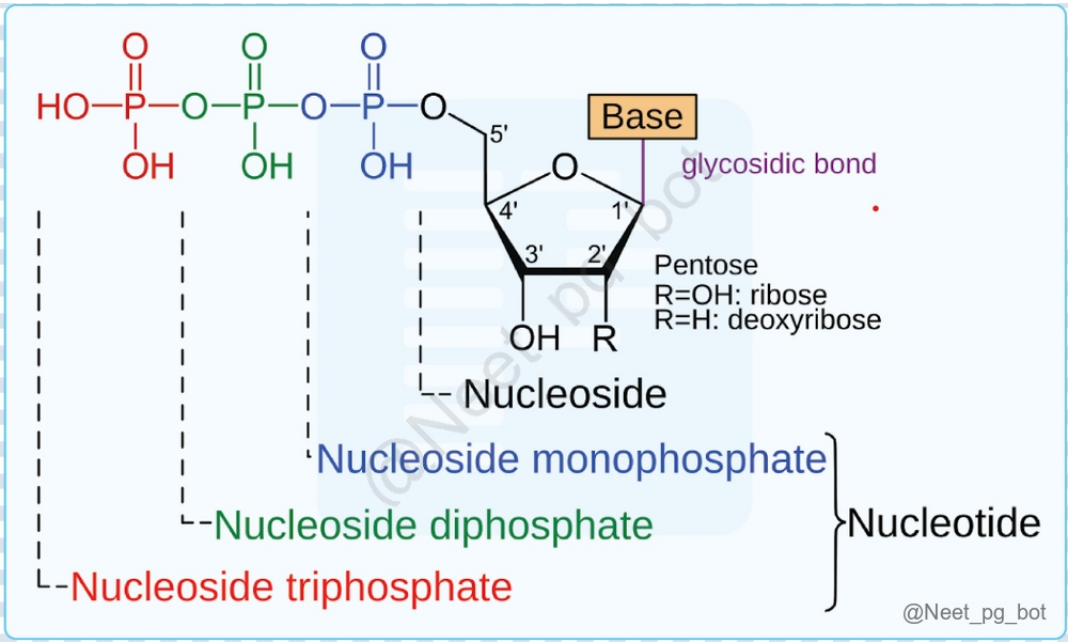
C. Phosphodiester bond

D. Hydrogen bond

Correct Answer: B. β-N-glycosidic bond

■ *Listen to Audio 1*

Explanation: β-N-glycosidic bond is the bond that connects the nitrogenous base to the pentose sugar in nucleosides. It links the 1' carbon of the sugar (ribose or deoxyribose) to the nitrogen atom of the base- N9 of purines or N1 of pyrimidines. It is called a β linkage because the OH group at the 1' position of ribose or deoxyribose is above the plane of the sugar ring. This orientation determines the β configuration of the glycosidic bond. {{caption_text}} α-N-glycosidic bond (Option A) : This bond is not found between nitrogenous bases and pentose sugars in nucleosides. Phosphodiester bond (Option C) : This bond links nucleotides together to form the backbone of nucleic acids (DNA and RNA). It connects the 5' phosphate group of one nucleotide to the 3' hydroxyl group of the next nucleotide. Hydrogen bond (Option D) : Hydrogen bonds are responsible for the base pairing between complementary nitrogenous bases in DNA or RNA. They do not connect the base to the sugar in a nucleoside.



References:

1. Harper's Illustrated Biochemistry, 32nd Edition, Page 330,348,249 2. Textbook of Biochemistry- Vasudevan, 7th Edition, Page 560

↑ *Back to this question in Part A*

@Neet_pg_bot

Q26: Which of the following is a pyrimidine base found in nucleic acids? (Q843147)

A. Uracil

B. Hypoxanthine

C. Xanthine

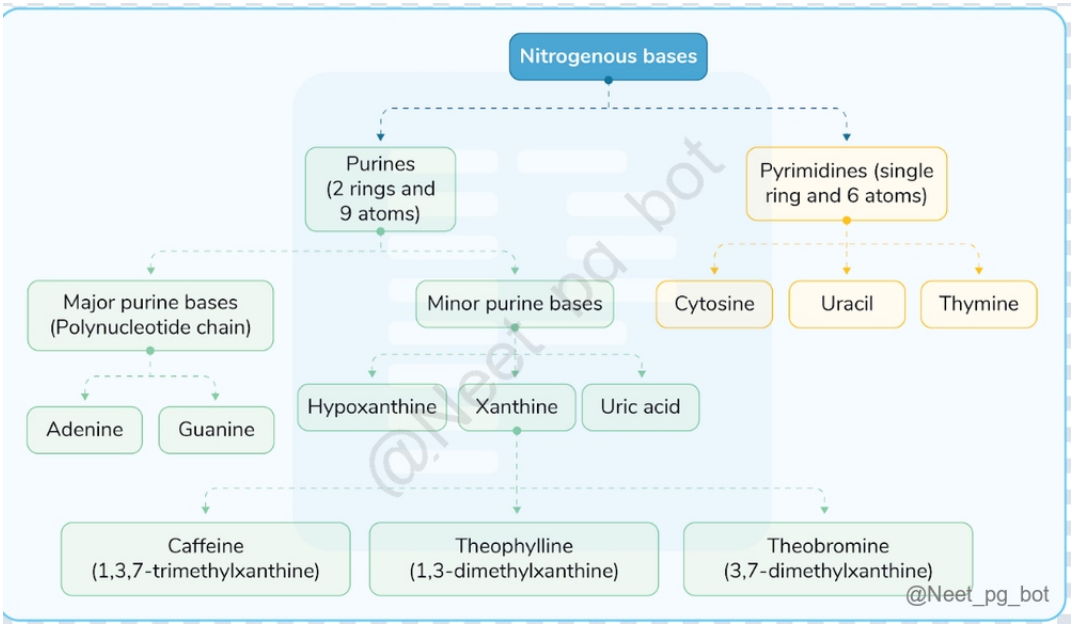
D. Guanosine

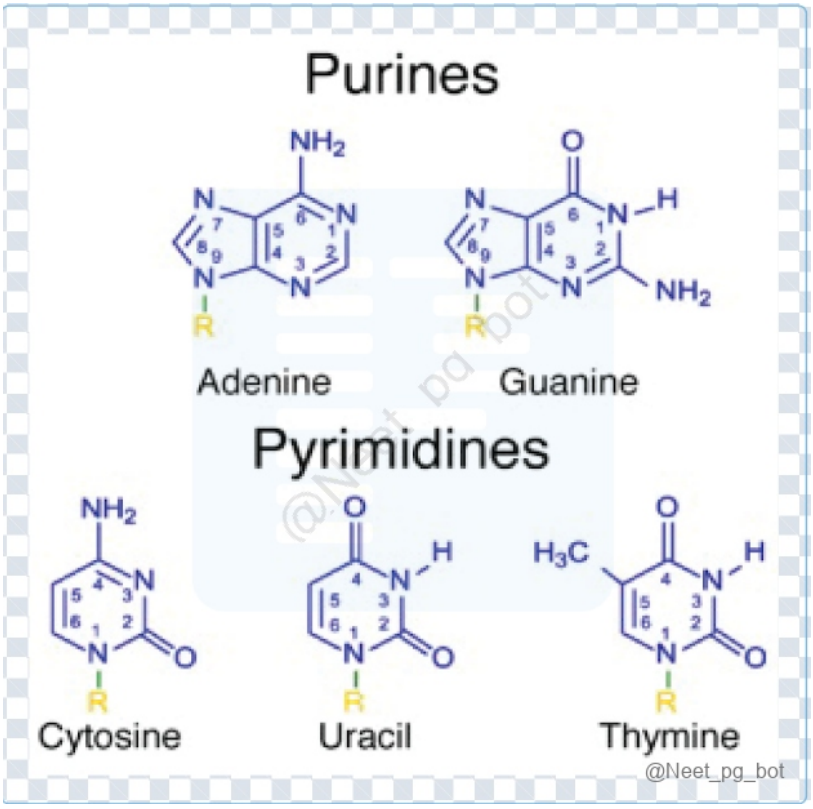
Correct Answer: A. Uracil

■ *Listen to Audio 1*

Explanation: Uracil is a pyrimidine base found in RNA , where it replaces thymine as a complementary base to adenine. It is a single-ring nitrogenous base, characteristic of pyrimidines. Classification of Nitrogenous Bases {{caption_text}} Hypoxanthine (Option B): It is a purine derivative , not a primary component of DNA or RNA. It appears in tRNA and purine metabolism as an intermediate. Xanthine (Option C): It is a purine base involved in purine degradation but not directly incorporated into nucleic acids. It is also a precursor for caffeine, theophylline, and theobromine. Guanosine (Option D): It is a nucleoside , consisting of guanine (a purine) attached to ribose.

While guanine is found in DNA and RNA, guanosine itself is not a base but a nucleoside.





References:

1. Harper's Illustrated Biochemistry, 32nd Edition, Page 330,331 2. Textbook of Biochemistry- Vasudevan, 7th Edition, Page 560,561

↑ *Back to this question in Part A*

@Neet_pg_bot

Q27: Which of the following is not caused by a defect of ds DNA repair? (Q842892)

A. Cockayne syndrome (CS)

B. Fanconi’s anemia

C. Severe Combined Immunodeficiency (SCID)

D. Bloom's syndrome

Correct Answer: A. Cockayne syndrome (CS)

■ *Listen to Audio 1*

Explanation: Cockayne syndrome (CS) is caused by a defect in Transcription-Coupled Nucleotide Excision Repair (TC-NER) , which is a type of Nucleotide Excision Repair (NER) . It does not involve defects in double-strand DNA (dsDNA) repair mechanisms like Homologous Recombination (HR) or Non-Homologous End Joining (NHEJ) . Presents with bird-like facies , severe growth retardation (cachectic dwarfism) , photosensitivity, microcephaly, and progressive neurodegeneration. Does not involve defects in global genome repair → do not exhibit the massive increase in skin cancer risk. Conditions Associated with Nucleotide Excision Repair: Xeroderma Pigmentosum (XP): Defect in Global Genome NER, characterized by extreme UV sensitivity, early-onset freckling, and a very high risk of basal/squamous cell carcinoma and melanoma. Trichothiodystrophy (TTD): Defect in NER (often TFIIH complex). Characterized by the sulfur-deficient triad: Brittle hair (tiger-tail banding) Ichthyosis Developmental delay/intellectual disability. Disorders Associated with dsDNA Repair Defects Disorder Defective Repair Mechanism Ataxia telangiectasia Non-Homologous End Joining (NHEJ) Bloom’s syndrome (Option D ruled out) Homologous DNA Repair (HDR) Severe Combined Immunodeficiency (SCID) (Option C ruled out) Non-Homologous End Joining (NHEJ) Fanconi’s anemia (Option B ruled out) Fanconi Anemia Repair Pathway (Interacts with HDR) HBOC (Human Breast and Ovarian Cancer Syndrome) Homologous DNA Repair (HDR) (BRCA1/BRCA2 mutation)

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 380

↑ *Back to this question in Part A*

@Neet_pg_bot

Q28: Which of the following statements about Homologous DNA Repair (HR) is correct? (Q842890)

A. HR is active during G0/G1 phases of the cell cycle.

B. HR is an error-prone process compared to nonhomologous end joining (NHEJ).

C. HR uses a homologous DNA template for repair, making it highly accurate.

D. Defects in HR-related proteins decrease Ovarian and breast cancers

Correct Answer: C. HR uses a homologous DNA template for repair, making it highly accurate.

■ *Listen to Audio 1*

Explanation: HR uses enzymes involved in genetic recombination between homologous chromosomes and uses a homologous DNA template for repair, making it highly accurate. Homologous DNA Repair (HR) Definition: HR is a type of Double-strand DNA break repair mechanism used to fix double-strand breaks (DSBs) by utilizing a homologous DNA template for error-free repair. Cell Cycle Phase : HR is active during the S and G2 phases of the cell cycle. (Option A ruled out) Process: Uses enzymes involved in genetic recombination between homologous chromosomes during meiosis. DNA damage is recognized, and the homologous sequence is used as a template for repair. Accuracy: HR is less error-prone compared to nonhomologous end joining (NHEJ), making it an "error-free" repair process. (Option B ruled out) Clinical Relevance: Mutations in HR-related proteins (e.g., BRCA1/BRCA2) increase the risk for breast and ovarian cancers . (Option D ruled out) Fanconi's anemia HBOC (Human Breast and Ovarian Cancer Syndrome) Advantages: Ensures high fidelity in DNA repair by accurately restoring the original DNA sequence using a homologous template. Holliday junction is a cross-shaped structure formed during the process of genetic r ecombination when two double-stranded DNA molecules become separated into four strands to exchange segments of genetic information.

References:

1. Harper's Illustrated Biochemistry, 32nd Edition, Page 380 2. Lippincott Illustrated Reviews: Biochemistry, 7th Edition, Page 1219-1222 3. https://www.britannica.com/science/Holliday-junction

↑ *Back to this question in Part A*

@Neet_pg_bot

• [07_ Genetics] 022 Rna Transcription Genetic Code Mutation Translation Inhibitors

« Back to Folder Directory

[07_ Genetics] 022 Rna Transcription Genetic Code Mutation Translation Inhibitors — Quick Jump

Q1 (p. 1043)	*Q2 (p. 1044)*	*Q3 (p. 1045)*	*Q4 (p. 1046)*	*Q5 (p. 1047)*	*Q6 (p. 1048)*
Q7 (p. 1049)	*Q8 (p. 1049)*	*Q9 (p. 1051)*	*Q10 (p. 1051)*	*Q11 (p. 1052)*	*Q12 (p. 1053)*
Q13 (p. 1054)	*Q14 (p. 1055)*	*Q15 (p. 1055)*	*Q16 (p. 1057)*	*Q17 (p. 1058)*	*Q18 (p. 1059)*
Q19 (p. 1060)	*Q20 (p. 1062)*	*Q21 (p. 1063)*	*Q22 (p. 1064)*	*Q23 (p. 1064)*	*Q24 (p. 1065)*
Q25 (p. 1066)	*Q26 (p. 1068)*	*Q27 (p. 1069)*	*Q28 (p. 1071)*		

Q1: Which of the RNA Polymerase is responsible for synthesis of rRNA? (Q843574)

A. RNA polymerase I

B. RNA polymerase II

C. RNA polymerase III

D. RNA polymerase IV

Correct Answer: A. RNA polymerase I

Explanation: RNA polymerase I: Responsible for the synthesis of rRNA (ribosomal RNA) in eukaryotic cells. Transcribes rRNA genes in the nucleolus. Produces the 45S rRNA precursor, which is processed into 18S, 5.8S, and 28S rRNAs, key components of ribosomes. RNA polymerase II: Synthesizes mRNA (messenger RNA) and some small nuclear RNAs. RNA polymerase III: Responsible for synthesizing tRNA (transfer RNA) and 5S rRNA. RNA polymerase IV: Found in plants. Involved in the synthesis of small interfering RNAs (siRNAs). Classification of RNAPs based on amanitin sensitivity: RNA Polymerase Sensitivity to α-Amanitin Primary Products RNA Polymerase I Resistant rRNA (28S, 18S, 5.8S) RNA Polymerase II Highly sensitive mRNA (messenger RNA) snRNA (small nuclear RNA) miRNA (microRNA) RNA Polymerase III Moderately sensitive tRNA (transfer RNA) 5S rRNA snRNA (U6)

References:

Harper’s Biochemistry 32e page 387, 388

↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: Original DNA sequence: 5'-ATG GCA TCG GAA UAC-3'. Mutated sequence: 5'-ATG GCA TCG GAA UAA-3'. Identify the mutation. (Q843045)

A. Frameshift mutation

B. Nonsense mutation

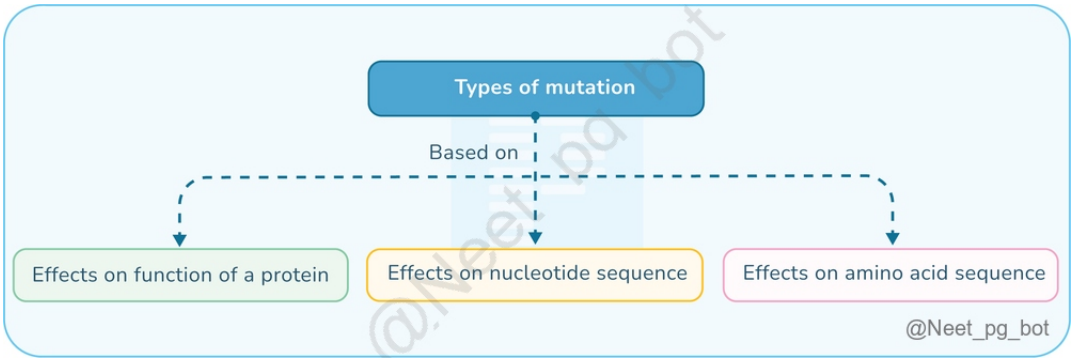
C. Silent mutation

D. Missense mutation

Correct Answer: B. Nonsense mutation

■ *Listen to Audio 1*

Explanation: UAC mutates to a termination codon (UAA or UAG), leading to premature termination of the protein and destroying the functional activity. This is called Nonsense mutation. Basis Type of Mutation Description Based on the Effects on Amino Acid Sequence Silent Mutation (Option C ruled out) Mutation changes the codon to a synonymous codon for the same amino acid ; no effect on phenotype due to degeneracy of the genetic code. Missense Mutation (Option D ruled out) Different amino acid incorporated due to nucleotide change; may be acceptable, partially acceptable, or unacceptable depending on effect on protein function. Nonsense Mutation (Option B) Codon mutated to a termination codon (UAA or UAG) causing premature termination of translation . Based on Nucleotide Sequence Point Mutation Single base substitution in DNA sequence. Transition Purine ↔ Purine (A↔G) or Pyrimidine ↔ Pyrimidine (C↔T) substitution. Transversion Purine ↔ Pyrimidine (A/G ↔ C/T) substitution. Frameshift Mutation (Option A ruled out) Insertion or deletion not in multiples of 3 , causing shift in reading frame and altered downstream amino acids. Based on Protein Function Acceptable Mutation Amino acid substitution does not significantly affect protein function . Example: Hb Sydney . Partially Acceptable Mutation Amino acid substitution affects protein function but is compatible with life . Example: Sickle Cell Disease (Glu → Val; HbS). Unacceptable Mutation Amino acid substitution severely affects protein function and may be life-threatening . Example: Congenital Methemoglobinemia (Histidine → Tyrosine substitution).



References:

Harper Biochemistry, 32nd Edition, Page 407, 408

↑ *Back to this question in Part A*

Q3: Match the following phases of translation (Column A) with the events involved (Column B). Column A Column B 1. Elongation a. RF1 binds with RF3 and GTP 2. Initiation b. Binding of aminoacyl-tRNA to the ribosome's A site 3. Termination c. Methionine tRNA scans mRNA for AUG codon (Q843048)

A. 1-b, 2-c, 3-a

B. 1-c, 2-a, 3-b

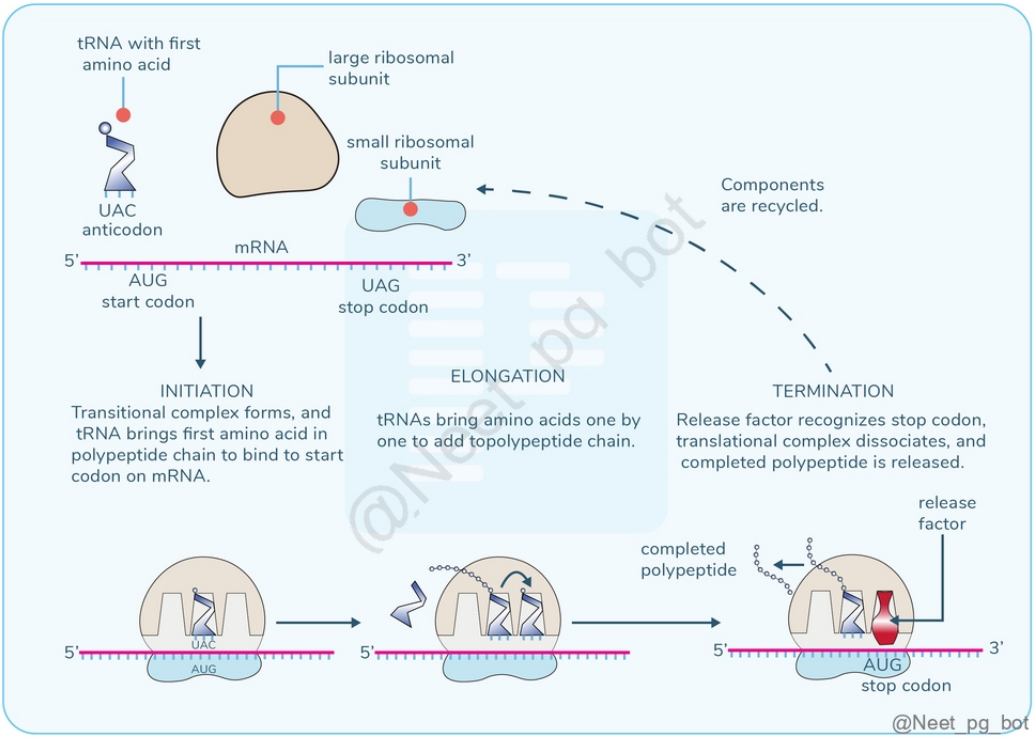
C. 1-b, 2-a, 3-c

D. 1-a, 2-c, 3-b

Correct Answer: A. 1-b, 2-c, 3-a

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Explanation: Phases of translation Key events Initiation Dissociation of 80s ribosome into 60s and 40s, and stabilized by various initiation factors. Formation of the 43S Preinitiation Complex: The 40S ribosomal subunit, in association with initiation factors, binds to the initiator tRNA (carrying methionine in eukaryotes or formyl-methionine in prokaryotes)forming the 43S preinitiation complex. Formation of 48S initiation complex: The 43S complex binds to the 5' end of the mRNA, and scans for start codon which upon recognition, the 48S initiation complex is formed. Formation of the 80S Initiation Complex: Binding of the 60S ribosomal subunit to the 48S initiation. The met-tRNA on the P site of the ribosome is ready for the elongation cycle. Elongation Binding of Aminoacyl-tRNA to the A Site Peptide Bond Formation Elongation factor 2 (EF2) binds to and displaces the peptidyl tRNA from the A site to the P site Termination Ribosome reaches the terminator codon sequence (UAA, UAG or UGA) Releasing factor(RF1), (RF3) and GTP complex promotes hydrolysis releasing the protein and the tRNA from the P site.



References:

Harper's Biochemistry, 32nd Edition, Page 410-414

↑ *Back to this question in Part A*

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Q4: Which of the following best describes the DNA segment from which the primary mRNA transcript is copied or transcribed? (Q876005)

A. Coding strand

B. Initiator methionine domain

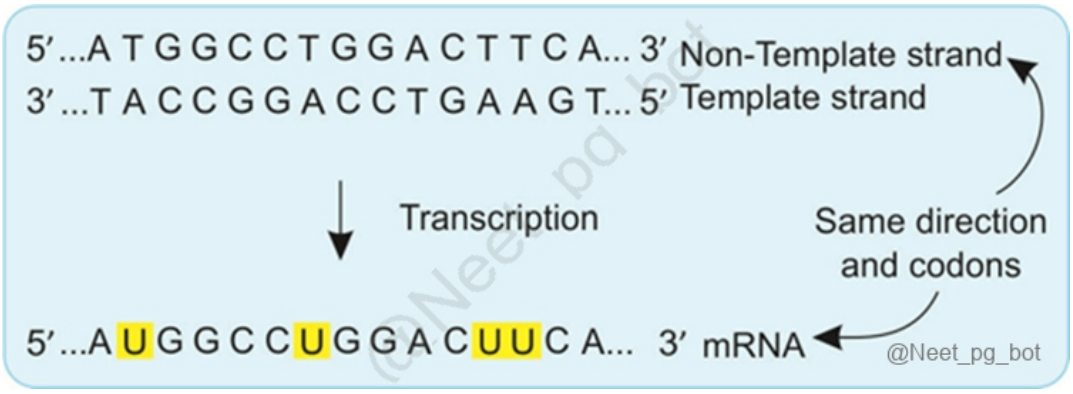
C. Template strand

D. Translational Unit

Correct Answer: C. Template strand

Explanation: Correct option C: Template strand. The DNA segment from which the primary mRNA transcript is copied or transcribed is called as Template strand Template strand: The sequence of ribonucleotides in an RNA molecule is complementary to the sequence of deoxyribonucleotides in one strand of the double-stranded DNA

molecule. The strand that is transcribed or copied into an RNA molecule is referred to as the template strand of the DNA. The other DNA strand, the non-template strand, is frequently referred to as the coding strand of that gene. It is called this because, except T for U changes, it corresponds exactly to the sequence of the RNA primary transcript, which encodes the (protein) product of the gene. In the case of a double-stranded DNA molecule containing many genes, the template strand for each gene will not necessarily be the same strand of the DNA double helix. Thus, a given strand of a double-stranded DNA molecule will serve as the template strand for some genes and the coding strand of other genes. Note that the nucleotide sequence of an RNA transcript will be the same (except for U replacing T) as that of the coding strand. The information in the template strand is read out in the 3′ to 5′ direction. Incorrect options Option A: Coding strand Also known as non-template strand. It does not transcribe to mRNA. Option B: Initiator methionine domain Initiator tRNAs (tRNAi), a special class of tRNAs, carry methionine (or its derivative, formyl-methionine) to ribosomes to start an enormous energy-consuming but highly regulated process of protein synthesis. It recognizes the AUG codon on mRNA which is present at the start of every protein-coding gene. These genes can be identified by the presence of this initiator domain. Option D: Translation unit Translational unit of a gene contains other domains of DNA such as binding sites for translational factors upstream and downstream of the gene. These regions are necessary for ribosome attachment and assembly for the start of translation.



References:

Harper 30th Ed., p.397; Fig. 36-5

↑ *Back to this question in Part A*

@Neet_pg_bot

Q5: Which of the following statements are false? (Q843580)

A. Ribosome is an example of ribozyme

B. Both ribozymes and protein enzymes exhibit specificity

C. Ribozymes exhibit greater structural complexity compared to protein enzymes

D. rRNA is directly involved in catalyzing the formation of peptide bonds

Correct Answer: C. Ribozymes exhibit greater structural complexity compared to protein enzymes

Explanation: Correct answer: Option C- Ribozymes exhibit greater structural complexity compared to protein enzymes Ribozymes are RNA molecules that can act as catalysts, accelerating biochemical reactions. A well-known example of a ribozyme is the ribosome , a complex found in all living cells responsible for protein synthesis. (Option A ruled out) rRNA is directly involved in catalysing the formation of peptide bonds which makes it a ribozyme (Option D ruled out) Ribozyme Protein enzymes Key similarities Both act as catalysts Both exhibit specificity, meaning they act on specific molecules (substrates) and catalyse specific reactions. (Option B ruled out) Both types of enzymes work by lowering the activation energy of reactions Key differences Made up of RNA molecules Made up of amino acids Lesser structural complexity Greater structural complexity Less diverse in terms of the types of reactions More diverse in terms of the types of reactions

References:

Harper's Biochemistry 32e page 69, 70, 354, 402

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: Which of the following belong to the group of Small non-coding RNAs? (Q843579)

A. mRNA

B. tRNA

C. rRNA

D. None of the above

Correct Answer: B. tRNA

Explanation: RNA is classified into two major classes: protein-coding and non-protein coding Protein-coding Messenger RNA (mRNA) is the only protein-coding RNA. mRNA carries genetic information from DNA to the protein-synthesizing machinery of the cell . (Option A ruled out) Non-protein coding Large non-coding RNAs: Ribosomal RNAs (rRNAs) are a major component of ribosomes, the molecular machines responsible for protein synthesis. (Option C ruled out) Long non-coding RNAs (lncRNAs) do not code for proteins and are involved in various cellular processes, including the regulation of gene expression. Small non-coding RNAs: Transfer RNAs (tRNAs) act as adapter molecules during protein synthesis, delivering amino acids to the ribosome based on the mRNA code. (Option B) Small nuclear RNAs (snRNAs) play crucial roles in RNA processing, particularly mRNA splicing, where they help remove non-coding sequences (introns) from mRNA precursors. MicroRNAs (miRNAs) and silencing RNAs (siRNAs) are involved in regulating gene expression by altering mRNA function, either inhibiting translation or inducing its degradation.

References:

Harper's Biochemistry 32e page 384, 385

↑ *Back to this question in Part A*

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Q7: Which of the following accurately describes the role of snRNAs in the spliceosome? (Q843578)

A. snRNAs bind to the poly-A tail of mRNA to stabilize the mature transcript.

B. snRNAs form ribonucleoprotein complexes with proteins to recognize and excise introns.

C. snRNAs bind to the 5' cap of mRNA to initiate translation.

D. snRNAs transport pre-mRNA from the nucleus to the cytoplasm for translation.

Correct Answer: B. snRNAs form ribonucleoprotein complexes with proteins to recognize and excise introns.

Explanation: snRNAs are crucial components of the spliceosome, forming ribonucleoprotein complexes (snRNPs) with proteins. These complexes recognize introns and facilitate their removal from the pre-mRNA and joins exons together. Spliceosome Processes pre-mRNA into mature mRNA by removing non-coding introns and joining coding exons. Components Pre-mRNA: Pre-mRNA is a primary RNA transcript that contains both introns (non-coding) and exons (coding).. Larger than mature mRNA. Spliceosome: Multi-component complex involved in splicing, including snRNAs and associated proteins. snRNAs: There are five types of snRNAs—U1, U2, U4, U5, and U6—that are crucial for the splicing process. Spliceosome Formation snRNAs: U1, U2, U4, U5, U6 are small nuclear RNAs that form snRNPs (small nuclear ribonucleoproteins) with proteins. snRNPs: Recognize splice sites and catalyze splicing reactions. Pre-mRNA Binding: snRNPs help position pre-mRNA for splicing. Spliceosome complex formation through interactions between snRNPs and pre-mRNA. Splicing Reaction U1 snRNP binds to the 5' exon-intron junction. U2 snRNP binds to the branch site of the intron. U4, U5, U6 snRNPs: Join complex, bring 5' and 3' splice sites close together for splicing. Splicing removes the intron and joins the exons to produce mature mRNA.

References:

Harper’s biochemistry Pg 398-399

↑ *Back to this question in Part A*

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Q8: Which of the following statements accurately describes a difference between eukaryotic and prokaryotic transcription? (Q843577)

A. Eukaryotic transcription occurs in the cytoplasm.

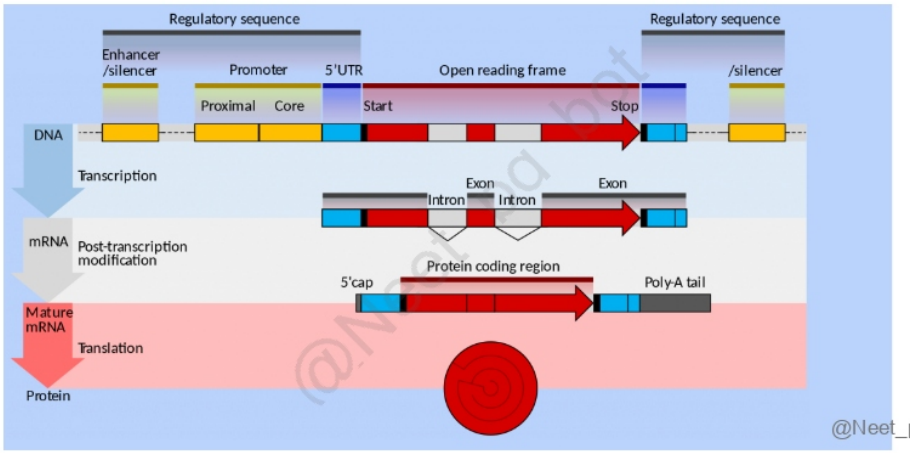
B. Eukaryotic transcription uses multiple RNA polymerases.

C. Eukaryotic transcription lacks chromatin remodeling.

D. Eukaryotic transcription does not involve mRNA processing.

Correct Answer: B. Eukaryotic transcription uses multiple RNA polymerases.

Explanation: Eukaryotic transcription involves three different RNA polymerases (I, II, and III), each responsible for specific types of RNA synthesis, whereas prokaryotic transcription uses a single RNA polymerase. Eukaryotic transcription Eukaryotic transcription is a complex process involving three RNA polymerases, extensive regulation, and post-transcriptional modifications. RNA Polymerases 1. RNA Polymerase I : Synthesizes rRNA (except 5S rRNA). 2. RNA Polymerase II : Synthesizes mRNA, snRNA, and miRNA (not snoRNA). 3. RNA Polymerase III : Synthesizes tRNA, 5S rRNA, and other small RNAs. Chromatin Remodeling Chromatin relaxation permits DNA access. Involves histone modifications (e.g., acetylation) and ATP-dependent nucleosome repositioning. Transcription Factors 1. Cis-acting Elements : DNA sequences regulating gene expression on the same chromosome. 2. Trans-acting Factors : Regulatory molecules (proteins or RNAs) that can act on genes located elsewhere in the genome. 3. General Transcription Factors (GTFs) : Required for transcription initiation complex assembly. 4. Specific Transcription Factors (STFs) : Bind to regulatory elements and mediate responses to signals (e.g., hormones). mRNA Processing Extensive co- and post-transcriptional modifications, including 5′ capping, 3′ polyadenylation, and intron splicing. Termination of Transcription Polyadenylation assists in termination but does not directly signal it. A poly-A tail is added to the 3′ end of pre-mRNA after cleavage downstream of an AAUAAA consensus sequence.



References:

https://slcc.pressbooks.pub/app/uploads/sites/20/2020/12/Gene_structure_eukaryote_2_annotated.svg-1024x551.png Lipincott Biochem chapter 31 RNA Structure, Synthesis, and Processing IV. EUKARYOTIC GENE TRANSCRIPTION

↑ *Back to this question in Part A*

@Neet_pg_bot

Q9: What is the primary role of the 7-methylguanosine cap added to the 5' end of eukaryotic mRNA? (Q843576)

A. To signal the termination of transcription

B. To aid in the transport of mRNA into the mitochondria

C. To protect the mRNA from exonucleases and facilitate ribosome recognition

D. To mark the mRNA for immediate degradation

Correct Answer: C. To protect the mRNA from exonucleases and facilitate ribosome recognition

Explanation: The primary functions of the 7-methylguanosine cap include protecting the mRNA from degradation by exonucleases and facilitating the recognition of the mRNA by the ribosome for translation initiation. Post-Transcriptional Modifications- 1. 5' Capping: The addition of a 7-methylguanosine cap to the 5' end of the pre-mRNA. Protects mRNA from degradation by exonucleases. Facilitates ribosome binding and initiation of translation. Aids in the export of mRNA from the nucleus to the cytoplasm. 2. 3' Polyadenylation: Addition of a Poly(A) tail (a sequence of adenine nucleotides) to the 3' end of the pre-mRNA. Increases the stability of the mRNA by protecting it from enzymatic degradation. Assists in the export of mRNA from the nucleus. It plays a role in the initiation of translation. 3. Splicing: Removal of non-coding sequences (introns) from the pre-mRNA. Joining coding sequences (exons) together. It is conducted by the spliceosome, a complex of small nuclear RNAs (snRNAs) and proteins. Alternative splicing allows for the production of different protein isoforms from a single gene. To signal the termination of transcription (Option A): Transcription termination is mediated by specific sequences and proteins, not the 7-methylguanosine cap. Other sequences and mechanisms mediate termination of transcription at the 3' end of the gene. To aid in the transport of mRNA into the mitochondria (Option B): Most mRNAs are not transported into mitochondria; mitochondrial proteins are encoded by nuclear genes and translated in the cytoplasm. To mark the mRNA for immediate degradation (Option D): The 7-methylguanosine cap protects the mRNA from exonuclease activity and promotes stability.

References:

Harper’s Biochemistry 32e page 398, 399, 400

↑ *Back to this question in Part A*

@Neet_pg_bot

Q10: Which of the following elements is typically found within the promoter region of a eukaryotic gene and is crucial for the initiation of transcription? (Q843575)

A. Pribnow box

B. TATA box

C. CAAT box

D. GC Box

Correct Answer: B. TATA box

Explanation: Promoter Element Location Function TATA Box (Option B) ~25-35 base pairs upstream of the transcription start site (TSS) Binds transcription factors and RNA polymerase II to initiate transcription. CAAT Box (Option C ruled out) ~50-100 base pairs upstream of the TSS Enhances promoter activity through binding of transcription factors. GC Box (Option D ruled out) Variable locations, often found in multiple copies Binds transcription factors such as SP1 to regulate gene expression. Additional promoter elements: Element Location Function Initiator Sequence (Inr) Positioned at the transcription start site (TSS), often spanning the TSS Works with the TATA box or independently to initiate transcription. It binds to transcription factors such as TFIID to recruit RNA polymerase II. Downstream Promoter Element (DPE) Downstream of the transcription start site, typically within 30 base pairs (around +28 to +32 relative to the TSS) Functions in synergy with the TATA box or Inr to enhance transcription Essential for binding of the transcription machinery, particularly in TATA-less promoters

References:

Harper’s Biochemistry 32e page 390, 391

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: Which technique is commonly used for DNA sequencing to identify mutations by determining the precise order of nucleotides in a DNA sample? (Q843150)

A. Fluorescence in situ hybridization (FISH)

B. Multiplex ligation-dependent probe amplification (MLPA)

C. Sanger technique

D. Single Strand Conformational Polymorphism (SSCP)

Correct Answer: C. Sanger technique

■ *Listen to Audio 1*

Explanation: The Sanger technique is a DNA sequencing method that uses modified nucleotides to stop DNA synthesis, allowing the sequence of DNA to be read by analyzing fragment lengths. There are cytogenetics and molecular methods for detection of mutations. Methods Uses Advantages/ disadvantages Cytogenetics Karyotype Detecting numerical and gross structural aberrations Low-resolution Time consuming FISH (Fluorescence in situ hybridization) (Option A ruled out) Detects copy number variations, structural alterations like translocations and microdeletion Detects Limitation: Can be used only for known defects CGH (Comparative genomic hybridization)

Detects copy number variations of genetic material Can be used to detect unknown chromosomal aberrations Used only for losses and gains Molecular RFLP (Restriction fragment length polymorphism) Restriction fragments are separated by electrophoresis Requires availability of enzyme with restriction site at the mutation site DNA Sequencing Sanger technique (Option C) is the most widely used. Directly determines the nucleotide sequence of a DNA fragment. Detects point mutations, small deletions and insertions. DNA microarray Microscopic DNA spots attached to a solid surface Multiple mutations can be detected. ARMS PCR (Amplification refractory mutation system) Allele-specific amplification of mutant and normal allele, determination of the genotype of an individual Highly sensitive May increase time and costs Multiplex PCR (Polymerase chain reaction) Amplification of more than one target simultaneously Reduces time and labour requirements Lower sensitivity and specificity Real-time PCR Amplification, detection, and quantification of target Increased specificity Eliminates post-amplification analyses More expensive MLPA (Multiplex ligation-dependent probe amplification) (Option B ruled out) Deletions and duplications Identifies very small single gene aberrations (50-70 nt) DGGE (Denaturing Gradient Gel Electrophoresis) Based on migration within gradient gel electrophoresis Detects close to 100% of point mutations SSCP (Single Strand Conformational Polymorphism) (Option D ruled out) Based on migration of single-stranded conformation on gel electrophoresis Detects about 80–90% of point mutations Hetero-duplex analysis Based on homoduplices and heteroduplices motilities in gel electrophoresis Detects nearly 80% of mutations OLA (Oligonucleotide ligation assay) It is based on the ligation of two flanked primers annealed with target sequences Detects all base exchanges PTT (Protein truncation test) It is based on a combination of PCR, transcription, and translation Detects translation-terminating mutations. Missense mutations are not detected.

References:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3883366/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q12: Which class of antibiotics inhibits bacterial protein synthesis by blocking the attachment of aminoacyl-tRNA to the ribosomal A site? (Q843149)

A. Tetracyclines

B. Chloramphenicol

C. Macrolides

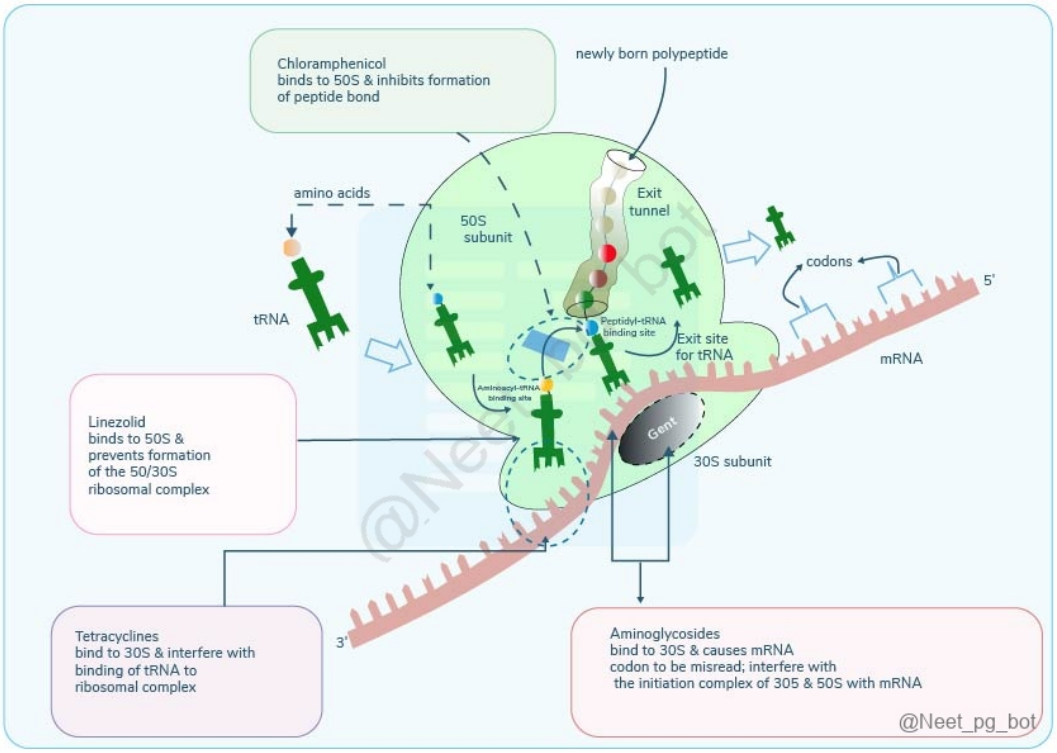
D. Puromycin

Correct Answer: A. Tetracyclines

■ *Listen to Audio 1*

Explanation: Tetracyclines inhibit the EF1A(prokaryotic elongation factor 1A) and also the attachment of aminoacyl tRNA to the A site. Inhibitors of Protein Synthesis Mechanism of action Reversible Inhibitors in Bacteria (bacteriostatic) Tetracyclines (Option A) Binds to the 30S ribosome of bacteria and inhibit attachment of aminoacyl tRNA to the A site. Chloramphenicol (Option B ruled out) Inhibits the peptidyl transferase activity of

bacterial ribosomes Erythromycin (macrolides) (Option C ruled out) and clindamycin Inhibits the translocation process. Irreversible Inhibitors in Bacteria (bactericidal) Aminoglycoside Bind to 30S subunit of bacterial ribosomes. Misreading of mRNA. Inhibit the initiation complex formation and totally inhibit protein synthesis. Inhibitors of Protein Synthesis in Mammals Puromycin (Option D ruled out) Analogue of tyrosine tRNA and attaches to the “A” site of the ribosome causing premature termination. Cycloheximide Inhibits peptidyl transferase in 60S subunit Diphtheria toxin Inactivation of EF-2 by attachment of ADP to EF-2



References:

Textbook of Biochemistry for Medical Students , Vasudevan, 7th Edition, Page 605

↑ *Back to this question in Part A*

@Neet_pg_bot

Q13: What is the total number of ATPs required for activating amino acids by aminoacyl tRNA synthetase? (Q843148)

A. 3

B. 2

C. 4

D. 1

Correct Answer: B. 2

■ *Listen to Audio 1*

Explanation: Energetics of translocation of Amino acids: Steps Number of ATPs needed Activating amino acids by aminoacyl tRNA synthetase (Option B) 2 Activating of eEF1 (A attaches amino acyl tRNA to A site) 1 Activating eEF2 (helps in translocation of ribosomes) 1 Total 4

References:

Textbook of Biochemistry for Medical Students , Vasudevan, 7th Edition, Page 602

↑ *Back to this question in Part A*

@Neet_pg_bot

Q14: Which of the following statements is true about aminoacyl tRNA? (Q843146)

A. Attaches to the A site of 60s ribosomes

B. Needs 3 high-energy phosphates for attachment

C. It is guided by eIF2C

D. None of the above

Correct Answer: A. Attaches to the A site of 60s ribosomes

■ *Listen to Audio 1*

Explanation: Initiation methionine tRNA (imet-tRNA) Aminoacyl tRNA (aa-tRNA) Involved in the initiation of translation. Involved in the elongation step of translation. Guided by eIF2C (Option C ruled out) Guided by eEF1A Attaches to the P site of 60s ribosome Attaches to the A site of 60s ribosome (Option A) Utilises 3 high energy phosphates for each peptide bond formation (Option B ruled out) Utilises 4 high energy phosphates for each peptide bond formation

References:

Harper's Biochemistry, 32nd Edition, Page 410, 413 Textbook of Biochemistry for Medical Students , Vasudevan, 7th Edition, Page 602

↑ *Back to this question in Part A*

@Neet_pg_bot

Q15: Which of the following is NOT TRUE regarding the initiation of translation? (Q843145)

A. eIF3 and 1A stabilize 60s ribosomal subunit to prevent reassociation with 40s.

B. Methionine tRNA scans mRNA to identify the initiation AUG codon

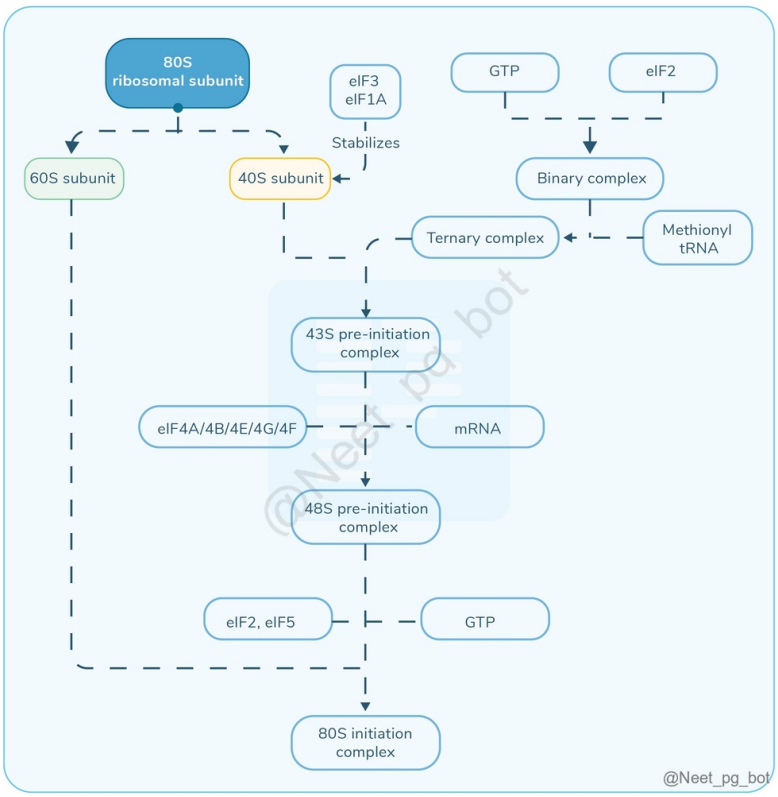
C. ATP dependent helicase eIF4A/4B breaks hydrogen bonds to remove the hairpin loops

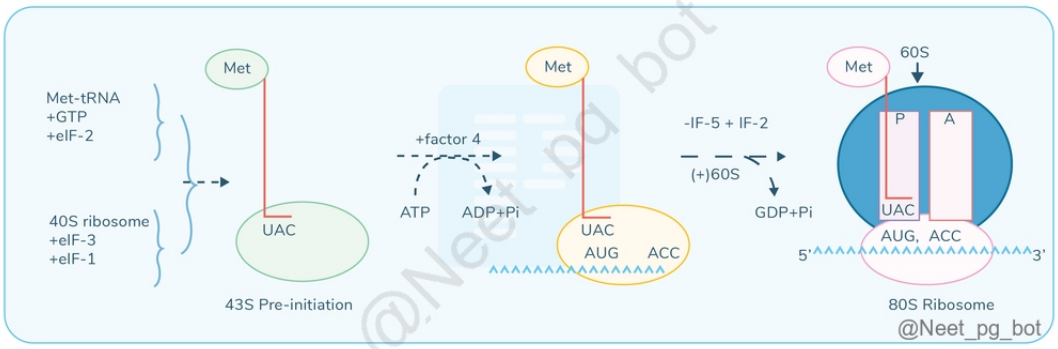
D. Association of mRNA with the 43S preinitiation complex forms the 48S initiation complex

Correct Answer: A. eIF3 and 1A stabilize 60s ribosomal subunit to prevent reassociation with 40s.

■ *Listen to Audio 1*

Explanation: eIF3 and 1A stabilize 40s (not the 60s subunit) ribosomal subunit to prevent reassociation with 60s. Steps of initiation: It involves tRNA, rRNA, mRNA, eukaryotic initiation factors (eIFs), GTP, ATP, and amino acids. 1. Ribosomal Dissociation: 80S ribosomes dissociate into 40S and 60S subunits. eIF3 and 1A stabilize 40s ribosomal subunit to prevent reassociation with 60s, allowing other translation initiation factors to associate with the 40S subunit. (Option A) 2. Formation of the 43S Preinitiation Complex: Binary complex is formed by binding of GTP and eIF-2. This binary complex then binds to methionyl tRNA. The GTP-eIF-2-tRNAi ternary complex binds to the 40S ribosomal subunit to form the 43S preinitiation complex. 3. Formation of the 48S Initiation Complex: The 5′ termini of mRNA molecules in eukaryotic cells are “capped.” The binding of mRNA to the 43S preinitiation complex is facilitated by 7meG-cap. 4G/4A attaches to the cap through 4E, which forms 4F. ATP-dependent helicase eIF4A/4B breaks hydrogen bonds to remove the hairpin loops and make mRNA single-stranded. (Option C ruled out) Methionine tRNA scans the mRNA to identify the initiation AUG codon, and attach the tRNA to AUG at the P site. (Option B ruled out) The association of mRNA with the 43S preinitiation complex forms the 48S initiation complex. (Option D ruled out) 4. Formation of the 80S Initiation Complex: The 60S ribosomal subunit binds to the 48S initiation complex by hydrolysis of the GTP bound to eIF-2 by eIF-5. This results in the release of the initiation factors bound to the 48S initiation complex. The rapid association of the 40S and 60S subunits forms the 80S ribosome. At this point, the met-tRNAi is on the P site of the ribosome, ready for the elongation cycle to commence





References:

Harper's Biochemistry, 32nd Edition, Page 410-412

↑ *Back to this question in Part A*

@Neet_pg_bot

Q16: Which of the following statements accurately reflects a difference between DNA and RNA, considering their roles in genetic information processing and stability? (Q843028)

A. RNA's 2'-hydroxyl group enhances its stability compared to DNA.

B. RNA polymerases need a primer; DNA polymerases do not.

C. RNA forms complex structures; DNA remains a double helix

D. DNA degrades more rapidly than RNA due to the presence of a 2'-hydroxyl group.

Correct Answer: C. RNA forms complex structures; DNA remains a double helix

■ *Listen to Audio 1*

Explanation: RNA Structure: RNA is single-stranded but can fold into complex three-dimensional structures due to intrachain base pairing (A-U, G-C). DNA is typically double-stranded with a stable double-helix structure. Aspect DNA RNA Sugar Deoxyribose (lacks 2' hydroxyl group) (Option D ruled out) Ribose (has 2' hydroxyl group) Bases Adenine (A), Guanine (G), Cytosine (C), Thymine (T) Adenine (A), Guanine (G), Cytosine (C), Uracil (U) Structure Double-stranded helix, antiparallel strands, complementary base pairing (A-T, G-C) DNA follows both Chargaff rule of base pairing and Watson and Crick base pairing rule. Single-stranded, can fold into complex structures with intrachain base pairing (A-U, G-C) (Option C) Do not follow Chargaff's Rule of Base Pairing. Watson and Crick Base pairing rule is followed here. Function Stores genetic information Various roles in gene expression: Messenger RNA (mRNA) - Carries genetic information from DNA to ribosomes for protein synthesis Ribosomal RNA (rRNA) - Major component of ribosomes, involved in protein synthesis Transfer RNA (tRNA) - Transports amino acids to ribosomes during protein synthesis Stability More stable due to lack of 2'-hydroxyl group B (Option A ruled out) Less stable, more susceptible to hydrolysis Degradation Susceptibility Less prone to degradation, stable over generations More prone to degradation, shorter lifespan, important for regulating gene expression Synthesis Primer Requirement Requires RNA primer for initiation (Option B ruled out) Can initiate synthesis de novo, no primer needed Proofreading and Fidelity High fidelity with 3' to 5' exonuclease activity for proofreading Higher error rate but can backtrack and cleave incorrect nucleotides.

References:

Harper’s Biochemistry, 32nd Edition, Page 335,352- 357

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: Sickle cell anaemia is suspected in a 25-year-old patient who presents with episodes of abdominal pain and fatigue. Genetic analysis reveals GAG in the 6th position is replaced with GTG. Which type of mutation here results in haemoglobin that is still functional but altered? (Q843047)

A. Unacceptable mutation

B. Partially acceptable mutation

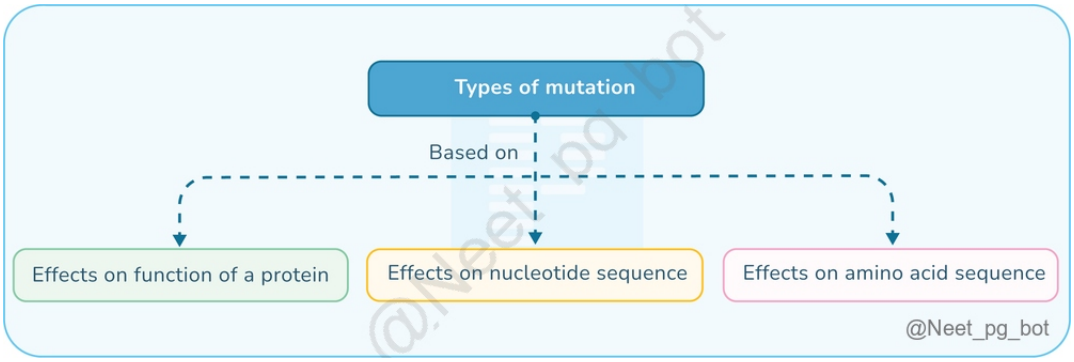
C. Acceptable mutation

D. Nonsense mutation

Correct Answer: B. Partially acceptable mutation

Listen to Audio 1

Explanation: The mutation described is a missense mutation , where the GAG codon (which codes for glutamic acid) is replaced by the GTG codon (which codes for valine) at the 6th position of the hemoglobin gene resulting in the production of hemoglobin S (HbS) instead of the normal hemoglobin A (HbA). This partially acceptable mutation still allows the hemoglobin to carry oxygen, but with altered efficiency (accumulation of globin chains and causes sickling crisis on exposure to deoxygenation.), leading to symptoms of sickle cell anemia. Sickle cell anemia is an example of point mutation, transversion, missense and partially acceptable mutation. Effects on the functioning of the protein: Based on the Effects on the function of protein Acceptable mutation: (Option C ruled out) Partially acceptable mutation: (Option B) Unacceptable mutation: (Option A ruled out) Change in the nucleotide sequence causes alteration in the amino sequence that does not affect the functioning of the protein. Example: Hb Sydney Change in the nucleotide sequence causing alteration in the amino sequence that affects the functioning of the protein but it is not immediately life-threatening. Example: HbS - GAG is replaced by GTG which replaces the glutamic acid to valine. Change in the nucleotide sequence causes alteration in the amino sequence that affects the functioning of the protein so extensively that it is immediately life-threatening. Example: Congenital methemoglobinemia (HbM) - Histidine (CAU) is replaced with tyrosine (UAU) in alpha-chain. Nonsense mutation (Option D) : Codon for an amino acid is mutated to a termination codon (UAA or UAG) causing premature termination of translation.



References:

Textbook of Biochemistry for Medical Students,Vasudevan, 7th Edition, Page 613

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7510211/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q18: A DNA sequence originally reads 5'-ATG GCA TCG TAC-3'. After a mutation, it reads 5'-ATG GCT CGT AC-3'. Identify the mutation that has occured? (Q843043)

A. Frameshift mutation

B. Point mutation

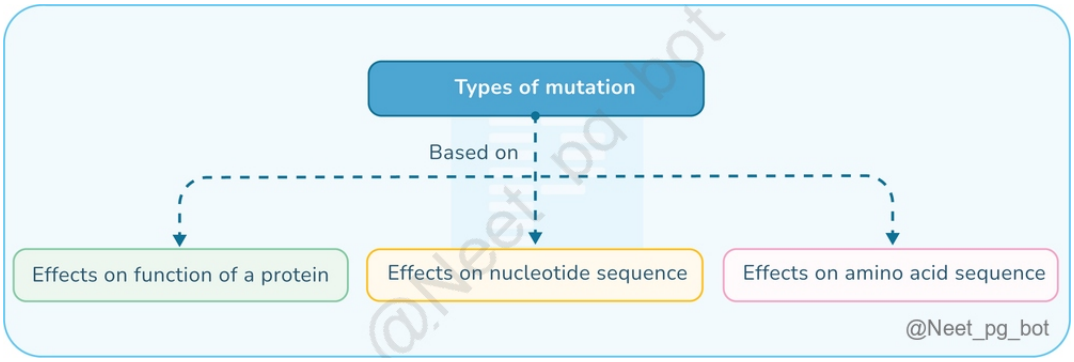
C. Transition mutation

D. Insertion mutation

Correct Answer: A. Frameshift mutation

■ *Listen to Audio 1*

Explanation: There is deletion of adenine at the 6th position from the DNA sequence, leading to a shift in the reading frame. This is a frameshift mutation. A mutation is defined as a change in the nucleotide sequence of DNA. Based on the Effects on Nucleotide sequence Point mutation (Option B ruled out) Frameshift mutation (Option A) Substitution of one nucleotide by another. Transition (Option C ruled out) : A purine is replaced by a purine, and a pyrimidine is replaced by a pyrimidine. Transversion : Purine is replaced by a pyrimidine and vice versa. Deletion (Option A ruled out) or insertion (Option D ruled out) of nucleotides leads to an altered reading frame in the mRNA.



References:

Harper's Biochemistry, 32nd Edition, Page 407-409

↑ *Back to this question in Part A*

@Neet_pg_bot

Q19: Identify the type of mutation illustrated in the following DNA sequences? Starting sequence: Mutated sequence: (Q843039)

A. Point mutation

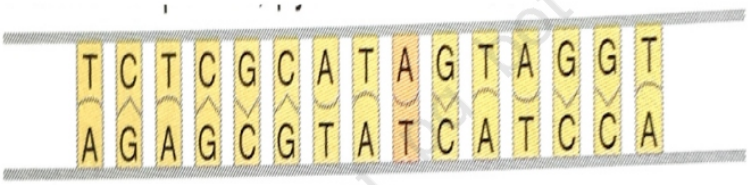
B. Frameshift mutation

C. Deletion mutation

D. Insertion mutation



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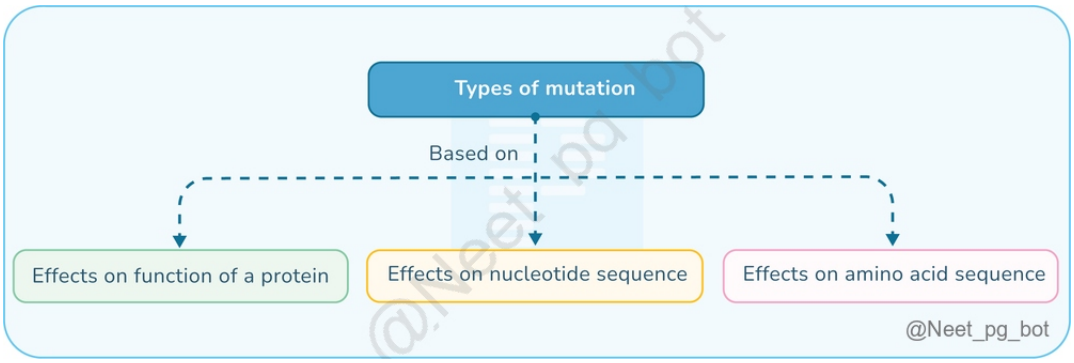


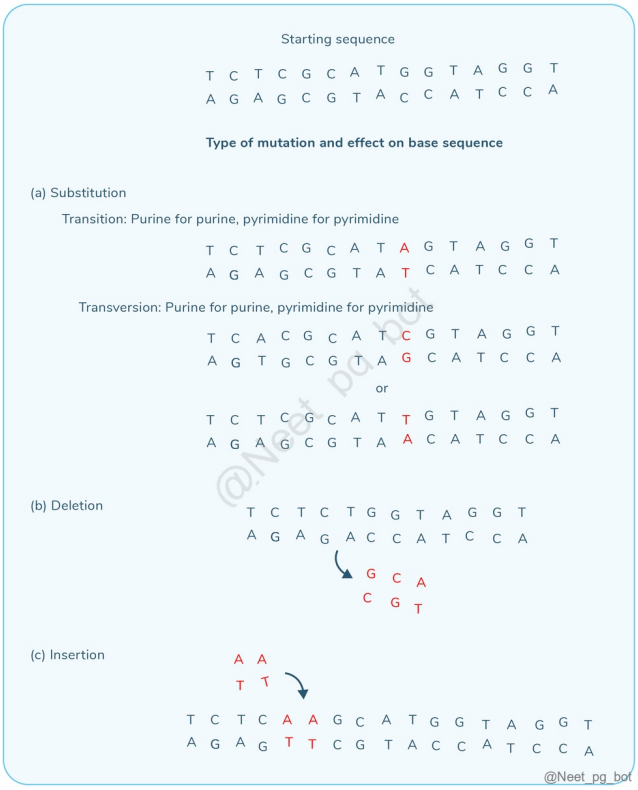
@Neet_pg_bot

Correct Answer: A. Point mutation

■ *Listen to Audio 1*

Explanation: There is substitution of guanine by adenine and cytosine by thymine from the starting sequence (one nucleotide is substituted by the other), which is a point mutation. A mutation is defined as a change in nucleotide sequence of DNA. Based on the Effects on Nucleotide sequence Point mutation (Option A) Frameshift mutation (Option B ruled out) Substitution of one nucleotide by another. Transition : Purine is replaced by a purine and a pyrimidine is replaced by a pyrimidine. Transversion : Purine is replaced by a pyrimidine and vice versa. Deletion (Option C ruled out) or insertion (Option D ruled out) of nucleotides leading to altered reading frame in the mRNA.





References:

Harper's Biochemistry, 32nd Edition, Page 407-409

↑ *Back to this question in Part A*

@Neet_pg_bot

Q20: Which of the following statements are true regarding codons? A, G, T, C are RNA nucleotides A, G, T, C are DNA nucleotides A, G, U, C are RNA nucleotides A, G, U, C are DNA nucleotides (Q843037)

A. 1 and 3 are correct

B. 2 and 3 are correct

C. 1 and 4 are correct

D. 2 and 4 are correct

Correct Answer: B. 2 and 3 are correct

■ *Listen to Audio 1*

Explanation: A, G, T, and C are nucleotides forming DNA codons, while A, G, U, and C are those forming RNA codons. Aspect Details Codon Three-nucleotide sequence in mRNA Codon Functions Specifies an amino acid sequence in proteins. Start codon (AUG): Signals the initiation of protein synthesis. Stop codons(UAA, UAG, and UGA) : Signals termination of protein synthesis Total Codons 64 (4 nucleotides, each position can be A, U, G, or C) DNA Nucleotides A, G, T, C (Options A, C, D ruled out) RNA Nucleotides A, G, U (replaces T), C (Options A, C, D ruled out) Amino Acid Codons 61 codons encode 20 amino acids Reading Frame mRNA sequence read from start codon to stop codon (Open Reading Frame)

References:

Harper's Biochemistry, 32nd Edition, Page 404, 405, 414, 420

↑ *Back to this question in Part A*

@Neet_pg_bot

Q21: A 23-year-old medical student is studying molecular biology. She learns that a single tRNA molecule can recognize more than one codon for the same amino acid, because base-pairing at the third codon position is less stringent. Which of the following best explains this phenomenon? (Q843036)

A. Inosine forming non-standard base pairs at the third codon position.

B. The exact matching of all three nucleotides between codon and anticodon.

C. Single tRNA binds only to one specific codon.

D. The rigid Watson-Crick base pairing at the third position of the codon.

Correct Answer: A. Inosine forming non-standard base pairs at the third codon position.

■ *Listen to Audio 1*

Explanation: Wobble phenomenon = flexibility in base-pairing at the 3rd codon position (5’ end of anticodon) . Inosine in the anticodon can pair with U, C, or A , allowing one tRNA to recognize multiple codons. The first two codon bases follow strict Watson-Crick pairing. Clinical relevance: explains the degeneracy of the genetic code

and why some mutations at 3rd base are silent. Takeaway: Wobble pairing (via inosine) allows one tRNA to decode multiple codons for the same amino acid.

References:

Harper's Biochemistry, 32nd Edition, Page 407

↑ *Back to this question in Part A*

@Neet_pg_bot

Q22: In the context of genetic code degeneracy, which of the following accurately describes its impact on protein synthesis? (Q843035)

A. Single codon specifies one amino acid.

B. Multiple codons can code for the same amino acid.

C. Flexibility occurs at the 2nd codon position.

D. Inosine in mRNA contributes to the flexibility in pairing.

Correct Answer: B. Multiple codons can code for the same amino acid.

■ *Listen to Audio 1*

Explanation: Degeneracy allows multiple codons to specify the same amino acid, based on flexible base-pairing rules. Degeneracy in the Genetic Code : Multiple codons can encode the same amino acid. (Option A ruled out) (Option B) There are 64 possible codons but only 20 amino acids. Example: Six codons (UCU, UCC, UCA, UCG, AGU, AGC) all code for serine. Causes of Degeneracy : Wobble Hypothesis : Flexibility in base-pairing rules between the third nucleotide of mRNA codon and the tRNA anticodon . (Option C ruled out) Inosine (I) Role : Inosine in t RNA anticodons can pair with U, C, and A, enhancing code flexibility. (Option D ruled out)

References:

Harper's Biochemistry, 32nd Edition, Page 405, 407.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q23: Which of the following best describes the primary mechanisms and applications of RNA interference (RNAi)? (Q843034)

A. Degradation of mRNA; used for gene manipulation and therapy.

B. Enhancement of mRNA synthesis; used for genetic modifications.

C. Conversion of mRNA to DNA; used for protein structure studies.

D. Stabilisation of mRNA; used for treating infections.

Correct Answer: A. Degradation of mRNA; used for gene manipulation and therapy.

Listen to Audio 1

Explanation: RNAi primarily functions through mRNA degradation and inhibiting protein synthesis, with gene manipulation and therapy applications. RNA Interference (RNAi) Definition : Biological process where double-stranded RNA (dsRNA) silences specific gene expressions. Mechanisms : Dicing: Long dsRNA entering the cell is cleaved by the enzyme Dicer (an RNase III-type enzyme) into short, 21–25 nucleotide fragments called siRNAs (small interfering RNAs). RISC Loading: These siRNAs are loaded into the RISC (RNA-Induced Silencing Complex). The duplex is unwound, leaving the guide strand to direct the complex. Silencing (Slicing): The guide strand directs the RISC to the target complementary mRNA. The Argonaute protein (the catalytic component of RISC) cleaves the target mRNA, leading to its degradation and preventing protein synthesis. siRNA vs. miRNA Differentiation: siRNA: Exhibits perfect complementarity to the target mRNA, causing direct endonucleolytic cleavage (degradation). miRNA: Often shows imperfect complementarity , leading primarily to translational repression (inhibiting protein synthesis without immediate mRNA cleavage). Applications : Functional Genomics: RNAi is the standard tool for gene knockdown - temporarily reducing protein levels to study gene function (reverse genetics). Therapeutic Potential: Used to treat diseases by silencing disease-causing genes. For example, Patisiran is an FDA-approved siRNA therapeutic for Transthyretin amyloidosis. Enhancement of mRNA synthesis; used for genetic modifications (Option B) does not enhance mRNA synthesis; it silences gene expression. Conversion of mRNA to DNA; used for protein structure studies (Option C) does not involve converting mRNA to DNA; it targets mRNA for degradation Stabilisation of mRNA; used for treating infections (Option D) causes degradation of mRNA, not stabilisation.

References:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1686061/

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8538544/

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC309050/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q24: A 25-year-old student is reviewing the roles of small RNAs in cellular processes. She learns about small nuclear RNAs (snRNAs) and small interfering RNAs (siRNAs). Which of the following statements about snRNAs and/or siRNAs is true? (Q843033)

A. Both snRNAs and siRNAs are involved in the degradation of mRNA.

B. snRNAs form complexes with the RNA-induced silencing complex (RISC).

C. snRNAs are primarily involved in mRNA splicing and are located in the nucleus.

D. snRNAs are typically found in the cytoplasm, while siRNAs are found in the nucleus.

Correct Answer: C. snRNAs are primarily involved in mRNA splicing and are located in the nucleus.

■ *Listen to Audio 1*

Explanation: Small nuclear RNAs (snRNAs) are essential components of the spliceosome, a complex responsible for removing introns from pre-mRNA (hnRNA) during splicing, which occurs in the nucleus. Feature snRNAs siRNAs Main Function RNA processing Gene regulation Location (Option C) (Option D ruled out) Nucleus Cytoplasm Involvement (Option A ruled out) mRNA splicing, transcription elongation mRNA degradation Specific Types U1, U2, U4, U5, U6 (splicing); U7 (histone mRNAs); 7SK (transcription) Typically 21-25 nucleotides long Role in mRNA Removes introns, generates correct 3' ends of histone mRNAs Silences gene expression Source Endogenous snRNAs Endogenous/exogenous dsRNAs processed into siRNAs Processing Mechanism Forms complexes with proteins (snRNPs) for splicing Processed by Dicer enzyme into siRNA duplexes Associated Complex (Option B ruled out) Small nuclear ribonucleoproteins (snRNPs) RNA-induced silencing complex (RISC) Length Varies; generally 100-300 nucleotides 21-25 nucleotides Stability Generally stable, involved in essential RNA processes Short-lived, rapidly degrades target mRNA Biogenesis Transcribed by RNA polymerase II or III Derived from longer dsRNA precursors Function in Disease Defects can lead to splicing disorders (e.g., spinal muscular atrophy) Utilized in RNAi therapies for gene silencing (e.g., in cancer) Example Applications Studying splicing mechanisms, understanding transcription regulation Therapeutic gene silencing, functional genomics studies

References:

Harper's Biochemistry, 32nd Edition, Pages 356, 357, 358, 359, 402.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q25: Which of the following statements is true regarding the synthesis of mRNA? (Q843032)

A. Introns are the coding sequence in a mRNA

B. 7-methyl guanosine gets added to the 3’ end of mRNA

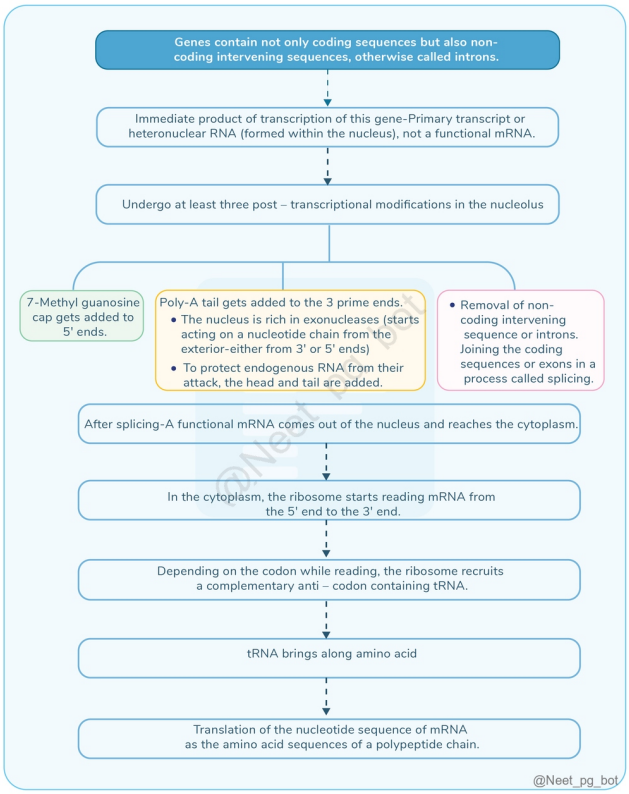
C. Process of splicing occurs in the cytoplasm

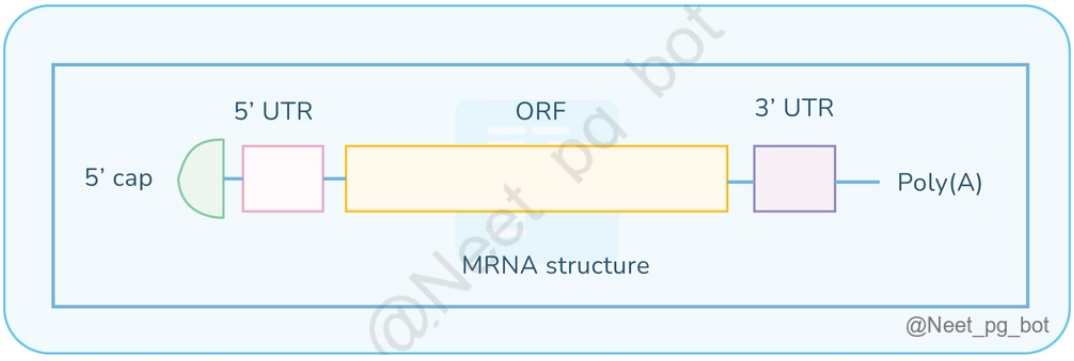
D. Heteronuclear RNA is not a functional mRNA

Correct Answer: D. Heteronuclear RNA is not a functional mRNA

■ *Listen to Audio 1*

Explanation: Heteronuclear RNA (hnRNA) (Primary transcript) is the immediate product of transcription and is the precursor to mRNA , containing introns and exons. It undergoes processing like splicing, capping, and polyadenylation to become functional mRNA. Synthesis of mRNA: Introns are the coding sequence in a mRNA (Option A): Exons are the coding sequences, while introns are the non-coding sequences in a mRNA. 7-methyl guanosine gets added to the 3’ end of mRNA (Option B): 7-methyl guanosine gets added to the 5’ end of mRNA, while Poly-A tail gets added to the 3’ end of mRNA. Process of splicing occurs in the cytoplasm (Option C): Process of splicing occurs in the nucleus.





References:

Harper's Biochemistry, 32nd Edition, Page 382, 384, 397 - 402

↑ *Back to this question in Part A*

@Neet_pg_bot

Q26: Which of the following steps in the process of tRNA synthesis occurs in the cytoplasm? (Q843031)

A. Rearrangement of glycosidic linkages giving rise to pseudouridine

B. Deamination creating inosine

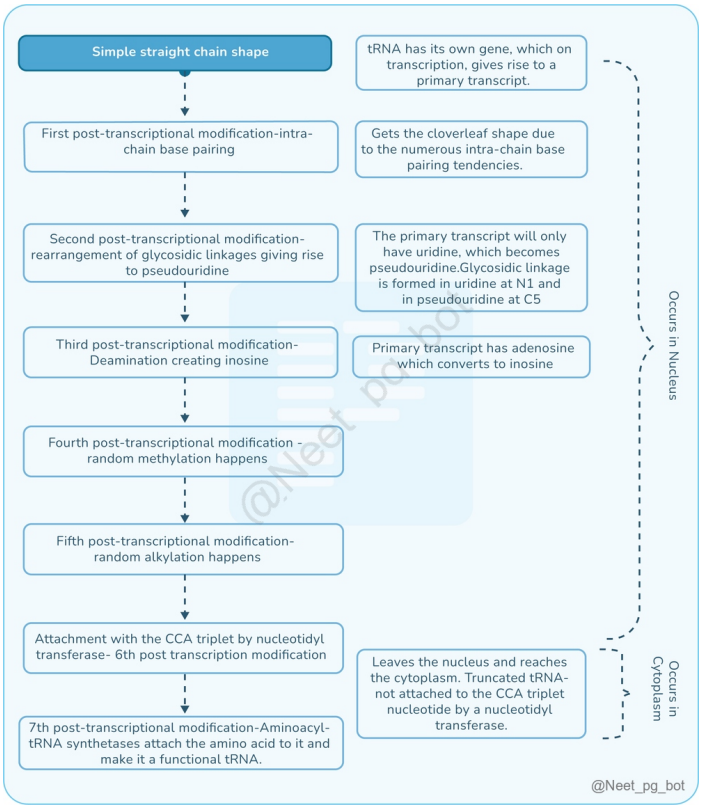
C. Fourth post-transcriptional modification of random methylation

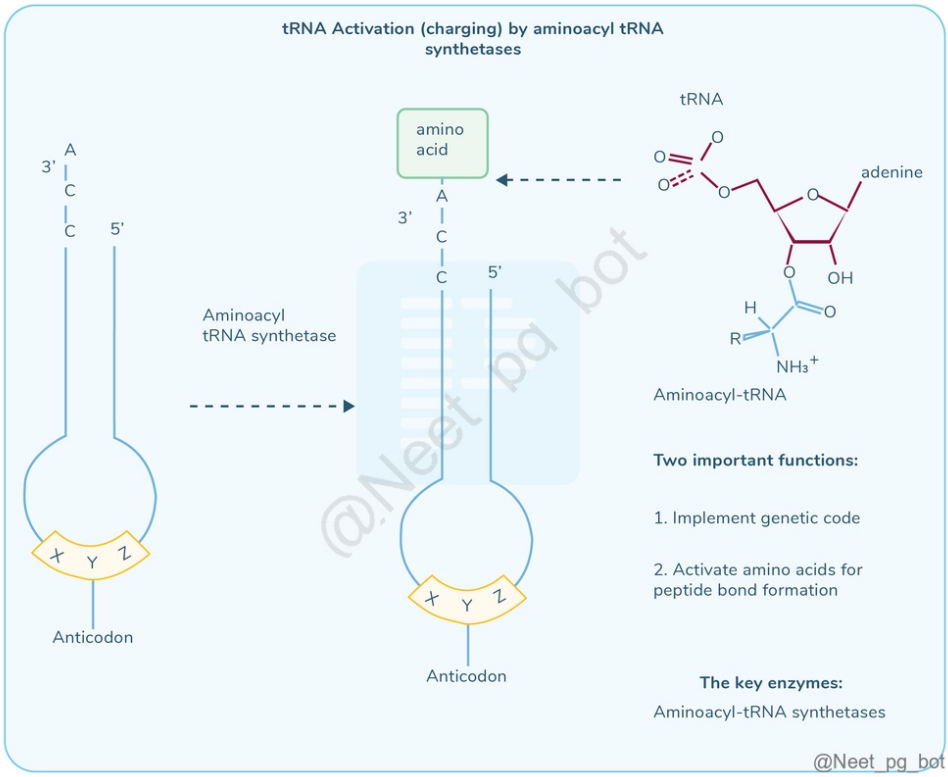
D. Aminoacyl-tRNA synthetases attach the amino acid

Correct Answer: D. Aminoacyl-tRNA synthetases attach the amino acid

■ *Listen to Audio 1*

Explanation: The 7th post-transcriptional modification involves aminoacyl-tRNA synthetases attaching the amino acid to the tRNA, making it functional in the cytoplasm. Synthesis of tRNA: Rearrangement of glycosidic linkages giving rise to pseudouridine (Option A) is incorrect because pseudouridine formation occurs in the nucleus , as part of post-transcriptional modifications of tRNA. Deamination creating inosine (Option B) is incorrect because inosine is formed in the nucleus by the deamination of adenosine during tRNA maturation. Inosine is important for wobble base pairing in codon recognition. Fourth post-transcriptional modification of random methylation (Option C) is incorrect because methylation of tRNA (as part of modifications like methylation of ribose or bases) occurs in the nucleus , not the cytoplasm.





References:

Harper's Biochemistry, 32nd Edition, Page 400, 402, 406, 407

↑ *Back to this question in Part A*

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Q27: Which statement best describes the wobble hypothesis in relation to tRNA's function? (Q843030)

A. Allows multiple tRNAs to bind to a single codon.

B. Explains tRNA's ability to bind to any codon with perfect complementarity.

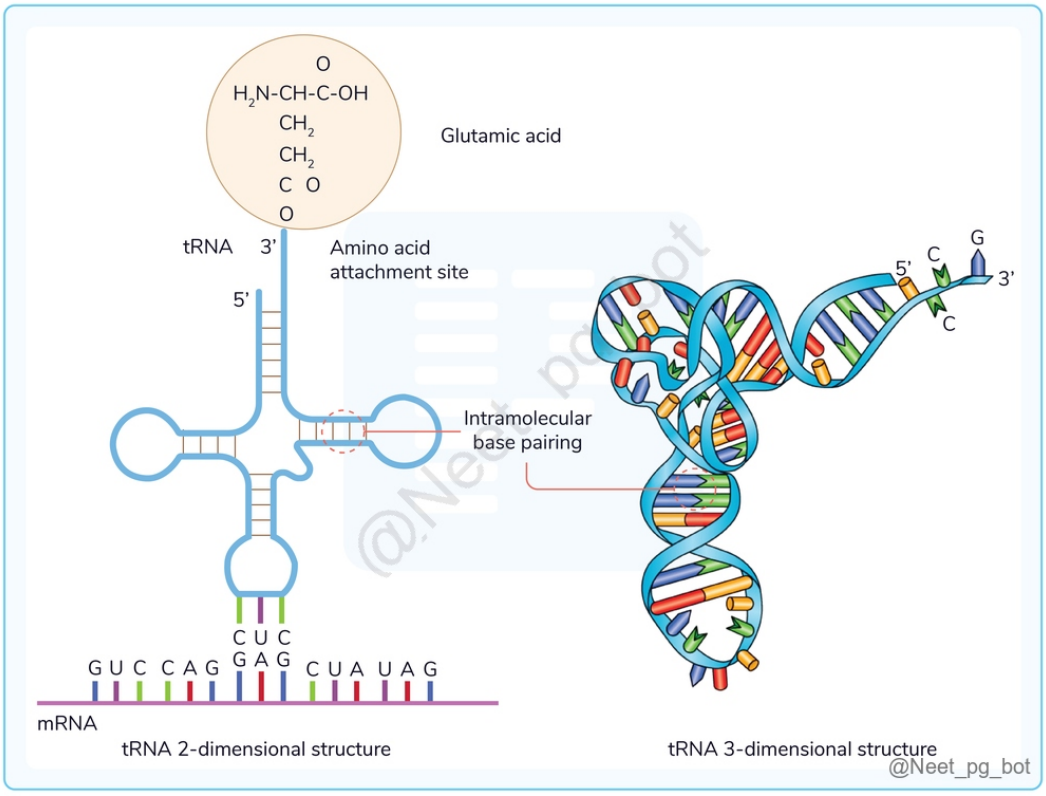
C. Single tRNA recognizes multiple codons due to flexible base pairing.

D. Accounts for varying tRNA lengths matching different mRNA sequences.

Correct Answer: C. Single tRNA recognizes multiple codons due to flexible base pairing.

■ *Listen to Audio 1*

Explanation: The Wobble Hypothesis explains that a single tRNA can bind to multiple codons because of flexible base pairing at the third position of the codon. tRNA / transfer RNA: Component Description Function Amino Acid Acceptor Arm Located at the 3' end, ending in CCA, which is added post-transcriptionally. The amino acid is attached to the 3′-OH group of the A moiety. Charging : Amino acids are attached to the tRNA by aminoacyl-tRNA synthetases for protein synthesis. D Arm Contains dihydrouridine, a modified base. Contributes to the overall structure and recognition of tRNA by its corresponding aminoacyl-tRNA synthetase. Recognition : Ensures correct aminoacylation by interacting with aminoacyl-tRNA synthetase. TψC Arm Named for its ribothymidine (T), pseudouridine (ψ), and cytidine (C) content. Involved in binding tRNA to the ribosome. Binding : Positions tRNA correctly at the ribosome for protein synthesis. Anticodon Loop Opposite the amino acid acceptor arm, contains a sequence of three nucleotides (anticodon) complementary to the codon on mRNA. Wobble phenomenon Third nucleotide pair formation is non-specific (more than one codon coding for a single amino acid) (Or) Reduced stringency when the third nucleotide pair is formed between codon and anti-codon. (Or) When tRNA is written in 5', 3' direction, the first nucleotide of the anticodon of tRNA is not specific Degeneracy More than one codon coding for a single amino acid because of the wobble phenomenon. Codon Recognition : Ensures the correct amino acid is added to the polypeptide chain during translation. Variable Arm Located between the TψC arm and the anticodon loop. Its length varies among different tRNA species and contributes to the tertiary structure of tRNA. Structural Flexibility : Aids in interactions with the ribosome and other translation factors. Allows multiple tRNAs to bind to a single codon (Option A): The wobble hypothesis focuses on how a single tRNA can recognize and bind to several codons that code for the same amino acid, not how multiple tRNAs interact with one codon. Explains tRNA's ability to bind to any codon with perfect complementarity (Option B): The wobble hypothesis involves flexible or non-standard base pairing. It does not imply that tRNAs bind with perfect complementarity to all codons. Accounts for varying tRNA lengths matching different mRNA sequences (Option D): The hypothesis does not address differences in tRNA lengths or their relationship with mRNA sequences.



References:

Harper’s biochemistry, 32nd Edition, Page 355

↑ *Back to this question in Part A*

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Q28: Which of the following types of rRNA is associated with the 40S ribosomal subunit in eukaryotic cells? (Q843029)

A. 5S rRNA

B. 18S rRNA

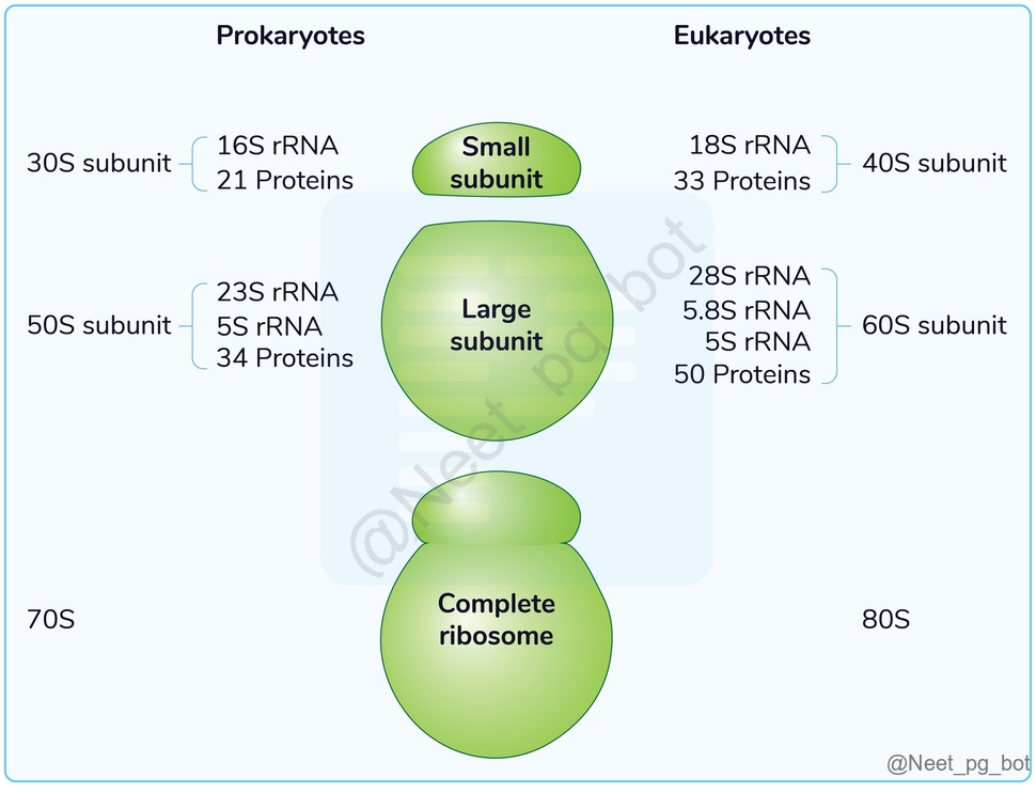
C. 28S rRNA

D. 16S rRNA

Correct Answer: B. 18S rRNA

■ *Listen to Audio 1*

Explanation: ■■■■■In eukaryotic cells, the ribosome is composed of two subunits: the small 40S subunit and the large 60S subunit. The 40S subunit contains the 18S rRNA, which is essential for the structural integrity and function of the ribosome during protein synthesis. The 60S subunit, on the other hand, includes the 5S, 5.8S, and 28S rRNAs (Options A and C ruled out) The 16S rRNA is found in the small subunit of prokaryotic ribosomes, not in eukaryotic ribosomes (Option D ruled out)



References:

Harper's Biochemistry, 32nd Edition, Page 384, 345, 400, 401, 402

↑ *Back to this question in Part A*

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• [07_ Genetics] 023 Pcr

« Back to Folder Directory

[07_ Genetics] 023 Pcr — Quick Jump

Q1 (p. 1073) *Q2 (p. 1073)* *Q3 (p. 1075)* *Q4 (p. 1075)* *Q5 (p. 1077)* *Q6 (p. 1077)*
Q7 (p. 1079) *Q8 (p. 1080)* *Q9 (p. 1081)* *Q10 (p. 1082)* *Q11 (p. 1082)*

Q1: In PCR cloning, the use of proofreading DNA polymerases is often preferred because: (Q843241)

A. They amplify DNA at a much faster rate than regular Taq polymerase

B. They have a lower error rate during DNA synthesis

C. They can amplify longer DNA fragments more efficiently

D. They are more tolerant to variations in PCR cycling conditions

Correct Answer: B. They have a lower error rate during DNA synthesis

■ *Listen to Audio 1*

Explanation: Proofreading DNA polymerases have a lower error rate during DNA synthesis. Proofreading DNA polymerases, such as Pfu, Phusion, or Q5, are often preferred in PCR cloning for the following reasons: Lower error rate: These enzymes possess 3'-5' exonuclease activity, which allows them to detect and remove misincorporated nucleotides during DNA synthesis. This proofreading ability significantly reduces the error rate compared to non-proofreading polymerases like regular Taq. Blunt-end products: Many proofreading polymerases generate blunt-end PCR products, which can be advantageous for certain cloning strategies. They amplify DNA at a much faster rate than regular Taq polymerase (Option A): Proofreading polymerases are often slower than Taq polymerase due to their proofreading activity. They can amplify longer DNA fragments more efficiently (Option C): While some proofreading polymerases can amplify longer fragments, this is not their primary advantage in PCR cloning. They are more tolerant to variations in PCR cycling conditions (Option D): Proofreading polymerases are generally not more tolerant to variations in PCR conditions; in fact, they often require more precise optimization.

References:

Harper's Biochemistry, 32nd Edition, Page 452.

↑ *Back to this question in Part A*

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Q2: Which of the following steps is NOT part of the standard PCR cloning process? (Q843242)

A. Denaturation

B. Annealing

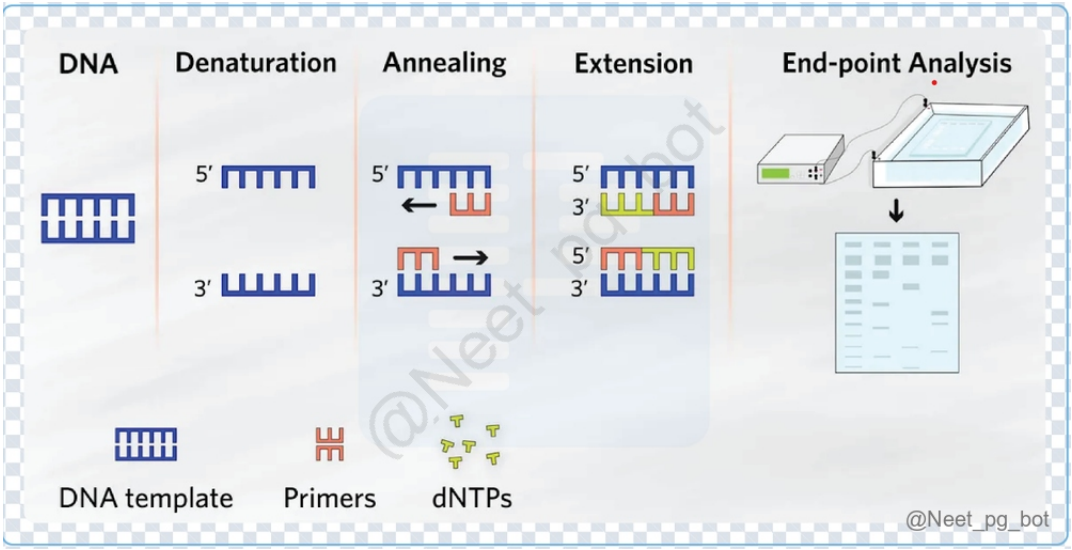
C. Ligation

D. Elongation

Correct Answer: C. Ligation

■ *Listen to Audio 1*

Explanation: Ligation is not a step involved in PCR .It is a step that occurs after PCR when inserting the amplified DNA fragment into a cloning vector. It involves the joining of the DNA fragment with the vector using DNA ligase, and it is not part of the actual PCR amplification process itself. PCR steps include: Step 1- Denaturation The double-stranded DNA is heated to around 95°C to separate into 2 single strands. Step 2- Annealing Addition of 2 primers, which will anneal to the flanking sequence of the desired fragments. An optimal temperature(5 degrees less than the average melting temperature of the primers) should be maintained to avoid non-specific amplification. Step 3- Elongation The enzyme used is thermostable Taq(Thermus Aquaticus)DNA polymerase. DNA polymerase elongates new strand in 5'3' direction and optimal temperature required for elongation is 72°. If PCR goes through 'N'number of cycle,then '2n' number of products formed



References:

Harper biochemistry ed 32,pg 452&453

↑ *Back to this question in Part A*

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Q3: Which of the following applications is a typical use of Polymerase Chain Reaction (PCR)? (Q843243)

A. Identifying specific genetic mutations

B. Determining protein structure

C. Measuring cell growth rates

D. Analyzing metabolite levels

Correct Answer: A. Identifying specific genetic mutations

■ *Listen to Audio 1*

Explanation: PCR applications: Medical Diagnostics: PCR can identify pathogens like bacteria and viruses by amplifying their DNA or RNA, making it possible to diagnose diseases such as COVID-19, tuberculosis, and HIV. Forensic Science/DNA Fingerprinting: PCR helps amplify DNA samples from crime scenes, enabling identification and comparison of genetic profiles to match suspects or victims. Detect Allelic Polymorphisms: PCR can identify allelic polymorphisms, which include single nucleotide polymorphisms (SNPs), insertions and deletions (indels), and gene amplifications. Biodiversity Studies: PCR can detect and identify species from environmental samples, helping in the study of biodiversity and monitoring endangered species. Determining protein structure (Option B): This process typically involves techniques like X-ray crystallography or nuclear magnetic resonance (NMR) spectroscopy, not PCR. Measuring cell growth rates (Option C): Cell growth is usually measured using methods such as cell counting, growth curves, or assays that assess cell proliferation. Analyzing metabolite levels (Option D): This involves techniques like mass spectrometry or chromatography to measure the concentrations of metabolites.

References:

Harper's Biochemistry, 32nd Edition, Page 453

↑ *Back to this question in Part A*

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Q4: What is the primary purpose of Reverse Transcription Polymerase Chain Reaction (RT-PCR) in molecular biology? (Q843244)

A. To amplify DNA sequences directly from the genome.

B. To convert RNA into complementary DNA (cDNA) and then amplify specific DNA sequences.

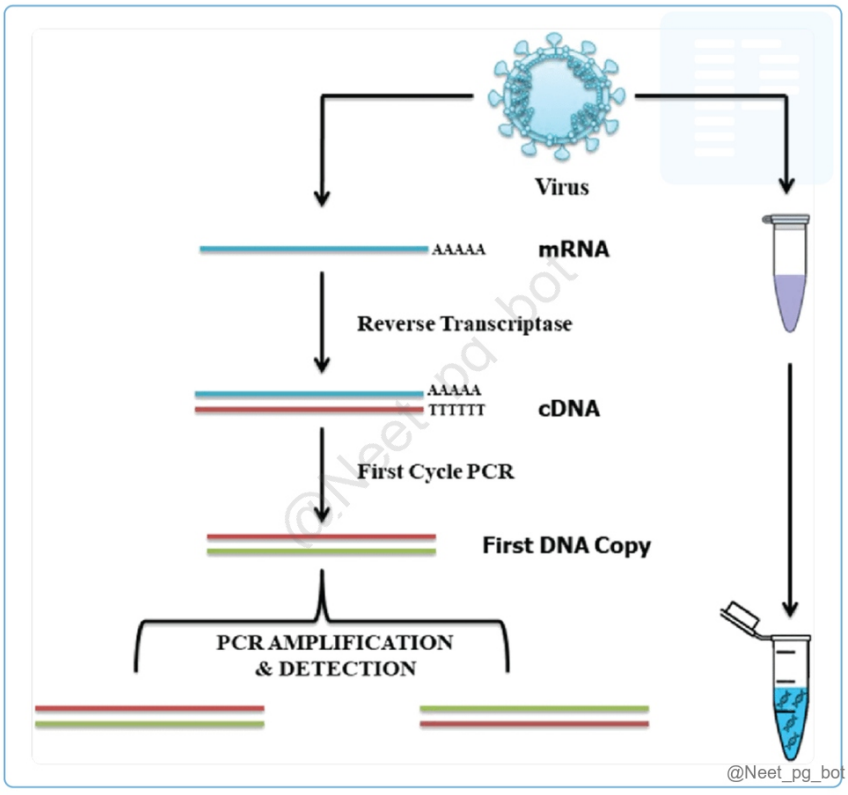
C. To sequence the entire genome of an organism.

D. To separate DNA fragments by size.

Correct Answer: B. To convert RNA into complementary DNA (cDNA) and then amplify specific DNA sequences.

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Explanation: RT-PCR is a technique used to detect and quantify RNA. It involves two main steps: Step 1- Conversion of RNA into complementary DNA (cDNA) using the enzyme reverse transcriptase. Step 2- Amplification of specific DNA sequences using PCR. To amplify DNA sequences directly from the genome (Option A): This option describes a process related to PCR but not specifically RT-PCR. Standard PCR amplifies DNA sequences directly from a sample, without the intermediate step of converting RNA to cDNA. To sequence the entire genome of an organism (Option C): Sequencing the entire genome of an organism is done through techniques such as whole-genome sequencing. To separate DNA fragments by size (Option D) : This describes gel electrophoresis, not RT-PCR. Gel electrophoresis is used to separate DNA fragments based on size, whereas RT-PCR is used for amplifying and quantifying specific DNA sequences derived from RNA.



References:

Harper's Biochemistry, 32nd Edition, Page 460

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5087796/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q5: What is the primary advantage of using real-time PCR (qPCR) over traditional PCR techniques? (Q843245)

A. It allows for the detection of multiple targets in a single reaction.

B. It enables the quantification of DNA as the amplification progresses.

C. It eliminates the need for a thermal cycler.

D. It provides a higher specificity in amplifying DNA.

Correct Answer: B. It enables the quantification of DNA as the amplification progresses.

■ *Listen to Audio 1*

Explanation: Real-time PCR, also known as quantitative PCR (qPCR), provides the advantage of quantifying DNA or RNA as the amplification process occurs in real time. This is achieved by measuring the fluorescence emitted by DNA-binding dyes or probes that are incorporated during the PCR amplification. Traditional PCR only allows for the detection of the PCR product at the end of the process, without the capability to quantify the initial amount of target nucleic acid. It allows for the detection of multiple targets in a single reaction (Option A): While real-time PCR can indeed be used to detect multiple targets in a single reaction through the use of different fluorescent probes or dyes, this is not its primary advantage. The main benefit of real-time PCR is its ability to quantify nucleic acids as the amplification occurs. It eliminates the need for a thermal cycler (Option C): Real-time PCR still requires a thermal cycler to facilitate the cycling of temperatures necessary for the PCR process. It provides a higher specificity in amplifying DNA (Option D): Real-time PCR does not inherently provide higher specificity compared to traditional PCR. Specificity is largely dependent on the design of primers and probes, not the real-time nature of the assay.

References:

Harper's Biochemistry, 32nd Edition, Page 460

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9778061/

↑ *Back to this question in Part A*

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Q6: Which of the following best describes the main principle behind Sanger sequencing? (Q843597)

A. Amplification of DNA segments to facilitate sequencing.

B. Incorporation of dideoxynucleotides to terminate DNA synthesis at specific bases.

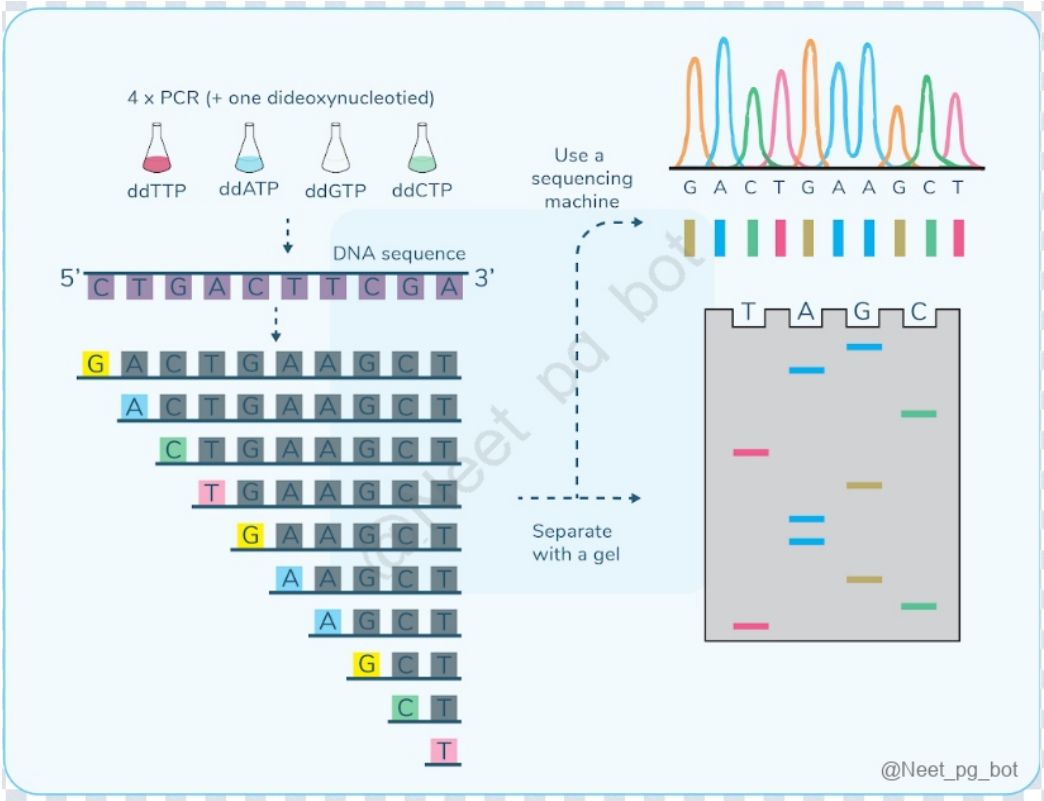
C. High-throughput sequencing of millions of DNA fragments simultaneously.

D. Insertion of a DNA fragment into a plasmid vector for replication and sequencing.

Correct Answer: B. Incorporation of dideoxynucleotides to terminate DNA synthesis at specific bases.

■ *Listen to Audio 1*

Explanation: Sanger sequencing is a method based on the principle of chain termination , where dideoxynucleotides (ddNTPs) are used to terminate DNA synthesis at specific points , allowing for the determination of the DNA sequence. Sanger sequencing was developed by Frederick Sanger in 1977. The technique is based on the principle of chain termination and involves several key steps: Preparation: The DNA of interest is first amplified to produce a large enough quantity for sequencing. Reaction Mix: The DNA is then mixed with DNA polymerase, a primer, and a mixture of deoxynucleotides (dNTPs) and dideoxynucleotides (ddNTPs). The ddNTPs are fluorescently labeled and act as chain terminators. Each ddNTP corresponds to one of the four DNA bases (A, T, C, G). Chain Termination: As the DNA polymerase extends the DNA strand, it occasionally incorporates a ddNTP instead of a dNTP. This incorporation causes the chain to terminate because ddNTPs lack the 3' hydroxyl group needed for further DNA elongation. Separation: The resulting mixture of terminated DNA fragments of varying lengths is then separated by size using capillary electrophoresis. Detection and Analysis: The fragments are detected as they pass through a laser, and the fluorescent labels indicate the identity of the terminating ddNTP. This information is used to reconstruct the DNA sequence. Sanger sequencing is known for its accuracy and was historically used for the Human Genome Project . However, next-generation sequencing techniques for high-throughput sequencing have largely supplanted it. Amplification of DNA segments to facilitate sequencing (Option A) is done by Polymerase Chain Reaction (PCR). High-throughput sequencing of millions of DNA fragments simultaneously (Option C) is done by Next-Generation Sequencing. Insertion of a DNA fragment into a plasmid vector for replication and sequencing (Option D) is done by Gene Cloning.



References:

Harper's Biochemistry, 32nd Edition, Page 451,452

Vasudevan’s Textbook of Biochemistry for Medical Students, 7th edition, Page 643

↑ *Back to this question in Part A*

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Q7: A 45-year-old patient diagnosed with COVID-19 has a real-time PCR test result showing a cycle threshold (Ct) value of 35. How should this Ct value be interpreted in the context of their infectivity and clinical management? (Q843598)

A. The patient has a high viral load and is highly infectious.

B. The patient has a low viral load and is less likely to be infectious.

C. The patient’s Ct value indicates they should be immediately admitted to the ICU.

D. The Ct value alone is sufficient to determine the patient’s disease severity and infectivity.

Correct Answer: B. The patient has a low viral load and is less likely to be infectious.

■ *Listen to Audio 1*

Explanation: The patient has a low viral load and is less likely to be infectious. Higher Ct values (>30) suggest a lower viral load, indicating reduced infectivity. Cycle Threshold (Ct) Value Definition The number of cycles required for the fluorescent signal to exceed a threshold, indicating detectable target nucleic acid (e.g., viral RNA). Significance of Ct Values Viral Load Estimation: Low Ct (≤30): High viral load. High Ct (>30): Low viral load. Infectivity: Ct >24 → reduced; Ct >34 → usually noninfectious. Use: Guides clinical decisions on isolation, treatment, and reporting. Limitations and Considerations Ct Value Variability: Across tests: Varies with platform, reagents, and target genes—each lab sets its own reference range. Sample quality: Affected by collection, timing, and storage; nasal vs. throat samples may differ. Interpretation: Ct alone can't define infectivity or severity; must consider clinical context. Option A: Incorrect. A Ct value of 35 indicates a lower viral load, not a high one. Option C: Incorrect. A Ct value alone does not necessitate ICU admission without considering other clinical factors. Option D: Incorrect. Ct values should be interpreted alongside other clinical factors, not in isolation.

References:

Reference : https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8362640/
https://pubmed.ncbi.nlm.nih.gov/35576147/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q8: A forensic scientist is analyzing a DNA sample from a crime scene. The DNA is digested with restriction enzymes, separated by gel electrophoresis, and transferred to a nitrocellulose membrane. Labeled DNA probes are then used to detect specific fragments. Which technique is the scientist most likely using? (Q843599)

A. Restriction Fragment Length Polymorphism (RFLP)

B. Polymerase Chain Reaction (PCR)

C. Single-Strand Conformation Polymorphism (SSCP)

D. DNA Sequencing

Correct Answer: A. Restriction Fragment Length Polymorphism (RFLP)

■ *Listen to Audio 1*

Explanation: RFLP involves DNA digestion with restriction enzymes, gel electrophoresis, transfer to a nitrocellulose membrane, hybridization with labeled probes, and visualization of fragments. Restriction Fragment Length Polymorphism (RFLP) Variation in DNA fragment lengths due to differences in DNA sequences when digested with restriction enzymes are used as genetic markers. Applications: Parenthood Disputes, Population Genetics, Genetic Disease Diagnosis, Genetic Mapping, Cloning, Forensic Medicine. Sequential Steps involved using RFLP technique 1. DNA Digestion DNA is cut by restriction enzymes at specific sites. 2. Electrophoresis DNA fragments are separated by size using gel electrophoresis. 3. Denaturation To enable probe binding, the

separated fragments are denatured 4. Southern Blotting Fragments are transferred from the gel to a nitrocellulose membrane. 5. Hybridization DNA fragments hybridize with labeled DNA probes. 6. Visualization Hybridized fragments are visualized to reveal the presence or absence of restriction sites. Option B: PCR is used to amplify DNA, not to analyze fragment length variations. Option C: SSCP is used to detect variations in single-stranded DNA based on their conformational differences, not the length of restriction fragments. Option D: DNA sequencing determines the nucleotide order, not the length variation of fragments after restriction enzyme digestion.

References:

Vasudevan biochemistry 7th edition pg: 637-638

↑ *Back to this question in Part A*

@Neet_pg_bot

Q9: Which of the following statements about Denaturing Gradient Gel Electrophoresis (DGGE) is false? (Q843600)

A. DGGE can separate DNA fragments based on their melting characteristics.

B. DGGE is effective for analyzing DNA segments of 200-300 base pairs.

C. DGGE requires the use of a denaturant gradient within a polyacrylamide gel.

D. DGGE is commonly used for detecting translocations.

Correct Answer: D. DGGE is commonly used for detecting translocations.

■ *Listen to Audio 1*

Explanation: DGGE is not used used for detecting translocations. Denaturing Gradient Gel Electrophoresis (DGGE) Purpose: Separates DNA fragments by melting behavior. Range: Best for 200–300 bp (e.g., 16S rRNA genes). Mechanism: DNA migrates through a denaturant gradient; differing melt points cause separation. Uses: Detects SNPs, profiles microbial communities, and aids genetic/clinical diagnostics. Sequential Steps in Denaturing Gradient Gel Electrophoresis (DGGE) 1. DNA Extraction Isolate DNA from the sample. 2. PCR Amplification Amplify the target DNA region with specific primers. 3. Gel Preparation Create a polyacrylamide gel with a denaturant gradient. (Option C) 4. Electrophoresis Load DNA samples into the gel and apply an electric field for separation. 5. Visualization Stain the gel and visualize bands under UV light.

References:

https://pubmed.ncbi.nlm.nih.gov/23913290/

https://pubmed.ncbi.nlm.nih.gov/8199599/

https://pubmed.ncbi.nlm.nih.gov/10572650/

https://pubmed.ncbi.nlm.nih.gov/12828664/

↑ *Back to this question in Part A*

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Q10: Which of the following methods is specifically designed to detect differences in DNA sequences based on variations in the electrophoretic mobility of single-stranded DNA fragments? (Q843602)

A. RFLP (Restriction Fragment Length Polymorphism)

B. SSCP (Single-Strand Conformational Polymorphism)

C. Sequencing

D. All of the above methods are equally effective for detecting DNA sequence variations.

Correct Answer: B. SSCP (Single-Strand Conformational Polymorphism)

■ *Listen to Audio 1*

Explanation: SSCP (Single-Strand Conformational Polymorphism) is the method that directly addresses variations in electrophoretic mobility of single-stranded DNA fragments. This technique is specifically designed to detect sequence variations by analyzing the conformational changes of single-stranded DNA. When DNA is denatured into single strands and then subjected to electrophoresis, differences in the DNA sequence can cause different folding patterns or conformations. These conformational changes affect the migration of the DNA through a non-denaturing gel, making SSCP highly effective for detecting small mutations or variations in DNA sequences. RFLP (Restriction Fragment Length Polymorphism) (Option A): Detects DNA sequence variations by comparing fragment lengths after restriction enzyme digestion and gel electrophoresis; does not assess single-stranded DNA mobility. Sequencing (Option C): Directly determines DNA sequence but is not based on electrophoretic mobility changes of single-stranded DNA.

References:

Harper biochemistry, 32nd Edition, Pages 68, 460.

https://www.sciencedirect.com/science/article/abs/pii/S0002929707603730

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↑ *Back to this question in Part A*

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Q11: A researcher is experiencing issues with the accuracy and efficiency of DNA amplification during a PCR experiment. They suspect the problem might be related to magnesium ion concentration. What role does magnesium play in PCR, and how might its concentration affect the experiment? (Q843603)

A. Magnesium ions serve only to stabilize the DNA template and do not affect enzyme activity.

B. Excessive magnesium concentration enhances DNA polymerase activity, reducing the likelihood of errors.

C. Insufficient magnesium concentration can lead to decreased enzyme activity and lower DNA amplification efficiency.

D. Magnesium ions are not essential for the nucleophilic attack of the primer's 3' hydroxyl group during DNA synthesis.

Correct Answer: C. Insufficient magnesium concentration can lead to decreased enzyme activity and lower DNA amplification efficiency.

■ *Listen to Audio 1*

Explanation: Magnesium ions (Mg^{2}■) are critical in PCR, acting as essential cofactors for DNA polymerases and insufficient concentration can decrease enzyme activity and amplification efficiency. Role of Magnesium in PCR 1. Enzymatic Function Cofactor: Positions nucleotides and stabilizes negative charges. Catalysis: Aligns substrates, maintains active-site geometry, and stabilizes the transition state for accurate DNA synthesis. 2. Stability and Fidelity: Maintains DNA strand stability, minimizes replication errors, and requires optimal levels for efficient, accurate amplification. Magnesium ions are essential for enzyme activity, not just DNA template stabilization.(Option A ruled out) Excessive magnesium concentration can lead to non-specific amplification and errors..(Option B ruled out) Magnesium ions are necessary for the nucleophilic attack of the primer's 3' hydroxyl group on the incoming nucleotide during DNA synthesis.(Option D ruled out)

References:

pubmed.ncbi.nlm.nih.gov/11295157/

www.ncbi.nlm.nih.gov/pmc/articles/PMC1868394/

↑ *Back to this question in Part A*

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• [07_ Genetics] 024 Advanced Molecular Techniques Recombinant Dna Technology Microarray Crispr Lac Operon

« Back to Folder Directory

[07_ Genetics] 024 Advanced Molecular Techniques Recombinant Dna Technology Microarray Crispr Lac Operon — Quick Jump

Q1 (p. 1084)	*Q2 (p. 1085)*	*Q3 (p. 1086)*	*Q4 (p. 1087)*	*Q5 (p. 1088)*	*Q6 (p. 1089)*
Q7 (p. 1090)	*Q8 (p. 1091)*	*Q9 (p. 1092)*	*Q10 (p. 1093)*	*Q11 (p. 1094)*	*Q12 (p. 1095)*
Q13 (p. 1097)	*Q14 (p. 1098)*	*Q15 (p. 1099)*	*Q16 (p. 1100)*	*Q17 (p. 1101)*	*Q18 (p. 1102)*

Q1: Which of the following best describes a mechanism involved in epigenetic changes that lead to heterochromatin formation? (Q843426)

A. Histone acetylation and DNA methylation of adenine residues

B. Histone deacetylation and methylation in CG islands

C. DNA replication

D. Histone phosphorylation

Correct Answer: B. Histone deacetylation and methylation in CG islands

■ *Listen to Audio 1*

Explanation: Epigenetic changes that lead to heterochromatin formation are primarily driven by histone deacetylation and methylation of cytidine residues in CG islands. Epigenetic changes involve stable alterations in gene expression that do not involve changes in the nucleotide sequence. Epigenetic Modifications: DNA Methylation: Represses gene expression by methylation of CpG islands. Involved in ageing, cancer, genomic imprinting, X chromosome inactivation, and dysregulation linked to diseases like Fragile X syndrome. Histone Methylation: Suppress or activate transcription depending on the location of methyl groups. Methylation of lysine and arginine residues on histones. Histone Acetylation: Activates transcription (Option A) The addition of acetyl groups neutralises histone charges, relaxing DNA coiling. Generally increases gene expression; affects processes like thyroid hormone synthesis. Histone Deacetylation: Represses transcription. Removal of acetyl groups tightens DNA coiling. Typically, it decreases gene expression and is linked to diseases such as Huntington's disease. Sumoylation: Addition of a SUMO (Small Ubiquitin-like Modifier) protein; typically represses transcription by promoting the recruitment of corepressor complexes or blocking transcriptional activators. Ubiquitination: Histone H2A or H2B ubiquitination plays a dual role, acting either as a signal for transcriptional silencing (e.g., Polycomb group proteins) or as a precursor for transcriptional activation. Histone Phosphorylation: Highly dynamic modification linked to chromosome condensation during mitosis and rapid response to cellular stress signals. Chromatin Remodeling: ATP-dependent remodeling complexes (e.g., SWI/SNF) physically move, slide, or restructure nucleosomes to physically regulate access of RNA polymerase to promoter DNA. DNA replication and Histone phosphorylation(Option C & D): Not associated with Epigenetic modification.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 92

↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: Which of the following statements about the lac operon is/are correct? Lac I is constitutively expressed. Lac Y is responsible for lactose transport into the cell. The structural genes are expressed when the CAP site is free. The ZYA genes are expressed when the operator site is free. (Q843437)

A. 1 and 2

B. 1, 2, and 3

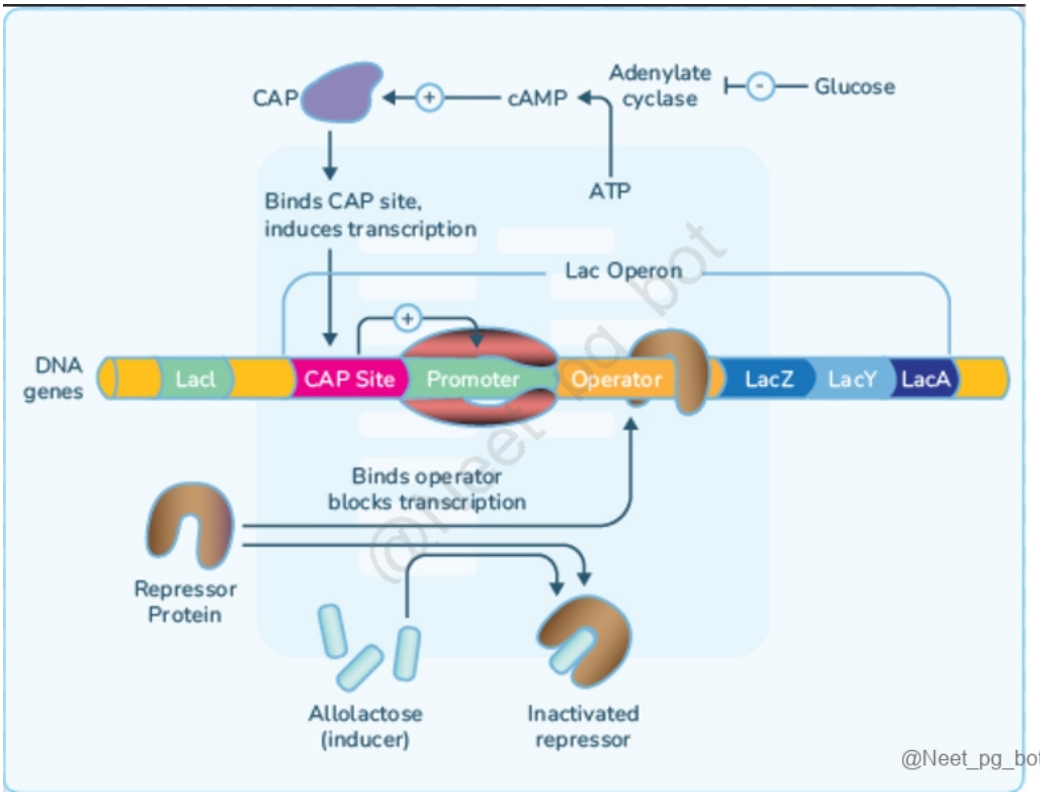
C. 1, 2, and 4

D. 1, 2, 3, and 4

Correct Answer: C. 1, 2, and 4

■ *Listen to Audio 1*

Explanation: The structural genes are expressed when the CAP site is Occupied. Lac Operon: The lac operon enables E. coli to metabolise lactose by producing enzymes necessary for lactose uptake and breakdown in the absence of glucose. Components: Structural Genes: lacZ: Encodes β-galactosidase (Breaks down lactose into glucose and galactose) lacY: Encodes lactose permease (Helps lactose enter the cell) (Statement 2) lacA: Encodes thiogalactoside transacetylase Regulatory Elements : lacI Gene: Encodes the LacI repressor, which binds to the operator to block transcription when lactose is absent. (Lactose unbinds repressor from Operator site) Constitutively Expressed (Statement 1) Promoter (P) : The binding site for RNA polymerase, which initiates transcription of the lac genes. Operator (O) : A DNA sequence that the lac repressor protein binds to, blocking transcription when lactose is absent. CAP (Catabolite Activator Protein): Bind to the CAP binding site near the promoter and enhance transcription (Statement 3). This requires requires cAMP (Glucose inhibits Adenylate cyclase which converts ATP to cAMP) Mechanism: Lactose unbinds repressor from Operator site ➜ Operator site becomes free ➜ Increases Transcription (Statement 4)



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page: 422,423

↑ *Back to this question in Part A*

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Q3: Which of the following statements about cloning vectors is correct? Plasmids can carry DNA inserts up to 10 kilobases in size. Phage vectors can carry DNA inserts up to approximately 50 kilobases. Cosmids are hybrid vectors that combine features of plasmids and phages. Artificial chromosomes are used to clone very large DNA fragments. (Q843435)

A. 1, 2, and 3

B. 1 and 4

C. All of the above

D. 1, 3, and 4

Correct Answer: D. 1, 3, and 4

■ *Listen to Audio 1*

Explanation: Phage vectors can carry DNA inserts up to approximately 20 kilobases, not 50 kilobases. Cloning Vectors are DNA molecules that carry and replicate foreign DNA segments within host cells. They are useful in molecular biology for gene cloning and manipulation. Vector Size (Kb) Features Plasmids <10 Small, circular DNA molecules found in Bacteria Commonly used for cloning and expressing small DNA fragments Replicate independently of the bacterial chromosome. Bacteriophages 10-20 Bacterial viruses with linear DNA genomes Useful for cloning slightly larger DNA fragments Infect bacterial cells to propagate the cloned DNA Bacterial Artificial Chromosomes (BACs) 50-250 BACs are vectors based on the F-plasmid of E. coli. They can accommodate large DNA inserts Used for constructing genomic libraries Yeast Artificial Chromosomes (YACs) >500 YACs are vectors that can accommodate very large DNA inserts, even larger than BACs More prone to chimerism and instability compared to BACs Cosmids 35 - 50 Hybrid vectors combining plasmid and phage features Viral Vectors Derived from viruses that infect mammalian cells Include adenoviral, adenovirus-associated viral (AAV), and retroviral vectors. Phagemids ~5-10 Hybrid plasmid + phage f1 origin Generates single-stranded DNA for sequencing/mutagenesis. Expression Vectors Contains promoter, operator, and Shine-Dalgarno; used for high-level protein synthesis. Shuttle Vectors Contains two origins of replication; allows replication in two different host organisms (e.g., bacteria + yeast). Ti-Plasmid (Binary) ~200 Derived from Agrobacterium tumefaciens; used for plant transformation/transgenic plants.

References:

Harper’s Biochemistry, 32nd edition, Page 448

↑ *Back to this question in Part A*

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Q4: In their CRISPR-Cas9 experiment with a mammalian cell line, the research team observes the repair of DNA double-strand breaks. Which repair mechanisms are they most likely observing? Non-Homologous End Joining (NHEJ) Homologous End Joining (HEJ) Nucleotide Excision Repair (NER) Base Excision Repair (BER) (Q843432)

A. 1 and 2

B. 1, 2, 3

C. 1 and 4

D. All of the above

Correct Answer: A. 1 and 2

■ *Listen to Audio 1*

Explanation: In a CRISPR-Cas9 experiment , the Cas9 enzyme introduces double-strand breaks (DSBs) in DNA. The cell then uses two primary repair mechanisms for these DSBs: Non-Homologous End Joining (NHEJ): A

quick, error-prone repair mechanism that directly ligates the broken DNA ends without a template. Commonly results in insertions or deletions (indels) , making it useful for gene knockouts . Homologous Recombination / Homologous End Joining (HEJ): A high-fidelity repair mechanism that uses a homologous DNA template (often supplied by the experimenter) to repair the break accurately. Used in gene knock-in strategies. DNA Repair Mechanisms in CRISPR Context Mechanism Role in CRISPR Repair DNA Damage Type Handled Non-Homologous End Joining Yes Double-strand breaks (DSBs) Homologous End Joining (HR) Yes Double-strand breaks (DSBs) Nucleotide Excision Repair No Bulky lesions (e.g. UV damage) Base Excision Repair No Small base damage or single-strand breaks 1, 2, 3 (NHEJ, HEJ, NER) (Option B): Incorrect because NER does not participate in double-strand break repair. 1 and 4 (NHEJ and BER) (Option C): Incorrect because BER handles single-strand lesions, not DSBs. All of the above (Option D): Incorrect because NER and BER are not involved in repairing Cas9-induced DSBs.

References:

Harper’s Illustrated Biochemistry 32nd Edition - 454

↑ *Back to this question in Part A*

@Neet_pg_bot

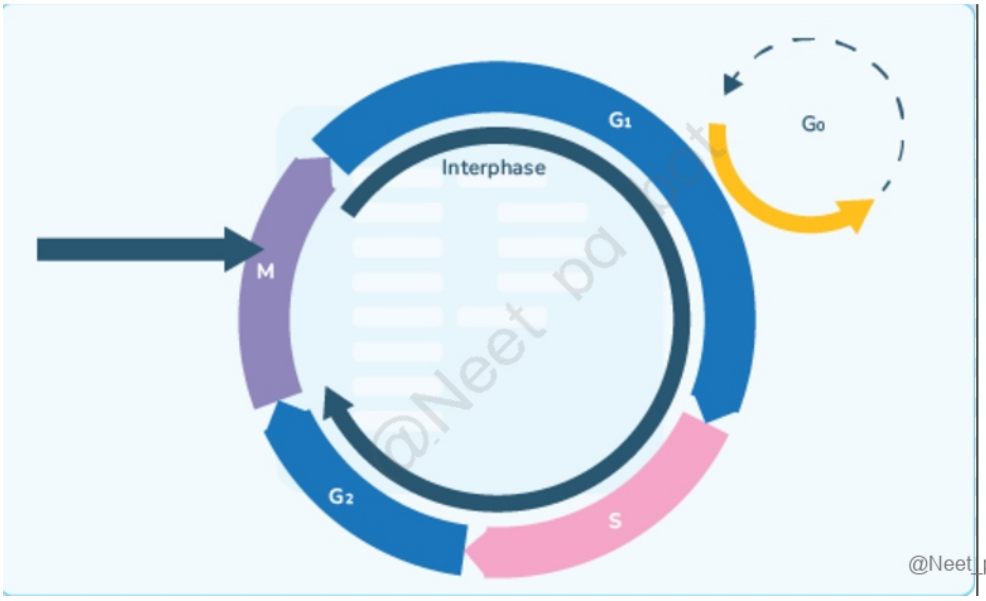
Q5: Which of the following is the feature of the ‘M’ phase in the image of the Cell cycle? (Q843431)

A. DNA replication occurs to duplicate the genome.

B. Chromosomes are highly condensed to facilitate segregation.

C. Cell growth and preparation for DNA synthesis take place.

D. Proofreading and repair of DNA replication errors are conducted.



Correct Answer: B. Chromosomes are highly condensed to facilitate segregation.

■ *Listen to Audio 1*

Explanation: This is a key feature of the M phase, where chromosomes become highly condensed to ensure proper segregation during mitosis . They are compressed to about 8000 fold during metaphase in M-Phase. Cell Cycle: The cell cycle is a series of stages that a cell goes through to divide and produce two daughter cells. Phases of the Cell Cycle: Phases Chromosome State Features Gap 1 (G1) Phase Pre-synthetic phase Uncondensed Cell growth and synthesis of proteins and organelles. (Option C) DNA proofreading and repair. Synthesis (S) Phase DNA replication occurs (Option A) Uncondensed Replication of the entire genome and formation of sister chromatids. Increase in the levels of DNA polymerases and other replication enzymes. Gap 2 (G2) Phase Post-synthetic or pre-mitotic phase Uncondensed Final proofreading and repair. (Option D) Mitosis (M) Phase Division of the nucleus and cytoplasm to produce two daughter cells. Condensed (ensure proper segregation). Chromosome condensation, alignment, and segregation. Cytokinesis, where the cytoplasm divides, resulting in two separate cells. Interphase The combined phases of G1, S, and G2 are where the cell grows, duplicates its DNA and prepares for division. Gap 0 (G0) Phase (Resting phase) Condensed Permanent or Temporary phase based on Cell type.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 365,379

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: A 32-year-old patient presents with a family history of progressive muscle weakness, cognitive decline, and involuntary movements. Genetic testing reveals a specific type of microsatellite instability disorder. Which of the following trinucleotide repeat sequences is most likely associated? (Q843430)

A. CAG

B. CTG

C. CGG

D. GAA

Correct Answer: A. CAG

■ *Listen to Audio 1*

Explanation: The clinical presentation is consistent with Huntington's Disease, which is characterised by progressive muscle weakness, cognitive decline, and involuntary movements (chorea). The specific trinucleotide repeat expansion associated with this condition is CAG. Microsatellite instability (MSI) Disorders: Genetic conditions are characterised by the accumulation of mutations in microsatellite regions of DNA due to failure in the DNA mismatch repair system. Anticipation is seen (Disease severity increases in successive generations) Trinucleotide Repeat Sequences and Associated Disorders: Trinucleotide repeat sequences are short DNA sequences in which a specific set of three nucleotides is repeated multiple times in tandem beyond a normal range, resulting in instability and dysfunction. Disease Trinucleotide Repeats Mode of Inheritance Features Huntington's Disease CAG AD Caudate nucleus atrophy (Box-car ventricles); Chorea, psychiatric symptoms. Kennedy Syndrome CAG XLR Bulbar/spinal muscular atrophy and gynecomastia. Myotonic Dystrophy CTG (Option B) AD Myotonia , distal muscle wasting, cataracts , cardiac arrhythmias. Fragile X Syndrome CGG (Option C) XLD Macroorchidism , long face, large everted ears, intellectual disability. Friedreich's Ataxia GAA (Option D) AR Hypertrophic cardiomyopathy , pes cavus, ataxia, diabetes. Note: AD: Autosomal Dominant; AR: Autosomal Recessive; XLD: X-Linked Dominant

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 36

↑ *Back to this question in Part A*

@Neet_pg_bot

Q7: A 5-year-old boy presents with frequent falls, difficulty climbing stairs, and an unusual way of getting up from the floor. Genetic testing reveals a mutation in a gene associated with a severe muscle disorder. Which of the following statements about the involved gene is true? (Q843429)

A. Located on chromosome 7

B. Largest in the human genome.

C. Associated with a condition that primarily affects females

D. The Gene’s product is a component of the sarcomere.

Correct Answer: B. Largest in the human genome.

■ *Listen to Audio 1*

Explanation: The clinical presentation with frequent falls, difficulty climbing stairs and unusual way of getting up (Gower sign) points towards Duchenne muscular Dystrophy. Inherited in X-linked Recessive pattern - Primarily affects males (Option C). The gene affected is Dystrophin , which is the largest gene in the human genome, located on the X chromosome (Option A). Dystrophin is a crucial protein found in muscle cells that helps maintain the muscle fibre's structural integrity. It acts as a bridge between sarcolemma and the internal cytoskeleton, which helps in stabilization. Option D: Dystrophin is not a component of the sarcomere but is crucial for anchoring the cytoskeleton to the muscle cell membrane. Sarcomere components involved in contraction include actin and myosin.

References:

Vasudevan Textbook of Biochemistry 7th Edition Pg - 637

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4767260/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q8: Satellite DNA has various applications due to its unique repetitive sequences. Which of the following is not an application of satellite DNA? (Q843428)

A. Forensic identification and criminal investigations

B. Determining genetic susceptibility to cardiovascular diseases

C. Resolving paternal dispute cases

D. Studying evolutionary relationships and genetic diversity

Correct Answer: B. Determining genetic susceptibility to cardiovascular diseases

■ *Listen to Audio 1*

Explanation: Satellite DNA is not used to identify susceptibility to cardiovascular diseases, as it mainly provides information about repetitive DNA sequences rather than disease susceptibility. Satellite DNA refers to repetitive DNA sequences that are found in specific regions of the genome, notably in centromeric and telomeric regions. Types: Highly Repetitive Sequences (HRS) : These sequences repeats >1,000,000 times. Simple Sequence Repeats (SSR) : Also known as microsatellites, these are short, repetitive sequences. Minisatellite Repeats : Known as Variable Number Tandem Repeats (VNTR), these are longer repetitive sequences compared to microsatellites. Applications: Forensic Identification (Option A ruled out) : Satellite DNA is used in forensic science to identify individuals due to its high variability between people uniquely. Paternal Dispute Resolution (Option C ruled out) :

It helps resolve paternal disputes by matching genetic profiles through repetitive DNA sequences. Principle: A child inherits two alleles for every STR locus: one from the mother and one from the father. Example: If a child has alleles (12, 16) at a specific locus, and the mother has (12, 14), the child must have inherited the 16 allele from the biological father. If the alleged father lacks a 16 allele at that locus, he is excluded as the father. Genetic Studies : To study evolutionary relationships and genetic diversity among different species. (Option D ruled out)

References:

Vasudevan Textbook of Biochemistry 7th Edition Pg - 578

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5219911/

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8122249/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q9: Which of the following statements is not accurate regarding post-transcriptional modifications of eukaryotic RNA? (Q843427)

A. The addition of a poly-A tail at the 3′ end.

B. The 5′ capping involves a 5′ to 5′ triphosphate bridge.

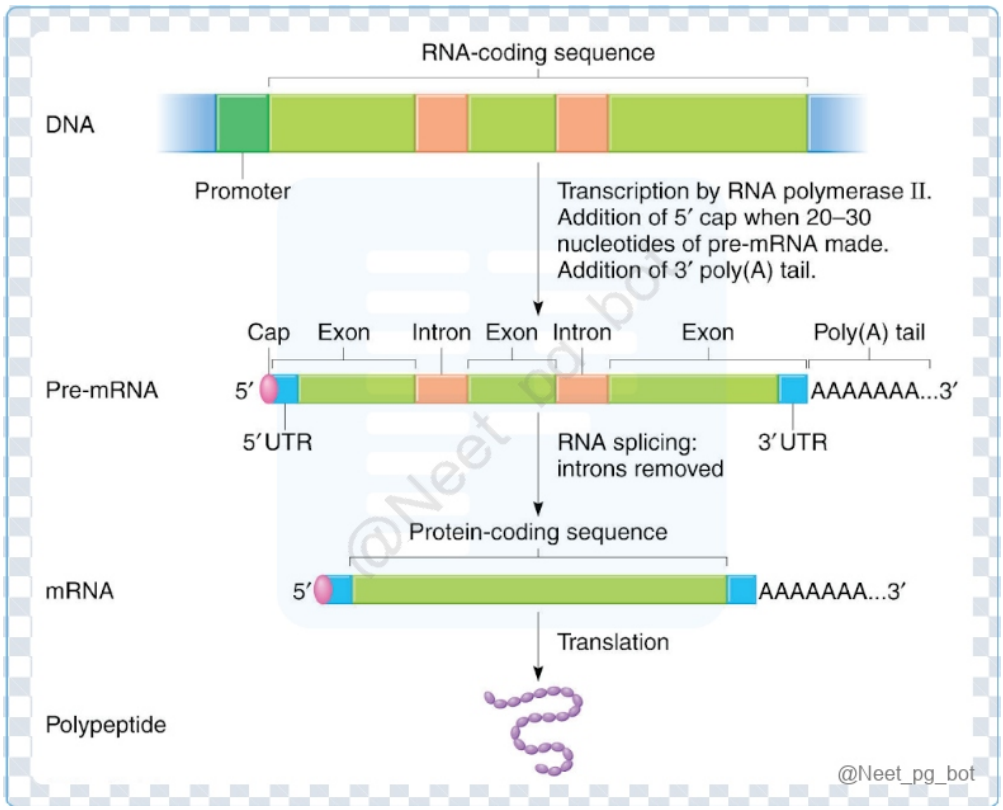
C. Methylation of the N6 position of adenine.

D. Introns are removed, and exons are spliced within the cytoplasm.

Correct Answer: D. Introns are removed, and exons are spliced within the cytoplasm.

■ *Listen to Audio 1*

Explanation: Introns are removed, and exons are spliced together in the nucleus, not the cytoplasm. Post-Transcriptional Modification of Eukaryotic RNA: Primary Transcript (hnRNA): Newly synthesised mRNA directly from DNA; includes both coding (exons) and non-coding (introns) regions. Key Modifications: Poly-A Tailing (3′ End) Addition of a poly-A tail (20-250 nucleotides) in the nucleoplasm. Protects mRNA from degradation. 5′ Capping Addition of a 7-methyl guanosine cap at the 5′ end. Involves a 5′ to 5′ triphosphate bridge. Enhances mRNA stability and translation initiation. Splicing Introns (non-coding regions) are removed, and exons (coding regions) are joined together. This splicing occurs in the nucleus and requires energy. Methylation Methylation of adenine residues and ribose 2′-hydroxyl groups. Primarily occurs in the cytoplasm for tRNA and rRNA. Bacterial mRNA: Not subject to extensive post-transcriptional modifications. Translation can begin before transcription is complete.



References:

Vasudevan Textbook of Biochemistry 7th Edition Pg - 591

↑ *Back to this question in Part A*

@Neet_pg_bot

Q10: A researcher aims to clone a large eukaryotic gene fragment (~600 kb) for use in functional genomics studies. Which of the following cloning vectors is the most appropriate for this purpose? (Q843416)

A. Plasmid

B. Bacteriophage λ

C. Yeast Artificial Chromosome (YAC)

D. Cosmid

Correct Answer: C. Yeast Artificial Chromosome (YAC)

■ *Listen to Audio 1*

Explanation: Yeast Artificial Chromosomes (YACs) are ideal for cloning large DNA fragments (100–1000 kb), making them suitable for large eukaryotic gene fragments (~600 kb) Cloning Type Key Features Insert Size Applications Plasmid-Based Cloning (Option A) Small, circular DNA in bacteria, independent replication, antibiotic resistance genes ~10 kb Gene cloning, bacterial expression Phage-Based Cloning (Option B) Uses bacteriophage (e.g., λ phage), foreign DNA inserted into phage genome ~20 kb High-efficiency cloning, phage display Cosmid-Based Cloning (Option D) Hybrid of plasmid & phage λ, contains cos sites for efficient packaging ~100-300 kb Genome sequencing, gene mapping Yeast Artificial Chromosome (YAC) Functions as a eukaryotic chromosome, contains centromere & telomere ~100-1000 kb Cloning large eukaryotic DNA fragments Mammalian Viral Vectors Used in gene therapy, adenoviral (DNA-based), retroviral (RNA-based) Varies Gene expression in mammalian cells, stable integration

References:

Harper's Biochemistry, 32nd edition, Pages 447, 448

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: In human somatic cells, which of the following statements accurately describes the chromatin and its characteristics? (Q843425)

A. Constitutive heterochromatin is always active

B. Facultative heterochromatin includes the Barr body

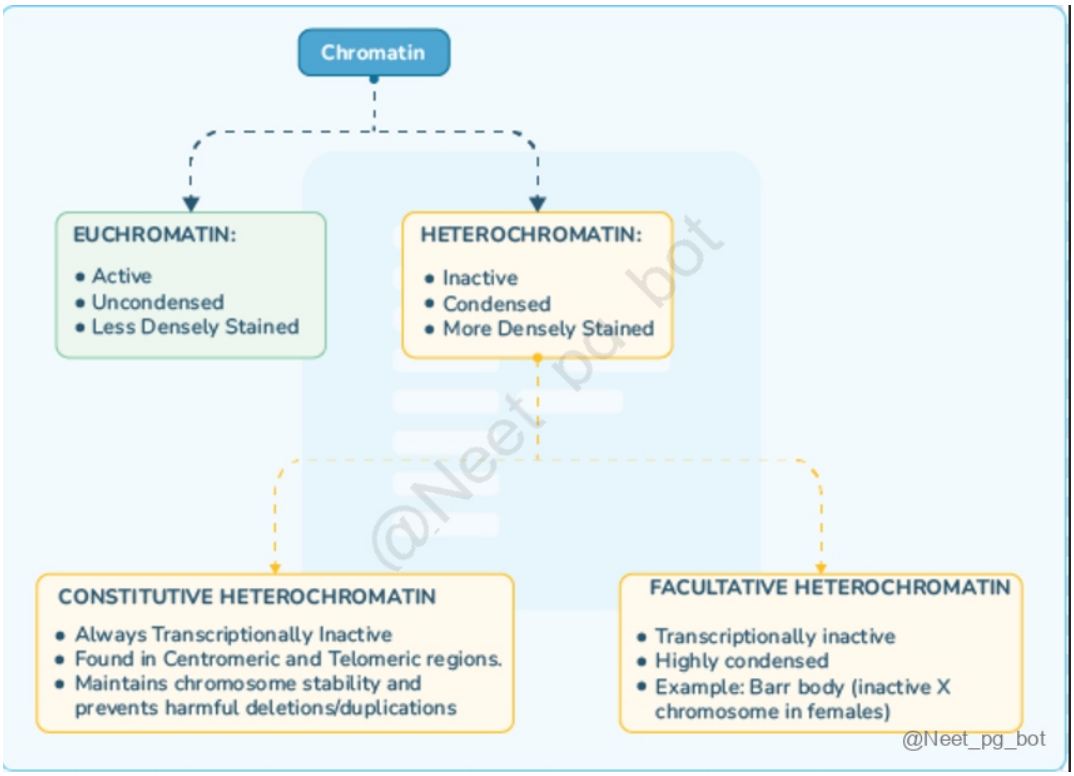
C. Euchromatin is found in centromeric regions

D. Heterochromatin is active and less stained

Correct Answer: B. Facultative heterochromatin includes the Barr body

■ *Listen to Audio 1*

Explanation: Facultative heterochromatin includes the Barr body. In somatic cells of females, one of the two X-chromosomes is transcriptionally active while the other is transcriptionally inactive and highly condensed, known as a Barr body. Chromatin is a complex of DNA and proteins found in the nucleus of eukaryotic cells. It plays a crucial role in packaging DNA into a more compact, dense structure, allowing it to fit within the cell nucleus and regulating gene expression. Types of Chromatin: Euchromatin (Transcriptionally Active) Heterochromatin (Transcriptionally Inactive)



References:

Harper's Illustrated Biochemistry 32nd Edition Pg - 364

↑ *Back to this question in Part A*

@Neet_pg_bot

Q12: In a laboratory setting, E. coli bacteria are cultured in a medium where glucose and lactose is available. To increase the expression of the lac operon, which levels of lactose and cAMP would you expect to be present? (Q843424)

A. High lactose and low cAMP

B. Low lactose and high cAMP

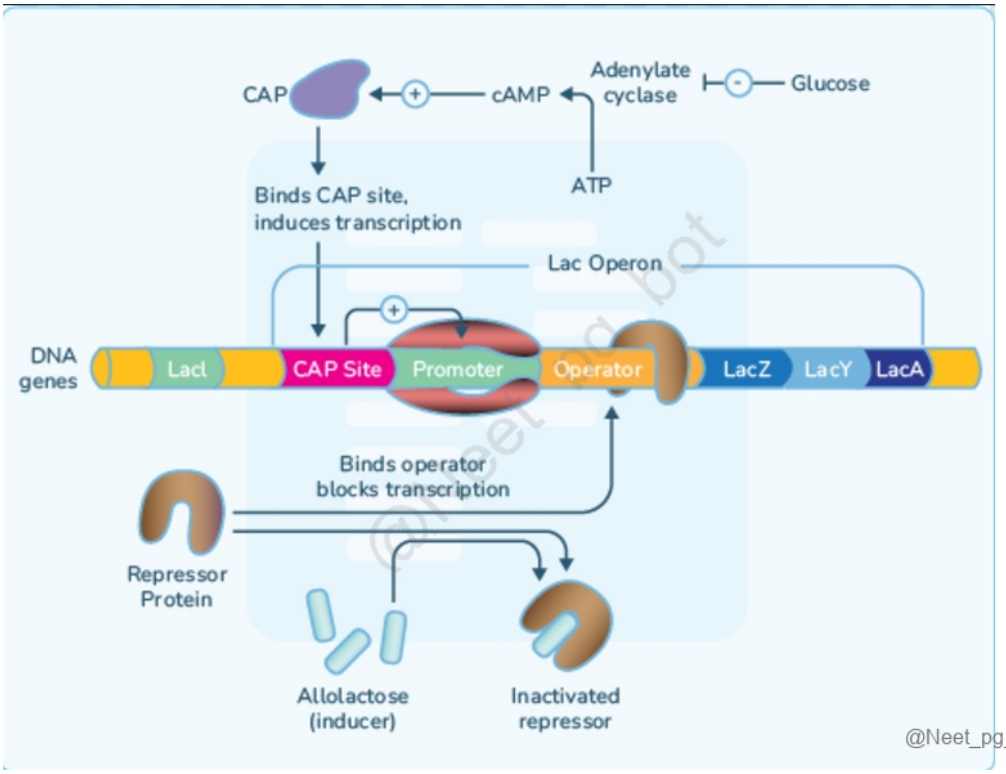
C. High lactose and high cAMP

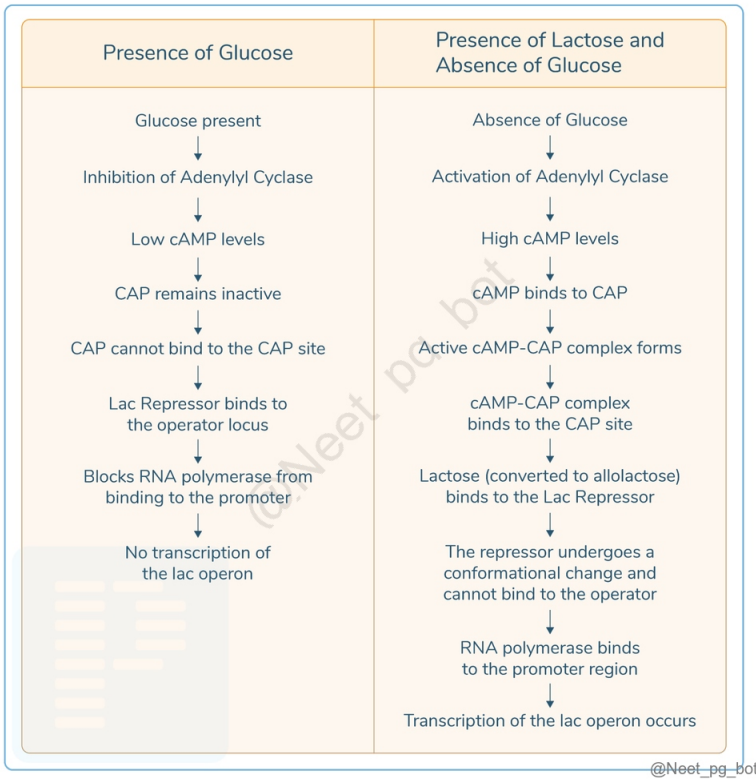
D. Low lactose and low cAMP

Correct Answer: C. High lactose and high cAMP

■ *Listen to Audio 1*

Explanation: In a laboratory setting aiming to maximise the expression of the lac operon, optimal transcription would be expected under conditions with high lactose and high cAMP. Lac Operon: The lac operon enables E. coli to metabolise lactose by producing enzymes necessary for lactose uptake and breakdown in the absence of glucose. Basic components: lacZ: Encodes β-galactosidase, which breaks down lactose into glucose and galactose. lacY: Encodes lactose permease, which helps lactose enter the cell. lacA: Encodes thiogalactoside transacetylase. lacI: Encodes the LacI repressor, which binds to the operator to block transcription when lactose is not present. (Lactose unbinds repressor from Operator site) CAP (Catabolite Activator Protein): Requires cAMP to bind to the CAP binding site (CRE) near the promoter and enhance transcription. (Glucose inhibits Adenylate cyclase, which converts ATP - - - - > cAMP) Regulation:





References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 422,423

↑ *Back to this question in Part A*

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Q13: Which of the following techniques is specifically designed to introduce specific genetic modifications, such as gene knock-ins or knock-outs, into the genome of an organism? (Q843423)

A. FISH (Fluorescence In Situ Hybridization)

B. CRISPR-Cas

C. Recombinant DNA Technology

D. Comparative Genomic Hybridization

Correct Answer: B. CRISPR-Cas

■ *Listen to Audio 1*

Explanation: CRISPR-Cas is a genome-editing tool that can be used to introduce specific genetic modifications, including gene knock-ins (adding new genetic material) and gene knock-outs (disrupting or deleting existing genes). CRISPR-Cas Mechanism: Guide RNA : Directs the Cas enzyme to the specific DNA sequence. Cas Enzyme : Cuts the DNA at the target location. DNA Repair : Knock-out: NHEJ (Non-Homologous End Joining), repairs the break by directly ligating the DNA ends, often leading to gene knockout. Knock-in: HDR (Homologous Directed Repair), uses a template to repair the break accurately, allowing for gene knock-in. RNA Interference (RNAi): A mechanism for post-transcriptional gene knockdown . Pathway: Long dsRNA → Dicer (RNase III enzyme) processes it into 21-25 bp siRNA . RISC Complex: The siRNA loads into RISC (RNA-induced silencing complex), where Argonaute (Slicer) protein cleaves the target mRNA, leading to degradation and gene silencing. FISH (Fluorescence In Situ Hybridization) (Option A) is used for detecting and visualizing specific nucleic acid sequences in cells but does not modify genes. Recombinant DNA Technology (Option C) involves creating new DNA combinations but is not as precise as CRISPR-Cas for making specific gene knock-ins or knock-outs. Comparative Genomic Hybridization (CGH) (Option D) is used to detect chromosomal abnormalities and does not involve gene editing .

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 454

↑ *Back to this question in Part A*

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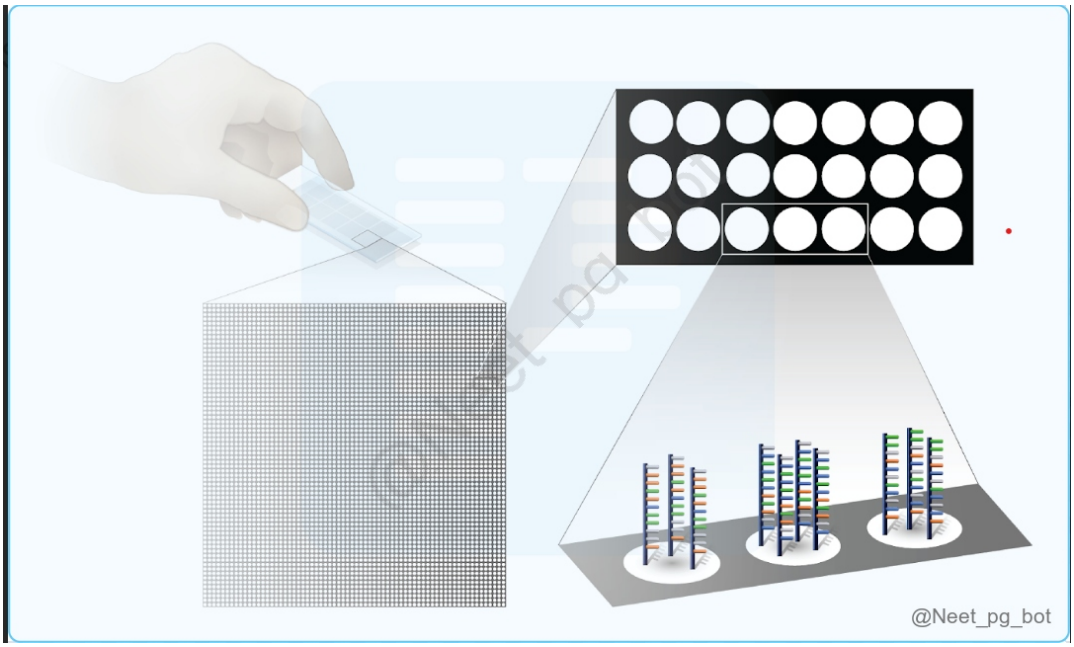
Q14: Which of the following is true about the significance of technology in the following image? {{caption_text}} (Q843422)

A. Used to study multiple gene expressions simultaneously

B. Not used to deciding the course of treatment

C. They have been completely replaced by next-generation sequencing.

D. Not used in proteomics and genotyping.



Correct Answer: A. Used to study multiple gene expressions simultaneously

■ *Listen to Audio 1*

Explanation: The given image represents Microarray Technique which can analyze thousands of genes at once facilitating multiple gene expression studies. Microarray consist of thousands of microscopic DNA spots (probes) on a slide, used for hybridizing cDNA or cRNA samples. Each spot on the microarray chip represents a specific gene, and by measuring the intensity of the signal at each spot, researchers can determine how much of each gene is expressed in the sample. Microarrays are crucial for gene expression profiling and can assist in making treatment decisions by helping us to understand the pathogenesis of a disorder (Option B) Types: DNA Microarray cDNA Microarray Protein Microarray Array comparative genomic hybridisation (CGH) Applications of Microarray Technique: Genotyping Gene sequencing Gene Expression Profiling: Used for cancer treatment decisions (e.g., identifying need for chemotherapy). Protein Detection by monoclonal antibody or aptamer microarrays, useful in personalised medicine and Proteomics (Option D) Clinical Uses: Diagnosing cancer, infectious diseases, and allergies. Microarrays are still being used alongside next-generation sequencing (Option C).

References:

Vasudevan Textbook of Biochemistry, 7th Edition, Page 635

↑ *Back to this question in Part A*

@Neet_pg_bot

Q15: Which of the following vectors is most suitable for cloning large DNA fragments up to 300 kb in size? (Q843421)

A. Plasmids

B. Bacteriophages

C. Bacterial Artificial Chromosomes (BACs)

D. Yeast Artificial Chromosomes (YACs)

Correct Answer: C. Bacterial Artificial Chromosomes (BACs)

■ *Listen to Audio 1*

Explanation: Vector Size Description Plasmids (Option A) Up to 10 kb Plasmids are small, circular DNA molecules commonly used in recombinant DNA technology. They are easy to manipulate and introduce into bacterial cells. However, they typically can only accommodate small to medium-sized DNA inserts. They are not suitable for very large DNA fragments. Bacteriophages (Option B) 10-20 kb Bacteriophages, particularly lambda phage vectors, are used for cloning DNA fragments. While larger than plasmids, they are still not suitable for very large DNA fragments of 300 kb. Bacterial Artificial Chromosomes (BACs) (Option C) 50-250 kb BACs are vectors based on the F-plasmid of E. coli. They can accommodate large DNA inserts and are widely used for constructing genomic libraries. Yeast Artificial Chromosomes (YACs) (Option D) >500 YACs are vectors that can accommodate very large DNA inserts, even larger than BACs. While they can certainly handle 300 kb inserts, they are more prone to chimerism and instability compared to BACs.

References:

Harper’s Biochemistry, 32nd edition, Pg 448

↑ *Back to this question in Part A*

@Neet_pg_bot

Q16: Which type of blotting technique is used to detect specific RNA sequences within a sample? (Q843420)

A. Southern Blotting

B. Northern Blotting

C. Western Blotting

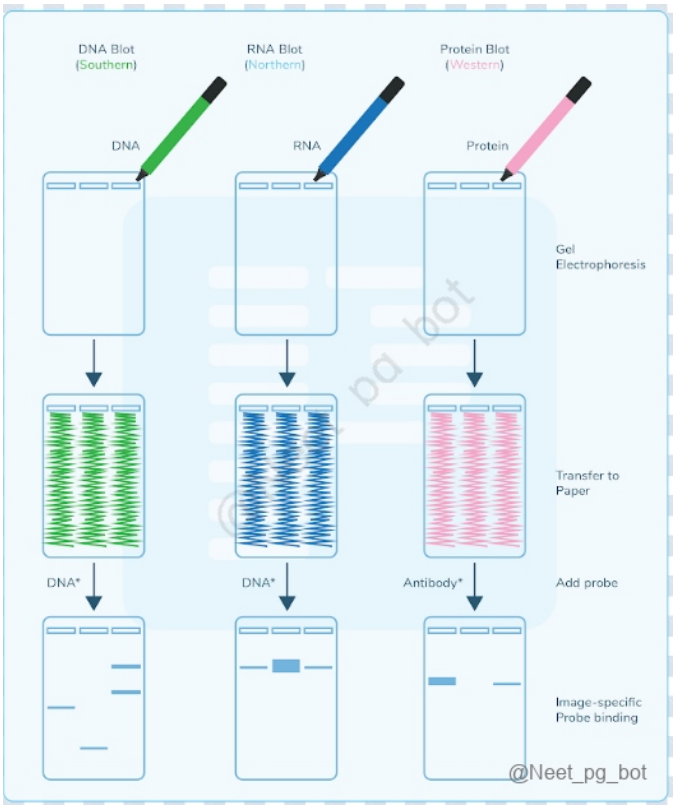
D. Eastern blotting

Correct Answer: B. Northern Blotting

■ *Listen to Audio 1*

Explanation: Types Target Features Southern blotting (Option A) DNA sequences It involves separating DNA fragments by gel electrophoresis, transferring them to a membrane, and then using labelled probes to identify

specific sequences Northern blotting (Option B) RNA sequences The process is similar to DNA blotting but focuses on RNA. Western blotting (Option C) Proteins This technique specifically detects and analyses proteins in a complex mixture. It involves separating proteins by gel electrophoresis, transferring them to a membrane, and then using specific antibodies to detect the proteins. Eastern blotting (Option D) Post-translational modifications It refers to specialised Western blotting techniques that detect specific post-translational modifications of proteins, such as phosphorylation or glycosylation. South-Western blot DNA-binding Proteins Uses a labeled double-stranded DNA probe to detect proteins like transcription factors/steroid receptors.



References:

Harper’s Illustrated Biochemistry, 32nd edition, Pages 450, 451

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: Which type of restriction endonuclease creates staggered cuts in the DNA, producing "sticky ends"? (Q843418)

A. Type I restriction enzymes

B. Type II restriction enzymes

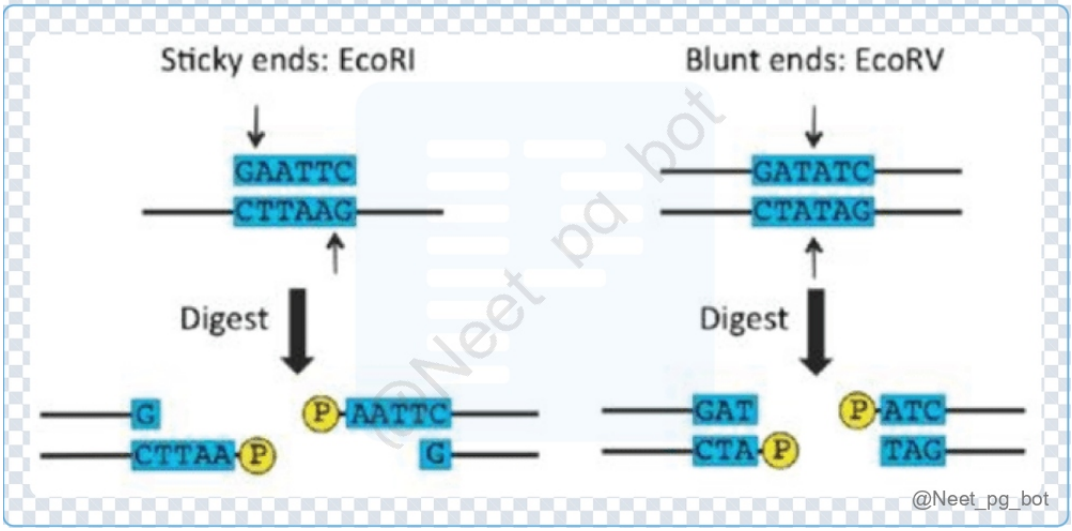
C. Type III restriction enzymes

D. Type IV restriction enzymes

Correct Answer: B. Type II restriction enzymes

Listen to Audio 1

Explanation: Restriction endonucleases are enzymes that cut DNA at specific recognition sequences. Characteristic Type I Type II Subunit structure Complex (3 subunits) Simple (1-2 subunits) ATP requirement Yes No Cleavage site Random, far from recognition site At or near the recognition site Recognition sequence Asymmetric Usually symmetric Type II Restriction Enzyme Subtypes : Sticky End Blunt End Cuts both the DNA strands at two different sites. Cuts both the strands at the same site. Forms two complexes with overhanging ends. Forms two complexes with blunt ends. Type III restriction enzymes(Option C): Recognise specific DNA sequences but cut at a short distance from the recognition site. They do not produce sticky ends and also require ATP. Type IV restriction enzymes(Option D): Target modified DNA, such as methylated DNA, rather than specific sequences. They do not produce sticky ends and are involved in the recognition of modified bases rather than sequence-specific cleavage.



References:

Textbook of Biochemistry by DM Vasudevan, 7th edition, Pg 625

↑ *Back to this question in Part A*

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Q18: Which of the following represents the correct sequence of steps in recombinant DNA technology? (Q843417)

A. Isolation of DNA → Insertion of recombinant DNA into host → Ligation → Cutting of DNA with restriction enzymes → Selection and screening.

B. Cutting of DNA with restriction enzymes → Ligation → Isolation of DNA → Insertion of recombinant DNA into host → Selection and screening.

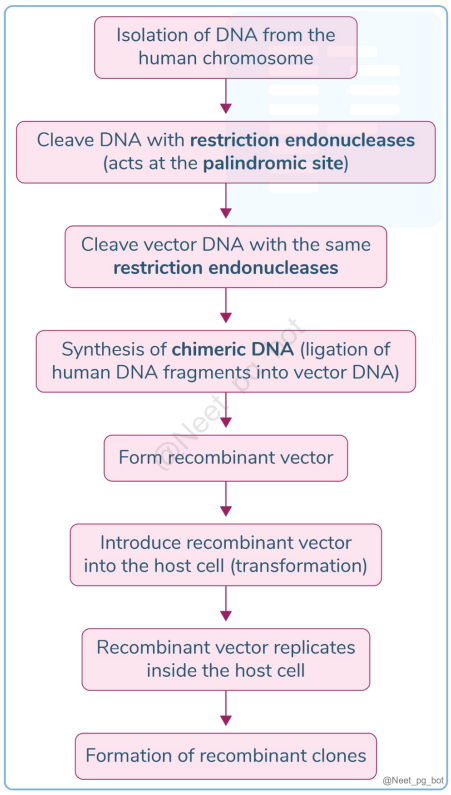
C. Isolation of DNA → Cutting of DNA with restriction enzymes → Ligation → Insertion of recombinant DNA into host → Selection and screening.

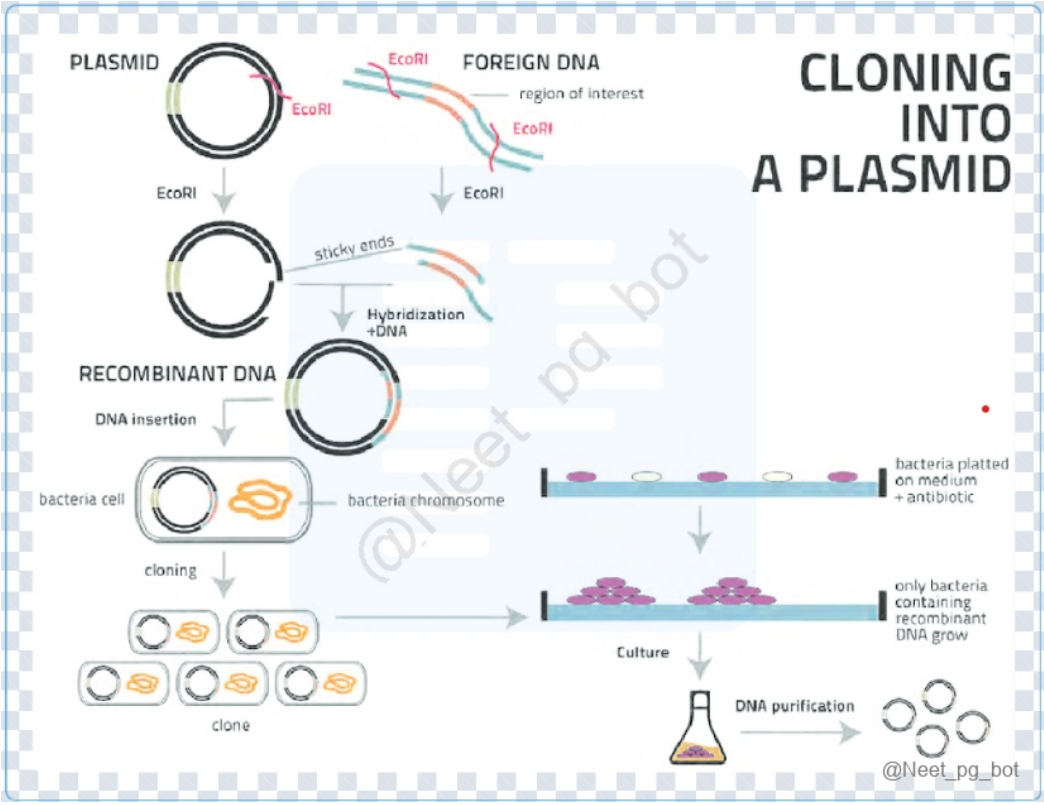
D. Ligation → Isolation of DNA → Cutting of DNA with restriction enzymes → Insertion of recombinant DNA into host → Selection and screening.

Correct Answer: C. Isolation of DNA → Cutting of DNA with restriction enzymes → Ligation → Insertion of recombinant DNA into host → Selection and screening.

■ *Listen to Audio 1*

Explanation: Steps of recombinant DNA Technology





References:

Harper's Biochemistry, 32nd Edition, Page 444-448

↑ *Back to this question in Part A*

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• [07_ Genetics] 025 Previous Year Questions

« Back to Folder Directory

[07_ Genetics] 025 Previous Year Questions — Quick Jump

Q1 (p. 1105)	*Q2 (p. 1106)*	*Q3 (p. 1107)*	*Q4 (p. 1107)*	*Q5 (p. 1108)*	*Q6 (p. 1109)*
Q7 (p. 1110)	*Q8 (p. 1110)*	*Q9 (p. 1111)*	*Q10 (p. 1112)*	*Q11 (p. 1113)*	*Q12 (p. 1114)*
Q13 (p. 1117)	*Q14 (p. 1118)*	*Q15 (p. 1118)*	*Q16 (p. 1119)*	*Q17 (p. 1120)*	*Q18 (p. 1121)*
Q19 (p. 1121)	*Q20 (p. 1122)*	*Q21 (p. 1123)*	*Q22 (p. 1124)*	*Q23 (p. 1124)*	*Q24 (p. 1125)*
Q25 (p. 1126)	*Q26 (p. 1127)*	*Q27 (p. 1128)*	*Q28 (p. 1129)*	*Q29 (p. 1130)*	*Q30 (p. 1131)*
Q31 (p. 1132)	*Q32 (p. 1132)*	*Q33 (p. 1133)*	*Q34 (p. 1134)*	*Q35 (p. 1135)*	*Q36 (p. 1135)*
Q37 (p. 1136)	*Q38 (p. 1137)*	*Q39 (p. 1139)*	*Q40 (p. 1139)*	*Q41 (p. 1140)*	*Q42 (p. 1141)*
Q43 (p. 1142)	*Q44 (p. 1144)*	*Q45 (p. 1144)*	*Q46 (p. 1146)*	*Q47 (p. 1147)*	*Q48 (p. 1147)*
Q49 (p. 1148)	*Q50 (p. 1149)*	*Q51 (p. 1149)*	*Q52 (p. 1150)*	*Q53 (p. 1151)*	*Q54 (p. 1151)*
Q55 (p. 1153)	*Q56 (p. 1153)*	*Q57 (p. 1154)*	*Q58 (p. 1155)*	*Q59 (p. 1156)*	*Q60 (p. 1156)*
Q61 (p. 1157)	*Q62 (p. 1158)*	*Q63 (p. 1159)*	*Q64 (p. 1160)*		

Q1: Ascent to high altitude causes ? (Q865438)

A. Metabolic acidosis

B. Metabolic alkalosis

C. Respiratory acidosis

D. Respiratory alkalosis

Correct Answer: D. Respiratory alkalosis

Explanation: At high altitudes, hypoxia triggers hyperventilation, leading to enhanced CO■ removal and results in respiratory alkalosis. Changes during Acclimatization Respiratory At high altitudes, lower oxygen levels stimulate peripheral chemoreceptors, increasing pulmonary ventilation. This leads to enhanced CO2 removal, causing respiratory alkalosis. Kidneys respond by excreting more bicarbonate to stabilize blood pH, causing a sustained increase in ventilation and supporting prolonged acclimatization. Haematological At high altitudes, lower PO2 boosts erythropoietin production, increasing red blood cell count. This also raises 2,3-diphosphoglycerate (2,3-DPG) levels in red blood cells. Initially, alkalosis shifts the oxyhemoglobin dissociation curve to the left but increased 2,3-DPG shifts it to the right, improving oxygen release to tissues. Cardiovascular Hypoxia activates the sympathoadrenal axis, increasing heart rate, cardiac output, and blood pressure. Increased cardiac output leads to an increase in blood flow, and hypoxia causes vasodilation in the systemic circulation, further contributing to increased blood flow and oxygen supply to the tissues. Tissue In acclimatization, skeletal muscles undergo several changes, including an increase in the number of capillaries and mitochondria and an increase in myoglobin

content. Further, chemicals released from the hypoxic tissues stimulate angiogenesis. Metabolic acidosis (Option A) involves decreased bicarbonate levels due to conditions like diarrhea or renal failure, not high altitude. Metabolic alkalosis (Option B) can occur later as a compensatory renal response to respiratory alkalosis but is not the initial effect. Respiratory acidosis (Option C) results from hypoventilation and CO■ retention .

References:

Guyton and Hall Textbook of Medical Physiology, 14th Edition, Page 554,555

Ganong's Review of Medical Physiology, 26th Edition, Page 637,638

GK Pal Comprehensive Textbook of Medical Physiology, Page. 948

↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: A patient who has been diagnosed with multiple gastrointestinal polyps and carcinoma, with a positive family history/history of hereditary non-polyposis colon carcinoma has a defect in which of the following? (Q855998)

A. Mismatch repair

B. Nucleotide excision repair

C. Base excision repair

D. Point mutation

Correct Answer: A. Mismatch repair

Explanation: Mismatch repair defects are primarily associated with HNPCC (Lynch syndrome), leading to an increased risk of colorectal and other cancers. Hereditary Nonpolyposis Colon Cancer (HNPCC), also known as Lynch syndrome: Characterized by microsatellite instability. The most common gene mutation associated with Lynch syndrome is in the human MutS homolog 2 (hMSH2) gene. Defects in mismatch repair genes, particularly hMSH1 and hMSH2, lead to ineffective repair of DNA replication errors. These gene defects are located on chromosome 2. The inability to correct mismatches during DNA replication increases the risk of mutations, contributing to the development of colon cancer. Incorrect Options: Nucleotide excision repair (Option B) associated with xeroderma pigmentosum (XP),Cockayne syndrome (CS) and Trichothiodystrophy (TTD). Base excision repair (Option C) associated with genetic disorders like some forms of inherited breast and ovarian cancer, Alzheimers and Parkinson disease. Point mutation (Option D) While they can contribute to cancer, hereditary non-polyposis colon carcinoma is related to mismatch repair pathway dysfunction, not general point mutations.

References:

Vasudevan 7th edition, pg 585,583

Lippincott 7th edition, 1217,1219

Harper Biochemistry Pg 715

Hereditary cancer syndromes

↑ *Back to this question in Part A*

@Neet_pg_bot

Q3: DNA methylation is not related to which of the following processes? (Q856919)

A. Mismatch repair

B. Chromatin remodeling

C. Gene silencing

D. DNA replication

Correct Answer: D. DNA replication

Explanation: DNA methylation involves the addition of a methyl group (–CH■) to cytosine residues (typically at CpG islands) and plays key epigenetic and regulatory roles . It affects gene expression , chromatin structure , and DNA repair — but it is not directly involved in the mechanical process of DNA replication . Process Role of DNA methylation Mismatch repair Helps differentiate parental (methylated) and newly synthesized (unmethylated) DNA strands in prokaryotes . Chromatin remodeling Methylation promotes heterochromatin formation , leading to tighter chromatin and reduced transcription. Gene silencing Methylation at CpG islands in promoter regions inhibits transcription factor binding → gene silencing . DNA replication Methylation does not directly regulate or participate in DNA polymerase activity or replication fork progression.

References:

DNA methylation https://pmc.ncbi.nlm.nih.gov/articles/PMC133775/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q4: Klenow fragment lacks the activity of? (Q857029)

A. 3’→5’ Exonuclease

B. 5’→3’ Exonuclease

C. 5’→3’ DNA polymerase

D. 3’→5’ DNA polymerase

Correct Answer: B. 5’→3’ Exonuclease

Explanation: Klenow fragment is produced by cleaving DNA polymerase I and retains 5’→3’ polymerase and 3’→5’ exonuclease (proofreading) activities. It lacks the 5’→3’ exonuclease function, which is normally used for primer removal. Klenow fragment: Origin Derived from DNA Polymerase I (Kornberg's enzyme) after removal of the 5′ to 3′ exonuclease domain Functions Retained Polymerization and 3′ to 5′ exonuclease (proofreading) activities Exonuclease Removed 5′ to 3′ exonuclease activity Applications Widely used in recombinant DNA technology Source Isolated from Escherichia coli

References:

Vasudevan’s Textbook of Biochemistry, 7th Edition, Page 580

↑ *Back to this question in Part A*

@Neet_pg_bot

Q5: DNA methylation is not related to which of the following processes? (Q856487)

A. Mismatch repair

B. Chromatin remodeling

C. DNA replication

D. Gene silencing

Correct Answer: C. DNA replication

Explanation: DNA methylation involves the addition of a methyl group (–CH■) to cytosine residues (typically at CpG islands) and plays key epigenetic and regulatory roles . It affects gene expression , chromatin structure , and DNA repair — but it is not directly involved in the mechanical process of DNA replication . Process Role of DNA methylation Mismatch repair Helps differentiate parental (methylated) and newly synthesized (unmethylated) DNA strands in prokaryotes . Chromatin remodeling Methylation promotes heterochromatin formation , leading to tighter chromatin and reduced transcription. Gene silencing Methylation at CpG islands in promoter regions inhibits transcription factor binding → gene silencing . DNA replication Methylation does not directly regulate or participate in DNA polymerase activity or replication fork progression.

References:

DNA methylation

↑ *Back to this question in Part A*

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Q6: The correct order of steps in polymerase chain reaction (PCR) is:- (Q856494)

A. Hybridization, annealing, elongation, denaturation

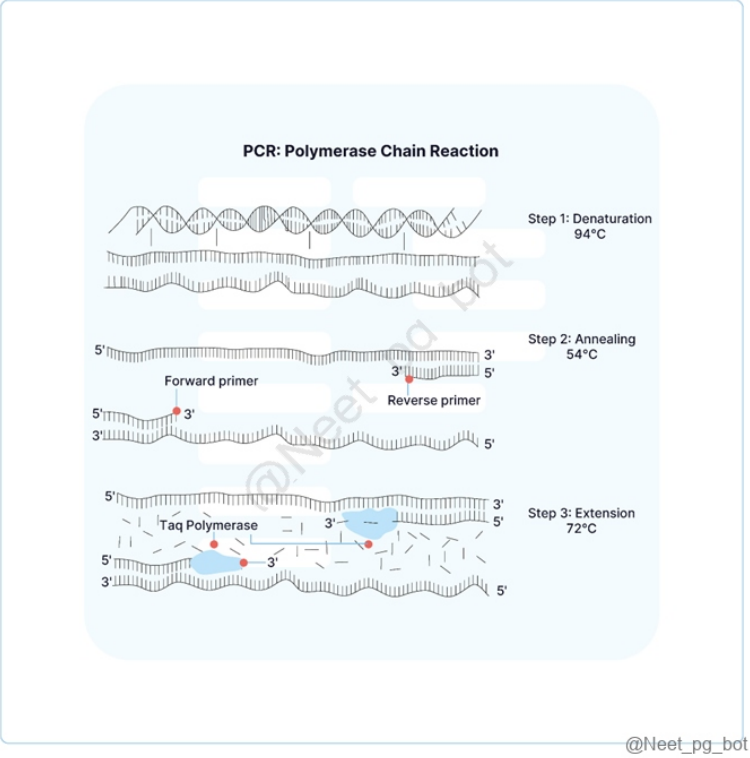
B. Elongation, denaturation, Hybridization, annealing,

C. Denaturation, annealing, extension.

D. Annealing, extension, denaturation

Correct Answer: C. Denaturation, annealing, extension.

Explanation: Steps of PCR: Step 1: Denaturation unwinding the two strands of double-stranded DNA to form 2 single strands. It is carried out at 94/95 degrees (at this temperature, the hydrogen bonds will be broken). Step 2: Annealing Adding primer which will anneal to the flanking sequences of the desired fragments (the temperature should neither be too high nor too low- it should be optimal) It should be 5 degrees < melting temperature of the primers. Step 3: Elongation It should be done by the DNA polymerase, which is thermostable. The enzyme that is used in elongation is Taq. (Thermus aquaticus) DNA polymerase.



References:

PCR

↑ *Back to this question in Part A*

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Q7: Which type of DNA repair mechanism involves the removal and replacement of a damaged DNA strand with the help of an undamaged complementary strand as a template? (Q856514)

A. Base Excision Repair

B. Nucleotide Excision Repair

C. Mismatch Repair

D. Homologous Recombination

Correct Answer: D. Homologous Recombination

Explanation: : Removes and replaces damaged bases by excising a single base and filling the gap using the original strand as a template. Nucleotide Excision Repair (Option B): Removes bulky lesions (e.g., thymine dimers) by excising a short DNA segment and filling the gap using the original strand as a template. Mismatch Repair (Option C): Fixes base mismatches that occur during replication using the parental strand as a template, not homologous DNA.

References:

Homologous recombination

↑ *Back to this question in Part A*

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Q8: If the percentage of thymine residues in DNA is 28%. What is the % of cytosine? (Q858457)

A. 28%

B. 44%

C. 36%

D. 22%

Correct Answer: D. 22%

Explanation: Chargaff's rule , which states that in DNA, the amount of adenine (A) is equal to the amount of thymine (T), and the amount of guanine (G) is equal to the amount of cytosine (C). DNA consists of four deoxyribonucleotides: deoxyadenylate (A), deoxyguanylate (G), deoxycytidylate (C), and thymidylate (T). The two strands of DNA are complementary, meaning adenine pairs with thymine, and guanine pairs with cytosine. Since the total percentage of nucleotides should add up to 100%, and 28% + 28% is already accounted for by thymine and adenine, the remaining percentage for guanine and cytosine is 44%. Since guanine and cytosine are present in equal amounts, the percentage of cytosine would be half of the remaining percentage, which is 22%.

References:

https://pmc.ncbi.nlm.nih.gov/articles/PMC7986665/

↑ *Back to this question in Part A*

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Q9: DNA packing is done by which of the following? (Q855513)

A. Histone

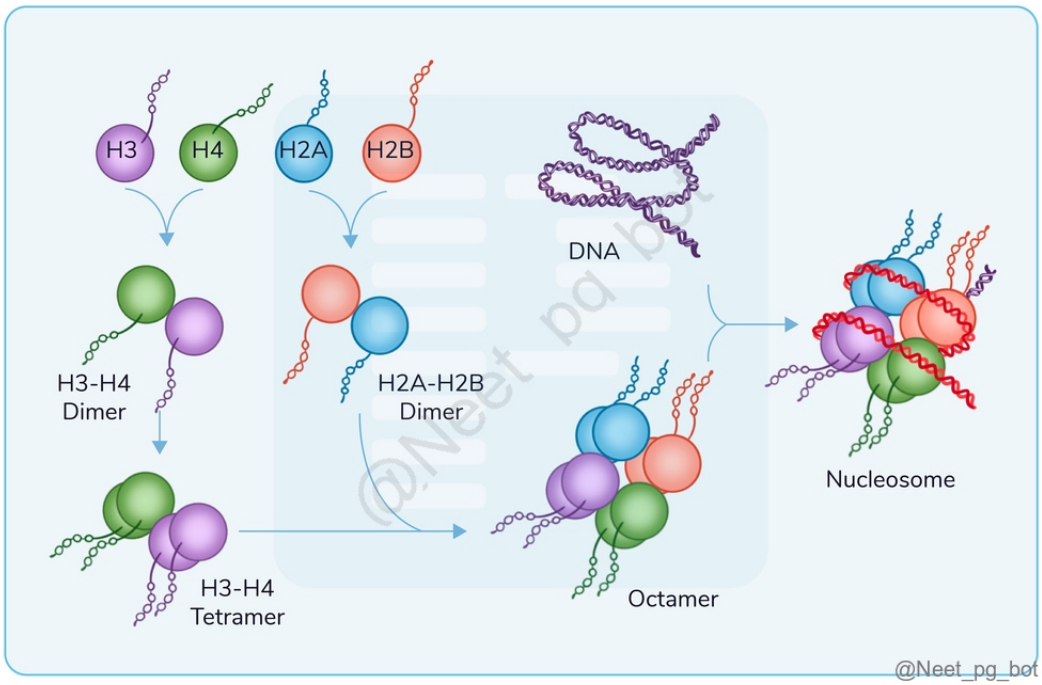
B. Glycoprotein

C. Nucleus acid

D. Adenine

Correct Answer: A. Histone

Explanation: DNA packing is done by histone Histones: Histones are a small family of closely related basic proteins Essential for the organization and function of chromatin. They play a critical role in DNA packaging within the nucleus, forming the structural unit of chromatin known as the nucleosome. Histones are also involved in regulating gene expression through various posttranslational modifications (PTMs) eg. Acetylation , Methylation, Phosphorylation, ADP-Ribosylation , Monoubiquitylation, and Sumoylation. Rich in lysine and arginine. Types Histones can be categorized into core histones and linker histones: Core Histones: H2A, H2B, H3, and H4 Linker Histone: H1 These histones are highly conserved across species, indicating their essential role in cellular processes. The core histones form the nucleosome by creating a histone octamer around which DNA is wrapped. The carboxyl-terminal two-thirds of the histone molecules are hydrophobic, while the amino-terminal thirds are rich in basic amino acids. H1 is the least tightly bound to chromatin and can be easily removed with a salt solution, making chromatin more soluble. H1 interacts with DNA as it enters and exits the nucleosome, playing a role in the higher-order structure of chromatin. Histone Octamer The histone octamer is the fundamental unit of the nucleosome and consists of eight histone molecules: Two molecules each of H2A, H2B, H3, and H4. These histones interact specifically to form a structure with the composition (H3–H4)■–(H2A–H2B)■. H3 and H4 form a tetramer (H3–H4)■, and H2A and H2B form dimers (H2A–H2B). This octamer binds approximately 145-150 base pairs of DNA, creating 1.75 superhelical turns around it. nucleosome assembly" data-author="" data-hash="11565" data-license="" data-source="" data-tags="May2025" src="" />



References:

Harper 32nd edition. Pg. 361, 363

↑ *Back to this question in Part A*

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Q10: Which of the subsequent disorders, exhibit only autosomal recessive inheritance pattern? (Q855527)

A. Huntington’s disease

B. Treacher collins syndrome

C. Cystic fibrosis

D. Achondroplasia

Correct Answer: C. Cystic fibrosis

Explanation: Cystic fibrosis is autosomal recessive mode of inheritance. Mutation in CFTR gene that causes formation of thick mucus that blocks airway. CFTR gene encodes protein that regulates transfer of salt and water into and out of cells. Autosomal Dominant Autosomal Recessive Huntington’s disease (Option A) Marfan syndrome Achondroplasia (Option D) Familial hypercholesterolemia Neurofibromatosis type 1 Polycystic kidney disease (ADPKD) Cystic fibrosis (Option C) Sickle cell anemia Tay-Sachs disease Phenylketonuria (PKU)

Thalassemia Albinism,Wilson’s disease Treacher collins syndrome: Have autosomal dominant, autosomal recessive or sporadic inheritance patterns due to mutation in various genes affecting facial bone and tissue development.

References:

Inheritance

↑ *Back to this question in Part A*

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Q11: What is the term for a single mutation in a nucleotide base pair that leads to formation of termination codon? (Q855533)

A. Missense mutation

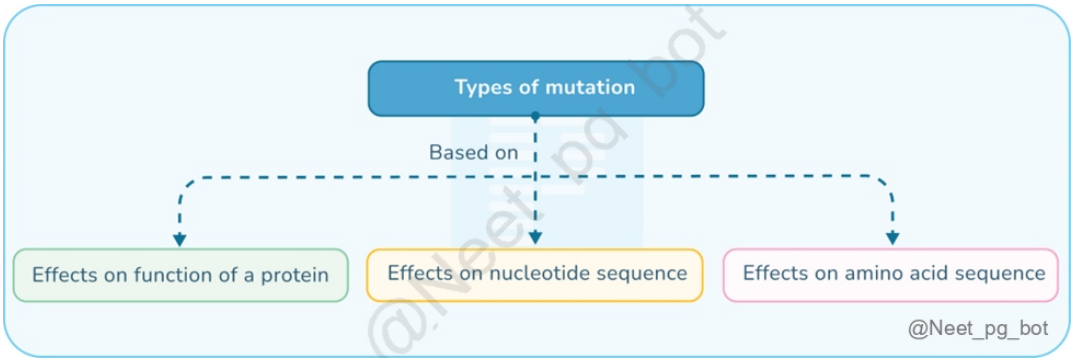
B. Nonsense mutation

C. Stop mutation

D. Silent mutation

Correct Answer: B. Nonsense mutation

Explanation: Nonsense mutation leading to premature termination of the protein, and destroying the functional activity. Based on the Effects on Amino acid sequence Silent Mutation (Option D) Missense Mutation (Option A) Nonsense Mutation (Option B) Mutation may change the codon for one amino acid to a synonym for the same amino acid. It has no effect on the phenotype. Degeneracy of the code. Different amino acid is incorporated due to change in the nucleotide sequence. It can be acceptable, partially acceptable, and unacceptable. Codon for an amino acid is mutated to a termination codon (UAA or UGA or UAG) causing premature termination of translation. Stop mutation (Option C): Refers to premature termination of protein synthesis, but rarely used in genetics.



References:

Harper 32e, pg 407, 408

↑ *Back to this question in Part A*

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Q12: A 5-year-old child is brought to the clinic with abdominal pain, irritability, and developmental delay. On examination, the child has pallor and a bluish line on the gums. Laboratory tests reveal microcytic anemia, and a peripheral blood smear shows red blood cells with basophilic stippling. Which of the following is the most likely diagnosis? (Q866662)

A. Iron deficiency anemia

B. Sideroblastic anemia

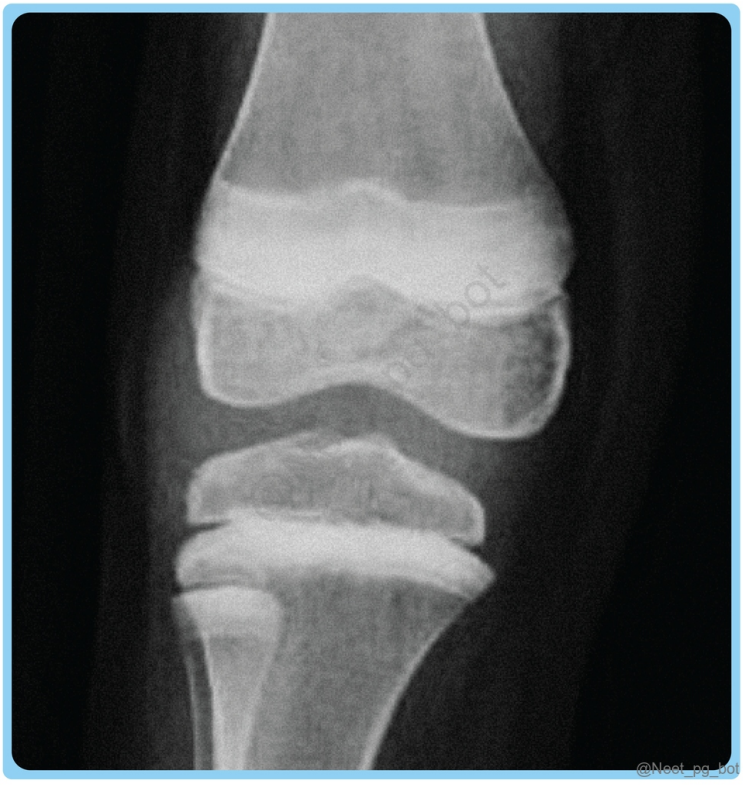
C. Lead poisoning

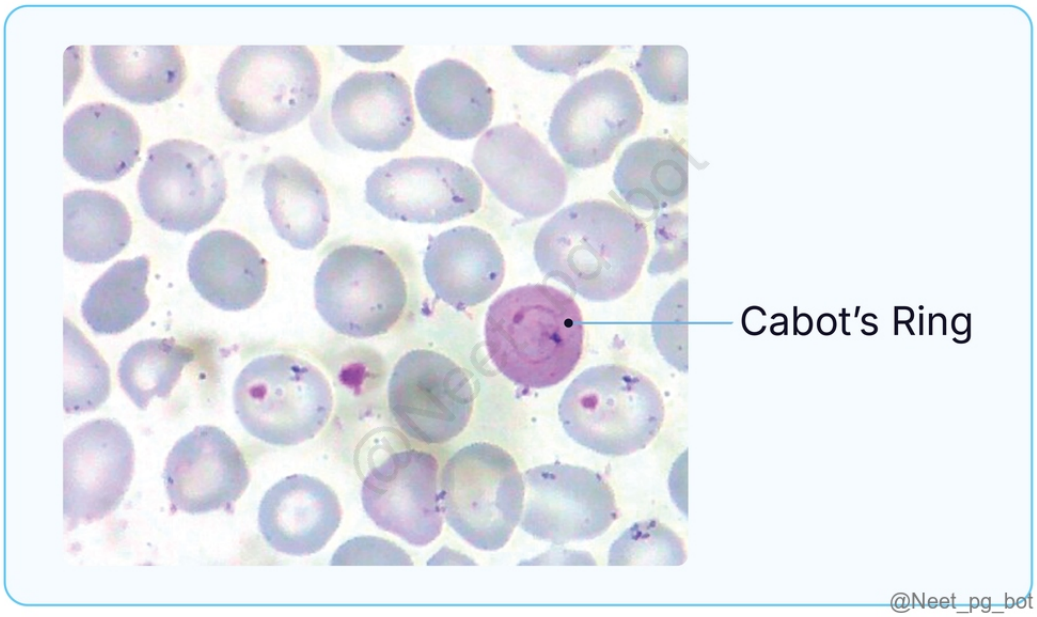
D. Thalassemia minor

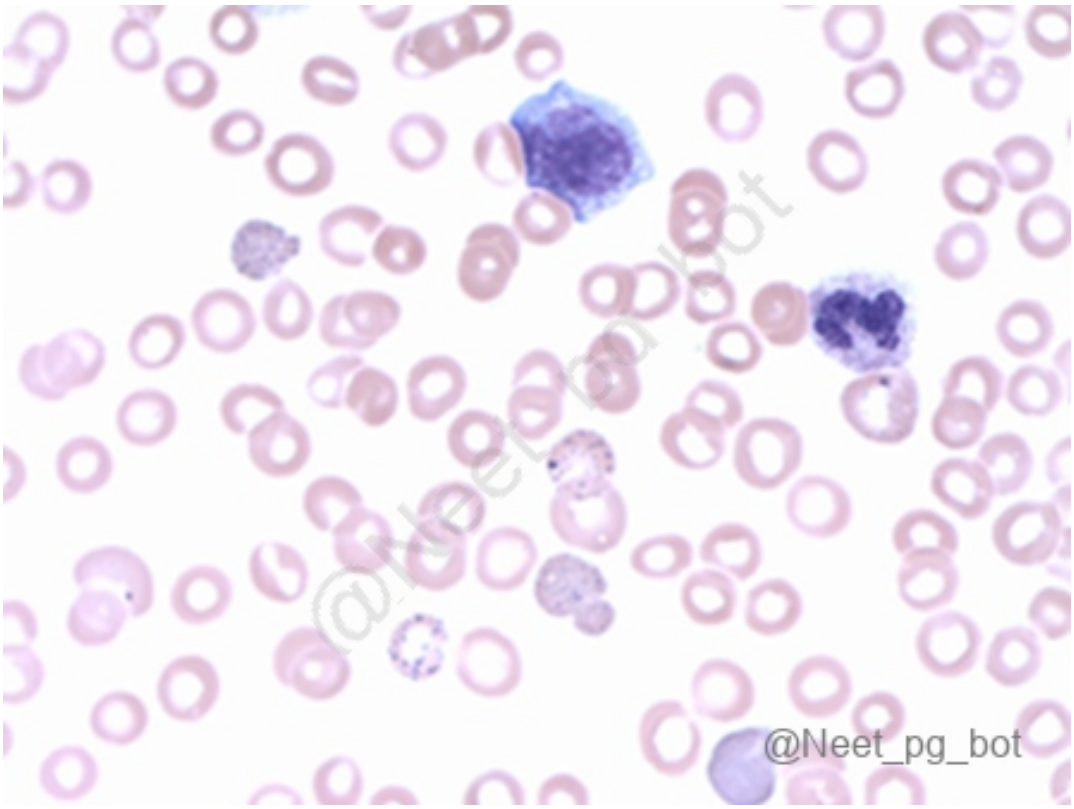
Correct Answer: C. Lead poisoning

Explanation: Classic signs of Lead Poisoning include abdominal pain, irritability, developmental delay, pallor, and a bluish line on the gums (Burtonian’s or Burton’s line), along with laboratory findings of microcytic anemia and basophilic stippling on the peripheral smear. Source Exposure to lead from paint, contaminated soil, or old pipes. Enzymes inhibited Ferrochelatase ALA-dehydratase Coproporphyrinogen oxidase). Clinical features Facial pallor: Circumoral (vasospasm) → Earliest and most specific feature Lead line or Burtonian lines: Stippled blue line observed on the gums, resulting from a subepithelial deposit of granules. Lead osteopathy/Lead line (Bone-line): Calcium deposition occurs at the growth plates of bones (specifically the lower end of the femur and the upper end of the tibia). Lead palsy: Predominantly motor features → (dry belly ache, wrist drop - radial nerve), foot drop - common peroneal nerve). Blood picture: Microcytic-hypochromic anemia, Cabot rings in RBCs, Sideroblasts (nucleated red cells), Anisocytosis, Poikilocytosis, Polycythemia Basophilic stippling or Punctate basophilia Colic and constipation (Painter's Colic or Devonshire colic) → Spasmodic pain relieved by pressure (at night). Saturnine gout (due to decreased excretion of uric acid) Encephalopathy Reproductive system (amenorrhea, dysmenorrhea, abortion, sterility derangments) CVS and Kidney (Hypertension, arteriolar degeneration, nephritis). Treatment Remove lead exposure and chelation therapy (e.g., EDTA, dimercaprol). Iron deficiency anemia (Option A) does not present with basophilic stippling or Burton’s line. Sideroblastic anemia (Option B) shows ring sideroblasts in bone marrow but not Burton’s line or neurological symptoms. ■■■■■■■Thalassemia minor (Option D) has microcytic anemia but lacks lead poisoning features.


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References:

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↑ *Back to this question in Part A*

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Q13: In a village of Assam, a man is observed walking with the help of a stick and has genu valgum. Many people in the same village present with similar symptoms. What is the most probable cause of this condition? (Q866664)

A. Calcium deficiency

B. Vitamin C deficiency

C. Skeletal fluorosis

D. Vitamin D deficiency (Osteomalacia)



Correct Answer: C. Skeletal fluorosis

Explanation: The presence of genu valgum and multiple affected individuals in an Assam village suggests Skeletal Fluorosis , a condition common in areas with high fluoride levels in drinking water, leading to bone pain, stiffness, joint deformities, and walking difficulty. Cause: Chronic ingestion of high levels of fluoride, usually through drinking water. Endemic Areas: Common in parts of India, especially Assam, Andhra Pradesh, and Rajasthan. Symptoms: Bone pain and joint stiffness Genu valgum (knock knees), difficulty walking May progress to crippling deformities X-ray findings: I ncreased bone density (osteosclerosis) Prevention: Use of fluoride-free water, defluoridation techniques Treatment: Primarily supportive; address deformities and improve nutrition. Fluorine content in water Related symptoms <0.5mg/L Dental caries >1.5mg/L Dental fluorosis 3-6mg/L Skeletal fluorosis >10mg/L Crippling fluorosis Calcium deficiency (Option A) and Vitamin D deficiency (Osteomalacia)

(Option D) cause bone pain and weakness, but are less likely to occur in clusters. Vitamin C deficiency (scurvy) (Option B) affects collagen synthesis and presents with bleeding gums, petechiae, not typically genu valgum.

References:

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↑ *Back to this question in Part A*

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Q14: Which of the following substances, at the given concentration, makes water unsuitable for human consumption according to safety standards? (Q866677)

A. Fluoride – 0.8 mg/L

B. Cadmium – 0.3 mg/L

C. Chloride - 200 mg/L

D. Calcium – 7 mg/L

Correct Answer: B. Cadmium – 0.3 mg/L

Explanation: The unsafe substance here is cadmium at 0.3 mg/L , because it exceeds permissible limits for drinking water. WHO & BIS (Bureau of Indian Standards) limits: Fluoride : desirable ~1.0 mg/L (max 1.5 mg/L) → 0.8 mg/L is safe. Cadmium : safe limit is 0.003 mg/L (3 µg/L) → 0.3 mg/L is 100× higher , toxic. Chloride : permissible up to 250 mg/L → 200 mg/L is safe. Calcium : desirable up to 75 mg/L (max 200 mg/L) → 7 mg/L is safe. Toxicity of cadmium in water: Chronic exposure → kidney damage (proximal tubular dysfunction), bone demineralisation (Itai-Itai disease in Japan), carcinogenic effects. Often from industrial waste, batteries, and electroplating runoff.

↑ *Back to this question in Part A*

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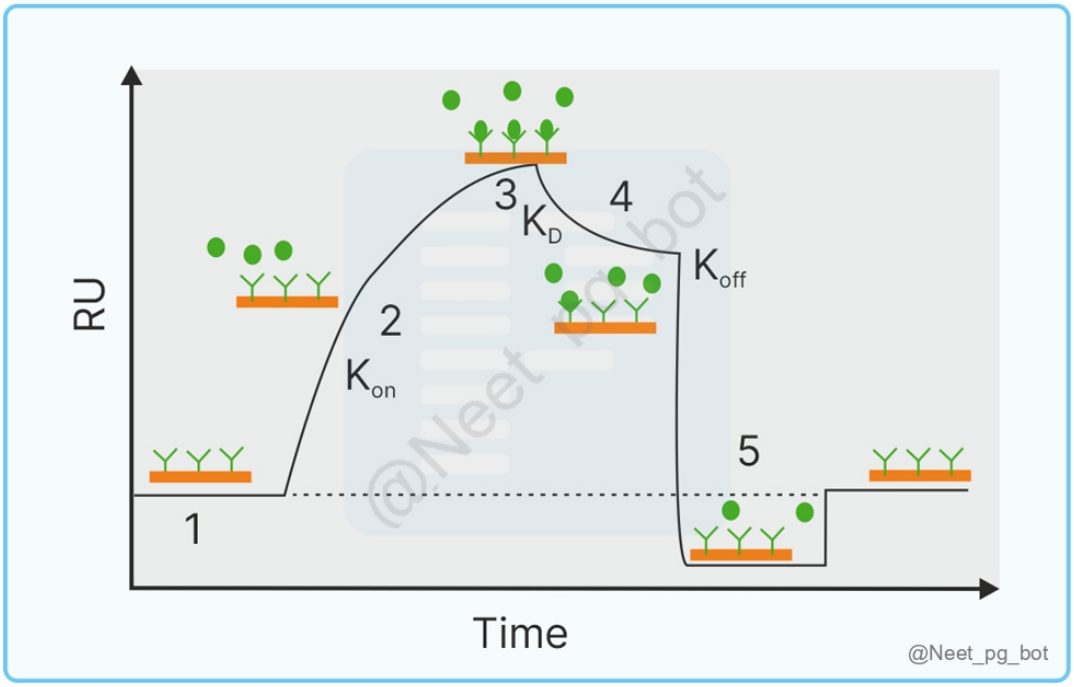
Q15: Name the graph and mention its application. (Q865396)

A. ELISA, Antibody concentration

B. ELISA, Antibody affinity

C. Plasma resonance, Ab concentration

D. Plasma resonance, Ab affinity



Correct Answer: D. Plasma resonance, Ab affinity

Explanation: The given graph is Plasma resonance or Surface Plasmon Resonance (SPR) , which is used to determine the determine the strength (affinity) and speed (kinetics) of antibody-antigen interactions. ELISA (Option A, B) for antibody concentration typically produces a standard curve with a sigmoidal shape, plotting absorbance against antibody concentration. This method uses colorimetric detection to quantify the amount of antibody or antigen present in a sample, making it a reliable and widely used tool in diagnostics and research

References:

Full article: Determining the affinities of high-affinity antibodies using KinExA and surface plasmon resonance

↑ *Back to this question in Part A*

@Neet_pg_bot

Q16: A child presents with hepatomegaly and a cherry-red spot on the macula. Neurological regression is also noted. These findings are suggestive of a lysosomal storage disorder. Which of the following enzyme deficiencies is most likely responsible? (Q865412)

A. Arylsulfatase A

B. Hexosaminidase A

C. Sphingomyelinase

D. Galactocerebrosidase

Correct Answer: C. Sphingomyelinase

Explanation: A child presenting with hepatomegaly, a cherry-red spot on the macula, and neurological regression is indicative of sphingomyelinase deficiency, the enzyme deficiency responsible for Niemann-Pick Disease. Disease Enzyme Deficiency Lipid Accumulating Clinical Symptoms Tay-Sachs disease (Option B ruled out) Hexosaminidase A, S Cer—Glc—Gal(NeuAc) GalNAc GM2 Ganglioside Mental retardation, blindness, muscular weakness Fabry disease α-Galactosidase Cer—Glc—Gal—Gal Globotriaosylceramide Skin rash, kidney failure (symptoms manifest fully only in males; X-linked recessive) Metachromatic leukodystrophy (Option A ruled out) Arylsulfatase A Cer—Gal—OSO3 3-Sulfogalactosylceramide Mental retardation and psychological disturbances in adults; demyelination Krabbe disease (Option D ruled out) Galactocerebrosidase Cer—Gal Galactosylceramide Mental retardation; myelin almost absent Gaucher disease β-Glucosidase Cer—Glc Glucosylceramide Enlarged liver and spleen, erosion of long bones, mental retardation in infants Niemann-Pick disease types A, B (Option C) Sphingomyelinase Cer—P—choline Sphingomyelin Enlarged liver and spleen, mental retardation; fatal in early life Farber disease Ceramidase Acyl—Sphingosine Ceramide Hoarseness, dermatitis, skeletal deformation, mental retardation; fatal in early life Abbreviations: Cer, ceramide; Gal, galactose; Glc, glucose; NeuAc, N-acetylne

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 245.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: Wobble hypothesis explains which of the following concepts of the genetic code? (Q856607)

A. Universal

B. Unambiguous

C. Degeneracy

D. Non-punctuated

Correct Answer: C. Degeneracy

Explanation: The wobble phenomenon is due to inosine's ability to form non-standard base pairs (with U, C, and A) at the third position of the codon, allowing a single tRNA to recognize multiple codons . Wobble Phenomenon Definition : Flexible/non-standard base-pairing between the third nucleotide of a codon (mRNA) and the corresponding nucleotide of the tRNA anticodon. Function : Allows one tRNA molecule to bind to multiple codons coding for the same amino acid (Degenerate). Inosine (I): A modified nucleotide in some tRNA anticodons, pairs with U, C, and A. Non-standard pairing is more flexible than Watson-Crick base pairing, which

is strict for the first two codon nucleotides. Codon Position Standard Base Pairing Wobble Pairing Example 1st and 2nd Watson-Crick rules Strict AG A and AG G code for Arginine. 3rd Non-standard pairing Flexible AGA/AGG with UCU, GGU/GGC/GGA with CCI

References:

Harper 32e, Pg 407

↑ *Back to this question in Part A*

@Neet_pg_bot

Q18: Cyanide and carbon monoxide act on which of the heme proteins: (Q865454)

A. Cytochrome c oxidase

B. Myoglobin

C. Catalase

D. NADH dehydrogenase

Correct Answer: A. Cytochrome c oxidase

Explanation: Cyanide (CN■) and carbon monoxide (CO) are potent inhibitors of cytochrome c oxidase (Complex IV of the mitochondrial electron transport chain). Cyanide: Binds irreversibly to the heme iron (Fe^{3}■) in cytochrome c oxidase, blocking electron transfer to oxygen. Carbon monoxide: Binds competitively to the heme iron (Fe^{2}■) in cytochrome c oxidase, displacing oxygen (O■). Myoglobin (Option B) CO binds to myoglobin (causing cherry-red tissues), but this is not the primary lethal mechanism. Catalase (Option C) Not a target of cyanide/CO toxicity (catalase breaks down H■O■, not involved in O■ utilization). NADH dehydrogenase (Complex I) (Option D) Inhibited by rotenone, not cyanide/CO.

References:

Harper’s Illustrated Biochemistry, 31st Edition, Page 123.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q19: If you need to diagnose a syndromic illness such as meningitis, which of the following diagnostic techniques is most appropriate? (Q866328)

A. Multiplex PCR

B. Uniplex PCR

C. Attributed PCR

D. Nested PCR

Correct Answer: A. Multiplex PCR

Explanation: When diagnosing a syndromic illness such as meningitis, which may be caused by various pathogens including bacteria, viruses, and fungi, Multiplex PCR is used because it can simultaneously detect multiple causative agents. Amplifies multiple DNA targets in one reaction using several primer sets. Saves time and reduces costs by testing multiple pathogens simultaneously. Commonly used in clinical diagnostics and genetic testing. Requires careful primer design to avoid interference. Offers high sensitivity and specificity when well optimized. Ideal for syndromic testing where multiple pathogens may be involved. Uniplex PCR (Option B) detects only one target per reaction, making it less efficient for syndromic testing. Attributed PCR (Option C) is not a commonly used term in molecular diagnostics. Nested PCR (Option D) is a two-step PCR designed to increase sensitivity and specificity but usually targets a single pathogen per test.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q20: A patient presents with a history of vomiting. Arterial blood gas analysis reveals the following: pH : 7.5 pCO■ : 48 mm Hg HCO■■ : 30 mm Hg What is the most likely acid-base abnormality? (Q852561)

A. Metabolic alkalosis

B. Metabolic acidosis

C. Respiratory acidosis

D. Respiratory alkalosis

Correct Answer: A. Metabolic alkalosis

Explanation: Parameter Value Normal Range Interpretation pH 7.5 7.35–7.45 ↑ → Alkalosis HCO■■ 30 mEq/L 22–26 mEq/L ↑ → Metabolic cause pCO■ 48 mmHg 35–45 mmHg ↑ → Respiratory compensation (retaining CO■) Since both pH and HCO■■ are elevated, the primary disturbance is metabolic alkalosis. The rise in pCO■ indicates compensatory hypoventilation by the lungs to retain CO■ (acid) and partially correct pH. These findings suggest a primary metabolic alkalosis with respiratory compensation . Common Causes of Metabolic Alkalosis: Vomiting or gastric suction → loss of H■ and Cl■ from stomach acid Diuretic use (loop or thiazide) → increased H■ and K■ loss Hyperaldosteronism → promotes H■ secretion Excess bicarbonate administration Mechanism in This Case: Vomiting leads to loss of H■ (as HCl) from the stomach. The body compensates by increasing bicarbonate (HCO■■) in plasma. Respiratory system responds by hypoventilation , raising pCO■ . Metabolic Acidosis (Option B) is incorrect because metabolic acidosis would present with a low pH (<7.35) and a low

HCO■■ , which is not the case here. Respiratory Acidosis (Option C) is incorrect because respiratory acidosis is characterized by low pH and high pCO■ , but in this case, the pH is elevated. Respiratory Alkalosis (Option D) is incorrect because respiratory alkalosis involves low pCO■ due to hyperventilation, whereas this patient has elevated pCO■ as a compensatory response.

References:

Ghai Textbook of Paediatrics Edition 10, Page 71, 72, 73, 74

↑ *Back to this question in Part A*

@Neet_pg_bot

Q21: The development of cataracts in patients with Diabetes Mellitus is primarily due to the accumulation of which of the following substances in the lens? (Q852565)

A. Sorbitol

B. Dulcitol

C. Mannitol

D. Galactitol

Correct Answer: A. Sorbitol

Explanation: Sorbitol accumulation in the lens is the primary cause of diabetic cataracts . In diabetes mellitus , chronic hyperglycaemia leads to the activation of the polyol pathway , where: Glucose is converted to sorbitol by aldose reductase . Sorbitol is slowly metabolised to fructose by sorbitol dehydrogenase . In the lens, sorbitol accumulates , causing: Osmotic stress → water influx → lens swelling Oxidative stress → protein damage → cataract formation Diabetic cataracts typically present as snowflake cataracts in younger individuals or as accelerated senile cataracts . Dulcitol (Option B) is incorrect because dulcitol (galactitol) accumulates in galactosemia , not diabetes. It causes galactose-induced cataracts due to galactokinase or GALT deficiency . Mannitol (Option C) is incorrect because mannitol is an exogenous osmotic diuretic and does not accumulate in the lens. Galactitol (Option D) is incorrect because it is another name for dulcitol , and its accumulation is linked to galactosemia , not diabetes.

References:

https://pmc.ncbi.nlm.nih.gov/articles/PMC2903955/

https://pmc.ncbi.nlm.nih.gov/articles/PMC11556822/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q22: Which of the following enzymes is typically elevated in prostate cancer? (Q852562)

A. Acid phosphatase

B. Alkaline phosphatase

C. Lactate dehydrogenase

D. Alpha-fetoprotein

Correct Answer: A. Acid phosphatase

Explanation: Acid phosphatase , specifically prostatic acid phosphatase (PAP) , is an enzyme that is elevated in prostate cancer , particularly in advanced or metastatic cases . PAP was historically used as a tumour marker for prostate cancer , though prostate-specific antigen (PSA) is now the preferred marker. Significance: Elevated in prostate cancer with bone metastases Can be used to monitor disease progression Less commonly used now due to the superior sensitivity of PSA Alkaline Phosphatase (Option B) is incorrect because while alkaline phosphatase (ALP) is elevated in bone metastases , it is not specific for prostate cancer and can also be seen in liver disease and bone disorders. Lactate Dehydrogenase (Option C) is incorrect because LDH is a non-specific marker of cell turnover and is elevated in various malignancies, not just prostate cancer. Alpha-Fetoprotein (Option D) is incorrect because AFP is primarily a marker for hepatocellular carcinoma and germ cell tumours , not prostate cancer.

References:

Robbins Pathology, 10th Edition, Page 976

↑ *Back to this question in Part A*

@Neet_pg_bot

Q23: Which test should be done for detecting metabolite levels in inborn errors of metabolism in neonates? (Q839926)

A. Tandem mass spectroscopy

B. Complete blood count (CBC)

C. Urinalysis

D. Genetic testing

Correct Answer: A. Tandem mass spectroscopy

Explanation: Correct Option A - Tandem mass spectroscopy: Tandem mass spectroscopy (MS/MS) is widely used for newborn screening for metabolic disorders. It allows for the detection of multiple metabolic diseases from a single blood sample. It is highly sensitive and can identify various metabolic abnormalities by analyzing specific metabolites and their precursors. Incorrect Options: Option B - Complete blood count (CBC): CBC is a common blood test used to evaluate overall health and detect a wide range of disorders, including anemia, infection, and many other diseases. However, it is not specifically designed for metabolic disease screening. Option C - Urinalysis: Useful for evaluating kidney function and urinary tract issues but not comprehensive enough for metabolic disease screening Option D - Genetic testing: Identifies specific genetic mutations, often used when there is a high suspicion of a particular disorder, but not a first-line screening tool due to cost and specificity.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q24: What is the order of the band in the sarcomere? (Q854616)

A. I-Z-M-A-H

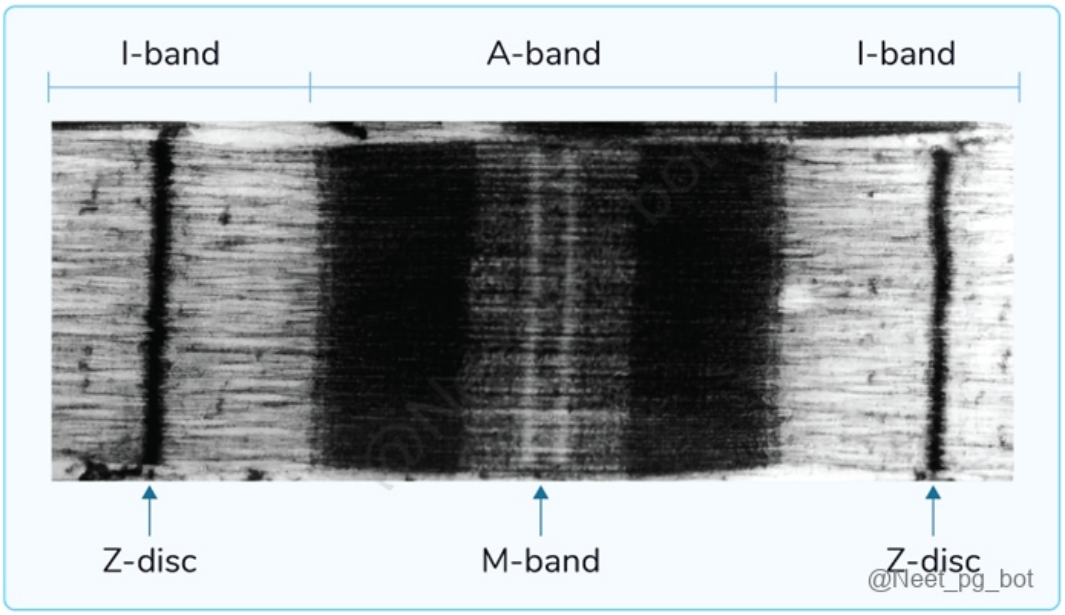
B. Z-H-M-A-I

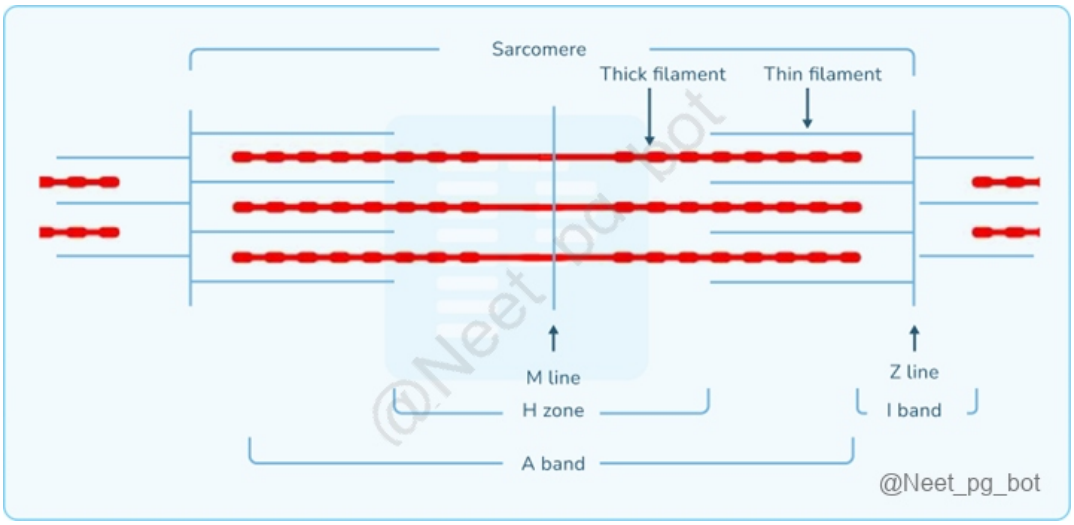
C. Z-I-A-H-M

D. Z-H-I-A-M

Correct Answer: C. Z-I-A-H-M

Explanation: {{caption_text}} Sarcomere The functional unit of a myofibril, composed of alternating dark A bands and light I bands. Z Line A dense, narrow line that bisects the I band; contains 2 actin molecules. I Band The light band alternating with the dark A band; bisected by the Z line. A Band The dark band in the sarcomere; includes the central H zone and is composed of thick filaments. H Zone The lighter region in the center of the A band. M Line The dark line in the middle of the H zone; contains M protein.





References:

Vasudevan's Textbook of Biochemistry for Medical Students, 7th edition, page 724

↑ *Back to this question in Part A*

@Neet_pg_bot

Q25: Nitric oxide is synthesized from (Q859428)

A. L-arginine

B. L-citrulline

C. Glycine

D. Lysine

Correct Answer: A. L-arginine

Explanation: Nitric oxide (NO■) is a highly reactive free radical with an unpaired electron, making it highly reactive. It is uncharged and has a short half-life (approximately 0.1 second). Synthesis of Nitric Oxide : NO■ is synthesized from arginine by the enzyme nitric oxide synthase (NOS) . NOS contains heme , FAD , FMN , and tetrahydrobiopterine . The process utilizes NADPH and molecular oxygen . Calmodulin is required to modulate NOS activity. The guanidino nitrogen of arginine is incorporated into NO■, while one oxygen atom from molecular oxygen is incorporated into NO■ and the other into citrulline . This makes NOS a di-oxygenase enzyme. Isoenzymes of NOS : Mechanism of Action of Nitric Oxide : NO■ diffuses to adjacent smooth muscle cells and activates guanylate cyclase . This leads to an increase in cyclic GMP levels, which activates protein kinase in smooth muscle. This causes the dephosphorylation of myosin light chains , resulting in relaxation of smooth muscle (vasodilation) in blood vessels, leading to lower blood pressure . L-citrulline (Option B) : L-citrulline is a byproduct of nitric oxide synthesis, as it is produced from L-arginine during the enzymatic conversion to NO■. It is not the direct precursor for nitric oxide. Glycine (Option C) : Glycine is an amino acid, but it is not involved in the synthesis of nitric oxide. It serves other functions in the body, such as being a neurotransmitter. Lysine (Option D) : L-lysine is another amino acid, but it does not play a direct role in the synthesis of nitric oxide.

References:

Vasudevan 7th edition Pg no. 227, 228

↑ *Back to this question in Part A*

@Neet_pg_bot

Q26: A child with mental retardation, blindness, and muscle weakness was investigated and was found to be deficient for Hexosaminidase-A enzyme. She is most likely suffering from .. (Q859572)

A. Krabbe's disease

B. Fabry's disease

C. Tay-sachs disease

D. Gaucher's disease

Correct Answer: C. Tay-sachs disease

Explanation: The child in the clinical scenario is showing signs of mental retardation, blindness, and muscle weakness , which strongly suggest a lysosomal storage disorder . Upon investigation, the child was found to be

deficient in hexosaminidase A enzyme. The disease most likely causing these symptoms is Tay-Sachs disease .It is an inborn error of metabolism due to the failure of degradation of gangliosides, which is caused by a deficiency in the enzyme hexosaminidase A. Lipid Storage Diseases (Sphingolipidoses): They are a group of lysosomal storage diseases. Disease Enzyme deficiency Features Tay-Sachs disease (Option C) Hexosaminidase A Mental retardation Blindness Muscular weakness Cherry red spots in the macula Fabry disease (Option B ruled out) α-Galactosidase Skin rash Kidney failure (full symptoms only in males X-linked recessive) Krabbe disease (Option A ruled out) β-Galactosidase Mental retardation Near absence of myelin Predominant neurological manifestation and cranial nerves causing deafness and blindness. Gaucher disease (Option D ruled out) β-Glucosidase Enlarged liver and spleen Erosion of long bones Mental retardation in infants Anemia Thrombocytopenia Niemann-Pick disease Sphingomyelinase Enlarged liver and spleen Mental retardation Fatal in early life Cherry red spot in macula Farber disease Ceramidase Hoarseness Dermatitis Skeletal deformation Mental retardation Fatal in early life

References:

Harper's illustrated Biochemistry 32e pg 245

https://www.ncbi.nlm.nih.gov/books/NBK564432/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q27: In a 14-year-old male, a medical condition has been identified as a result of a defect in a gene responsible for DNA repair. The individual is experiencing increased pigmentation on the skin and is at a heightened risk of developing skin cancers, according to the physician. Which of the following mechanisms is most likely to be impaired in this particular condition? (Q859573)

A. Nonhomologous end joining repair defect

B. Nucleotide excision repair defect

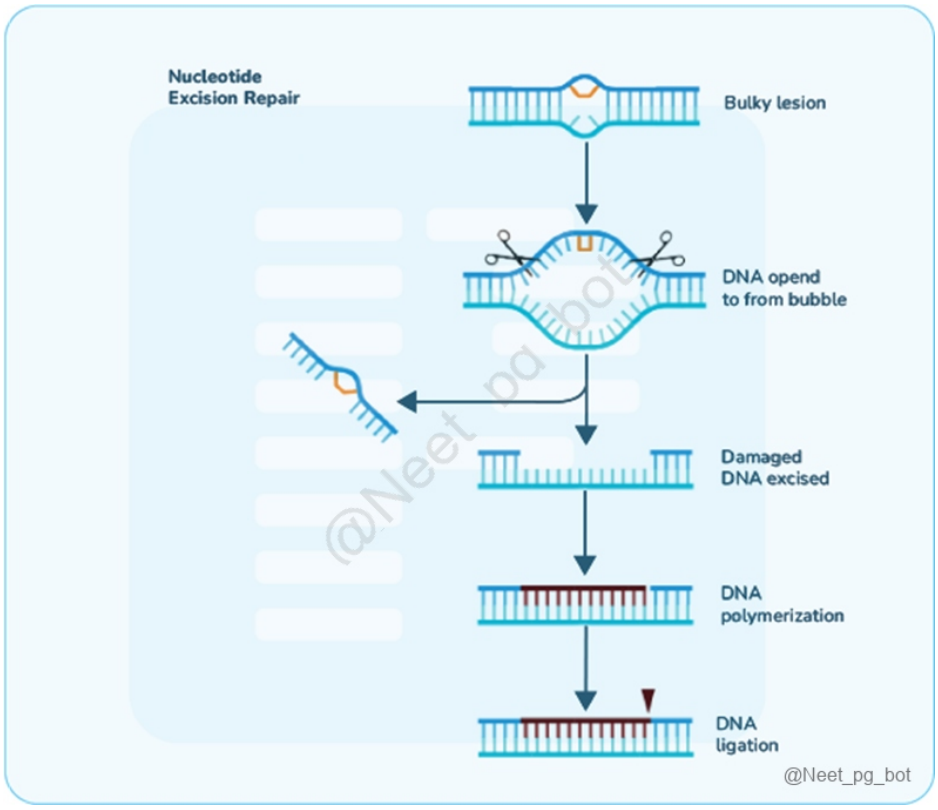
C. DNA base excision repair defect

D. DNA mismatch repair defect

Correct Answer: B. Nucleotide excision repair defect

Explanation: In this 14-year-old male, the condition described involves increased pigmentation on the skin and a heightened risk of skin cancers. These symptoms are consistent with Xeroderma Pigmentosum (XP) , a disorder caused by an inability to repair UV-induced DNA damage. UV radiation can cause the formation of pyrimidine dimers (thymine dimers) in the skin cells, which, if not repaired, lead to mutations and an increased risk of cancer. The key defective mechanism in XP is Nucleotide Excision Repair (NER) . Nucleotide excision repair: Nucleotide excision repair is required when chemical damage to a segment occurs. UV mediated Pyr Pyr dimerization is observed in a segment. 30 bases are removed and then the correct bases are added Recognition and Excision: In

bacteria, UvrABC proteins (uvrABC excinuclease) recognize these dimers and cleave the damaged strand on both the 5′ and 3′ sides of the lesion. A short oligonucleotide containing the dimer is excised, leaving a gap in the DNA strand. The gap is subsequently filled in by DNA polymerase I and sealed by DNA ligase. Nucleotide Excision Repair in Humans: A related nucleotide excision repair (NER) pathway exists in humans, addressing UV-induced damage encountered during DNA synthesis. Transcription-coupled repair, a type of NER, specifically targets lesions that obstruct RNA synthesis. Clinical application: Pyrimidine dimers can form in human skin cells exposed to unfiltered sunlight. In xeroderma pigmentosum (XP), a genetic disorder, the inability to repair UV-induced DNA damage leads to the accumulation of mutations and a heightened risk of developing early and multiple skin cancers. XP results from defects in genes coding for proteins necessary for NER of UV damage {{caption_text}}



References:

Vasudevan 7th edition, pg 583

Lippincott 7th edition, 1219-1222

↑ *Back to this question in Part A*

@Neet_pg_bot

Q28: Nitrogenous waste is excreted from the body in the form of? (Q859710)

A. Urea

B. Nitric oxide

C. Ammonia

D. Glutamine

Correct Answer: A. Urea

Explanation: Ammonia is produced during the breakdown of amino acids and by gut bacteria in humans. However, ammonia is highly toxic, especially to the brain, if its levels in the blood rise too high. To prevent this toxicity, the body converts ammonia into urea through the urea cycle . This process occurs in the liver, where ammonia is safely transformed into urea, a less toxic substance. Urea is then excreted from the body through the urine, effectively removing excess nitrogenous waste from the body. Incorrect Options: Nitric oxide (Option B): A signaling molecule involved in vasodilation and other processes, not nitrogenous waste excretion. Ammonia (Option C): While ammonia is produced during amino acid breakdown, it is toxic at high levels and is converted into urea for excretion. Glutamine (Option D): An amino acid involved in nitrogen transport but not directly excreted as a nitrogenous waste product.

References:

The Urea Cycle Step by Step

↑ *Back to this question in Part A*

@Neet_pg_bot

Q29: A scientist treats experimental cells with a histone acetylase inhibitor. Which of the following is the most immediate epigenetic change of this treatment? (Q879279)

A. Increased expression of H3K9me2/3

B. Increased acetylation of H3K9ac

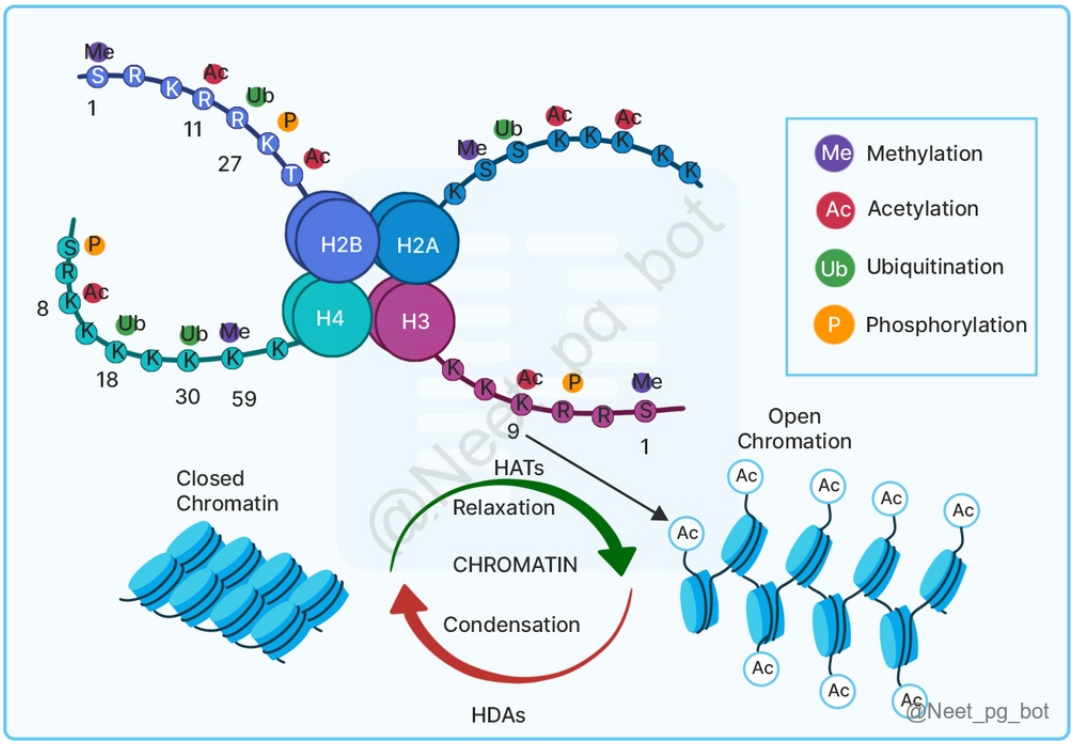
C. Chromatin condensation

D. DNA methylation

Correct Answer: C. Chromatin condensation

Explanation: Histone acetylases (HATs) add acetyl (Ac) groups to histones → relax chromatin → increase transcription . Histone deacetylases (HDACs) remove acetyl groups → condense chromatin → decrease transcription. Effects of Inhibition of HATs : Immediate effects: ↓ Histone acetylation ↑ Chromatin condensation (most immediate epigenetic change) ↓ Transcriptional activity Delayed downstream effects: ↑ H3K9me2/3 (methylation of lysine-9 residue on histone-3) occurs later because condensed chromatin recruits histone methyltransferases → establishes stable heterochromatin. ↑ DNA methylation is also a later, reinforcing silencing mechanism. Incorrect Options: Increased expression of H3K9me2/3 (Option A): H3K9 methylation rises later, not immediately. Increased acetylation of H3K9ac (Option B) : HAT inhibition causes decreased , not increased,

acetylation. DNA methylation (Option D): Also promotes gene silencing but occurs later , not as the earliest response.



References:

Histone deacetylase inhibitors: molecular mechanisms of action and clinical trials as anti-cancer drugs - PMC
Histone acetylation and the role of histone deacetylases in normal cyclic endometrium | Reproductive Biology and Endocrinology

↑ *Back to this question in Part A*

@Neet_pg_bot

Q30: In rheumatoid arthritis, synovial tissue from affected patients exhibits elevated expression of TNF-α and IL-6, which contribute to the inflammatory process. Which epigenetic modification most likely accounts for this increased gene expression? (Q879280)

A. Activation of DNA methylation

B. Inhibition of DNA methylation

C. Activation of histone deacetylation

D. Inhibition of histone acetylation

Correct Answer: B. Inhibition of DNA methylation

Explanation: DNA methylation normally suppresses gene expression. Its inhibition (hypomethylation of gene promoters) leads to increased transcription of pro-inflammatory genes such as TNF-α and IL-6 in rheumatoid arthritis synovial tissue. While increased histone acetylation may also contribute, among the options provided, inhibition of DNA methylation is the primary epigenetic modification driving this upregulation. Incorrect Options: Activation of DNA methylation (Option A): Increases gene silencing, which would reduce, not enhance TNF-α and IL-6 expression. Activation of histone deacetylation (Option C): Condenses chromatin and decreases gene transcription. Inhibition of histone acetylation (Option D): Also leads to chromatin condensation and reduced gene expression.

References:

DM Vasudevan's Textbook of Biochemistry, 7th Edition, Page 622-623.

Epigenetic modifications of immune cells in rheumatoid arthritis - PMC

↑ *Back to this question in Part A*

@Neet_pg_bot

Q31: Cancer cells exhibit an increased length of a particular RNA transcript or fragment compared to normal cells. The most likely explanation for this is: (Q879281)

A. Polyadenylation

B. Alternative RNA splicing

C. Gene amplification

D. Gene silencing

Correct Answer: B. Alternative RNA splicing

Explanation: The most likely

References:

DM Vasudevan's Textbook of Biochemistry, 7th Edition, Page 592.

Alternative cleavage and polyadenylation: key regulatory mechanisms in health and disease - PMC

↑ *Back to this question in Part A*

@Neet_pg_bot

Q32: During B-cell development in the bone marrow, each B cell expresses only one immunoglobulin heavy chain allele to ensure that all antibodies produced by that cell possess a single antigen specificity. This process, known as allelic exclusion, is primarily mediated by: (Q879282)

A. V(D)J rearrangements

B. Alternate splicing of RNA transcripts

C. Alternate polyadenylation sites

D. Somatic hypermutation

Correct Answer: A. V(D)J rearrangements

Explanation: Allelic exclusion is mediated by V(D)J rearrangement , the somatic recombination of variable (V), diversity (D), and joining (J) gene segments that generates the variable region of the immunoglobulin heavy chain. This rearrangement occurs on both alleles, but once a productive rearrangement occurs on one allele, feedback mechanisms inhibit rearrangement and expression from the other allele. This ensures that each B cell expresses a heavy chain from only one allele , producing antibodies with a single antigen specificity. Incorrect Options: Alternate splicing of RNA transcripts (Option B): Alters mRNA isoforms but does not control allele expression. Alternate polyadenylation sites (Option C): Affects mRNA stability or isoform length, not allelic choice. Somatic hypermutation (Option D): Introduces mutations after B-cell activation, not during initial heavy chain selection.

References:

Allelic exclusion of immunoglobulin genes: models and mechanisms - PMC

↑ *Back to this question in Part A*

@Neet_pg_bot

Q33: Which type of mutation results in the reversal to the wild type of phenotype when the mutant gene is suppressed? (Q839921)

A. Frameshift of the mutatedgene

B. Addition of another normal gene

C. Deletion of mutanted gene

D. Mutation in tRNA

Correct Answer: D. Mutation in tRNA

Explanation: Correct Option D - Mutation in tRNA: Mutation in tRNA: This represents a suppressor mutation , where a second mutation occurs in a tRNA gene . The altered tRNA can recognize the mutant codon (often a premature stop codon) and insert an appropriate amino acid, allowing translation to continue. This restores the wild-type phenotype even though the original mutation remains in the coding gene. Example: A nonsense mutation (UAG) introduces a premature stop codon → a tRNA suppressor mutation alters the anticodon to recognize UAG and insert an amino acid → translation continues → wild-type phenotype restored. Incorrect Options: A) Frameshift of the mutated gene: Alters the reading frame, producing a nonfunctional protein. Such mutations are deleterious and irreversible by a single suppressor mutation. B) Addition of another normal gene: Represents gene therapy or complementation , not a mutation. While it may restore function, it is not a genetic suppression event. C) Deletion of the mutated gene: Removes the defective gene but does not restore the wild-type allele or function through mutation.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q34: Which of the following enzymes is responsible for the immortality of cancer cells? (Q866654)

A. Telomerase

B. Topoisomerase

C. Helicase

D. RNA polymerase

Correct Answer: A. Telomerase

Explanation: Telomerase maintains telomere length , preventing chromosome degradation. Telomere shortening occurs with each cell division, contributing to cellular ageing and death . However, telomerase extends telomeres , enabling indefinite division . In cancer cells , its overexpression promotes uncontrolled proliferation and immortality . Incorrect Options: Topoisomerase (Option B) is responsible for manipulating the structure of DNA by inducing and resolving DNA strand breaks. Helicase (Option C) unwinds the DNA double helix, allowing other enzymes to access and replicate the DNA. RNA polymerase (Option D) synthesises RNA from a DNA template during transcription, essential for gene expression and protein synthesis. While its dysregulation can occur in cancer, it is not directly linked to cancer cell immortality.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q35: Which of the following genetic mutations is most commonly associated with familial cases of Amyotrophic Lateral Sclerosis (ALS)? (Q866660)

A. CFTR gene mutation

B. SOD1 gene mutation

C. HTT gene mutation

D. FMR1 gene mutation

Correct Answer: B. SOD1 gene mutation

Explanation: Familial Amyotrophic Lateral Sclerosis (ALS) is primarily associated with mutations in the SOD1 gene, which cause a toxic gain-of-function leading to motor neuron degeneration. Definition: Progressive neurodegenerative disorder affecting motor neurons. Genetics: Most commonly caused by mutations in the SOD1 (superoxide dismutase 1) gene. Pathophysiology: SOD1 mutation leads to toxic gain-of-function, causing motor neuron death. Clinical features: Muscle weakness, atrophy, fasciculations, spasticity, and eventual paralysis. Inheritance: Autosomal dominant pattern in familial cases. Diagnosis: Clinical signs plus genetic testing for SOD1 mutation. Treatment: Mainly supportive; no cure currently available. Incorrect Options: The CFTR gene mutation (Option A) causes cystic fibrosis. CFTR encodes a chloride/bicarbonate channel in epithelial cells. Impaired chloride secretion → dehydrated, thick mucus → chronic pulmonary infection/inflammation, bronchiectasis; pancreatic exocrine insufficiency (steatorrhea, failure to thrive); male infertility from congenital bilateral absence of the vas deferens; salty sweat (↑ Cl■). The HTT gene mutation (Option C) causes Huntington's disease. Expansion of CAG trinucleotide repeats in the HTT gene → polyglutamine (polyQ) toxic gain-of-function. The FMR1 gene mutation (Option D) causes Fragile X syndrome, which has CGG repeat expansion in the FMR1 gene. The most common inherited cause of intellectual disability; autistic traits, developmental delay, long face, large ears, macroorchidism, and connective-tissue laxity.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q36: A woman with a strong family history of breast and ovarian cancer is being evaluated for BRCA1/BRCA2 mutations. Which of the following is the most appropriate test to detect multiple mutations at different codons in these genes? (Q865400)

A. qPCR - Measures BRCA1/2 expression levels

B. RT-PCR - Detects RNA-level mutations

C. Northern blot - Analyzes RNA expression

D. Next-generation sequencing (NGS)

Correct Answer: D. Next-generation sequencing (NGS)

Explanation: The most appropriate test to detect multiple mutations at different codons in BRCA1/BRCA2 genes is Next-generation sequencing (NGS) or Digital Droplet PCR. It is a revolutionary method for rapid, high-throughput DNA and RNA sequencing. Enables massive parallel sequencing of hundreds to thousands of genes or whole genomes. Significantly faster and more comprehensive than traditional methods like Sanger sequencing. Integrates: Advanced sequencing chemistries Diverse sequencing platforms Robust bioinformatics tools Widely used for: Disease diagnosis and prognosis Therapy selection Patient monitoring qPCR (Option A) measures gene expression levels, not specific DNA mutations. RT-PCR (Option B) detects RNA-level changes, limited for comprehensive mutation scanning. Northern blot (Option C) analyzes RNA expression, not DNA mutations.

References:

Next-generation sequencing and its clinical application - PMC

Digital Droplet PCR: CNV Analysis and Other Applications - PMC

↑ *Back to this question in Part A*

@Neet_pg_bot

Q37: Which of the following techniques are correctly matched with their uses: (Q865401)

A. Southern blot - RNA Identification, Northern blot - DNA Identification, Western blot - Protein Identification

B. Southern blot - DNA Identification, Northern blot - Protein Identification, Western blot - RNA Identification

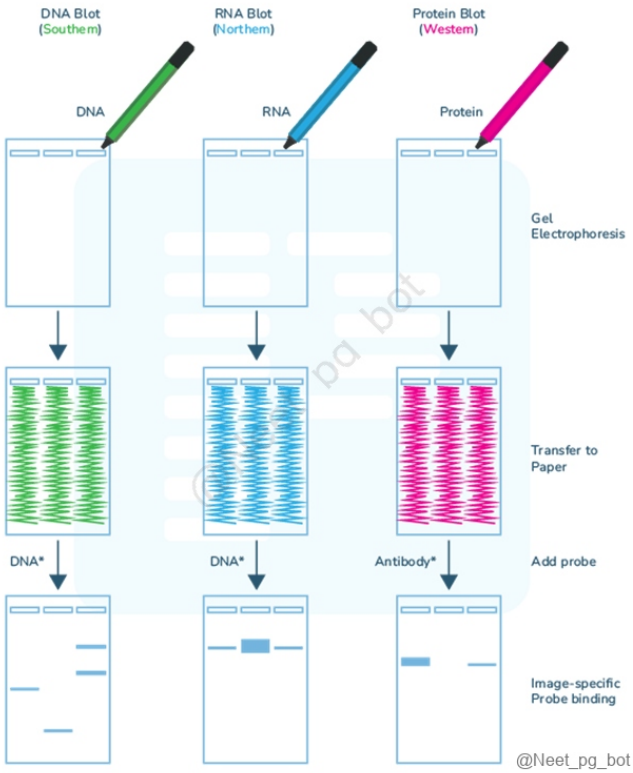
C. Southern blot - Protein Identification, Northern blot - RNA Identification, Western blot - DNA Identification

D. Southern blot - DNA Identification, Northern blot - RNA Identification, Western blot - Protein Identification

Correct Answer: D. Southern blot - DNA Identification, Northern blot - RNA Identification, Western blot - Protein Identification

Explanation: Correct match is Southern blot - DNA Identification, Northern blot - RNA Identification, Western blot - Protein Identification. Types of blotting: Types Target Features Southern blotting DNA sequences It involves separating DNA fragments by gel electrophoresis, transferring them to a membrane, and then using labelled probes to identify specific sequences Northern blotting RNA sequences The process is similar to DNA blotting but focuses on RNA . Western blotting Proteins This technique specifically detects and analyses proteins

in a complex mixture. It involves separating proteins by gel electrophoresis, transferring them to a membrane, and then using specific antibodies to detect the proteins. Eastern blotting Post-translational modifications It refers to specialised Western blotting techniques that detect specific post-translational modifications of proteins, such as phosphorylation or glycosylation. Southwestern blotting DNA-binding proteins Combines Southern and Western blotting elements to identify proteins that bind to specific DNA sequences.



References:

Harper's Textbook of Biochemistry, 32nd Edition, Page 450-451.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q38: After how many cycles of PCR, are we expected to have half the number of copies as found after 26 cycles? (Q865403)

A. 27

B. 13

C. 25

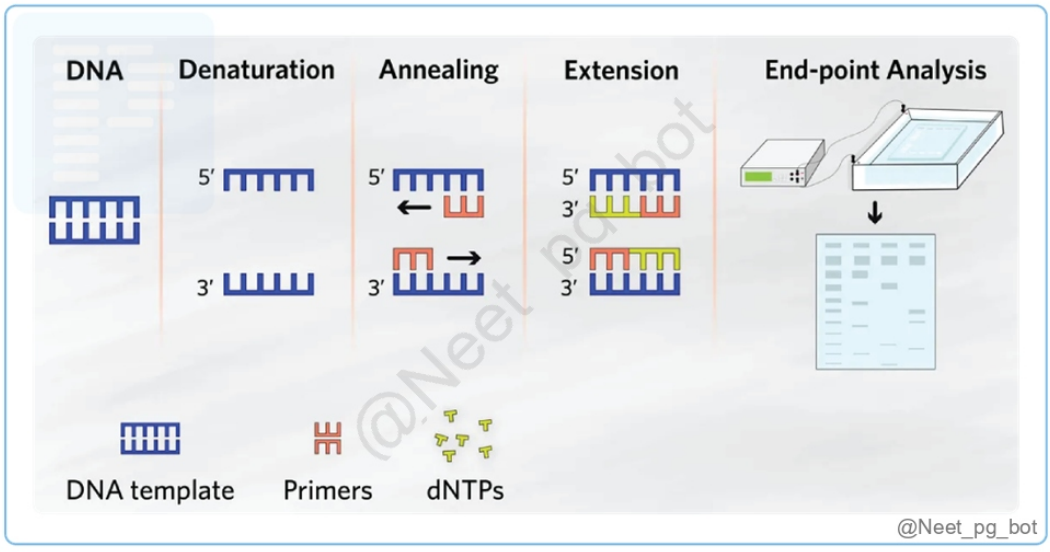
D. 35

Correct Answer: C. 25

Explanation: If PCR goes through ‘n’ number of cycles, Number of products formed: 2 n Half the number of copies compared to after 26 cycles will be: 25 Steps of PCR: Step 1- Denaturation The double-stranded DNA is heated to around 95°C to separate into 2 single strands. Step 2- Annealing Addition of two primers , which anneals the flanking sequence of the desired fragments. An optimal temperature (5 degrees less than the average melting temperature of the primers) should be maintained to avoid non-specific amplification. Step 3- Elongation The enzyme used is thermostable Taq (Thermus Aquaticus) DNA polymerase. DNA polymerase elongates new strands in 5’3’ direction and the optimal temperature required for elongation is 72°C. If PCR goes through ‘n’ number of cycles, then ‘2 n ’ number of products are formed.

$$\frac{1}{2} \times 2^{26} = 2^{25}$$

@Neet_pg_bot



References:

Harper’s Textbook of Biochemistry, 32nd Edition, Page 452-453.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q39: Cancer cells consume more glucose by which of the following process: (Q865442)

A. Increased GLUT-2 transporters, oxidative phosphorylation

B. Increased conversion of pyruvate to Acetyl CoA

C. Increased lactate is produced even in the presence of oxygen

D. Increased fatty acid oxidation

Correct Answer: C. Increased lactate is produced even in the presence of oxygen

Explanation: Cancer cells exhibit a phenomenon known as the Warburg effect, where they consume more glucose and convert it to lactate even when oxygen is plentiful (aerobic glycolysis). This process allows cancer cells to generate energy and biosynthetic precursors rapidly to support their uncontrolled growth, despite the inefficiency of producing only 2 ATPs per glucose molecule compared to the 36-38 ATPs from oxidative phosphorylation in normal cells. Increased GLUT-2 transporters, oxidative phosphorylation (Option A) - While increased glucose uptake via GLUT transporters is a feature of cancer cells, oxidative phosphorylation is reduced in cancer cells due to the Warburg effect. They rely more on glycolysis and lactate production rather than oxidative phosphorylation. Increased conversion of pyruvate to Acetyl CoA (Option B) - In normal cells, pyruvate is converted to Acetyl CoA for entry into the citric acid cycle under aerobic conditions. However, in cancer cells, pyruvate is preferentially converted to lactate due to the Warburg effect, not to Acetyl CoA. Increased fatty acid oxidation (Option D) - Fatty acid oxidation is more characteristic of normal cells or certain cancer types under specific conditions, but it is not the primary process for increased glucose consumption in cancer cells.

References:

Harper's Illustrated Biochemistry, 31st Edition, (Chapter 19, "Oxidative Phosphorylation and the Warburg Effect)

↑ *Back to this question in Part A*

@Neet_pg_bot

Q40: Gene pool refers to all the alleles in a: (Q865448)

A. Family

B. Individual

C. Population

D. Tissue

Correct Answer: C. Population

Explanation: The gene pool consists of all copies of every type of allele at every locus in all members of the population. This definition emphasizes that the gene pool encompasses genetic variation across an entire population, not just an individual or family. Family (Option A) A family represents only a small subset of a population and does not encompass all genetic diversity. Individual (Option B) An individual has only two alleles per gene (diploid), not the entire pool of alleles. Tissue (Option D) Tissue is a group of cells that have similar structure and that function together as a unit.

References:

Campbell Biology, 12th Edition, Page 484.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q41: A frameshift mutation occurs due to the insertion of a new nucleotide at the 4th position of an mRNA sequence with 900 nucleotides. What is the most likely outcome of this mutation? (Q866327)

A. No change in the final protein

B. Partial protein production

C. Complete change in protein production

D. No change due to RNA editing

Correct Answer: C. Complete change in protein production

Explanation: The insertion of a nucleotide at the 4th position of a 900-nucleotide mRNA sequence causes Frameshift Mutation , most likely resulting in a complete alteration of the protein produced. It occur when nucleotides are inserted into or deleted from the DNA or mRNA sequence, causing a shift in the reading frame of the genetic code. This usually results in: A completely different sequence of amino acids from the mutation site onward. Potential introduction of premature stop codons, leading to truncated proteins. Loss of the original protein function. Incorrect Options: No change in the final protein (Option A): Unlikely, because an insertion at position 4 shifts the reading frame early, changing most of the protein. Partial protein production (Option B): Possible if a premature stop codon is introduced, but the question emphasizes the insertion at the 4th position, so the frame shift affects nearly the entire protein. No change due to RNA editing (Option D): RNA editing is rare and usually doesn't correct frameshift mutations, so unlikely.

↑ *Back to this question in Part A*

Q42: A patient presents with skin cancer and hyperpigmentation that worsens with sunlight exposure. Which of the following DNA repair mechanisms is most likely defective in this condition? (Q866329)

A. Nucleotide excision repair

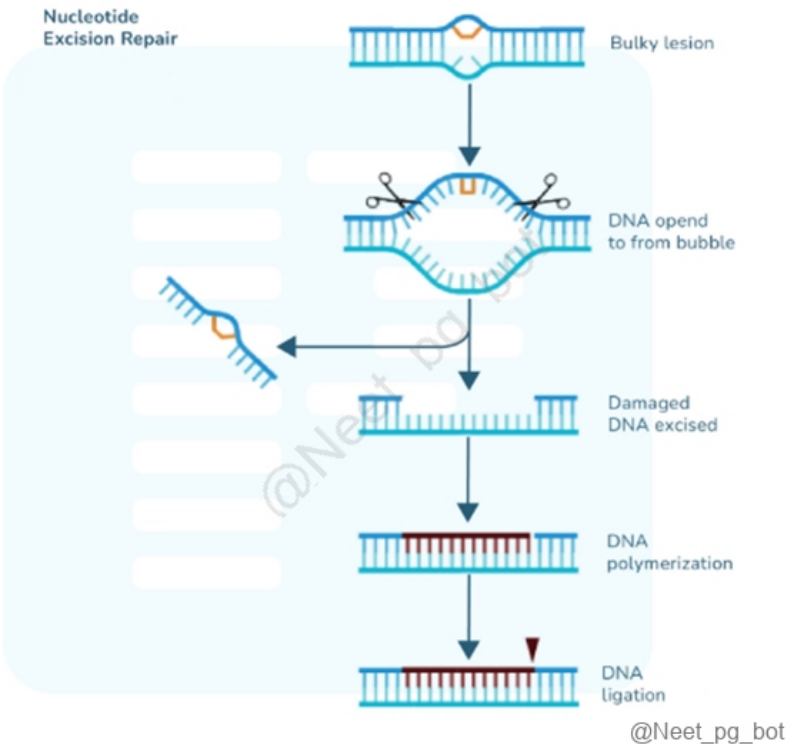
B. Base excision repair

C. Non-homologous end joining (NHEJ)

D. Mismatch repair

Correct Answer: A. Nucleotide excision repair

Explanation: Skin cancer and hyperpigmentation that worsen with sun exposure strongly indicate a defect in the Nucleotide Excision Repair (NER) Pathway , which normally repairs UV-induced bulky DNA lesions like thymine dimers; such defects are seen in conditions like xeroderma pigmentosum (XP), marked by extreme UV sensitivity, pigmentation abnormalities, and a high risk of skin cancer. This repair is required when, Chemical damage to a segment occurs. UV mediated Pyr Pyr dimerization is observed in a segment. 30 bases are removed and then the correct bases are added. A related nucleotide excision repair (NER) pathway exists in humans, addressing UV-induced damage encountered during DNA synthesis. Transcription-coupled repair, a type of NER, specifically targets lesions that obstruct RNA synthesis. Clinical application: Pyrimidine dimers can form in human skin cells exposed to unfiltered sunlight. In xeroderma pigmentosum (XP), a genetic disorder, the inability to repair UV-induced DNA damage leads to the accumulation of mutations and a heightened risk of developing early and multiple skin cancers. XP results from defects in genes coding for proteins necessary for NER of UV damage Incorrect Options: Base excision repair (Option B) fixes small, non-bulky base lesions (e.g., oxidative damage). Non-homologous end joining (NHEJ) (Option C) repairs double-strand breaks, unrelated to UV damage. Mismatch repair (Option D) corrects replication errors, not UV-induced damage.



↑ *Back to this question in Part A*

@Neet_pg_bot

Q43: A 22-year-old tall male presents with long limbs, increased arm span, hypermobile joints, and a high-arched palate. On examination, he has lens subluxation and a diastolic murmur suggestive of aortic root dilation. Which of the following genes is most likely mutated in this condition? (Q866331)

A. Fibrillin-1 (FBN1)

B. COL4A5

C. COL1A1

D. Elastin

Correct Answer: A. Fibrillin-1 (FBN1)

Explanation: The combination of tall stature, long limbs, increased arm span, hypermobile joints, high-arched palate, lens subluxation, and aortic root dilation strongly suggests Marfan Syndrome , a connective tissue disorder caused by mutations in the Fibrillin-1 (FBN1) gene affecting the cardiovascular, ocular, and skeletal systems.

Marfan Disease The most common Clinical Manifestation is Skeletal Abnormalities. Mitral Valve Prolapse (Most common Cardiac abnormality) Aortic Dissection (Most common cause of death) Retinal Detachment (eye problem) Freely moveable Joint (most common Thumb joint) Arachnodactyly (long fingers, spider fingers), High Arched Palate Nine Feet Tall, Pigeon chest (defect in sternum) Dislocation of the lens (Ectopia Lentis- Direction of dislocation is Supero Temporal) Steinberg Sign: Positive if the thumb tip extends beyond the palm when folded into a closed fist. Walker Sign: Positive if the thumb and fifth finger overlap when you grip your wrist with the opposite hand. Incorrect Options: COL4A5 (Option B) is associated with Alport syndrome (kidney disease, hearing loss, and eye abnormalities). COL1A1 (Option C) is linked to osteogenesis imperfecta (brittle bones). Elastin (Option D) mutations cause supravalvular aortic stenosis, not typical Marfan features.





References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q44: Which of the following is used in diagnosing aneuploidy? 1. FISH 2. Cytogenetics 3. Sangers 4. PCR (Q853000)

A. 1,2

B. 1,2,3

C. 2,3

D. 1,2,3,4

Correct Answer: A. 1,2

Explanation: Aneuploidy is a condition in which there is an abnormal number of chromosomes, due to the gain or loss of one or more chromosomes (not whole sets). Normal human cells: 46 chromosomes (23 pairs) Aneuploidy: 45, 47, etc 1. FISH (Fluorescence In Situ Hybridisation) → Correct Uses fluorescent probes that bind to specific chromosome regions Can count chromosome copies directly in cells Example: 3 signals for chromosome 21 → Trisomy 21 Hence, directly detects aneuploidy. 2. Cytogenetics (Karyotyping)→Correct (Gold standard) It involves visualising entire chromosomes under a microscope Detects: Extra chromosomes (trisomy) Missing chromosomes (monosomy) Gives a complete chromosomal picture (46, XX / 47, XY +21, etc.) Most definitive test for aneuploidy. 3. Sanger sequencing →Incorrect Reads the exact nucleotide sequence of DNA Detects: Point mutations Small insertions/deletions Cannot determine the number of whole chromosomes. So it misses aneuploidy. 4. PCR →Incorrect Amplifies specific DNA sequences Useful for: Detecting genes Mutations Does not measure chromosome number reliably. Hence, not used for diagnosing aneuploidy.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 444-457

↑ *Back to this question in Part A*

@Neet_pg_bot

Q45: Enzymes used in recombinant DNA technology are: 1. Isomerase 2. Phosphatase 3. Terminal transferase. 4. CRISPR CASS (Q853001)

A. 1,2,3,4

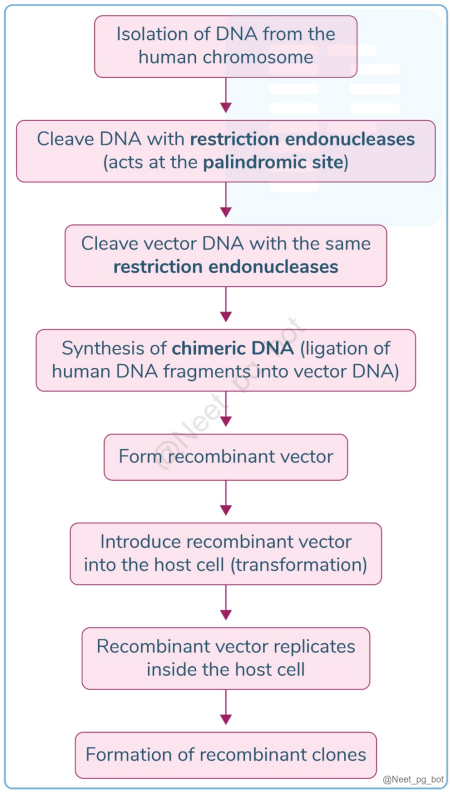
B. 1,2,3

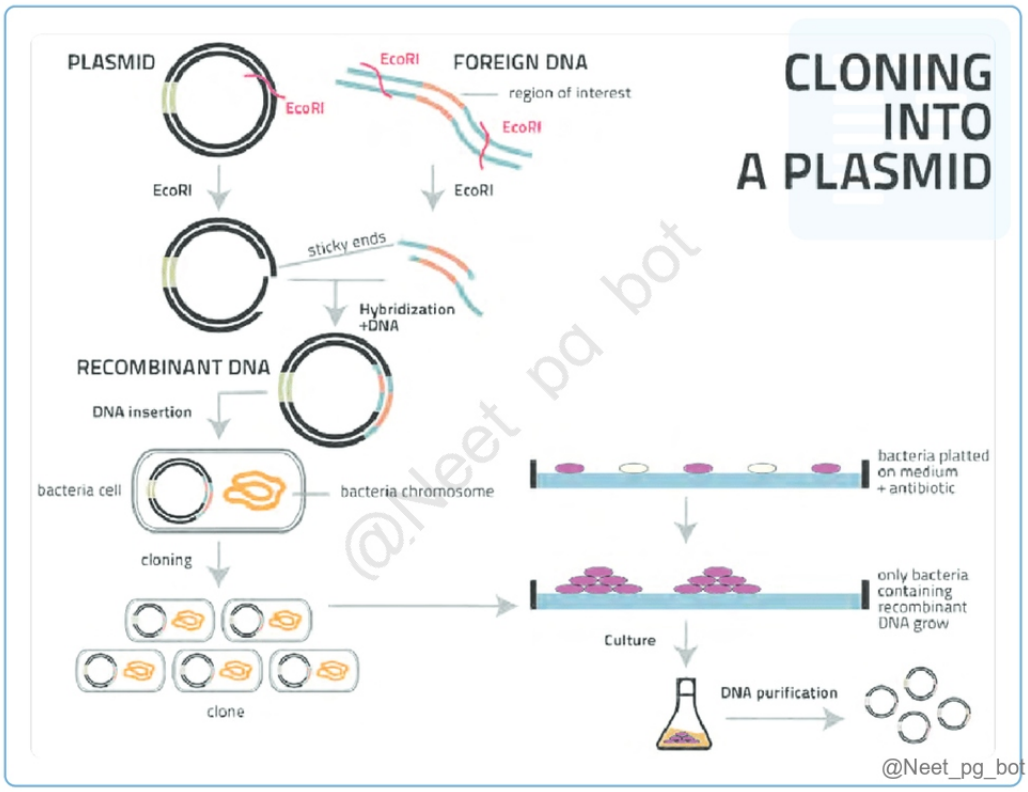
C. 2,3,4

D. 1,3,4

Correct Answer: C. 2,3,4

Explanation: Phosphatase, terminal transferase, and CRISPR CASS are used in rDNA technology Phosphatase Removes phosphate groups from the 5' ends of DNA to prevent self-ligation of vectors during cloning. Terminal Transferase Adds nucleotides to the 3' ends of DNA molecules, enabling the creation of sticky or blunt ends for cloning. CRISPR-CASS Precisely cuts DNA at specific sequences using a guide RNA, enabling targeted genome editing and manipulation. Steps of recombinant DNA Technology {{caption_text}} {{caption_text}}





References:

Harper's Biochemistry, 32nd edition, Pages 444-448

↑ *Back to this question in Part A*

@Neet_pg_bot

Q46: A child presents with micrognathia and low-set ears. These clinical features are commonly associated with which type of genetic abnormality? (Q852566)

A. Deletion

B. Translocation

C. Amplification

D. Inversion

Correct Answer: A. Deletion

Explanation: Deletion is the most common genetic abnormality associated with micrognathia (small jaw) and low-set ears . These features are commonly seen in chromosomal deletion syndromes , such as: Cri-du-chat

syndrome (5p deletion) Micrognathia, low-set ears, high-pitched cry ("cat-like cry") Intellectual disability and growth delay DiGeorge syndrome (22q11.2 deletion) Micrognathia, low-set ears, conotruncal heart defects (e.g., TOF) Thymic hypoplasia → immunodeficiency Treacher Collins syndrome (TCOF1 gene mutation, chromosomal microdeletion) Micrognathia, low-set ears, facial hypoplasia Conductive hearing loss Translocation (Option B) is incorrect because translocations usually cause genetic disorders by gene fusion or misregulation , often leading to cancers (e.g., CML t(9;22)) , rather than micrognathia and low-set ears. Amplification (Option C) is incorrect because gene amplification is associated with oncogenesis (e.g., HER2 in breast cancer, N-MYC in neuroblastoma) , not structural anomalies. Inversion (Option D) is incorrect because chromosomal inversions rarely lead to major congenital anomalies , unless a critical gene is disrupted.

References:

https://pubmed.ncbi.nlm.nih.gov/31509347/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q47: A mother brings her 14-year-old mentally retarded son to the clinic, complaining of joint pain and an irresistible urge for the boy to bite his fingers and lips. Serum uric acid is elevated. What is the most likely diagnosis? (Q844280)

A. Lesch-Nyhan syndrome

B. Gout

C. Hyperuricemia

D. Juvenile arthritis

Correct Answer: A. Lesch-Nyhan syndrome

Explanation: Correct Option A - Lesch-Nyhan syndrome: Lesch-Nyhan syndrome is caused by a deficiency of the enzyme hypoxanthine-guanine phosphoribosyltransferase (HGPRT). This enzyme is essential for the salvage pathway of purines, and its deficiency leads to the accumulation of uric acid in the body, causing hyperuricemia. The patient also presents with self-mutilating behaviour. Incorrect Options: Options B, C, and D do not show the given findings.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q48: Which of the following is not associated with post-transcriptional modification? (Q839917)

A. Methylation

B. Endonuclease cleavage

C. 5' capping

D. Glycosylation

Correct Answer: D. Glycosylation

Explanation: Glycosylation is a post-translational modification that occurs after translation, not during post-transcriptional modification. Post-transcriptional modification involves alterations to mRNA after it's transcribed but before it's translated into proteins. This includes adding a protective cap and tail to the mRNA, removing non-coding sections (introns) through splicing, and sometimes editing the mRNA's nucleotide sequence. These modifications affect mRNA stability, transport, and the proteins ultimately produced. Methylation (Option A): Methylation can occur as a post-transcriptional modification, particularly in eukaryotic mRNA. It can affect gene expression and mRNA stability. Endonuclease cleavage (Option B): Endonuclease cleavage typically occurs during RNA processing, where introns are removed from the pre-mRNA to produce mature mRNA. So, it is indeed associated with post-transcriptional modification. 5' capping (Option C): 5' capping is a crucial post-transcriptional modification in eukaryotic mRNA, where a 7-methylguanosine cap is added to the 5' end. This modification protects the mRNA from degradation and aids in translation initiation.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q49: A patient presents with multiple colonic polyps and has been diagnosed with colorectal carcinoma. There is a strong family history of Hereditary Non-Polyposis Colorectal Cancer (HNPCC). Which DNA repair mechanism is most likely defective in this condition ? (Q866643)

A. Nucleotide excision repair

B. Mismatch repair

C. Base excision repair

D. Double-strand break repair

Correct Answer: B. Mismatch repair

Explanation: A patient with multiple colonic polyps, colorectal carcinoma, and a family history of HNPCC most likely has a defect in the DNA mismatch repair mechanism. Hereditary Nonpolyposis Colon Cancer (HNPCC), also known as Lynch syndrome: Characterized by microsatellite instability. The most common gene mutation associated with Lynch syndrome is in the human MutS homolog 2 (hMSH2) gene. Defects in mismatch repair

genes, particularly hMSH1 and hMSH2, lead to ineffective repair of DNA replication errors. These gene defects are located on chromosome 2. The inability to correct mismatches during DNA replication increases the risk of mutations, contributing to the development of colon cancer. Incorrect Options: Nucleotide excision repair (Option A) fixes bulky DNA damage like thymine dimers and is defective in xeroderma pigmentosum, not HNPCC. Base excision repair (Option C) corrects small base lesions from oxidative damage and is not involved in HNPCC. Double-strand break repair (Option D) handles major DNA breaks and is linked to BRCA mutations, not mismatch repair defects seen in HNPCC.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q50: FISH technique helps to identify all except?? (Q839943)

A. Point mutation

B. Translocation

C. Deletion

D. Gene amplification

Correct Answer: A. Point mutation

Explanation: Correct Option A - Point mutation: FISH (Fluorescence In Situ Hybridization) is not useful for detecting point mutations. FISH is designed to detect larger genetic changes such as deletions, duplications, translocations, and amplifications. Point mutations involve a single nucleotide change, which is too small for FISH to detect. Incorrect Options: Option B - Translocation: FISH is useful for detecting translocations because it can identify specific chromosome rearrangements by using probes that bind to the translocated segments. Option C - Deletion FISH can detect deletions by using probes that bind to the region where the deletion occurs. If the probe does not bind, it indicates a deletion in that area. Option D - Amplification FISH can detect gene amplifications by showing an increased number of signals from the probe, indicating that there are multiple copies of a particular gene or chromosomal region.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q51: Which of the following is true about p43? (Q857848)

A. It increases telomere length in the liver

B. It synthesizes RNA

C. It stabilizes RNA

D. It retains the telomerase enzyme in the nucleus

Correct Answer: D. It retains the telomerase enzyme in the nucleus

Explanation: Correct Option: D) It retains the telomerase enzyme in the nucleus Telomerase is an enzyme that adds DNA repeats (nucleotides) to the ends of chromosomes , preventing them from shortening during DNA replication . p43 has been shown to play a role in regulating telomerase activity. It helps retain telomerase in the nucleu s, which can act on telomeres, thus maintaining their length and contributing to cellular lifespan. By retaining telomerase in the nucleus, p43 helps regulate the balance between cell proliferation and senescence or cell cycle arrest. It increases telomere length in the liver (Option A) Telomere lengthening in the liver is not attributed to p43. Telomeres shorten with cell division, and telomerase, not p43, maintains or lengthens them in specific cells, including stem cells and some cancer cells. It synthesizes RNA (Option B) While p43 regulates gene expression, it is not directly involved in RNA synthesis. RNA polymerases primarily carry out RNA synthesis. It stabilizes RNA (Option C) p43 can indirectly affect RNA levels through gene regulation, p43 does not stabilize RNA molecules.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q52: Which is the correct sequence of steps in isolating desirable protein using recombinant DNA technology? 1. Expression of protein and lysis of the bacterial cell 2. Incorporation of genes into bacteria 3. SDS PAGE 4. Protein elution 5. Hybridization (Q855531)

A. 2, 1, 3, 5, 4

B. 1, 2, 4, 3, 5

C. 1, 5, 2, 4, 3

D. 2, 4, 5, 3, 1

Correct Answer: A. 2, 1, 3, 5, 4

Explanation: The sequence of steps in the isolation of desirable protein using recombinant DNA technology are as follows: Gene incorporation: Desired genes are inserted into bacterial cells using plasmid vectors containing promoter sequences for transcription into mRNA. Protein expression and bacterial cell lysis: Chemical induction activates gene expression, leading to protein synthesis. Bacterial cells are lysed to release proteins, achieved through methods like sonication or enzymatic digestion. SDS-PAGE: Proteins are separated from cellular

components using SDS-PAGE, where samples are denatured with SDS, loaded onto a gel, and separated by size via electrophoresis. Hybridization : Protein identity confirmation is conducted using techniques such as Western blotting, mass spectrometry, or hybridization. Protein elution: Proteins of interest are eluted from the gel, typically through methods like electrolution or diffusion.

References:

pubmed.ncbi.nlm.nih.gov/36853452/#:~:text=This%20comprises%20six%20linked%20stages,creating%20a%20recombinant%20DNA%20molecule

↑ *Back to this question in Part A*

@Neet_pg_bot

Q53: Affinity maturation of antibodies is because of (Q855564)

A. Somatic hypermutation

B. Differential mRNA processing

C. CD40

D. Gene rearrangements

Correct Answer: A. Somatic hypermutation

Explanation: Affinity maturation of antibodies is the process by which B cells produce antibodies with increasingly higher affinity for their specific antigen during an immune response. This is primarily driven by somatic hypermutation (SHM). Somatic Hypermutation: Occurs in the germinal centers of lymphoid tissues. The enzyme Activation-Induced Cytidine Deaminase (AID) introduces random point mutations in the variable (V) regions of immunoglobulin genes, particularly in the complementarity-determining regions (CDRs) , which determine antigen binding. Selection for High Affinity: B cells with mutated antibodies are tested for their ability to bind the antigen presented on follicular dendritic cells. High-affinity B cells receive survival signals and proliferate. Low-affinity or non-functional B cells undergo apoptosis. Over successive rounds of SHM and selection, the antibody pool shifts towards higher affinity for the antigen, enhancing the effectiveness of the immune response.

References:

pmc.ncbi.nlm.nih.gov/articles/PMC4456294/#:~:text=Affinity%20maturation%20is%20the%20process,antigen%20binding%20(Figure%201).

↑ *Back to this question in Part A*

@Neet_pg_bot

Q54: Which of the following techniques is used to detect DNA protein interaction? (Q855047)

A. DNA footprinting

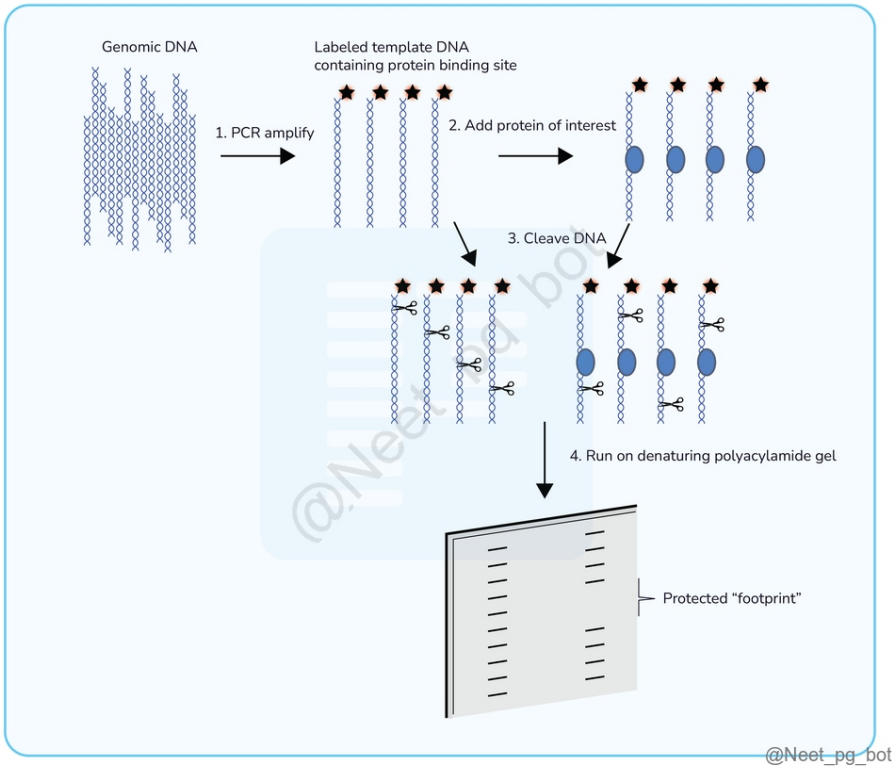
B. DNA fingerprinting

C. ELISA

D. Northern Blotting

Correct Answer: A. DNA footprinting

Explanation: DNA foot printing: This technique is used to detect DNA-protein interactions. It involves binding DNA with a protein and then using a nuclease to selectively cleave it at sites not bound by the protein. This creates a pattern of DNA fragments, which can be analyzed to determine the binding sites of the protein. Identify DNA binding protein sites like pinpointing both the protein and bound sequence. DNA finger printing (Option B): DNA fingerprinting identifies individuals based on their unique DNA profiles.It utilizes PCR and gel electrophoresis to generate a pattern of DNA fragments for comparison with known samples. ELISA (Option C): ELISA detects specific proteins or antigens in samples using antibodies linked to enzymes that produce a signal upon interaction with the target. Northern blotting (Option D): Northern blotting detects RNA molecules by separating them via gel electrophoresis, transferring them to a membrane, and probing with labeled DNA or RNA probes complementary to the target RNA.



References:

pmc.ncbi.nlm.nih.gov/articles/PMC342238/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q55: Misincorporation of uracil into DNA is seen in deficiency of (Q855066)

A. Vitamin B12

B. Folate

C. Vitamin B6

D. Vitamin E

Correct Answer: B. Folate

Explanation: Misincorporation of uracil into DNA is seen in folate deficiency due to impaired thymidine synthesis . Role of Folate in DNA Synthesis: Folate (in its active form, tetrahydrofolate) is crucial for the synthesis of thymidine, one of the four nucleotide bases in DNA. Folate acts as a methyl donor in the conversion of dUMP (deoxyuridine monophosphate) to dTMP (deoxythymidine monophosphate), which is required for DNA synthesis. Effect of Folate Deficiency: In the absence of adequate folate, the conversion of dUMP to dTMP is impaired. dUMP is a precursor to thymidine, but without sufficient folate, it accumulates and can be misincorporated into DNA instead of dTMP (which contains thymine). Uracil (which normally appears in RNA) can then be incorporated into DNA, because uracil can pair with adenine, unlike thymine (which normally pairs with adenine). Massive incorporation of uracil into DNA(4million per cell). Consequences of Uracil Misincorporation: This uracil misincorporation leads to DNA instability. The presence of uracil in the DNA can result in mutations or the activation of repair mechanisms, which may cause strand breaks or chromosomal abnormalities.

References:

https://pmc.ncbi.nlm.nih.gov/articles/PMC20362/#:~:text=Folate%20deficiency%20causes%20massive%20incorporation,into%20DNA%20by%20DNA%20polymerase.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q56: What is the pattern of inheritance for hemophilia A? (Q856955)

A. X linked recessive

B. X linked Dominant

C. Autosomal dominant

D. Autosomal recessive

Correct Answer: A. X linked recessive

Explanation: Hemophilia A is X linked recessive inheritance. Definition Hemophilia A is a genetic bleeding disorder that affects the blood's ability to clot properly due to a deficiency of clotting factor VIII. Inheritance Hemophilia A is an X-linked recessive disorder, which means it primarily affects males. Females can be carriers of the condition. Symptoms Excessive or prolonged bleeding after injuries or surgeries. Frequent nosebleeds. Easy bruising. Bleeding into joints, leading to pain and swelling. Diagnosis Blood tests to measure the level of clotting factor VIII. Genetic testing to identify the specific mutation. Treatment Replacement therapy: Regular infusion of clotting factor VIll concentrates. Desmopressin DDAVP): Stimulates the release of stored clotting factor VIII. Gene therapy: Emerging treatment option to introduce the missing clotting factor gene. Complications Joint damage: Repeated bleeding into the joints can cause permanent damage and limited mobility. Intracranial bleeding: Severe cases may result in bleeding inside the skull, which can be life-threatening.

References:

Hemophilia A

↑ *Back to this question in Part A*

@Neet_pg_bot

Q57: Which of the following enzymes is deficient in the autosomal recessive type of severe combined immunodeficiency? (Q856971)

A. Ornithine transcarbamylase

B. Phosphomannose isomerase

C. Adenosine deaminase

D. Hypoxanthine-guanine phosphoribosyl transferase

Correct Answer: C. Adenosine deaminase

Explanation: ADA deficiency cause Severe Combined Immunodeficiency (SCID) SCID is a group of genetic disorders characterized by profound immunodeficiency due to defective T-cell and B-cell function. Autosomal recessive SCID is commonly caused by a deficiency in adenosine deaminase (ADA), accounting for about 15-20% of SCID cases. Adenosine Deaminase (ADA) Function: ADA is an enzyme in the purine salvage pathway. Converts adenosine and deoxyadenosine into inosine and deoxyinosine. Important for maintaining the balance of nucleotide pools in cells. Pathophysiology of ADA Deficiency : Deficiency of ADA → Accumulation of deoxyadenosine and dATP. Deoxyadenosine toxicity Directly toxic to developing T-cells and B-cells. Inhibits ribonucleotide reductase, blocking DNA synthesis and cell division. Lymphocyte apoptosis: Lymphocyte precursors (especially T-cells) undergo apoptosis due to DNA damage and lack of nucleotide availability. Clinical

Features of ADA-SCID: Onset : Neonatal period (within the first few months of life). Severe recurrent infections: Fungal, bacterial, and viral infections. Persistent diarrhea. Failure to thrive. Chronic candidiasis. Absent lymphoid tissue: No tonsils or palpable lymph nodes. Severe lymphopenia: Low T-cells, B-cells, and NK cells (T■B■NK■ phenotype). Diagnosis: Lymphopenia on CBC. Flow cytometry: Absent T-cells, B-cells, and NK cells. Low ADA activity in leukocytes. Genetic testing: Mutation in the ADA gene. Treatment: Enzyme Replacement Therapy (ERT): Pegylated ADA (PEG-ADA). Hematopoietic Stem Cell Transplantation (HSCT).

References:

SCID

↑ *Back to this question in Part A*

@Neet_pg_bot

Q58: Which of the following molecular analysis methods is used to detect RNA? (Q855892)

A. Sanger’s method

B. G banding

C. Western blot

D. RT-PCR

Correct Answer: D. RT-PCR

Explanation: Reverse transcriptase PCR is used to study RNA as well as DNA. It is a quick, automated, and highly sensitive method to study RNA and also detect mutations. RNA with the help of reverse transcriptase enzyme forms complementary DNA (cDNA). This cDNA is amplified using the polymerase enzyme so that multiple copies can be made and studied. Sanger's method (Option A) is a DNA sequencing technique also called the dideoxy nucleotide method. It is the process of selective incorporation of chain-terminating dideoxynucleotide by DNA polymerase during in vitro DNA replication. G banding (Option B) is the karyotype technique best for detecting monosomy and trisomy. With this technique, Cells are arrested in the metaphase stage using colchicine. In metaphase, all the chromosomes lie at the equator so that they can be easily analyzed under the microscope for any change in their number Chromosomes are stained with Giemsa stain to observe a G -banding. Western blot (Option C) is the antigen-antibody reaction (immunoblotting), used for the detection of protein antigen .

References:

Harper biochemistry ed 32, pg 460

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5087796/

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3883366/

Table 5https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3883366/

↑ *Back to this question in Part A*

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Q59: Which of the following is the best test for quantitative analysis of an RNA sample? PCR: polymerase chain reaction RT PCR: Reverse transcriptase polymerase chain reaction (Q855940)

A. Real-time PCR

B. Reverse transcriptase PCR

C. PCR-restriction fragment length polymorphism

D. RTPCR followed by real-time PCR

Correct Answer: D. RTPCR followed by real-time PCR

Explanation: RT-PCR is used to convert RNA into cDNA, and then real-time PCR is performed to quantify the cDNA. Hence, this approach can be suitable for quantitative analysis of RNA. Real-time PCR (Option A) can analyze DNA as it is getting synthesized, enabling us to know if there is a problem in the amplification process. Reverse transcriptase PCR (Option B) is a technique used to amplify RNA by first converting it into complementary DNA (cDNA) using reverse transcriptase enzyme. While RT-PCR is useful for detecting and amplifying RNA, it does not provide direct quantitative analysis. Additional steps, such as gel electrophoresis or hybridization, would be required for quantification. PCR-restriction fragment length polymorphism (PCR-RFLP) (Option C) is a technique used to detect genetic variations by analyzing the length differences in PCR-amplified DNA fragments after digestion with specific restriction enzymes.

References:

Harper Biochemistry, 32 Edition, Page 460

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5087796/

Vasudevan Biochemistry, 7th Edition Page 637-638

↑ *Back to this question in Part A*

@Neet_pg_bot

Q60: Which of the following statements are true regarding mitochondrial DNA? Higher rate of mutations as compared to the nuclear genome Double-stranded circular DNA with about 16,000 base pairs A slight change in nucleotide sequence as compared to cellular DNA Encodes all electron transport chain proteins (Q855945)

A. 1 and 3

B. 1 and 2

C. 2 and 4

D. 2 and 3

Correct Answer: B. 1 and 2

Explanation: The mitochondrial DNA is circular ds DNA containing about 16,000 base pairs and 37 genes. Mitochondrial DNA has a high rate of mutations as compared to nuclear DNA due to no introns (introns prevent mutation). There is no DNA repair mechanism in mitochondria. Thus, 1 and 2 are correct. Mitochondria DNA: 1. Circular ds DNA contains around 16,000 bp, 37 genes (1% of total cellular DNA) 2. ETC requires 67 proteins. 13/67 = 19% of proteins are derived from mitochondrial DNA 3. Mitochondrial DNA is present only in eukaryotes and resembles prokaryotic DNA 4. mtDNA has a high rate of mutation as compared to nuclear DNA due to no introns (introns prevent mutation),No DNA repair enzyme in mitochondria and high OFR production from ETC operation 5. Mitochondria have their own ribosomes to synthesize their proteins

References:

Vasudevan biochemistry, 7th Edition, Page 605-606

↑ *Back to this question in Part A*

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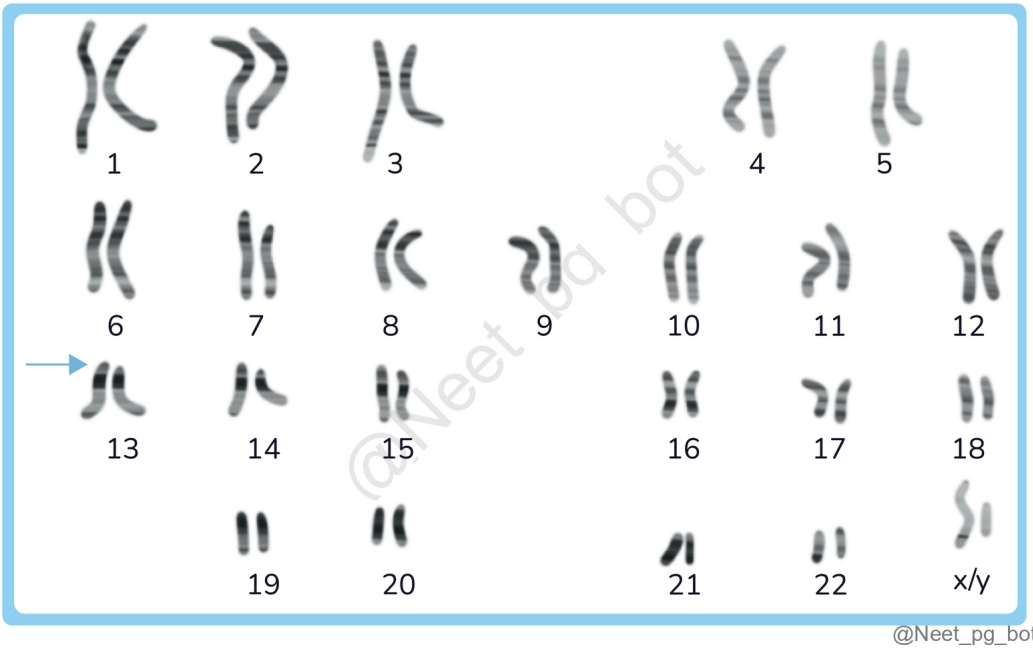
Q61: Identify the type of marked chromosome in the given karyotype. (Q856566)

A. Acrocentric

B. Submetacentric

C. Metacentric

D. Telocentric



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Correct Answer: A. Acrocentric

Explanation: An acrocentric chromosome has the centromere located near one end, resulting in a long arm and a very short (or almost nonexistent) short arm. Human chromosomes 13, 14, 15, 21, and 22 are acrocentric. Acrocentric chromosomes are characterized by having satellites or small structures called stalks attached to their short arms, which contain multiple copies of ribosomal RNA genes. Metacentric (Option B) chromosome has the centromere located approximately in the middle, resulting in two arms of roughly equal length. Human chromosomes 1, 3, 16, 19, and 20 are metacentric. Submetacentric (Option C) chromosome has the centromere slightly off-center, resulting in one arm being longer than the other. Human chromosomes 2, 4, 5, 6, 7, 8, 9, 10, 11, 12, 17, and 18 are submetacentric Telocentric(Option D) A telocentric chromosome has the centromere at one end, resulting in a chromosome that has only one arm. Humans do not have telocentric chromosomes , but they are found in some species such as mice.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q62: How does RNAi impact gene expression? (Q856571)

A. Knock out

B. Knockdown

C. Knock up

D. Knock in

Correct Answer: B. Knockdown

Explanation: RNAi primarily functions through mRNA degradation and inhibiting protein synthesis, with applications in gene manipulation and therapy. RNA Interference (RNAi) Definition : Biological process where double-stranded RNA (dsRNA) silences specific gene expressions. Mechanisms : Degradation of messenger RNA (mRNA) molecules corresponding to the target gene. Reduction of expression levels of the target gene. Applications : Tool in genetic research and biotechnology for gene knockdown. Understanding gene function. Developing therapeutic strategies.

References:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1686061/pdf/ajhg00189-0080.pdf

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8538544/

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC309050/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q63: miRNA binds to which part of the mRNA to inhibit translation? (Q856579)

A. Gene body

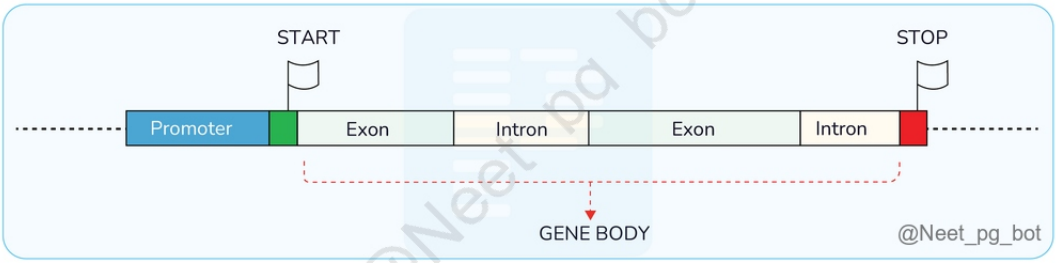
B. 3'UTR

C. Gene promoter

D. 5'UTR

Correct Answer: B. 3'UTR

Explanation: The miRNA along with the proteins forms the RNA-induced silencing complex (RISC complex) and binds to the 3’ end of the target mRNA to be degraded. Thus, miRNA is synthesized from DNA and it binds to 3’ UTR. Gene body (Option A) gene has regulatory regions such as the promoter where the enzymes bind that initiate the transcription. Furthermore, a gene has a start and a stop site, In between the start and stop site is the region that is transcribed into mRNA. This includes exons and introns. Introns and exons together are called the transcriptional region, or the gene body. Gene promoter(Option B) It is present upstream to the operator site and serves as the initiation site where RNA polymerase binds to and initiates the transcription of certain genes. 5’ UTR(Option D) is the region of a messenger RNA (mRNA) that is directly upstream from the initiation codon. This region is important for the regulation of the translation of a transcript by differing mechanisms in viruses, prokaryotes, and eukaryotes. Also known as the operator gene.



References:

Textbook of Biochemistry by DM Vasudevan, 7th edition, pg 594

↑ *Back to this question in Part A*

@Neet_pg_bot

Q64: What is the repair mechanism associated with CRISPR-cas9? (Q856605)

A. Non-homologous end joining

B. Base excision repair

C. Nucleotide excision repair

D. Mismatch repair

Correct Answer: A. Non-homologous end joining

Explanation: CRISPR-Cas is a genome-editing tool that can be used to introduce specific genetic modifications, including gene knock-ins (adding new genetic material) and gene knock-outs (disrupting or deleting existing genes). CRISPR-Cas Mechanism: Guide RNA : Directs the Cas enzyme to the specific DNA sequence. Cas Enzyme : Cuts the DNA at the target location. DNA Repair : NHEJ (Non-Homologous End Joining) (Option A) : Repairs the break by directly ligating the DNA ends, often leading to gene knockout. HDR (Homologous Directed Repair) : Uses a template to repair the break accurately, allowing for gene knock-in. Uses Can be adapted to be used in eukaryotes for purposes like Gene deletion Exogenous gene insertion Multigene editing possible Altering gene transcription and regulation Advantages: Cheap More accessible Simple Highly efficient Rapid technique Base excision repair (Option B) is a DNA repair mechanism that primarily deals with the repair of damaged or abnormal bases in the DNA molecule. Nucleotide excision repair (Option C) is a DNA repair pathway that primarily deals with the removal and repair of bulky DNA lesions, such as UV-induced thymine dimers or certain chemical adducts. Mismatch repair (Option D) is a DNA repair system that corrects errors that occur during DNA replication, such as mismatches or small insertions or deletions that can result in DNA loops.

References:

Harper’s Illustrated Biochemistry 32nd Edition - 454

↑ *Back to this question in Part A*

• [08_Lipid Metabolism] 026 Previous Year Questions

« Back to Folder Directory

[08_Lipid Metabolism] 026 Previous Year Questions — Quick Jump

Q1 (p. 1162)	*Q2 (p. 1162)*	*Q3 (p. 1163)*	*Q4 (p. 1164)*	*Q5 (p. 1164)*	*Q6 (p. 1166)*
Q7 (p. 1166)	*Q8 (p. 1168)*	*Q9 (p. 1168)*	*Q10 (p. 1169)*	*Q11 (p. 1170)*	*Q12 (p. 1171)*
Q13 (p. 1172)	*Q14 (p. 1173)*	*Q15 (p. 1174)*	*Q16 (p. 1176)*	*Q17 (p. 1176)*	*Q18 (p. 1177)*
Q19 (p. 1178)	*Q20 (p. 1178)*				

Q1: The enzyme that is not used in cholesterol synthesis is: (Q856879)

A. Thiolase

B. HMG CoA reductase

C. HMG CoA synthase

D. HMG CoA lyase

Correct Answer: D. HMG CoA lyase

Explanation: HMG CoA lyase: Cholesterol synthesis involves several key enzymes, but HMG CoA lyase is not one of them. HMG CoA lyase: This enzyme is involved in ketogenesis , where it converts HMG CoA to acetoacetate (a ketone body), and it is not involved in the cholesterol biosynthesis pathway. Enzymes involved in cholesterol synthesis: Thiolase : This enzyme catalyzes the first step in cholesterol synthesis, where two acetyl-CoA molecules are combined to form acetoacetyl-CoA. HMG CoA synthase: This enzyme catalyzes the next step, combining acetoacetyl-CoA with another acetyl-CoA to form HMG CoA (3-hydroxy-3-methylglutaryl-CoA). HMG CoA reductase : This is the rate-limiting enzyme in cholesterol biosynthesis. It converts HMG CoA to mevalonate, a crucial step in the pathway.

References:

Cholesterol synthesis

↑ *Back to this question in Part A*

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Q2: A 6-month-old infant is brought to the emergency department with lethargy, vomiting, and poor feeding. The episode occurred after prolonged fasting. Laboratory results reveal hypoglycemia, low ketone levels (hypoketosis), and mild hepatomegaly.

These findings suggest a defect in fat metabolism. Which of the following is the most likely underlying disorder? (Q866670)

A. Her’s disease

B. Medium-chain acyl-CoA dehydrogenase (MCAD) deficiency

C. Hereditary fructose intolerance

D. Glycogen storage disease type I (Von Gierke disease)

Correct Answer: B. Medium-chain acyl-CoA dehydrogenase (MCAD) deficiency

Explanation: This presentation is classic for medium-chain acyl-CoA dehydrogenase (MCAD) deficiency, a fatty acid β-oxidation defect. Prolonged fasting - Body normally switches to fatty acid oxidation for energy and ketone production. MCAD deficiency - Inability to break down medium-chain fatty acids → impaired ketogenesis and gluconeogenesis. Clinical Features: Hypoglycemia (due to no gluconeogenesis from fat-derived acetyl-CoA) Hypoketosis (unbale to synthesize ketone bodies from fatty acids) Mild hepatomegaly (fat accumulation in liver) Typical age: Symptoms often appear between 3–24 months, especially after illness or prolonged fasting. Incorrect Options: Her’s disease (GSD type VI) (Option A): Liver phosphorylase deficiency → fasting hypoglycemia + hepatomegaly, but ketones are normal because fat oxidation is intact. Hereditary fructose intolerance (Option C): Aldolase B deficiency → symptoms after fructose ingestion, not prolonged fasting; causes hypoglycemia, vomiting, jaundice. GSD type I (Von Gierke disease) (Option D): Severe fasting hypoglycemia, lactic acidosis, hyperuricemia, hyperlipidemia, but normal or high ketones .

↑ *Back to this question in Part A*

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Q3: A patient presents with elevated total cholesterol, subcutaneous xanthomas, and a positive family history of similar findings. Triglyceride levels are normal (<140 mg/dL). What is the most likely type of familial dyslipidemia? (Q866333)

A. Type I

B. Type IIa

C. Type IIb

D. Type II

Correct Answer: B. Type IIa

Explanation: The combination of elevated total cholesterol, normal triglyceride levels (<140 mg/dL), subcutaneous xanthomas, and a positive family history is characteristic of Type IIa Familial Dyslipidemia, also

known as Familial Hypercholesterolemia . Feature Type IIa (Familial Hypercholesterolemia) (Option B) Type IIb (Combined Hyperlipidemia) (Option C ruled out) Defect Defective LDL receptors or Apo B-100 ligand region mutation Increased Apo B, Apo C-II Lipoprotein Fraction Elevated LDL LDL and VLDL Main Lipid Abnormality Hypercholesterolemia Combined hyperlipidemia (elevated cholesterol and triglycerides) Plasma after 24 hours Clear Cloudy Features Atherosclerosis Coronary artery disease (early onset) Tendon xanthomas Corneal arcus. Management Statins PUFA Lifestyle modifications (diet, exercise) Statins PUFA Lifestyle modifications (diet, exercise) Incorrect Options: Type I (Option A) dyslipidemia is characterized by elevated chylomicrons, leading to severely high triglyceride levels, not elevated cholesterol. It typically presents with pancreatitis, rather than xanthomas or isolated high cholesterol. Type II (Option D) is a non-specific term that does not differentiate between Type IIa and IIb, and therefore is not adequate for precise diagnosis.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q4: A patient presents with orange-colored tonsils. Laboratory investigations reveal triglyceride level of 140 mg/dL and HDL cholesterol of 5 mg/dL. What is the most likely diagnosis? (Q866332)

A. Familial hypercholesterolemia

B. Tangier disease

C. Type I hyperlipoproteinemia

D. Abetalipoproteinemia

Correct Answer: B. Tangier disease

Explanation: The presence of orange-colored tonsils, extremely low HDL cholesterol (5 mg/dL), and normal triglyceride levels (140 mg/dL) are hallmark features of Tangier Disease , a rare inherited disorder of lipid metabolism. Incorrect Options: Familial hypercholesterolemia (Option A) is characterized by high LDL cholesterol, not low HDL, and typically presents with tendon xanthomas, not orange-colored tonsils. Type I hyperlipoproteinemia (Familial chylomicronemia) (Option C) involves markedly elevated triglyceride levels, often exceeding 1000 mg/dL, and is not associated with low HDL or orange tonsils. Abetalipoproteinemia (Option D) is marked by low or absent ApoB-containing lipoproteins such as VLDL, LDL, and chylomicrons, and presents with fat malabsorption, acanthocytosis, and neurological symptoms, but not orange-colored tonsils.

↑ *Back to this question in Part A*

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Q5: Hormone-sensitive lipase is activated by all of the following EXCEPT? (Q857872)

A. Glucagon

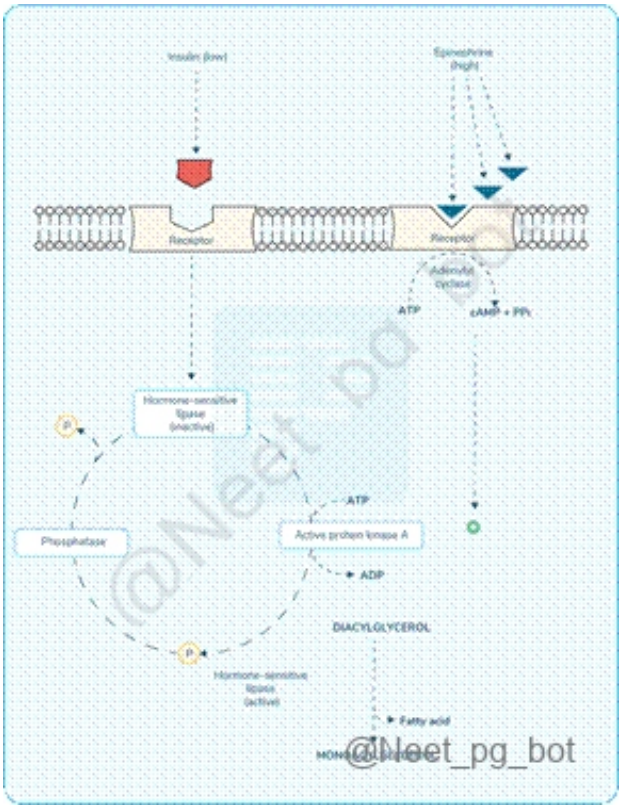
B. Epinephrine

C. ACTH

D. Insulin

Correct Answer: D. Insulin

Explanation: Insulin inhibits the activation of hormone-sensitive lipase Hormone-sensitive lipase is present in the adipose tissues which breaks down fats stored in the adipose tissue to FA + glycerol during fasting and starvation. Since it breaks fats, it is considered a catabolic enzyme. It is activated by the catabolic hormone like glucagon and inhibited by the anabolic hormone insulin. Peripheral lipolysis: It is a process in which triacylglycerols are hydrolyzed into free fatty acids (FFA) and glycerol. This process is mediated by hormone-sensitive lipase (HSL). This process occurs in adipose tissue The lipase acts on triacylglycerols and converts them into diacylglycerol , the preferred substrate for HSL. The glycerol released from the adipose tissue is transported to the liver and participates in glycolysis or gluconeogenesis. Free fatty acids bind to albumin and are transported to muscles, where they are activated into their CoA derivatives and oxidized in mitochondria for ATP. Regulation: HSL is activated when phosphorylated with Protein kinase A, which in turn is dependent on cAMP produced in the adipocyte when catecholamines activate adenylyl cyclase. Insulin dephosphorylates HSL and inactivates it.(Option D) The counter-regulatory hormones of insulin, such as glucagon (Option A), epinephrine (Option B), and cortisol, stimulate the activity of HSL. ACTH (Adrenocorticotropic Hormone) (Option C) While cortisol plays a role in metabolism, it does not directly activate hormone-sensitive lipase. Instead, cortisol can indirectly influence lipid metabolism by affecting other regulatory pathways.



References:

Lippincott Illustrated Reviews: Biochemistry 7e, pg no: 553-56

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: A 5-year-old girl is brought to the clinic by her mother due to the abnormalities in the genital area. On examination, there is the presence of pubic and axillary hair, indicating early androgen effects. Which of the following enzyme deficiencies is the least likely cause of her symptoms? (Q857557)

A. 17α-hydroxylase deficiency

B. 21-hydroxylase deficiency

C. 11β-hydroxylase deficiency

D. 3β-hydroxy steroid dehydrogenase deficiency

Correct Answer: A. 17α-hydroxylase deficiency

Explanation: 17α-hydroxylase deficiency results in low blood levels of estrogens, androgens, and cortisol. This deficiency causes a compensatory increase in adrenocorticotropic hormone (ACTH), which stimulates the production of 11-deoxycorticosterone and corticosterone. Elevated levels of these mineralocorticoids lead to volume expansion, hypertension, and hypokalemia. Clinically, females present with delayed puberty and primary amenorrhea, often with hypertension. In females, there may be hyperandrogenism or signs of precocious puberty . Treatment involves glucocorticoid replacement (like hydrocortisone) to suppress excess ACTH and normalize hormone levels. In addition, sex steroid hormone therapy (estrogen and progesterone) is used to induce secondary sexual characteristics and regulate menstrual cycles. Variants of congenital adrenal hyperplasia (CAH): Ambiguous genitalia in female Under virilization in Males With salt wasting & Hyperkalemia 21-hydroxylase deficiency (Option B ruled out) 3β-hydroxysteroid dehydrogenase deficiency (Option D ruled out) With Hypertension 11β-hydroxylase deficiency (Option C ruled out) 17α-hydroxylase deficiency (Option A)

References:

CAH

↑ *Back to this question in Part A*

@Neet_pg_bot

Q7: Enzyme deficient in Gaucher disease : (Q857482)

A. Betagalacto cerebrosidase

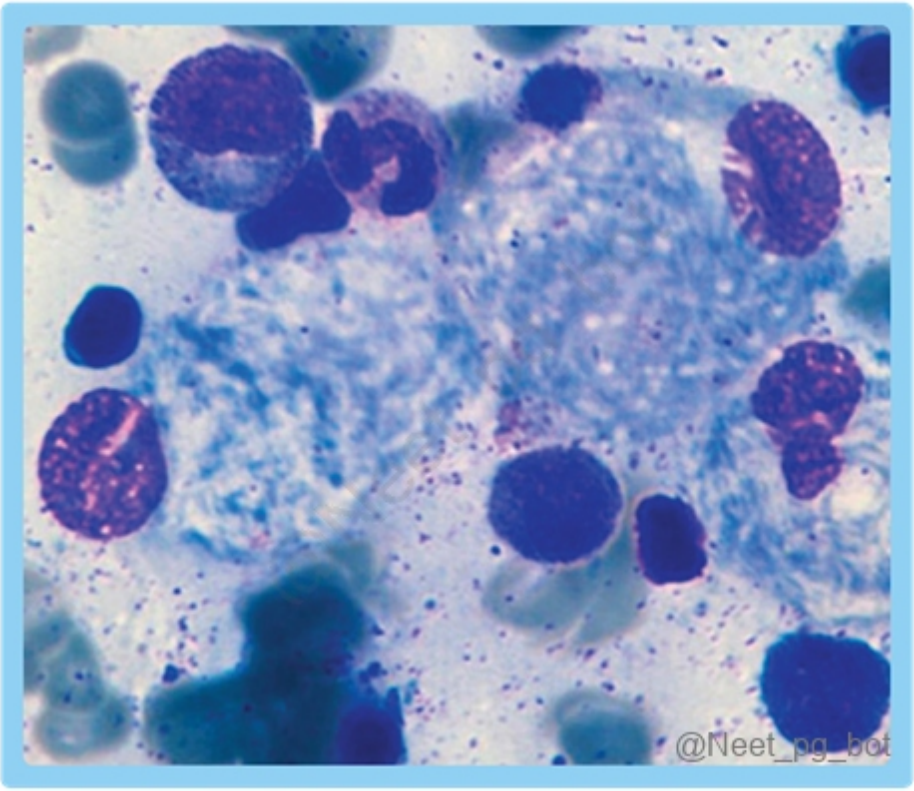
B. Glucose-6-phosphate

C. Betaglucocerebrosidase

D. Alpha glucocerebrosidase

Correct Answer: C. Betaglucocerebrosidase

Explanation: Gaucher disease , caused by a defect in beta- glucosidase (glucocerebrosidase), leads to the accumulation of glucosylceramide (glucocerebroside) within macrophages. Lysosomal storage disorder of autosomal recessive inheritance. These macrophages, engorged with glucosylceramide, are known as Gaucher cells. The accumulation affects the bone marrow and spleen, leading to anaemia and thrombocytopenia. Gaucher cells have characteristic appearance of crumbled tissue paper look due to accumulation of glucosylceramide in the cytoplasm of macrophages, forming fibril like structures. {{caption_text}}



References:

Gaucher disease

↑ *Back to this question in Part A*

@Neet_pg_bot

Q8: What is the primary source of cholesterol synthesis? (Q856980)

A. Malonyl-CoA

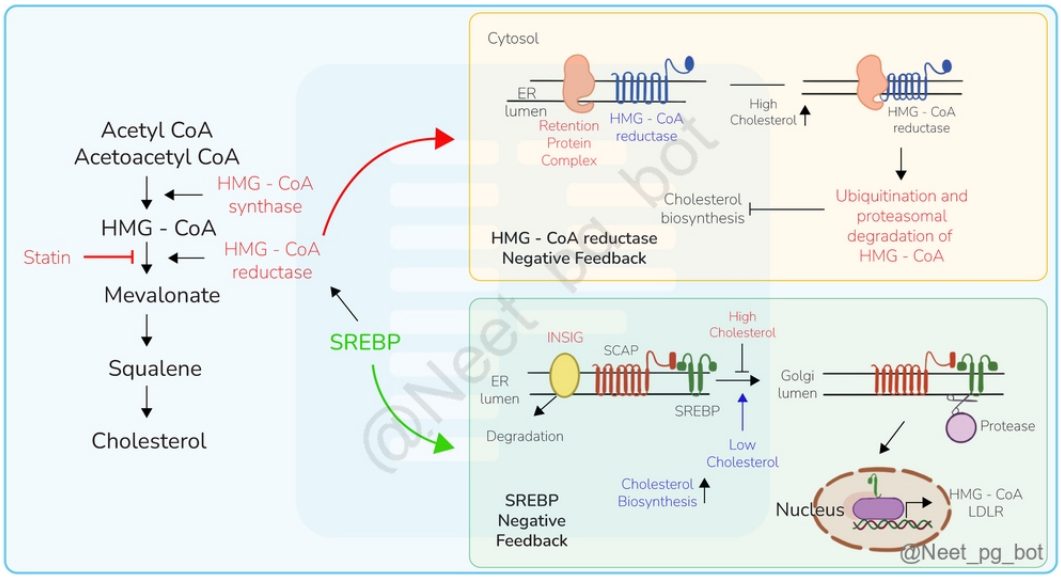
B. Pyruvate

C. Oxaloacetate

D. Acetyl-CoA

Correct Answer: D. Acetyl-CoA

Explanation: Cholesterol is synthesized from acetyl-CoA in the liver through a multi-step process called the mevalonate pathway. Acetyl-CoA → HMG-CoA (via HMG-CoA synthase) HMG-CoA → Mevalonate (via HMG-CoA reductase, the rate-limiting step) Mevalonate is further converted into isoprenoid units, which eventually lead to cholesterol formation. Malonyl CoA (Option A): Involved in fatty acid synthesis. Pyruvate (Option B): Amino acid, not involved in cholesterol synthesis. Oxaloacetate (Option C): Part of the TCA cycle, not cholesterol synthesis.



References:

Cholestrol synthesis

↑ *Back to this question in Part A*

@Neet_pg_bot

Q9: Which of the cycles listed below is absent in muscle tissue? (Q856932)

A. Glycogenolysis

B. Ketogenesis

C. Glycogenesis

D. Lipolysis

Correct Answer: B. Ketogenesis

Explanation: Ketogenesis is absent in muscle tissue because: It occurs exclusively in the liver (mitochondria of hepatocytes). The liver has the necessary enzyme HMG-CoA synthase, which is absent in muscle. Purpose of Ketogenesis: Converts acetyl-CoA into ketone bodies (acetoacetate, β-hydroxybutyrate). Ketone bodies are then released into the blood for use by peripheral tissues, including muscle, during fasting or low glucose states. Glycogenolysis (Option A): Muscle breaks down glycogen for its own energy needs during exercise. Glycogenesis (Option C): Muscle stores glycogen for later energy use. Lipolysis (Option D): Muscle can break down triglycerides to obtain fatty acids for energy.

References:

Ketogenesis

↑ *Back to this question in Part A*

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Q10: What is the primary source of reducing equivalents for liver fatty acid synthesis? (Q856924)

A. Hexose monophosphate shunt pathway

B. Glycolysis

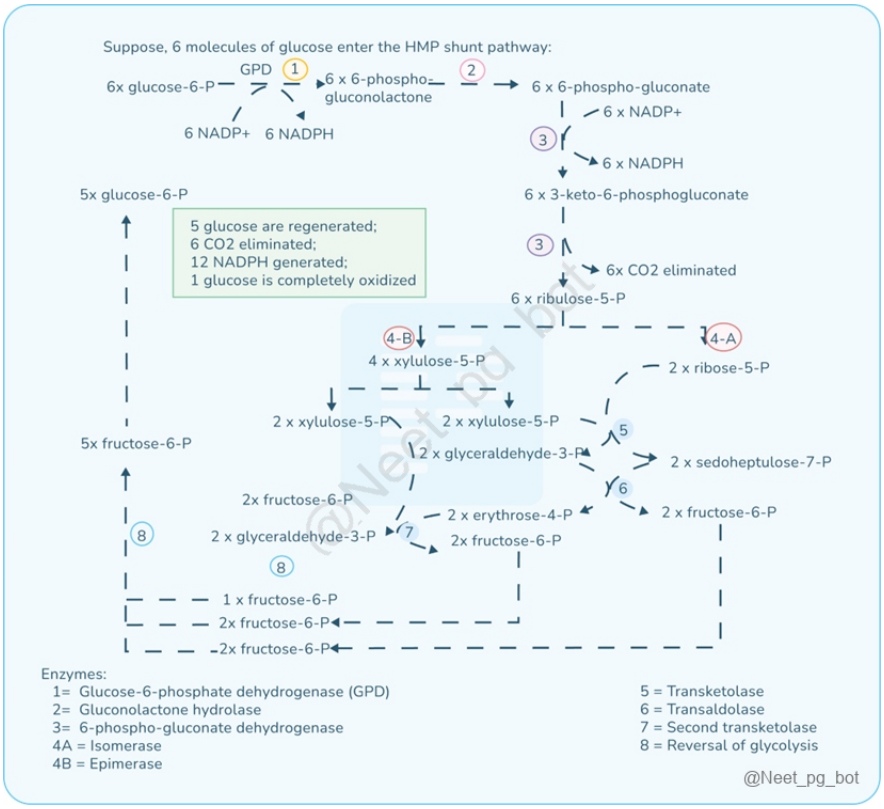
C. Tricarboxylic acid cycle

D. Uronic acid pathway

Correct Answer: A. Hexose monophosphate shunt pathway

Explanation: The primary source of reducing equivalents (NADPH) for fatty acid synthesis in the liver is the Hexose Monophosphate Shunt (HMP), also called the Pentose Phosphate Pathway (PPP). Fatty acid synthesis involves the conversion of acetyl-CoA into palmitate. This process requires reducing equivalents (NADPH) for the reduction reactions catalyzed by fatty acid synthase. NADPH provides the electrons needed for the reduction of carbonyl groups during fatty acid elongation. HMP Shunt Provides NADPH: Occurs in the cytosol.

Glucose-6-phosphate enters the pathway. Key enzyme: Glucose-6-phosphate dehydrogenase (G6PD). Produces 2 NADPH per glucose molecule oxidized. Ribose-5-phosphate (for nucleotide synthesis) is another product.



References:

HMP pathway

↑ *Back to this question in Part A*

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Q11: Which gene defect causes familial hypercholesterolemia? (Q839947)

A. Apo E

B. Apo C II

C. LDL receptor

D. Apo B48

Correct Answer: C. LDL receptor

Explanation: Correct Option C - LDL receptor: Familial hypercholesterolemia is most commonly caused by mutations in the gene coding for the LDL receptor. This receptor is responsible for the uptake of LDL cholesterol from the bloodstream into the liver. Defects in this receptor lead to high levels of LDL cholesterol in the blood. Incorrect Options: Option A - Apo E: Mutations in the Apo E gene are associated with different lipid disorders, such as familial dysbetalipoproteinemia, but not typically familial hypercholesterolemia. Option B - Apo C II: Apo C II is a cofactor for lipoprotein lipase, and defects in this gene are linked to hypertriglyceridemia, not hypercholesterolemia. Option D - Apo B48: Apo B48 is a form of apolipoprotein B that is involved in chylomicron formation and is not related to familial hypercholesterolemia. The defect in familial hypercholesterolemia involves Apo B100, not Apo B48.

↑ *Back to this question in Part A*

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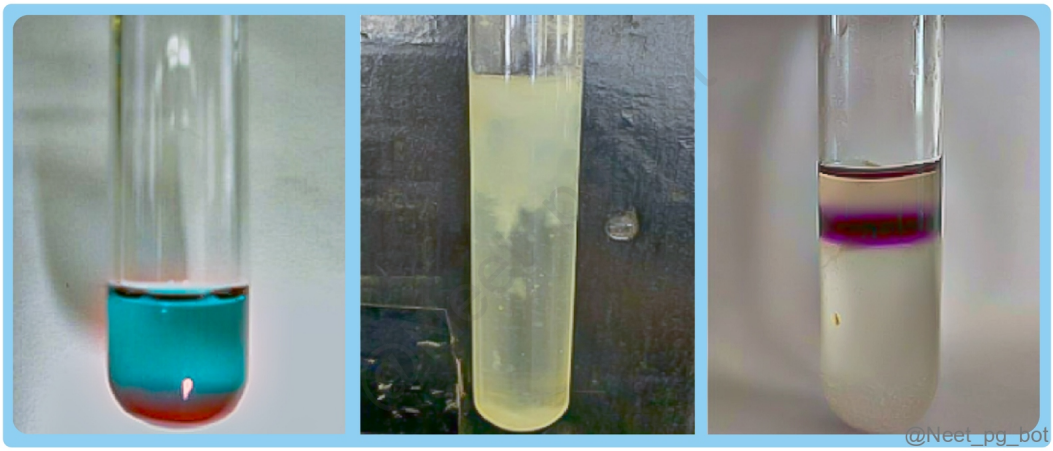
Q12: Which of the following tests will be positive in the urine of a patient who has undergone prolonged starvation? (Q855964)

A. 1 and 3

B. Only 3

C. 1, 2 and 3

D. 1 and 2



Correct Answer: B. Only 3

Explanation: Rothera's test is done for the detection of ketone bodies which are produced as a result of starvation/fasting . The purple color ring at the junction of 2 liquids is a positive test due to acetoacetate and acetone Benedict test (Image 1) : Used for the detection of reducing sugars in a complex Simple carbohydrates containing a free ketone or aldehyde functional group can be identified with this test. The test is based on Benedict’s reagent (also known as Benedict’s solution) When exposed to reducing sugars, the reactions undergone by Benedict’s reagent result in the formation of a brick-red precipitate, which indicates a positive Benedict test.

Heat coagulation test (Image 2) is done for the detection of proteins in a sample

References:

Vasudevan Biochemistry, 7th Edition, Page 166, 365

↑ *Back to this question in Part A*

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Q13: Insulin inhibits which of the following lipase enzymes? (Q855896)

A. Lipoprotein lipase

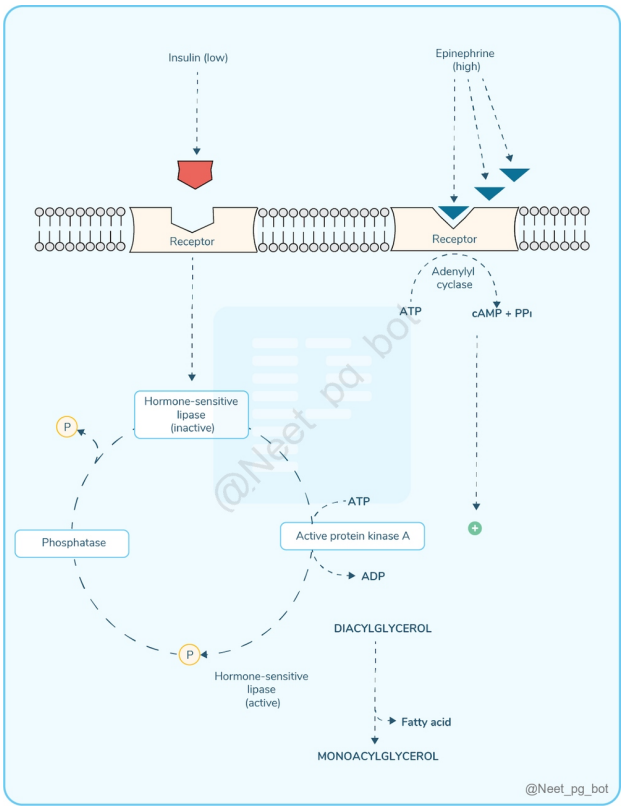
B. Pancreatic lipase

C. Gastric lipase

D. Hormone sensitive lipase

Correct Answer: D. Hormone sensitive lipase

Explanation: Hormone-sensitive lipase is present in the adipose tissues which breaks down fats stored in the adipose tissue to FA + glycerol during fasting and starvation. Since it breaks fats, it is considered a catabolic enzyme. It is activated by the catabolic hormone like glucagon and inhibited by the anabolic hormone insulin. Peripheral lipolysis: It is a process in which triacylglycerols are hydrolyzed into free fatty acids (FFA) and glycerol. This process is mediated by hormone-sensitive lipase (HSL). This process occurs in adipose tissue The lipase acts on triacylglycerols and converts them into diacylglycerol , the preferred substrate for HSL. The glycerol released from the adipose tissue is transported to the liver and participates in glycolysis or gluconeogenesis. Free fatty acids bind to albumin and are transported to muscles, where they are activated into their CoA derivatives and oxidized in mitochondria for ATP. Regulation: HSL is activated when phosphorylated with Protein kinase A, which in turn is dependent on cAMP produced in the adipocyte when catecholamines activate adenylyl cyclase. Insulin dephosphorylates HSL and inactivates it. The counter-regulatory hormones of insulin, such as glucagon, epinephrine, and cortisol, stimulate the activity of HSL. Lipoprotein lipase (Option A) is present in the endothelium of blood vessels. It breaks down fats in the blood to FA+ glycerol, which gets absorbed into adipose tissue and gets stored as triglycerides. It is considered an anabolic enzyme. It is activated by insulin and inhibited by glucagon. Pancreatic lipase (Option B) is a primary lipase enzyme that breaks down triglyceride substrates to monoglyceride + free fatty acid for absorption and storage. It is activated by insulin. Gastric lipase (Option C) is present in gastric juice where fats are broken down to triglycerides for absorption and storage. It is activated by the anabolic hormone insulin.



References:

Lippincott Illustrated Reviews: Biochemistry 7th Edition, Page no: 553-56

↑ *Back to this question in Part A*

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Q14: A patient presented with reduced levels of high-density lipoprotein and an ABCA1 mutation. On examination, tonsils appeared as shown in the image. What is the diagnosis? (Q855549)

A. Tangier's disease

B. Abetalipoproteinemia

C. Hyperlipoproteinemia

D. Wolman's disease



Correct Answer: A. Tangier's disease

Explanation: Tangier disease is a rare, autosomal recessive disorder caused by mutations in the ABCA1 (ATP-binding cassette, sub-family A, member 1) gene. Pathophysiology: The ABCA1 protein is crucial for the efflux of cholesterol and phospholipids to apolipoprotein A-I (ApoA-I), forming HDL particles. Mutations in the ABCA1 gene impair this process, leading to a shortage of HDL and cholesterol accumulation in tissues such as the tonsils, liver, spleen, and peripheral nerves. Clinical Features: Greyish-Orange Tonsils: (Hallmark feature) Tonsils, become enlarged and acquire greyish-orange hue due to the accumulation of cholesterol esters Hepatosplenomegaly: Due to cholesterol and phospholipid accumulation. Peripheral Neuropathy: Multiple nerves can be individually affected due to cholesterol deposition in peripheral nerves, leading to peripheral neuropathy.

References:

Reference:Vasudevan Textbook of Biochemistry, 7th Edition, Page 342.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q15: What is the enzyme defect if a child with anemia, thrombocytopenia, bony pain and hepatosplenomegaly shows a "crumpled tissue paper" appearance on light microscopy of bone marrow aspirate? (Q855548)

A. Beta- glucosidase

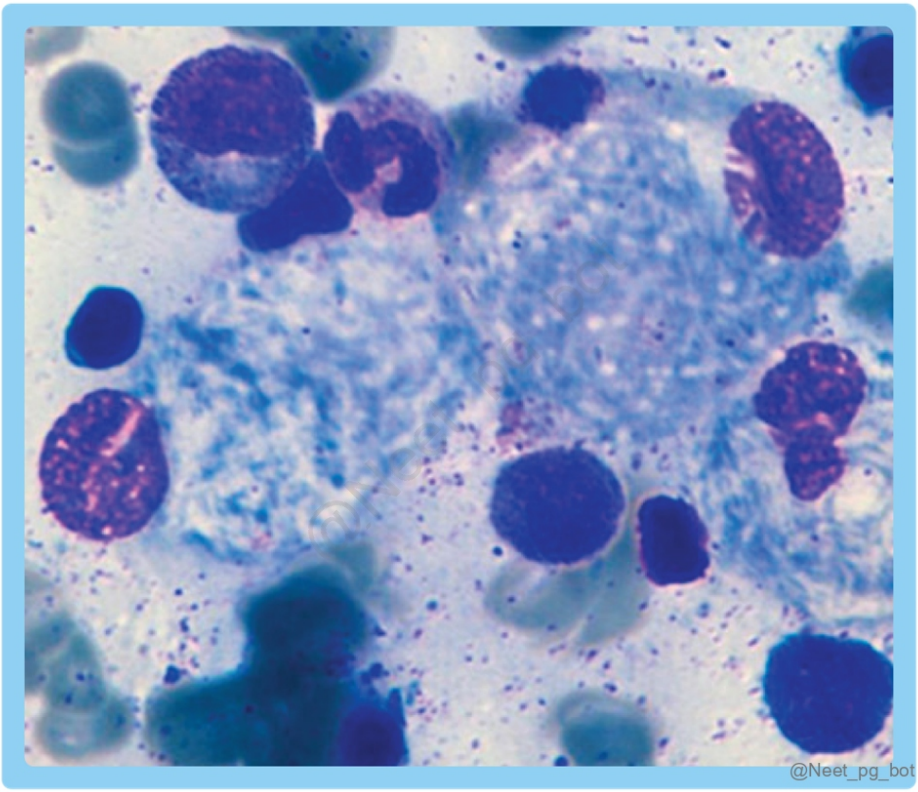
B. Sphingomyelinase

C. Hexosaminidase

D. Glucose6-phosphatase

Correct Answer: A. Beta- glucosidase

Explanation: Gaucher disease , caused by a defect in beta- glucosidase (glucocerebrosidase), leads to the accumulation of glucosylceramide (glucocerebroside) within macrophages. Lysosomal storage disorder of autosomal recessive inheritance. These macrophages, engorged with glucosylceramide, are known as Gaucher cells. The accumulation affects the bone marrow and spleen, leading to anaemia and thrombocytopenia. Gaucher cells have characteristic appearance of crumbled tissue paper look due to accumulation of glucosylceramide in the cytoplasm of macrophages, forming fibril like structures. {{caption_text}} Sphingomyelinase (Option B): Niemann-Pick disease results from a deficiency in sphingomyelinase.Its bone marrow findings involve foamy histiocytes with lipid-filled cytoplasm. Hexosaminidase (Option C): Tay-Sachs disease, characterized by hexosaminidase deficiency, doesn't typically cause anemia, thrombocytopenia, or bone pain.Its bone marrow findings may show an onion-skin appearance. Glucose 6-phosphatase (Option D): Von Gierke disease, due to glucose-phosphatase deficiency, may lead to hepatosplenomegaly and bone pain but not anemia or thrombocytopenia.



References:

Gaucher disease

↑ *Back to this question in Part A*

@Neet_pg_bot

Q16: Which of the options aids in the transportation of fatty acids through the inner mitochondria membrane? (Q855530)

A. Acyl carrier protein

B. Carnitine

C. Lecithin-Cholesterol acyltransferase

D. Albumin

Correct Answer: B. Carnitine

Explanation: The carnitine shuttle system transports long-chain fatty acids into the mitochondrial matrix for β-oxidation. It involves: Carnitine palmitoyltransferase I (CPT-I): Converts fatty acyl-CoA to fatty acyl-carnitine. Translocase : Moves fatty acyl-carnitine across the inner mitochondrial membrane. Carnitine palmitoyltransferase II (CPT-II): Converts fatty acyl-carnitine back to fatty acyl-CoA inside the matrix. Movement of fatty acids across inner mitochondrial membrane where they are oxidised to produce energy depends on carnitine. Acyl carrier protein (Option A): Involved in movement of fatty acids within the cytosol, but not in the movement of fatty acids across mitochondrial membrane. Lecithin-Cholesterol acyltransferase (Option C): Involved in metabolism of phospholipids and cholesterol. Albumin (Option D): Functions as carrier and aids in the transportation of numerous chemicals in the blood, including fatty acids, but not directly.

References:

Carnitine

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: The process of apolipoprotein B48 synthesis involves which of the following? (Q854877)

A. RNA alternate splicing

B. RNA editing

C. DNA editing

D. RNA interference

Correct Answer: B. RNA editing

Explanation: Apolipoprotein B (apoB) is crucial in lipid metabolism, transporting cholesterol and triglycerides in the bloodstream. It has two isoforms: apoB100 synthesized in the liver, part of VLDL, and apoB48 synthesized in the intestine, part of chylomicrons. ApoB48 synthesis involves "A-to-I RNA editing," where a specific cytosine is converted to uridine, creating a premature stop codon and producing apoB48. ApoB48 is found in chylomicrons, mediates the transport of dietary triglycerides from the intestine to peripheral tissues. Incorrect Options: RNA alternate splicing (Option A): While alternate splicing generates different mRNA isoforms, it's not directly related to apoB48 synthesis. DNA editing (Option C): ApoB48 synthesis involves RNA editing, not DNA editing. RNA interference (Option D): RNAi regulates gene expression but does not directly impact apoB48 synthesis.

References:

RNA editing

↑ *Back to this question in Part A*

@Neet_pg_bot

Q18: A patient with multiple tendon xanthomas is found to have a serum cholesterol level of 398 mg/dL and an LDL level of 220 mg/dL. What is the most likely defect? (Q844263)

A. Lipoprotein lipase deficiency

B. LDL receptor defect

C. Defective Apo B-100

D. PCSK9 gain-of-function mutation

Correct Answer: B. LDL receptor defect

Explanation: The most likely defect in a patient with multiple tendon xanthomas, elevated serum cholesterol levels, and LDL levels is an LDL receptor defect. LDL levels in the blood remain high because the liver and other cells cannot efficiently remove LDL from the bloodstream. This leads to elevated cholesterol levels and the formation of xanthomas, resulting from the accumulation of cholesterol in tissues. LDL receptor defect One allele affected – LDL receptor defective individual (LDL receptor activity <25% of normal) Gene dose effect: Milder Both alleles affected – LDL receptor-negative individual (LDL receptor activity <8% of normal) Gene dose effect: Severe Clinical features: Eruptive xanthoma, recurrent pancreatitis, and Lipemia retinalis. LPL (Lipoprotein lipase) activity is low. Lipoprotein lipase deficiency (Option A): Causes ↑ chylomicrons and ↑ triglycerides, not predominantly LDL . Defective Apo B-100 (Option C): Also causes increased LDL but tendon xanthomas and severe elevation are more typical with receptor defects. PCSK9 gain-of-function (Option D): Can cause high LDL but is much rarer than LDL receptor defects.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q19: A patient presents to the emergency room with severe abdominal pain, and serum triglyceride levels are 1500 mg/dL. What is the most likely underlying defect? (Q844261)

A. Apo B-48 defect

B. Apo B-100 defect

C. Apo C-II defect

D. LDL receptor defect

Correct Answer: C. Apo C-II defect

Explanation: Correct Option C - Apo C-II defect: Apo C-II is a cofactor for lipoprotein lipase (LPL) , the enzyme responsible for hydrolyzing triglycerides in chylomicrons and VLDL into free fatty acids. A deficiency of Apo C-II (or LPL itself) results in familial hyperchylomicronemia (Type I hyperlipoproteinemia) , characterized by: Massive hypertriglyceridemia (>1000 mg/dL) Recurrent episodes of acute pancreatitis (due to elevated chylomicrons) Eruptive xanthomas Lipemia retinalis (milky retinal vessels) Despite very high triglycerides, atherosclerosis risk is low , as LDL and cholesterol are usually normal. Incorrect Options: A) Apo B-48 defect: Leads to impaired chylomicron formation → abetalipoproteinemia , presenting with fat malabsorption , not hypertriglyceridemia. B) Apo B-100 defect: Causes defective LDL formation → Type IIb hyperlipoproteinemia , with elevated LDL, not chylomicrons. D) LDL receptor defect: Causes familial hypercholesterolemia (Type IIa) with elevated LDL and tendon xanthomas , not pancreatitis.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q20: A patient presents with xanthomas on the Achilles tendon. Which of the following is the most likely diagnosis? (Q844257)

A. Familial hypercholesterolemia

B. Tangier disease

C. Familial hyperchylomicronemia

D. Type III hyperlipoproteinemia

Correct Answer: A. Familial hypercholesterolemia

Explanation: Correct Option A - Familial hypercholesterolemia: Familial hypercholesterolemia (Type IIa hyperlipoproteinemia) is caused by a defect in the LDL receptor or ApoB-100 , leading to markedly elevated LDL cholesterol levels . Characteristic clinical findings include: Tendon xanthomas , especially on the Achilles tendon and extensor tendons of the hands , Xanthelasma , arcus cornealis , Premature atherosclerosis , myocardial infarction, and coronary artery disease. Tangier disease (Option B): Due to ABCA1 transporter defect , causes low HDL , orange tonsils , and peripheral neuropathy — not tendon xanthomas. Familial hyperchylomicronemia (Type I) (Option C): Caused by LPL or ApoC-II deficiency , presents with eruptive xanthomas and pancreatitis , not tendon xanthomas. Type III hyperlipoproteinemia (Option D): Caused by ApoE mutation , presents with palmar xanthomas and tuberoeruptive xanthomas , not Achilles tendon involvement.

References:

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↑ *Back to this question in Part A*

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• [09_Amino Acids Metabolism] 027 Previous Year Questions

« Back to Folder Directory

[09_Amino Acids Metabolism] 027 Previous Year Questions — Quick Jump

Q1 (p. 1180)	*Q2 (p. 1181)*	*Q3 (p. 1182)*	*Q4 (p. 1183)*	*Q5 (p. 1184)*	*Q6 (p. 1185)*
Q7 (p. 1187)	*Q8 (p. 1188)*	*Q9 (p. 1189)*	*Q10 (p. 1189)*	*Q11 (p. 1191)*	*Q12 (p. 1192)*
Q13 (p. 1192)	*Q14 (p. 1193)*	*Q15 (p. 1194)*	*Q16 (p. 1194)*	*Q17 (p. 1197)*	*Q18 (p. 1197)*
Q19 (p. 1198)	*Q20 (p. 1199)*	*Q21 (p. 1200)*	*Q22 (p. 1200)*		

Q1: Ammonia in the brain is detoxified to form (Q856874)

A. Urea

B. Glutamine

C. Ammonium ions

D. Alanine

Correct Answer: B. Glutamine

Explanation: Ammonia in Brian is detoxified to form Glutamine. Glutamine synthetase in astrocytes converts glutamate and ammonia to glutamine, effectively detoxifying ammonia in the brain. Ammonia Management in Neurons: Ammonia can be neurotoxic if accumulated. Glutamine Synthesis: Produced in neurons as a byproduct of amino acid metabolism and neurotransmitter turnover. Ammonia reacts with α-ketoglutarate and NADH to form glutamate, catalyzed by glutamate dehydrogenase. α-ketoglutarate + NH 3 ■ ■+ NADH → Glutamate + NAD + + H2■O Glutamate is converted to glutamine by glutamine synthetase, adding another ammonia molecule. Glutamine synthetase is predominantly found in astrocytes. Glutamate + NH 3 ■ + ATP → Glutamine + ADP + Pi This conversion reduces ammonia toxicity by storing it in a non-toxic form. The fate of glutamine: Release into the Bloodstream: Glutamine is released into the extracellular space and enters the bloodstream. This transport helps maintain nitrogen balance and prevents ammonia accumulation. Transport to the Liver: Glutamine carries ammonia from the brain to the liver, where it is deaminated to release ammonia. Ammonia enters the urea cycle in the liver for detoxification. Importance of Glutamine in Neuronal Health: Acts as a non-toxic ammonia carrier, preventing neurotoxicity. Serves as a precursor for neurotransmitters like GABA , maintaining neurotransmitter balance.

References:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8244593/

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4133642/

https://www.sciencedirect.com/science/article/pii/S019701862030200X

↑ *Back to this question in Part A*

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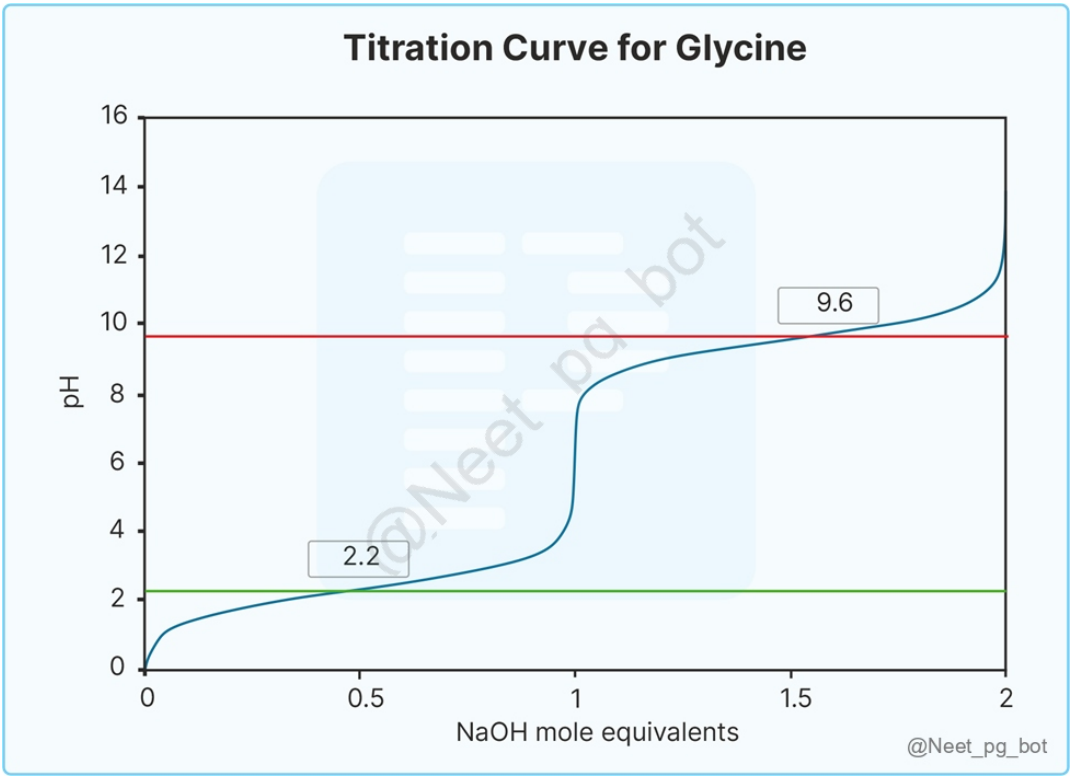
Q2: In the given glycine titration curve, calculate or identify the pH at which molecules exist in a zwitter ion form? (Q865428)

A. 4.4

B. 6

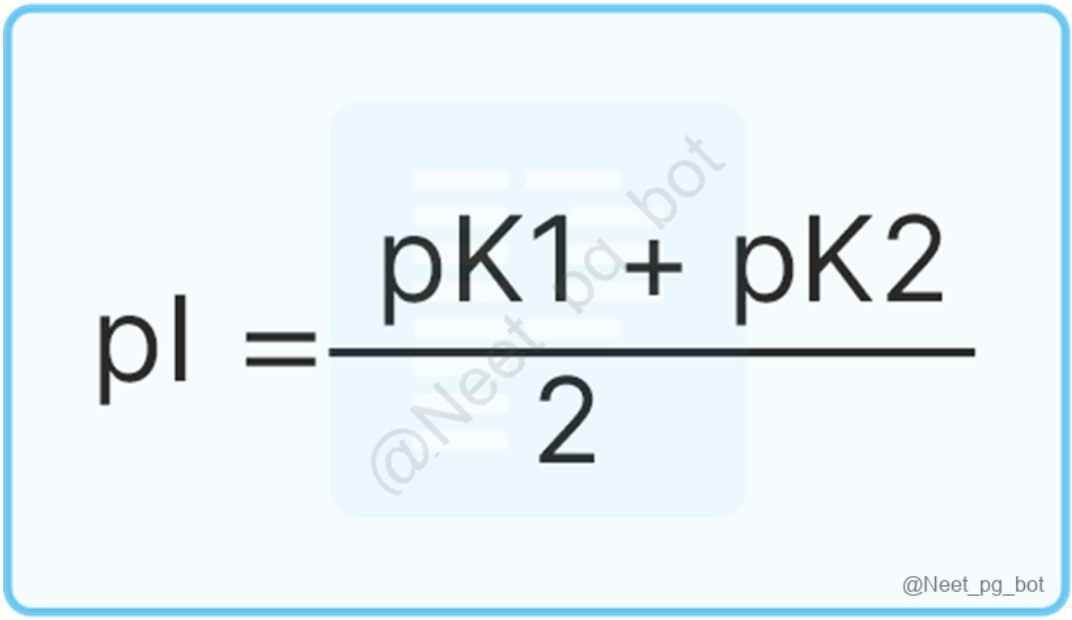
C. 9

D. 2.2



Correct Answer: B. 6

Explanation: In the given glycine titration curve, the pH at which the molecules predominantly exist in their zwitterionic form is 6. Isoelectric pH It is the pH at which a molecule carries no net electrical charge. For proteins and amino acids, this is the pH where the molecule exists as a “zwitter ion” with equal numbers of positive and negative charges, resulting in a net zero charge and no migration in an electric field. The isoelectric pH (pI) for monoamino monocarboxylic amino acids can be calculated: In the above question, it will be = (2.2 + 9.6)/2 = 5.9 ■ 6 .



References:

Vasudevan's Textbook of Biochemistry, 7th Edition, Page 28.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q3: All of the following are lipotropic factors except? (Q859698)

A. Choline

B. Lecithine

C. Arginine

D. Methionine

Correct Answer: C. Arginine

Explanation: Arginine is an amino acid involved in nitric oxide production and other functions but is not considered a lipotropic factor. Lipotropic factors Lipotropic factors are nutrients such as choline, betaine, and methionine that help improve liver function and detoxification. (Option A & D ruled out) Lecithin contains choline as its primary active ingredient and is considered a lipotropic agent. (Option B ruled out) These compounds promote the flow of fat and bile to and from the liver, aiding fat metabolism and liver health. They are used to treat liver disorders like hepatitis, cirrhosis, and chemical-induced liver disease. Lipotropic formulas may increase the liver's levels of S-adenosyl-methionine and glutathione, key compounds for liver function and detoxification. Common lipotropic agents include choline, methionine, betaine, folic acid, and vitamin B12. These formulas support detoxification, protect liver function, and enhance fat metabolism. They may also play a role in

methylation and epigenetics, influencing health at a molecular level. Carnitine , another lipotropic agent, helps prevent alcohol-induced fatty liver disease by improving fatty acid metabolism. Lipotropic formulas are also used in detoxification, especially in conditions like chronic candidiasis and liver damage. Daily recommended doses for choline and methionine are 1000 mg each . Lipotropic agents are often combined with other herbs and nutrients to support liver health and detoxification.

References:

Lipotropic Agent - an overview | ScienceDirect Topics

Lipotropic factors and oncogenesis - PubMed

https://www.healthline.com/health/lipotropic-injections

↑ *Back to this question in Part A*

@Neet_pg_bot

Q4: A patient came to the hospital with a complaint of abdominal pain. Routine investigations were unremarkable. On observation, the urine turned black on standing. Which of the following enzymes is likely to be defective in this patient? (Q858459)

A. Homogentisate oxidase

B. Xanthine oxidase

C. Phenylalanine hydroxylase

D. Dihydric orotate dehydrogenase

Correct Answer: A. Homogentisate oxidase

Explanation: The clinical presentation of black urine on standing, along with abdominal pain, points to alkaptonuria , a rare autosomal recessive metabolic disorder caused by a deficiency in the enzyme homogentisate oxidase. Alkaptonuria Homogentisate oxidase is an enzyme involved in the breakdown of the amino acids tyrosine and phenylalanine Its deficiency leads to the accumulation of homogentisic acid in the body Pathophysiology: Homogentisic acid is excreted in the urine. Upon exposure to air (oxidation), it polymerizes, causing the urine to turn black on standing Clinical Features Urine discoloration: The hallmark sign is black urine when exposed to air Abdominal pain: Likely due to deposition of homogentisic acid or associated complications like kidney stones Ochronosis : Long-term deposition of homogentisic acid in connective tissues, leading to bluish-black discoloration (typically seen in adulthood) Arthropathy : Degenerative joint disease due to ochronotic deposits in cartilage Diagnosis: Urine analysis: Elevated levels of homogentisic acid. Genetic testing: Mutations in the HGD gene (homogentisate oxidase gene) Management: Limit intake of tyrosine and phenylalanine. Vitamin C may slow ochronosis progression. Nitisinone : A newer drug that inhibits the production of homogentisic acid by blocking the enzyme upstream in the metabolic pathway Xanthine oxidase (Option B): Xanthine oxidase is an enzyme

involved in purine metabolism , converting hypoxanthine to xanthine to uric acid . Deficiency leads to xanthinuria , which presents with low uric acid levels and renal stones , but does not cause black discoloration of urine on standing . Therefore, this enzyme defect does not explain the findings in this patient. Phenylalanine hydroxylase (Option C): Phenylalanine hydroxylase converts phenylalanine to tyrosine . Deficiency causes phenylketonuria (PKU) , characterized by intellectual disability, seizures, fair skin, eczema, and a musty/mousy odor of urine due to phenylketones. Urine does not turn black on standing , making this option inconsistent with the clinical scenario. Dihydroorotate dehydrogenase (Option D): Dihydroorotate dehydrogenase is involved in de novo pyrimidine synthesis , converting dihydroorotate to orotate . Defects in pyrimidine metabolism are associated with orotic aciduria , which presents with megaloblastic anemia and developmental delay . It does not cause black urine on standing , so it does not match the presentation.

References:

https://pmc.ncbi.nlm.nih.gov/articles/PMC3978898/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q5: A 62-year-old male with CAD comes to the physician for a follow-up examination. His blood pressure is 130/80 mm Hg, height of 182 cm, weight of 85 kg, and a BMI of 25 kg/m². Serum studies show a glucose concentration of 95 mg/dL and homocysteine concentration of 19.3 μmol/L (N=5–15). Serum lipid concentrations are within the normal range. Which of the following enzyme deficiencies is most likely to be found in this patient? (Q857892)

A. Homogentisate oxidase

B. Cystathionine beta synthase

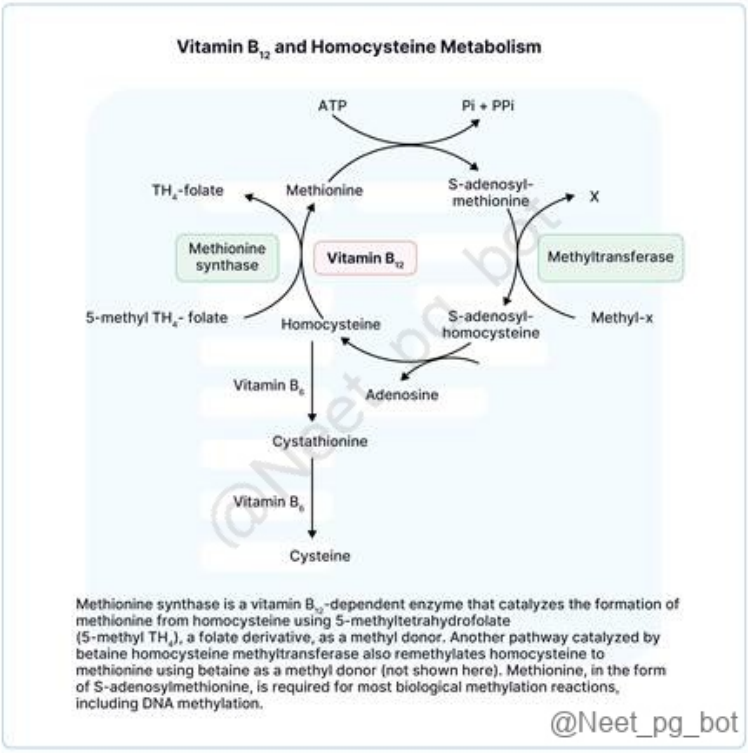
C. Cystathionine gamma synthase

D. HGPRTase

Correct Answer: B. Cystathionine beta synthase

Explanation: The clinical scenario CAD patient with high homocysteine levels is suggestive of homocystinuria caused by Cystathionine beta synthase deficiency is associated with homocystinuria. Homocystinuria: A genetic disorder characterized by elevated levels of homocysteine in the blood. The clinical manifestations of homocystinuria can include cardiovascular issues (such as early-onset atherosclerosis), skeletal abnormalities, intellectual disabilities, and other systemic complications. The patient in the question has an elevated homocysteine concentration, which is characteristic of homocystinuria. Types of Homocystinuria: Type 1 vs Type 2 Homocystinuria: Feature Type - I Homocystinuria Type - II Homocystinuria Enzyme Deficiency Cystathionine beta-synthase Methionine synthase Metabolic Pathway Affected Homocysteine to cystathionine Homocysteine to methionine Elevated Metabolites Homocysteine and methionine Homocysteine and elevated levels of methionine

Biochemical Diagnosis Low levels of cysteine. High levels of methionine. High levels of cysteine. Low levels of methionine. Treatment Vitamin B6 (pyridoxine) supplementation; low-methionine diet; cysteine supplementation Vitamin B12 and folate supplementation; low-methionine diet Homogentisate oxidase (Option A) deficiency leads to alkaptonuria, a condition characterized by the accumulation of homogentisic acid, causing darkening of urine and connective tissue. Cystathionine gamma synthase (Option C) deficiency is not a well-known enzyme deficiency associated with a specific clinical disorder. Cystathionine gamma synthase is involved in the transsulfuration pathway, converting cystathionine to cysteine. HGPRTase (Hypoxanthine-guanine phosphoribosyltransferase) (Option D) deficiency is seen in Lesch-Nyhan syndrome, which is characterized by neurological and behavioral abnormalities, along with uric acid overproduction leading to gout.



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg. 544

Vasudevan 7th edition , pg no 454,477,478,479,482,483,486

Adenosylcobalamin

Methylcobalamin

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: Which enzyme is likely to be deficient in a 4-year-old child who presents with a habit of biting their lips and fingers, and has a missing portion of fingers on their left hand? Further evaluation shows delayed developmental milestones. The parents report that two male cousins on the mother's side have similar symptoms and have been diagnosed with a genetic disorder. Laboratory reports indicate hyperuricemia. (Q857544)

A. Purine nucleoside phosphorylase

B. Hypoxanthine -guanine phosphoribosyl transferase.

C. Adenine phosphoribosyl transferase

D. Adenosine deaminase

Correct Answer: B. Hypoxanthine -guanine phosphoribosyl transferase.

Explanation: Lesch-Nyhan Syndrome (X-linked recessive disorder) Enzyme deficiency: HGPRT deficiency impairs the purine salvage pathway. Excess uric acid production (hyperuricemia) due to increased purine breakdown. Neurological symptoms due to the lack of purine recycling, affecting brain development. Key symptoms: Self-mutilation behavior (lip and finger biting) ■■■■ ■■ ■■ ■■ ■■ ■■ ■ ■ ■ ■ ■■ ■■ ■■ ■■ {{caption_text}} Developmental delay Hyperuricemia → Gout, nephrolithiasis X-linked inheritance: Symptoms in males, family history consistent with maternal transmission. Purine nucleoside phosphorylase (Option A): Inherited in an autosomal recessive pattern. Adenine phosphoribosyl transferase (Option C): Deficiency is characterised by excessive production and renal excretion of 2,8-dihydroxyadenine (DHA), which leads to kidney stone formation and crystal-induced kidney damage (DHA crystal nephropathy). Adenosine deaminase (Option D): Deficiency damages the immune system and causes severe combined immunodeficiency (SCID).



References:

Lesch-Nyhan syndrome

↑ *Back to this question in Part A*

@Neet_pg_bot

Q7: Which of the following does not contribute to the repair of DNA double-stranded breaks? (Q857361)

A. ATP

B. RAD-51

C. ATM

D. Ku 70

Correct Answer: A. ATP

Explanation: ATP is not directly involved in the repair of DNA double-stranded breaks (DSBs), but it provides the energy necessary for many cellular processes, including DNA repair. Homologous DNA Repair (HR) HR is a type

of Double-strand DNA break repair mechanism used to fix double-strand breaks (DSBs) by utilizing a homologous DNA template for error-free repair. HR is active during the S and G2 phases of the cell cycle. RAD 51 and ATM are involved in the G2 phase Nonhomologous End Joining (NHEJ) NHEJ is a DNA repair mechanism that fixes double-strand breaks (DSBs) by directly joining the broken ends without requiring a homologous template. Active throughout the entire cell cycle, including G0/G1 phases. Ku70 is involved in the G1 phase

References:

Harper 32nd edition, pg 380

Lippincott 7th edition, 1219-1222

Holliday junction

↑ *Back to this question in Part A*

@Neet_pg_bot

Q8: Which of the following is considered a universal methyl group donor in various biochemical reactions? (Q856961)

A. Glucose-6-phosphate

B. Acetyl CoA

C. S-Adenosyl Methionine

D. S-Adenosyl Serine

Correct Answer: C. S-Adenosyl Methionine

Explanation: S-Adenosyl Methionine (SAM) is the universal methyl group donor in various biochemical reactions. Key Features of SAM: Formed from methionine and ATP. Transfers methyl groups (-CH■) to a variety of molecules, including: DNA (methylation) Neurotransmitters (e.g., epinephrine synthesis) Phospholipids After donating its methyl group, it becomes S-adenosyl homocysteine (SAH), which can be recycled back to methionine via the methionine cycle. Glucose-6-phosphate (Option A): Involved in glycolysis and the HMP shunt, not methylation. Acetyl CoA (Option B): Donates acetyl groups (not methyl) in pathways like the TCA cycle and fatty acid synthesis. S-Adenosyl Serine (Option D): No significant role as a methyl donor.

References:

Methyl donor

↑ *Back to this question in Part A*

@Neet_pg_bot

Q9: What is the enzyme that is responsible for the defect causing Maple syrup urine disease? (Q856942)

A. Branched-chain a-ketoacid dehydrogenase

B. Isovaleryl CoA dehydrogenase

C. Pyruvate carboxylase

D. Acetyl CoA carboxylase

Correct Answer: A. Branched-chain a-ketoacid dehydrogenase

Explanation: Maple syrup urine disease (MSUD) is caused by a deficiency of the branched-chain α-ketoacid dehydrogenase (BCKD) complex , which is required for the oxidative decarboxylation of branched-chain amino acids (leucine, isoleucine, valine). Its deficiency leads to accumulation of these amino acids → sweet “maple syrup” odor in urine and severe neurotoxicity. Maple Syrup Urine Disease (MSUD): Autosomal recessive metabolic disorder caused by a deficiency of the enzyme branched-chain α-ketoacid dehydrogenase (BCKD) , Responsible for the breakdown of branched-chain amino acids (BCAAs): Leucine Isoleucine Valine Enzyme Function and Pathophysiology: BCKD is a mitochondrial multienzyme complex that catalyzes the oxidative decarboxylation of branched-chain α-keto acids. These α-keto acids are derived from the transamination of BCAAs. Deficiency Results In: Accumulation of BCAAs and their α-keto acid derivatives (e.g., α-ketoisocaproic acid). Toxic metabolites lead to severe neurological dysfunction due to their impact on brain metabolism. Clinical Features: Neonatal onset : Usually presents within the first few days of life. Poor feeding Vomiting Lethargy Maple syrup odor of urine (due to isoleucine metabolite sotolone) Seizures Neurological damage: Due to leucine accumulation affecting brain function. Treatment : Dietary restriction of BCAAs (low protein diet). Special medical formulas without leucine, isoleucine, and valine. Thiamine supplementation: As BCKD is a thiamine-dependent enzyme. Isovaleryl-CoA dehydrogenase (Option B): Defective in isovaleric acidemia. Pyruvate carboxylase (Option C): Defect causes lactic acidosis, not MSUD. Acetyl-CoA carboxylase(Option D): Involved in fatty acid synthesis, unrelated to BCAA metabolism.

References:

MSUD

↑ *Back to this question in Part A*

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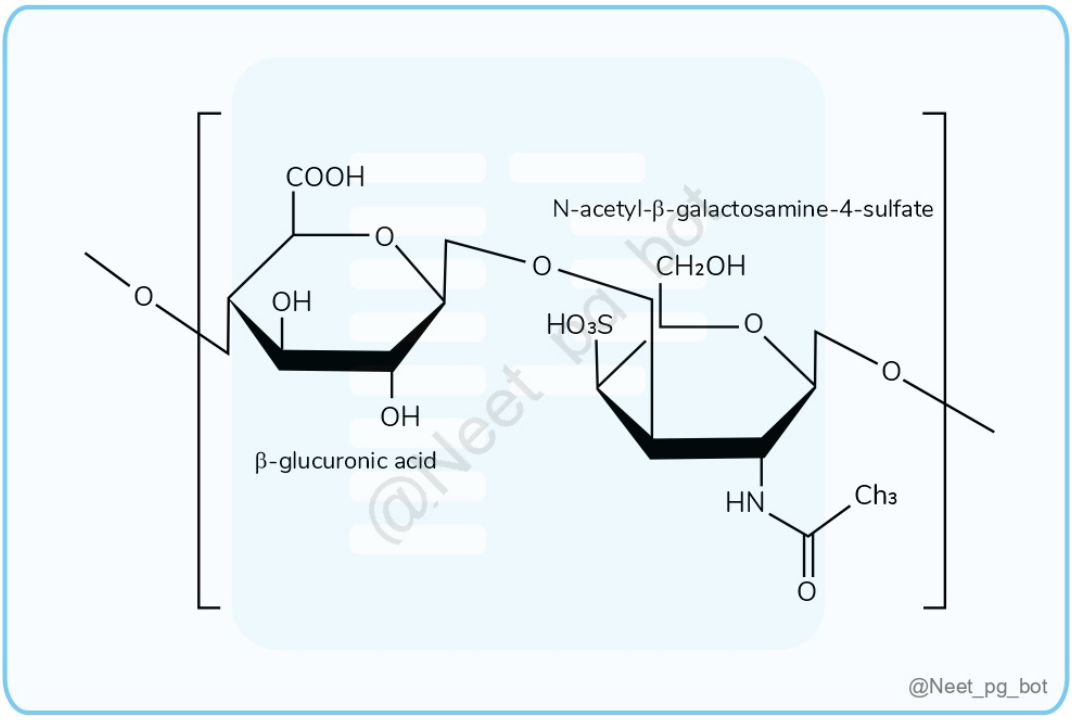
Q10: The given chemical structure is of: (Q856923)

A. Keratan sulfate

B. Chondroitin sulfate

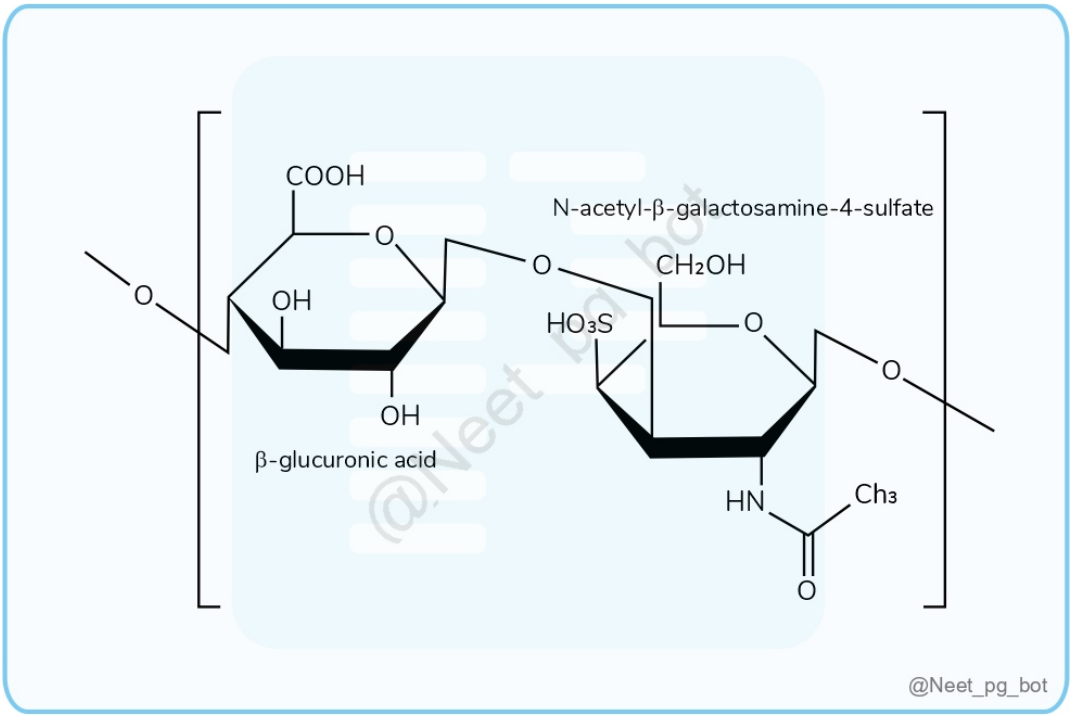
C. Heparin

D. Dermatan sulfate



Correct Answer: B. Chondroitin sulfate

Explanation: Chondroitin sulfate is a glycosaminoglycan (GAG) that is a major component of the extracellular matrix and connective tissues in the body. It consists of repeating units of N-acetylgalactosamine (GalNAc) and glucuronic acid (GIcA) or iduronic acid (Ido). Chondroitin sulfate provides structural support and elasticity to various tissues, including cartilage, tendons, ligaments, and skin. Keratan sulfate(Options A): Another type of glycosaminoglycan. It consists of repeating units of galactose and N-acetylglucosamine. It is commonly found in the cornea of the eye and cartilage, and it helps maintain the structural integrity of these tissues. Heparin(Options C): Different type of glycosaminoglycan that is highly sulfated. It consists of repeating units of glucuronic acid and N-acetylglucosamine. Heparin is an anticoagulant and is primarily produced and stored in mast cells. It plays a role in preventing blood clotting. Dermatan sulfate(Options D): Another glycosaminoglycan that is composed of repeating units of iduronic acid and N-acetylgalactosamine. It is found in various tissues, including skin, blood vessels, and heart valves.



References:

Chondritin sulfate

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: Which of the following is not a true statement regarding telomerase? (Q856880)

A. Has reverse transcriptase activity

B. Present only in Eukaryotes

C. Maintain the chromosome length

D. Involved in DNA repair

Correct Answer: D. Involved in DNA repair

Explanation: Telomerase is not involved in DNA repair process. Telomerase: An RNA-dependent DNA polymerase that counteracts telomere shortening. It is the enzyme responsible for telomere synthesis . Adds TTAGGG repeats to the 3′ ends of chromosomes. Contains its own RNA template, which is complementary to the telomeric DNA sequence, enabling it to extend telomeres. Has reverse transcriptase activity (Option A): Telomerase extends the telomeres using its reverse transcriptase activity. Present only in Eukaryotes (Option B): Telomerase is found in eukaryotes, as prokaryotes have circular DNA and do not require telomerase. Maintain the

chromosome length (Option C): Telomerase prevents the shortening of chromosomes during replication.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg. 365

Vasudevan, 7th Ed. Pg 585

Telomeres and telomere length

↑ *Back to this question in Part A*

@Neet_pg_bot

Q12: Nitric oxide is synthesized from: (Q854869)

A. L-Arginine

B. L-Citrulline

C. Glycine

D. Lysine

Correct Answer: A. L-Arginine

Explanation: Nitric oxide (NO) is synthesized from the amino acid L-arginine by a group of enzymes called nitric oxide synthases (NOS). The process involves the conversion of L-arginine to NO and L-citrulline. L-arginine: It is the primary precursor for the synthesis of nitric oxide in the body. Nitric oxide synthases convert L-arginine to nitric oxide and L-citrulline in a reaction that requires oxygen and cofactors. The synthesis of nitric oxide (NO) from L-arginine involves a reaction catalyzed by the enzyme nitric oxide synthase (NOS). The general equation for NO synthesis is as follows: L-arginine + NADPH + O 2 + H* → Nitric oxide + L-citrulline + NADP+ + H 2 O There are three isoforms of NOS: Endothelial NOS (eNOS) Neuronal NOS (NOS) Inducible NOS (iNOS). L-Citrulline (Option B): It is the product, not the substrate, of NO synthesis. Glycine (Option C) & Lysine (Option D): Not involved in NO production.

References:

NO

↑ *Back to this question in Part A*

@Neet_pg_bot

Q13: Which of the following options is false in a patient with advanced liver disease? (Q856619)

A. Hyperammonemia

B. Decreased ATP synthesis

C. Oxidative carboxylation of alpha-ketoglutarate

D. Increased synthesis of glutamine and glutamine from alpha-ketoglutarate

Correct Answer: C. Oxidative carboxylation of alpha-ketoglutarate

Explanation: In advanced liver disease, Hyperammonemia arises from impaired urea cycle and defective hepatic ammonia detoxification. (Option A ruled out) Mitochondrial dysfunction reduces ATP synthesis and depletes alpha-ketoglutarate, which is diverted for glutamine synthesis to detoxify ammonia. (Option B ruled out) Oxidative carboxylation of alpha-ketoglutarate does not occur; instead, glutamate undergoes oxidative deamination to regenerate alpha-ketoglutarate. (Option C) (Option D ruled out) This metabolic disruption contributes to energy failure and ammonia accumulation. Glutamate dehydrogenase (GDH): Glutamate dehydrogenase (GDH) converts glutamate to alpha-ketoglutarate This reaction occurs only in the Liver Glutamate is the only AA, that can undergo oxidative deamination to release amino groups in the liver Activators of GDH Inhibitors of GDH NAD ATP ADP GTP GDP NADH

References:

Harper’s biochemistry 32nd edition, Pg 282,283,291

Vasudevan 7th edition, Pg 201

↑ *Back to this question in Part A*

@Neet_pg_bot

Q14: The maximum thermic effect is seen with a meal that has (Q856500)

A. Protein

B. Fat

C. Carbohydrates

D. Mixed meal having all 3 in equal amount

Correct Answer: A. Protein

Explanation: F) refers to the increase in energy expenditure due to digestion, absorption, and metabolism of nutrients. Protein: Has the highest thermic effect (~20–30% of its energy content), as it requires significant energy for metabolism and nitrogen disposal. Fat: Has the lowest thermic effect (~0–3%), as it is metabolized more efficiently with minimal energy expenditure. Carbohydrates: Moderate thermic effect (~5–10%) , primarily for digestion and conversion into glycogen or fat. Mixed meal: The thermic effect of a mixed meal depends on its

macronutrient composition but will generally be lower than a protein-rich meal if all components are equal.

References:

TEF

↑ *Back to this question in Part A*

@Neet_pg_bot

Q15: A young girl presented to the OPD for an examination. She is tall and thin with slender fingers, and dislocated lenses. Workup revealed cystathione beta-synthase deficiency. Which amino acid supplement is required in this case? (Q855999)

A. Cysteine

B. Methionine

C. Serine

D. Tyrosine

Correct Answer: A. Cysteine

Explanation: The Clinical scenario of tall, thin and dislocated lens with cystathione beta synthase deficiency indicates Type 1 Homocystinuria. Cysteine is deficient in this patient, thus supplement is required in this case. Type 1 vs Type 2 Homocystinuria: Feature Type - I Homocystinuria Type - II Homocystinuria Enzyme Deficiency Cystathionine beta-synthase Methionine synthase Metabolic Pathway Affected Homocysteine to cystathionine Homocysteine to methionine Elevated Metabolites Homocysteine and methionine Homocysteine and elevated levels of Cysteine Biochemical Diagnosis Low levels of cysteine. High levels of methionine. High levels of cysteine. Low levels of methionine. Treatment Vitamin B6 (pyridoxine) supplementation; low-methionine diet; cysteine supplementation Vitamin B12 and folate supplementation; low-methionine diet

References:

Harper's Illustrated Biochemistry.32nd edition p.294, 295

Vasudevan Textbook of Biochemistry (7th Edition), Page 431.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q16: A patient presented with joint pain and on evaluation, urine turns black on standing. What is the likely enzyme deficiency? (Q855951)

A. Homogentisic acid 1,2-dioxygenase

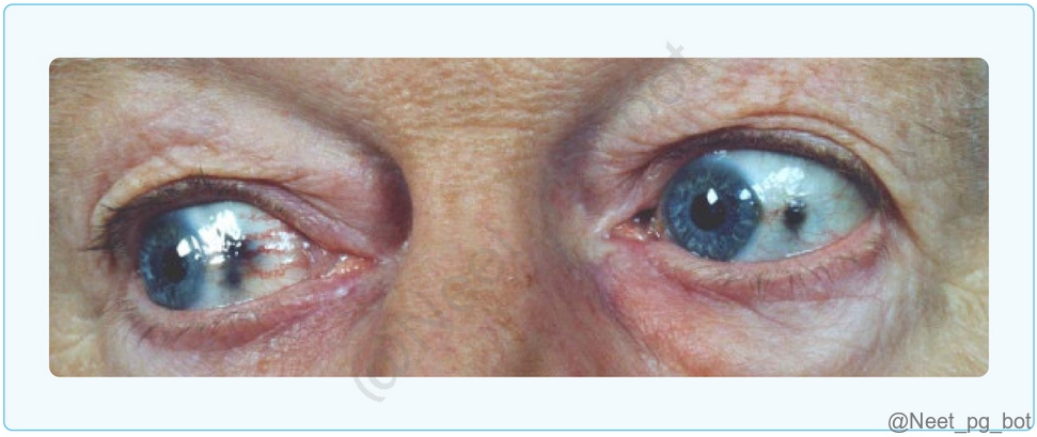
B. Tyrosine transaminase

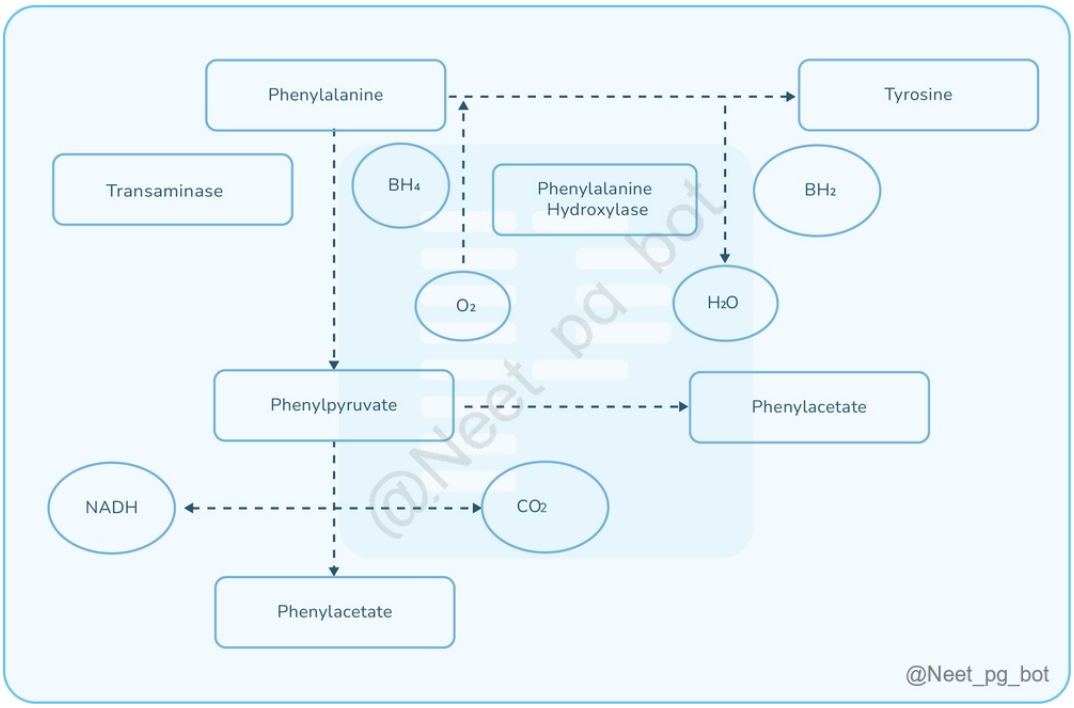
C. Fumarylacetoacetase hydrolase

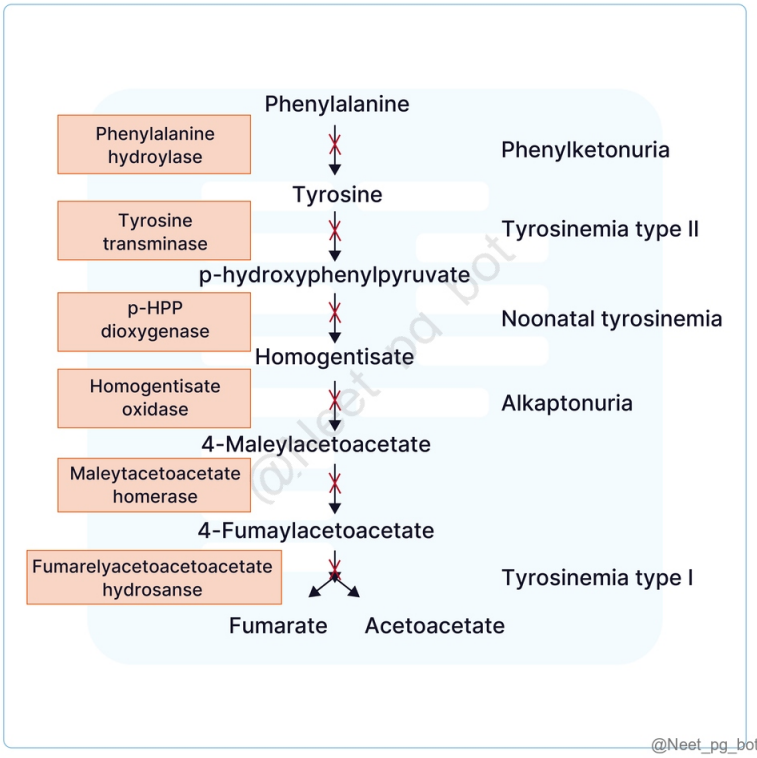
D. 4-hydroxy-phenyl pyruvate dioxygenase

Correct Answer: A. Homogentisic acid 1,2-dioxygenase

Explanation: The clinical scenario described, including darkening of urine upon standing and joint pain, is characteristic of Alkaptonuria , which is caused by a deficiency in the enzyme homogentisate oxidase. Garrod's tetrad in alkaptonuria 1. Homogentisic Aciduria Presence of homogentisic acid in the urine, which turns dark upon standing or when exposed to air. 2. Ochronosis {{caption_text}} Deposition of dark pigment in connective tissues, particularly noticeable in the ear cartilage, sclera, and other connective tissues, leading to a bluish-black discoloration.The image shows Osler’s sign. 3. Arthropathy Degenerative joint disease, especially affecting the spine and large joints, leading to chronic pain and stiffness. 4. Cardiac Involvement Includes heart valve calcification and atherosclerosis due to the deposition of pigment in cardiac tissues. Reaction Enzyme Associated Disorder Tyrosine → p-Hydroxyphenylpyruvate Tyrosine transaminase Tyrosinemia type II p-Hydroxyphenylpyruvate → Homogentisate p-Hydroxyphenylpyruvate dioxygenase Tyrosinemia type III Homogentisate → Maleylacetoacetate Homogentisate 1,2-dioxygenase Alkaptonuria Maleylacetoacetate → Fumarylacetoacetate Maleylacetoacetate isomerase - Fumarylacetoacetate → Fumarate + Acetoacetate Fumarylacetoacetate hydrolase Tyrosinemia type I







References:

Harper's Illustrated Biochemistry, 32nd Edition Pg. 296

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: Which of the following consumes the highest energy during assimilation? (Q855919)

A. Proteins

B. Carbohydrates

C. Mono-unsaturated fatty acids

D. Polyunsaturated fatty acids

Correct Answer: A. Proteins

Explanation: Proteins consume the highest energy during assimilation as they have the highest thermogenic effect and require maximum energy. Carbohydrates (Option B) utilizes less energy for assimilation 5%. MUFA (Option C) has a single double bond and consumes the least energy to be broken down. PUFA (Option D) are essential amino acids required by our body and require the least energy to be broken down.

References:

Vasudevan Textbook of Biochemistry, 7th Edition, Page 529

↑ *Back to this question in Part A*

@Neet_pg_bot

Q18: Which of the following options best describes the mutation seen in patients with cystic fibrosis? (Q855554)

A. Tryptophan at 708 position, chloride channel

B. Phenylalanine at 708 position, calcium channel

C. Phenylalanine at 508 position, chloride channel

D. Tryptophan at 508 position, calcium channel

Correct Answer: C. Phenylalanine at 508 position, chloride channel

Explanation: The mutation seen in cystic fibrosis (CF) is CFTR (Cystic Fibrosis Transmembrane Conductance Regulator) gene. Gene affected: CFTR gene, located on chromosome 7. Mutation type: The most common mutation is F508 (deletion of three nucleotides leading to the loss of phenylalanine at position 508). Effect : This

mutation leads to defective CFTR protein, which functions as a chloride channel in epithelial cells. Pathophysiology : Defective chloride transport results in thick, sticky mucus affecting lungs, pancreas, and other organs. Inheritance : Autosomal recessive disorder. Clinical Features of Cystic Fibrosis (CF): Respiratory : Chronic cough, recurrent infections, bronchiectasis, nasal polyps. Gastrointestinal : Pancreatic insufficiency, malabsorption, meconium ileus, liver disease. Nutritional : Vitamin deficiencies (A, D, E, K), failure to thrive.

References:

www.ncbi.nlm.nih.gov/books/NBK493206/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q19: An infant is brought by his parents with complaints that his urine turns black on standing. Which of the following metabolic disorders is most likely? (Q855263)

A. Phenylketonuria

B. Alkaptonuria

C. Homocystinuria

D. Maple syrup urine disease

Correct Answer: B. Alkaptonuria

Explanation: Alkaptonuria is Characterized by black urine upon standing due to the oxidation and polymerization of homogentisic acid (HGA). Alkaptonuria Definition Rare autosomal recessive genetic disorder caused by a deficiency of homogentisate 1,2 dioxygenase (HGD) , an enzyme in the tyrosine metabolism pathway. Mechanism and Pathophysiology Defective tyrosine metabolism → homogentisic acid (HGA) accumulation → ochronotic pigment deposition → systemic disease (ochronosis). Deficiency: HGD fails to convert HGA to maleylacetoacetate. Consequences: HGA oxidizes into melanin-like polymers → connective tissue deposition (ochronosis). Affects joints, heart valves, kidneys, and vessels. Clinical Features Triad: Homogentisic aciduria, ochronosis, ochronotic osteoarthropathy. Pediatric signs: Dark urine (oxidized HGA). Musculoskeletal : Arthritis (spine, hips, knees), kyphoscoliosis, spinal stenosis. Cardiac : A ortic stenosis, mitral valve disease, coronary artery disease. Renal/Metabolic: Stones (renal, gallbladder, prostate). Neurological : Peripheral neuropathy, stroke. Evaluation Clinical scoring: AKU Severity Score Index (AKUSSI). Diagnostic tests: Gold standard: Urinary HGA quantification via gas chromatography-mass spectrometry. Genetic testing for HGD mutations. Imaging (CT/MRI): Joint involvement, valvular calcification. Echocardiography: Valvular abnormalities. Management Symptomatic : Pain control, physiotherapy, joint surgery. Diet : Low-protein diet (limited efficacy). Medications: Vitamin C : Reduces HGA oxidation but not urinary excretion. Nitisinone : Inhibits tyrosine metabolism, reduces HGA >95%. Side effects: tyrosinemia, leukopenia, thrombocytopenia. Prognosis and Complications Prognosis : Normal life expectancy; reduced quality of life (pain, sleep issues, disability). Complications : Stones (renal, gallbladder, salivary). Tendon/ligament ruptures. Osteopenia, fractures. Aortic calcification/stenosis. Phenylketonuria (Option A): Caused by phenylalanine hydroxylase deficiency, leading to the accumulation of

phenylalanine. Symptoms include intellectual disability, seizures, and musty odor, but no black urine. Homocystinuria (Option C): Due to cystathionine beta-synthase deficiency. Presents with lens dislocation, thromboembolism, and developmental delay, but not black urine. Maple Syrup Urine Disease (Option D): Results from branched-chain alpha-keto acid dehydrogenase deficiency. Urine has a characteristic sweet odor but does not turn black on standing.

References:

Vasudevan Textbook of Biochemistry (7th Edition), Page 237, 238

https://www.ncbi.nlm.nih.gov/books/NBK560571/

Harper's Illustrated Biochemistry, 32nd Edition, Page 296, 304

↑ *Back to this question in Part A*

@Neet_pg_bot

Q20: A chronic alcoholic patient is diagnosed with gouty arthritis. What biochemical changes will be seen in this patient? (Q855230)

A. Decreased NADH/NAD+ ratio

B. Increased urea and urate levels

C. Increased urate and lactate levels

D. Increased level of alkaline phosphatase

Correct Answer: C. Increased urate and lactate levels

Explanation: Increased Uric Acid (Hyperuricemia): Chronic alcohol consumption is a significant contributor to hyperuricemia, a condition characterized by elevated levels of uric acid in the blood. This occurs due to: Increased turnover of purines from alcohol metabolism. Decreased renal excretion of uric acid as lactate competes with uric acid for excretion through the kidneys. These factors increase the likelihood of uric acid crystal deposition in joints, triggering gouty arthritis. Increased Lactate Levels (Lactic Acidosis): Alcohol metabolism, particularly through the enzyme alcohol dehydrogenase, generates an increased NADH/NAD+ ratio. This shift favors the conversion of pyruvate to lactate, leading to lactic acidosis. Elevated lactate levels further impair the kidney's ability to excrete uric acid, exacerbating hyperuricemia and contributing to gout attacks. Decreased NADH/NAD+ ratio (Option A): Alcohol metabolism increases the NADH/NAD+ ratio, which indirectly contributes to lactate production and hyperuricemia. Increased urea and urate levels (Option B): While urate levels increase, chronic alcohol use does not typically elevate urea levels unless there is concurrent kidney damage or protein metabolism disturbances. Increased level of alkaline phosphatase (Option D): This is associated with conditions like bone or liver disease but is not a key feature of gouty arthritis linked to alcohol consumption.

References:

Is lactic acid a misunderstood trigger of gout attack for a century? - ScienceDirect Gout Symptoms, Causes & Diet Recommendations | NIAMS

↑ *Back to this question in Part A*

@Neet_pg_bot

Q21: In a patient with Maple Syrup Urine Disease (MSUD), all of the following amino acids should be restricted in diet except? (Q855044)

A. Methionine

B. Valine

C. Leucine

D. Isoleucine

Correct Answer: A. Methionine

Explanation: Methionine breaks down in maple syrup urine disease. Maple syrup urine disease is a rare genetic disorder that affects the breakdown of the branched-chain amino acids (BCAAs) leucine, isoleucine, and valine. In people with maple syrup urine disease, they cannot be metabolized properly due to a deficiency in the enzyme branched-chain ketoacid dehydrogenase. Burnt sugar odour from urine Ketosis Mental retardation Abnormal muscle tone

References:

www.ncbi.nlm.nih.gov/books/NBK557773/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q22: In a patient with obstructive jaundice, which of the following outcomes may be observed in Van der Bergh's reaction? (Q854880)

A. Direct positive

B. Indirect positive

C. Both positive

D. Coproporphyrin

Correct Answer: A. Direct positive

Explanation: Van den Bergh’s reaction is a biochemical test used to differentiate types of bilirubin in serum by observing their reactivity with diazotized sulfanilic acid . Types of Reaction: Type of Jaundice Type of Bilirubin Elevated Van den Bergh Reaction Interpretation Hemolytic (Pre-hepatic) Unconjugated (Indirect) Indirect positive (requires alcohol to react) Due to excessive RBC breakdown Hepatocellular (Hepatic) Both conjugated & unconjugated Biphasic (Both positive) Liver cell injury affects conjugation & excretion Obstructive (Post-hepatic) Conjugated (Direct) Direct positive (reacts immediately) Due to bile duct obstruction Mechanism in Obstructive Jaundice: In obstructive jaundice, conjugated bilirubin (water-soluble) regurgitates into the bloodstream due to biliary obstruction. Since conjugated bilirubin reacts directly with diazo reagent without the need for alcohol, it produces a direct positive Van den Bergh reaction.

References:

Obstructive jaundice

↑ *Back to this question in Part A*

@Neet_pg_bot

• [10_Heme Metabolism] 028 Previous Year Questions

« Back to Folder Directory

[10_Heme Metabolism] 028 Previous Year Questions — Quick Jump

Q1 (p. 1202) *Q2 (p. 1202)* *Q3 (p. 1203)* *Q4 (p. 1204)* *Q5 (p. 1205)* *Q6 (p. 1206)* *Q7 (p. 1207)*

Q1: Decreased % transferrin saturation is seen in? (Q854613)

A. Hemochromatosis

B. Iron deficiency anemia

C. Megaloblastic anemia

D. Hemosiderosis

Correct Answer: B. Iron deficiency anemia

Explanation: Laboratory findings in iron deficiency anemia: Hemoglobin Indices Low Mean Corpuscular Hemoglobin (MCH), Low Mean Corpuscular Volume (MCV) Peripheral Blood Smear Microcytosis, Hypochromia, Anisocytosis Serum Ferritin Decreased Serum Iron Decreased Transferrin Saturation Decreased Total Iron-Binding Capacity Increased Stool for Occult Blood May be positive Mentzer Index >15 Hemoglobin Electrophoresis Normal Bone Marrow Aspiration/Biopsy Iron stores absent on staining In the rest of the conditions, the % transferrin saturation is normal or increased

References:

https://www.ncbi.nlm.nih.gov/books/NBK448065/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: Which part of the haemoglobin molecule does 2,3-Bisphosphoglycerate (2,3-BPG) bind to in order to regulate oxygen release? (Q854619)

A. Alpha chains of haemoglobin

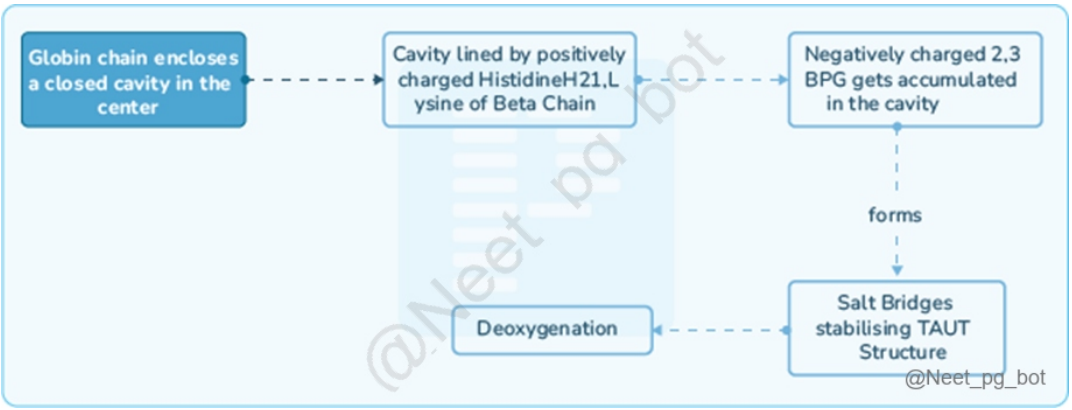
B. Heme iron centre

C. Beta chains of haemoglobin

D. Globin-heme interface

Correct Answer: C. Beta chains of haemoglobin

Explanation: 2,3-BPG binds within the central cavity of the deoxyhaemoglobin tetramer . It interacts with positively charged residues (mainly lysine and histidine) on the β-chains . This binding stabilises the T (tense) state , lowering haemoglobin's affinity for oxygen and promoting oxygen delivery to tissues. Effect of 2,3 BPG on Hb: Reduces the affinity of Hb for oxygen and helps unload oxygen to tissues. A by-product of glycolysis. (Rapaport Leubering Cycle) Alpha chains of haemoglobin (Option A) : Incorrect because 2,3-BPG binds primarily to residues on the β-chains , not the α-chains. Heme iron centre (Option B) : 2,3-BPG does not bind directly to heme or iron ; it acts allosterically , not at the active oxygen-binding site. Globin-heme interface (Option D) : This is the site of coordination between globin and heme, not where 2,3-BPG binds.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 55

↑ *Back to this question in Part A*

@Neet_pg_bot

Q3: What does iron deficiency anemia (IDA) stand for? (Q854624)

A. Low serum ferritin

B. Low transferrin saturation

C. Increased TIBC

D. All of the above

Correct Answer: D. All of the above

Explanation: Iron deficiency anemia (IDA) is characterized by laboratory evidence of depleted iron stores and compensatory changes in iron transport proteins: Low serum ferritin reflects depleted body iron stores. Low transferrin saturation indicates reduced circulating iron available for erythropoiesis. Increased total iron-binding capacity (TIBC) occurs as a compensatory rise in transferrin production by the liver to scavenge available iron. Laboratory findings in iron deficiency anemia: Hemoglobin Indices Low Mean Corpuscular Hemoglobin (MCH), Low Mean Corpuscular Volume (MCV) Peripheral Blood Smear Microcytosis, Hypochromia, Anisocytosis Serum Ferritin Decreased Serum Iron Decreased Transferrin Saturation Decreased Total Iron-Binding Capacity Increased Stool for Occult Blood May be positive Mentzer Index >15 Hemoglobin Electrophoresis Normal Bone Marrow Aspiration/Biopsy Iron stores absent on staining

References:

https://www.ncbi.nlm.nih.gov/books/NBK448065/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q4: Which of the following is the rate-limiting enzyme of heme synthesis pathway? (Q857891)

A. HMB synthase

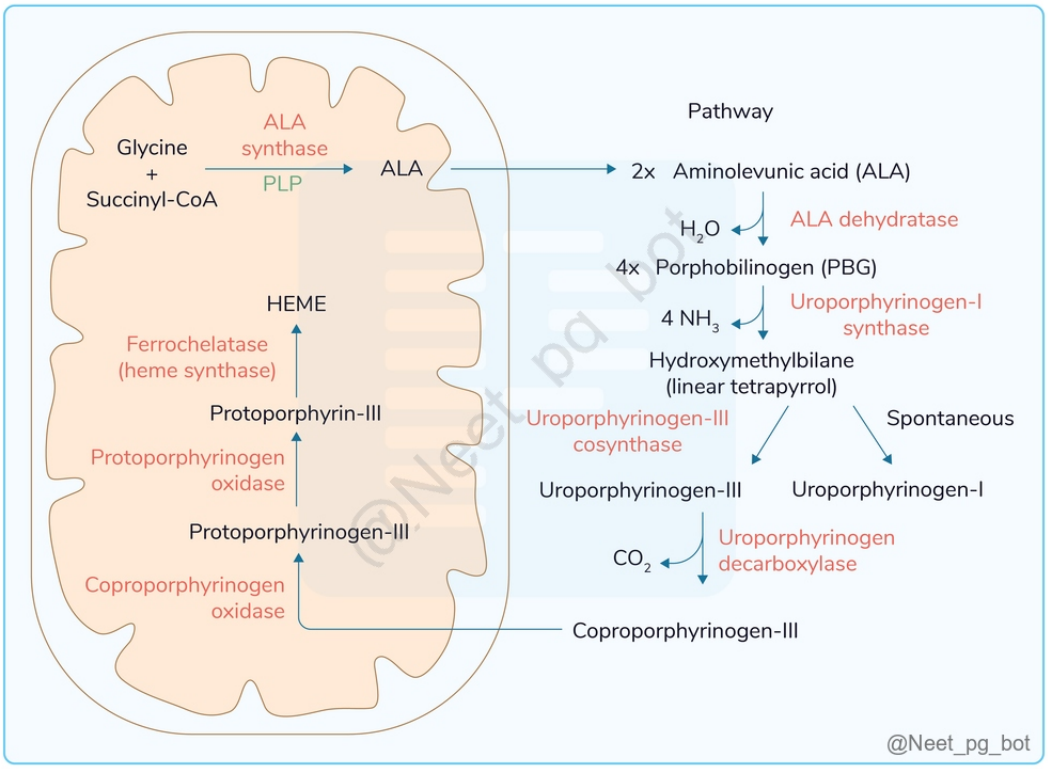
B. δ Amino-Levulinic Acid dehydratase

C. δ Amino-Levulinic Acid synthase

D. Ferrochelatase

Correct Answer: C. δ Amino-Levulinic Acid synthase

Explanation: This enzyme catalyzes the formation of ALA from glycine and succinyl-CoA. It is the rate-limiting enzyme of the heme synthesis pathway. Its activity is tightly regulated by feedback inhibition by heme itself. Heme Synthesis: Step Reaction Enzyme Location Key Points 1 Succinyl CoA + Glycine → Delta-aminolevulinic acid (ALA) ALA Synthase (Option C) Mitochondria Rate-limiting step ; requires pyridoxal phosphate as a cofactor 2 2 ALA → Porphobilinogen (PBG) ALA Dehydratase (Option B) Cytoplasm It involves removal of 2 molecules of water; the enzyme contains zinc and is inhibited by lead 3 4 PBG → Hydroxymethylbilane (HMB) Uroporphyrinogen I Synthase/ HMB synthase (Option A) Cytoplasm - 4a HMB → Uroporphyrinogen I Spontaneous cyclization Cytoplasm - 4b HMB → Uroporphyrinogen III Uroporphyrinogen III Synthase Cytoplasm - 5 Uroporphyrinogen III → Coproporphyrinogen III Uroporphyrinogen Decarboxylase Cytoplasm Decarboxylation, removal of 4 molecules of CO_2 6 Coproporphyrinogen III → Protoporphyrinogen III Coproporphyrinogen Oxidase Mitochondria 7 Protoporphyrinogen III → Protoporphyrin III Protoporphyrinogen Oxidase Mitochondria 8 Protoporphyrin III → Heme Ferrochelatase (Option D) Mitochondria Insertion of Fe^{2+}



References:

Vasudevan Textbook of Biochemistry Edition 7th Pg. 271, 272, 273

↑ *Back to this question in Part A*

@Neet_pg_bot

Q5: A female is diagnosed with lead poisoning . Which of the following enzyme defects is primarily responsible for the hematologic abnormalities observed in this condition? (Q857898)

A. ALA synthase

B. Uroporphyrinogen decarboxylase

C. Heme synthase

D. Coproporphyrinogen oxidase

Correct Answer: C. Heme synthase

Explanation: Lead (Pb^{2}■) inhibits ferrochelatase , the final enzyme in the heme biosynthesis pathway , which catalyzes: Protoporphyrin IX + Fe^{2}■ → Heme Inhibition prevents iron incorporation into protoporphyrin IX,

causing accumulation of free protoporphyrin and resulting in microcytic, hypochromic anemia . Although lead also inhibits ALA dehydratase , the hematologic effects (anemia) are primarily due to ferrochelatase inhibition ALA synthase (A): Rate-limiting step of heme synthesis, not affected by lead Uroporphyrinogen decarboxylase (B): Defective in porphyria cutanea tarda , unrelated to lead poisoning. Coproporphyrinogen oxidase (D): Defective in hereditary coproporphyria , also unrelated to lead toxicity.

References:

Vasudevan Textbook of Biochemistry Edition 7th Pg. 276

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: Heme is derived from which of the following amino acids? (Q860419)

A. Glutamine

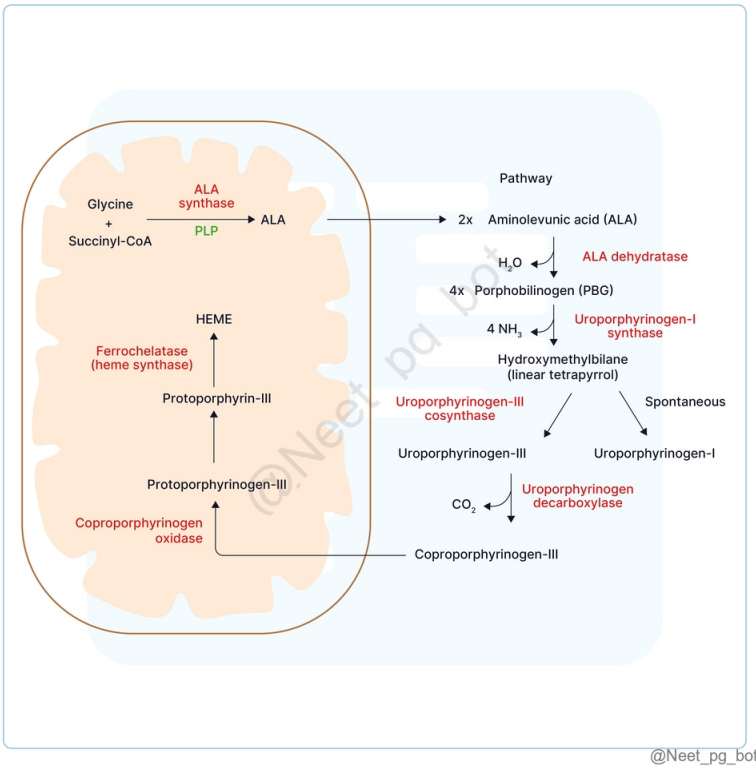
B. Glycine

C. Glutamate

D. Valine

Correct Answer: B. Glycine

Explanation: Glycine combines with succinyl-CoA in the first step of heme synthesis , catalyzed by the enzyme δ-aminolevulinic acid (ALA) synthase . This reaction is the rate-limiting step in heme biosynthesis. Glutamine (Option A): Glutamine is involved in nucleotide biosynthesis and ammonia transport but does not play a direct role in heme synthesis. Glutamate (Option C): Glutamate is primarily involved in the synthesis of glutathione and neurotransmitters but does not contribute to heme synthesis. Valine (Option D): Valine is a branched-chain amino acid involved in protein synthesis and energy production but not in heme biosynthesis.



References:

Vasudevan Textbook of Biochemistry Edition 7th Pg. 271, 272, 273

↑ *Back to this question in Part A*

@Neet_pg_bot

Q7: Which of the following amino acids are necessary for purine ring formation? (Q860424)

A. Glycine, aspartate, valine

B. Glycine, Aspartate, Glutamine

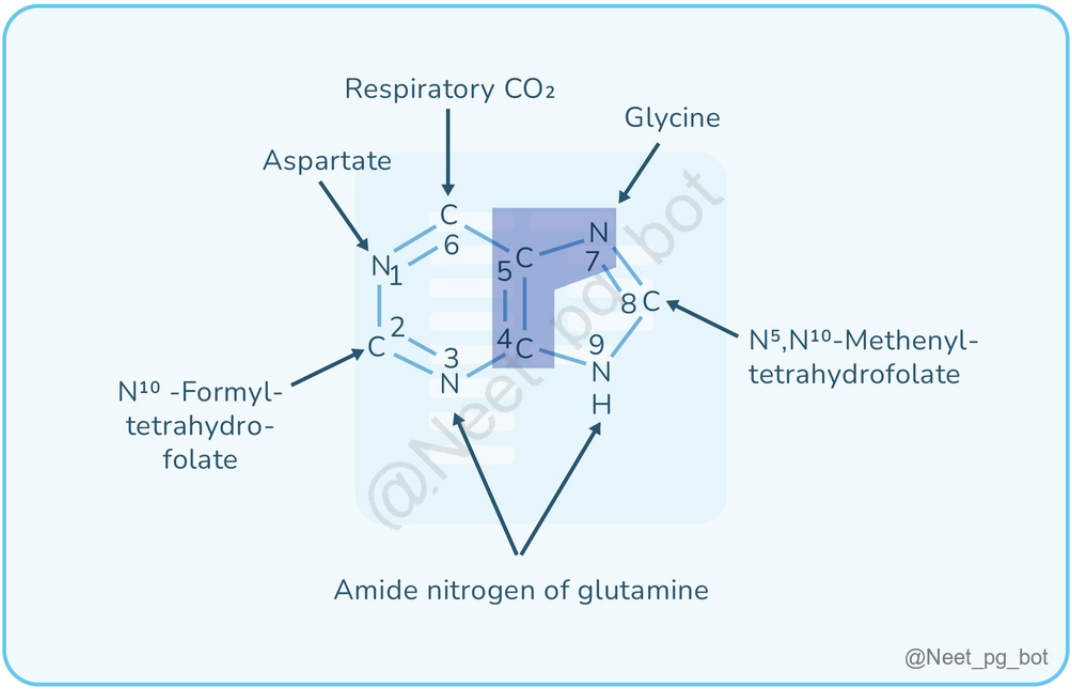
C. Glycine, Aspartate, Glutamate

D. Glycine, Asparagine, Glutamine

Correct Answer: B. Glycine, Aspartate, Glutamine

Explanation: Purine ring formation requires three amino acids - Glycine, Aspartate, Glutamine and the cofactor N10-formyl tetrahydrofolate (N10- formyl THF) for purine ring formation. (Option B) (Option A, C and D ruled

out) The sources of each atom in a purine were found by feeding isotopic precursors of uric acid to pigeons. The C-2 atom is from formyl THF The C-8 atom is from N5N10 methenyl THF The C-6 atom is from respiratory CO2 . N1 is from aspartate N3 and N9 are from Glutamine C4, C5 and N7 are from Glycine



References:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7869020/

Harper's Illustrated Biochemistry, 32nd edition, pg:338, 339

Vasudevan textbook of biochemistry, 7th edition, pg: 208

↑ *Back to this question in Part A*

@Neet_pg_bot

• [11_Vitamins] 029 Fat-Soluble Vitamins - I

« Back to Folder Directory

[11_Vitamins] 029 Fat-Soluble Vitamins - I — Quick Jump

Q1 (p. 1209)	*Q2 (p. 1210)*	*Q3 (p. 1211)*	*Q4 (p. 1211)*	*Q5 (p. 1212)*	*Q6 (p. 1213)*
Q7 (p. 1216)	*Q8 (p. 1217)*	*Q9 (p. 1218)*	*Q10 (p. 1219)*	*Q11 (p. 1221)*	*Q12 (p. 1222)*
Q13 (p. 1223)	*Q14 (p. 1223)*	*Q15 (p. 1224)*	*Q16 (p. 1225)*	*Q17 (p. 1225)*	*Q18 (p. 1226)*
Q19 (p. 1227)	*Q20 (p. 1228)*	*Q21 (p. 1229)*			

Q1: A 62-year-old male on warfarin therapy for atrial fibrillation presents with uncontrolled bleeding. Laboratory tests show prolonged PT and INR. Which of the following mechanisms best explains the effect of warfarin on coagulation? (Q844939)

A. Inhibition of Vitamin K epoxide reductase

B. Inhibition of thrombin activity directly

C. Binding to calcium ions

D. Direct inhibition of clotting factors

Correct Answer: A. Inhibition of Vitamin K epoxide reductase

■ *Listen to Audio 1*

Explanation: Warfarin Mechanism of action Inhibits Vitamin K epoxide reductase (VKORC1) (Option A) Prevents regeneration of reduced vitamin K Effect on coagulation factors Decreases synthesis of Vitamin K–dependent factors: II, VII, IX, X, and Proteins C & S Produces anticoagulation Biochemical effect Prevents γ-carboxylation of glutamate residues on clotting factors Clotting factors cannot bind Ca^{2}■ and phospholipid membrane Coagulation pathway affected Mainly extrinsic pathway PT and INR increase first Laboratory monitoring PT / INR Therapeutic INR is usually 2–3 Onset of action Delayed (24–72 hours) Existing clotting factors must degrade Early prothrombotic state Rapid fall in Protein C Can cause warfarin-induced skin necrosis Pharmacokinetics Oral drug, ~99% protein bound Highly susceptible to drug interactions Half-life 36–42 hours Long duration of action Metabolism Hepatic metabolism via CYP2C9 (major), CYP1A2, CYP3A4 Genetic variations affect dose CYP inhibitors (↑ bleeding) Amiodarone, metronidazole, azoles Increase warfarin levels CYP inducers (↓ effect) Rifampicin, carbamazepine, phenobarbital Reduce anticoagulant effect Elimination kinetics First-order kinetics Constant fraction eliminated per unit time Major adverse effects Bleeding, skin necrosis , teratogenicity Contraindicated in pregnancy Antidotes Vitamin K (phytonadione), Fresh frozen plasma (FFP), Prothrombin complex concentrate (PCC) Used in warfarin toxicity

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 541

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 475

↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: A 40 year old woman presents with complaints of persistent nausea, vomiting, and frequent urination over the past few weeks. She has a history of osteomalacia and has been taking supplements for several months without proper medical supervision. Her blood tests reveal elevated serum calcium levels. What is the most likely diagnosis? (Q844949)

A. Hyperparathyroidism

B. Vitamin D deficiency

C. Vitamin D toxicity

D. Hypercalcemia of malignancy

Correct Answer: C. Vitamin D toxicity

■ *Listen to Audio 1*

Explanation: Vitamin D toxicity aligns with the patient's history of high supplement intake, most probably containing Vitamin D, and her symptoms of nausea, vomiting, frequent urination, and hypercalcemia. Comparison of Vitamin D Toxicity and Deficiency: Aspect Vitamin D Deficiency Vitamin D Toxicity Causes Inadequate dietary intake, insufficient sun exposure, malabsorption syndromes, and certain medical conditions affecting metabolism. Excessive supplementation. Dietary intake and sun exposure rarely cause toxicity. Symptoms Bone pain, muscle weakness, and increased risk of fractures; in children, it leads to rickets; in adults, it leads to osteomalacia or osteoporosis. Nausea, vomiting, weakness, frequent urination, and hypercalcemia; can lead to kidney stones and kidney damage. Serum Levels Defined as 25-hydroxyvitamin D (25(OH)D) < 30 ng/mL , indicating insufficient levels. Occurs when 25(OH)D > 150 ng/mL , leading to adverse health effects. Treatment Managed with dietary supplementation via food or medications and increased sun exposure. Requires discontinuation of vitamin D intake, intravenous fluids, and medications, such as corticosteroids or bisphosphonates. Hyperparathyroidism (Option A) typically presents with elevated serum calcium due to increased parathyroid hormone but is less likely given the context of high supplement intake. Vitamin D deficiency (Option B) would not cause hypercalcemia; rather, it leads to bone pain, muscle weakness, and increased fracture risk. Hypercalcemia of malignancy (Option D) can cause elevated serum calcium but does not fit the clinical context of excessive Vitamin D supplementation.

References:

www.ncbi.nlm.nih.gov/books/NBK532266/

www.ncbi.nlm.nih.gov/books/NBK557876/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q3: Which of the following is not a commonly associated symptom with vitamin E deficiency? (Q844948)

A. Spinocerebellar ataxia

B. Megaloblastic anemia

C. Peripheral neuropathy

D. Hemolytic anemia

Correct Answer: B. Megaloblastic anemia

■ *Listen to Audio 1*

Explanation: Megaloblastic anemia is caused by a deficiency of vitamin B9 (folic acid) and B12 (cobalamin). Vitamin E Deficiency: Rare but seen in severe fat malabsorption, cystic fibrosis, and some forms of chronic liver disease, as these conditions are unable to absorb or transport vitamin E. Premature infants are born with inadequate vitamin E reserves. Symptoms: Hemolytic anemia: Due to lipid peroxidation and fragile erythrocyte membranes. (Option D ruled out) Neurological problems due to poor nerve conduction: Spinocerebellar ataxia (Option A ruled out) Retinopathy Peripheral neuropathy (Option C ruled out) Myopathies Infertility in both males and females Alopecia

References:

Lippincott Illustrated Reviews, Biochemistry, 7th Edition, Page 540

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 474

↑ *Back to this question in Part A*

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Q4: A 45-year-old man presents with fatigue, frequent urination, constipation, and confusion. His laboratory results show hypercalcemia and low serum parathyroid hormone level. He reports taking high-dose, over-the-counter vitamin D supplements for the past year. Which of the following conditions is most likely associated with his current presentation? (Q844947)

A. Increased risk of hypertension

B. Increased risk of colon cancer

C. Increased risk of breast cancer

D. Increased risk of insulin resistance

Correct Answer: A. Increased risk of hypertension

■ *Listen to Audio 1*

Explanation: The patient's symptoms and laboratory findings are consistent with vitamin D toxicity. Excessive vitamin D intake can lead to hypercalcemia, which can manifest as fatigue, frequent urination, constipation, and confusion. Hypercalcemia causes high blood pressure. Vitamin D toxicity also causes low serum parathyroid hormone levels. Vitamin D Toxicity: Vitamin D toxicity is defined as Adults > 4000 IU and children > 2000 IU. Calcium homeostasis: Hypercalcemia Thickening of the long bones Calcification of soft tissues (Calcinosis). Calcium deposition in Blood vessels (Monckeberg medial calcific sclerosis, atherosclerosis, etc) Hypercalcemia: Contraction of blood vessels and High blood pressure. (Option A) While excessive dietary vitamin D is toxic, overexposure to sunlight does not cause vitamin D poisoning. Beneficial effects of High Vitamin D status: Protects against prediabetes and metabolic syndrome. (Option D ruled out) Protect against various cancers: Prostate cancer Colorectal cancer (Option B ruled out) Breast cancer (Option C ruled out)

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 539-540.

↑ *Back to this question in Part A*

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Q5: A 38-year-old female with a history of chronic kidney disease presents with persistent fatigue, bone pain, and muscle weakness. Blood work reveals elevated alkaline phosphatase (ALP), high serum phosphorus, and low serum calcium. What will the result of 1,25 dihydroxy vitamin D likely reveal? (Q844946)

A. Elevated 1, 25 dihydroxy vitamin D levels.

B. Estimating 1, 25 dihydroxy vitamin D is unnecessary.

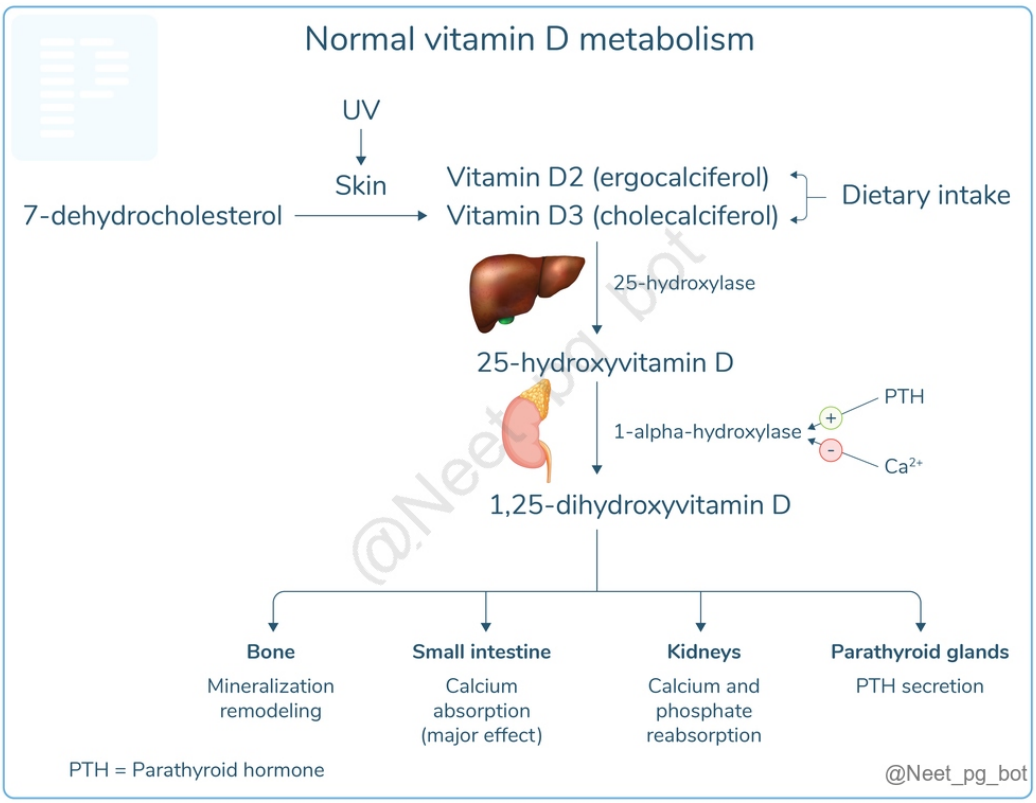
C. Decreased 1, 25 dihydroxy vitamin D.

D. Normal 1, 25 dihydroxy vitamin D.

Correct Answer: C. Decreased 1, 25 dihydroxy vitamin D.

■ *Listen to Audio 1*

Explanation: Patients with CKD often have reduced 1α-hydroxylase, leading to lower levels of active vitamin D: 1,25 dihydroxy vitamin D. 1,25 dihydroxy Vitamin D in Chronic kidney disease: In chronic kidney disease (CKD), estimating 1,25 dihydroxy vitamin D levels is essential due to the kidneys' role in vitamin D metabolism. (Option B ruled out) The kidneys are the primary site where 1α-hydroxylase converts inactive vitamin D (25 hydroxy cholecalciferol) into its active form, 1,25 dihydroxy cholecalciferol. Patients with CKD often have reduced 1α-hydroxylase, leading to lower levels of active vitamin D: 1,25 dihydroxy vitamin D. (Options A and D ruled out) Impact on Calcium Homeostasis : Low levels in CKD causes hypocalcemia. Secondary Hyperparathyroidism : To compensate for low calcium, PTH increases and has negative consequences for bone health. Renal Osteodystrophy : Abnormal bone turnover, as evidenced by the elevated alkaline phosphatase in the clinical scenario. Diagnostic Value : 1,25 dihydroxy cholecalciferol has a longer half-life and remains stable. Therefore, it provides a more reliable test for vitamin D status in CKD and helps guide appropriate treatment. Treatment: Dialysis. Calcimimetic drugs - Cinacalcet and Etelcalcetide.



References:

Vitamin D Supplementation for Patients with Chronic Kidney Disease: A Systematic Review and Meta-analyses of Trials Investigating the Response to Supplementation and an Overview of Guidelines - PMC (nih.gov)

↑ *Back to this question in Part A*

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Q6: A 2-year-old child presents with progressive bowing of the legs and tenderness in the lower limbs. The child has a history of limited outdoor play and a diet low in dairy products. Which of the following limb deformities is most likely present in this child? (Q844945)

A. Rickets

B. Blount’s disease

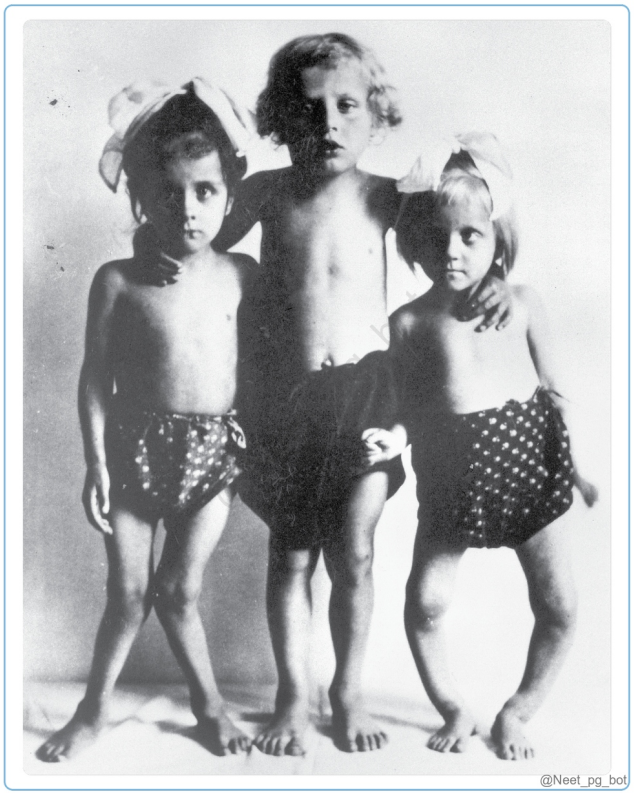
C. Osteomalacia

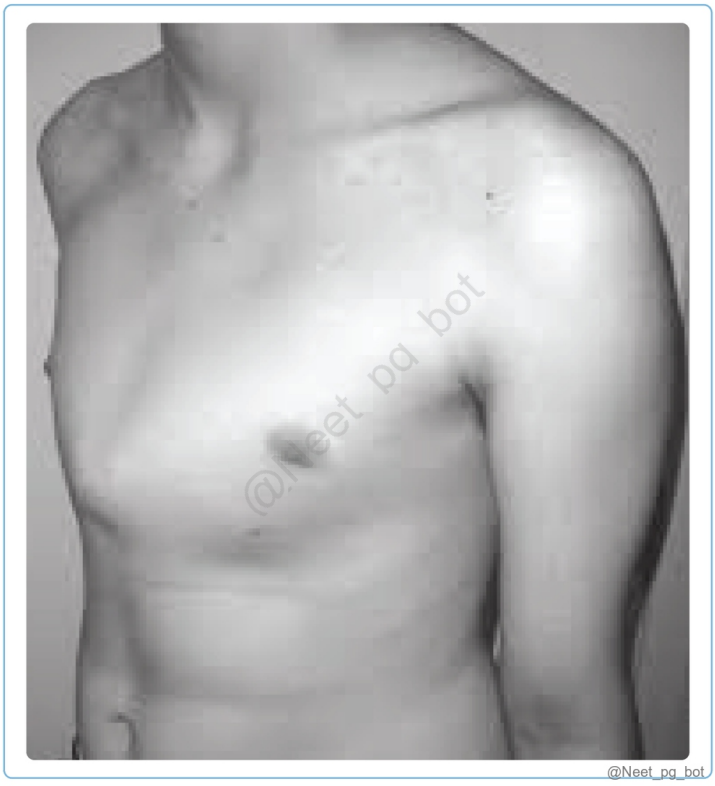
D. Osteogenesis imperfecta

Correct Answer: A. Rickets

■ *Listen to Audio 1*

Explanation: The scenario describes a child with classic signs of Rickets associated with vitamin D deficiency. This condition results in leg deformities in children with inadequate sun exposure and dietary intake of vitamin D. Clinical manifestations of Vitamin D deficiency Rickets (Option A) Occurs in children. Inadequate bone mineralization due to poor calcium absorption. Bones become soft and weak, leading to deformities under pressure. Clinical signs : Genu varum (bow legs), genu valgum, windswept deformity Craniotabes, frontal bossing Rickety rosary (costochondral swelling), pigeon chest Delayed walking, growth retardation Proximal muscle weakness Laboratory Findings Serum calcium: Decreased or normal Serum phosphate: Decreased Alkaline phosphatase: Markedly increased PTH: Increased Vitamin D levels: Decreased Radiological Finding: Metaphyseal cuppings Metaphyseal fraying Metaphyseal widening Generalized osteopenia Bowing of long bones Delayed ossification Management: Vitamin D supplementation: Vitamin D 2000 IU/day for 6–12 weeks followed by maintenance 400–600 IU/day Calcium supplementation Sunlight exposure Dietary correction Orthopedic correction Osteomalacia (Option C ruled out) Occurs in adults. Demineralization of bone, making them weak and prone to fractures. Risk factors: limited sun exposure, women after multiple pregnancies. Renal osteodystrophy Long-standing secondary hyperparathyroidism can result in renal osteodystrophy, increasing the risk of pathological fractures. Blount’s disease (Option B) (tibia vara) is an acquired genu varus deformity typically developing later in childhood, while nutritional rickets, caused by vitamin D deficiency from poor diet and limited sun exposure, is more likely in this young child. Osteogenesis imperfecta (Option D) is unlikely because it is a genetic disorder affecting collagen synthesis, leading to brittle bones and frequent fractures from birth, rather than the progressive bowing of the legs seen in nutritional rickets.





References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 539-540

www.ncbi.nlm.nih.gov/books/NBK560923/

www.ncbi.nlm.nih.gov/books/NBK536957/

↑ *Back to this question in Part A*

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Q7: A 35-year-old woman presents with fatigue, muscle weakness, and bone pain for the past several months. She has a history of hypothyroidism and is currently taking levothyroxine. She lives in North India and avoids dairy products. Physical examination reveals mild tenderness over her shins. Laboratory tests reveal: Serum calcium 8.2 mg/dL (normal range: 8.5-10.5 mg/dL) Serum phosphorus 2.0 mg/dL (normal range: 2.5-4.5 mg/dL) Parathyroid hormone (PTH) Elevated Which of the following is the most likely diagnosis? (Q844944)

A. Vitamin D deficiency

B. Primary hyperparathyroidism

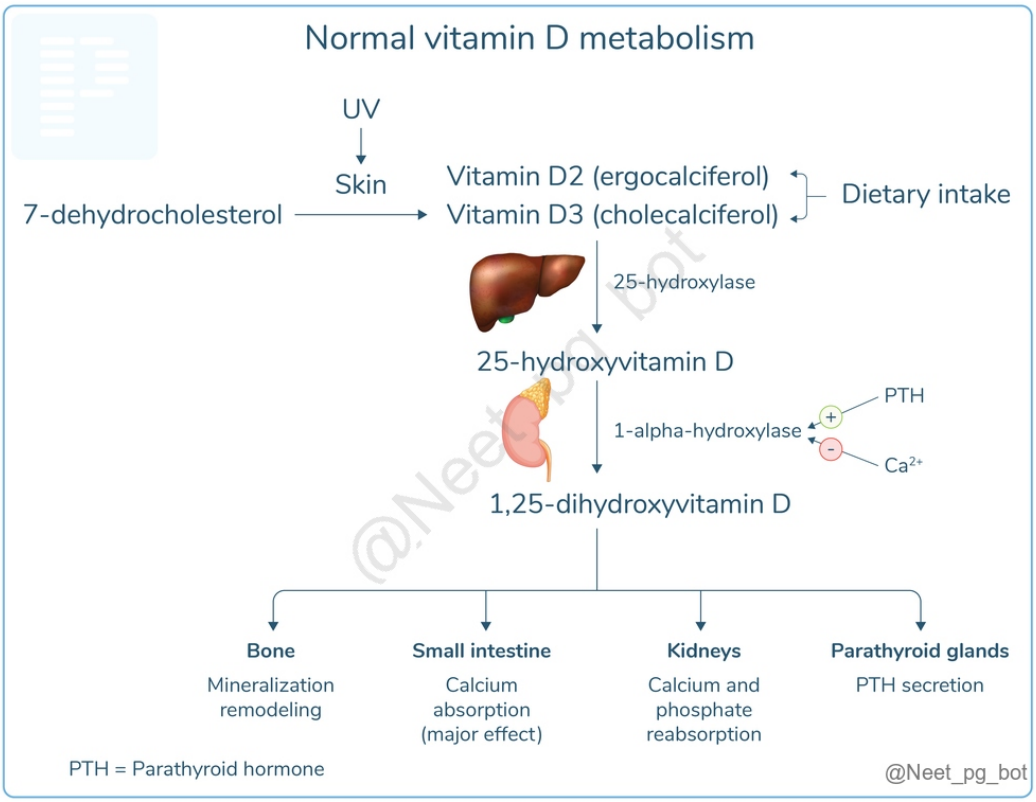
C. Hypoparathyroidism

D. Chronic kidney disease

Correct Answer: A. Vitamin D deficiency

■ *Listen to Audio 1*

Explanation: Based on the above scenario, the patient’s symptoms and laboratory findings are most likely to be Vitamin D deficiency. Findings of Vitamin D deficiency Hypocalcemia Due to decreased calcium absorption in the intestine. Hypophosphatemia Due to decreased phosphate absorption in the intestine and decreased reabsorption of phosphate in the kidneys. Secondary Hyperparathyroidism In response to low calcium levels, the parathyroid hormone (PTH) increases, which increases calcium reabsorption from bone and the kidneys. Elevated Alkaline Phosphatase (ALP) Due to Increased bone turnover as a result of increased PTH. 25-hydroxy cholecalciferol Decreased. Prolonged secondary hyperparathyroidism can cause renal osteodystrophy, which may lead to pathological fractures. Primary hyperparathyroidism (Option B) leads to excess PTH production, causing hypercalcemia. Hypoparathyroidism (Option C) presents with low PTH and hypocalcemia. Hypothyroidism is commonly associated with vitamin D deficiency, potentially due to immune dysfunction and impacts on calcium metabolism Chronic kidney disease (Option D) causes secondary hyperparathyroidism due to impaired phosphate excretion and low 1,25-dihydroxy vitamin D levels, it is typically associated with hyperphosphatemia.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 538.

↑ *Back to this question in Part A*

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Q8: A 52-year-old woman presents with muscle cramps and tingling in her hands and feet. She follows a strict vegetarian diet high in whole grains and has a history of fat malabsorption due to IBS. Lab tests reveal hypocalcemia and low vitamin D levels. What is the most likely cause of her hypocalcemia? (Q844943)

A. Low intake of oxalate

B. Low fatty acid in intestine

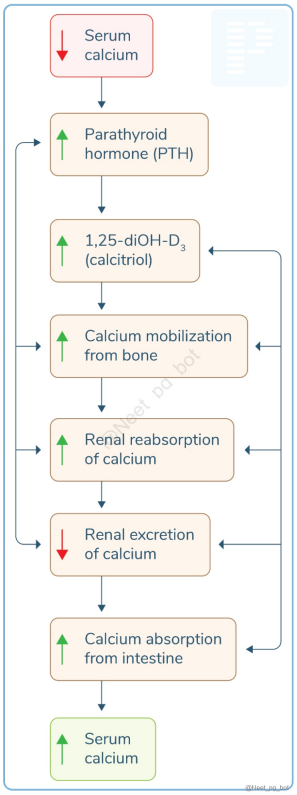
C. High phytate intake from cereals

D. Increased calbindin level

Correct Answer: C. High phytate intake from cereals

■ *Listen to Audio 1*

Explanation: Phytates, found in high amounts in whole grains and cereals, bind calcium in the intestine, preventing its absorption. Since the patient's diet is rich in these foods, the phytates are likely reducing her calcium absorption. Vitamin D and Calcium Absorption: Vitamin D is essential for intestinal calcium absorption. It induces the synthesis of calbindin, a protein crucial for calcium absorption. (Option D ruled out) Vitamin D recruits calcium transporters to the cell surface, enhancing absorption rapidly, independent of new protein synthesis. Factors Inhibiting Calcium Absorption: Phytic Acid: Found in cereals, it binds calcium in the intestine, preventing absorption. Unleavened Whole-Wheat Products: High in phytate, which binds to calcium, preventing absorption. (Option C) Fatty Acids: High levels in the intestine can form insoluble calcium salts, reducing absorption. (Option B ruled out) Oxalate: High intake can cause calcium deficiency by forming insoluble calcium oxalate. (Option A ruled out)



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 531

Lippincott Illustrated Reviews, Biochemistry, 7th Edition, Chapter 28: Micronutrients

↑ *Back to this question in Part A*

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Q9: Which of the following factors does not inhibit 1α-hydroxylase activity? (Q844942)

A. Hyperphosphatemia

B. Hypercalcemia

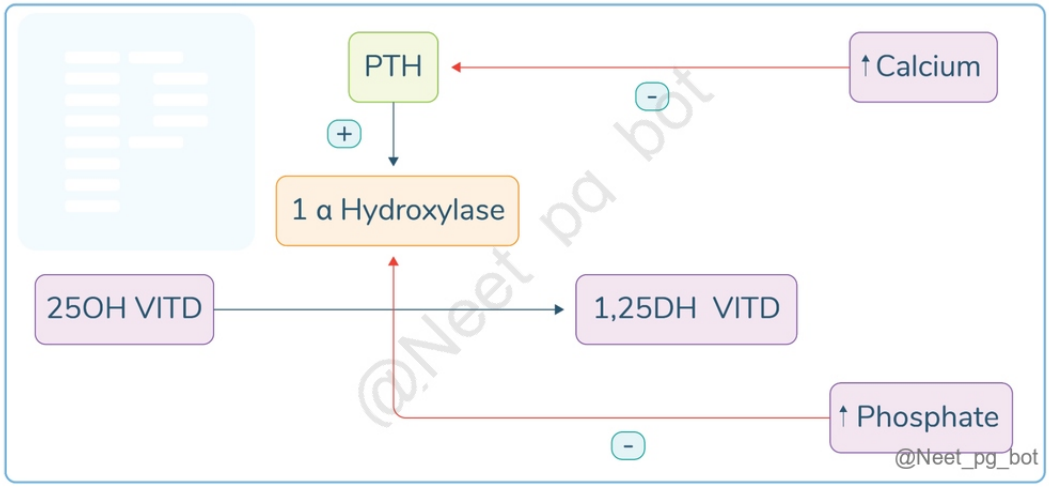
C. High parathormone

D. 1,25-dihydroxycholecalciferol

Correct Answer: C. High parathormone

■ *Listen to Audio 1*

Explanation: The parathyroid hormone has a positive effect on 1 alpha-hydroxylase enzyme, which enhances the conversion of 25(OH)2-D3 to 1,25(OH)2-D3. Regulation of Vit-D synthesis: The rate-limiting step of vitamin D synthesis is the conversion of 25(OH)2-D3 to 1,25(OH)2-D3 via 1- α hydroxylase enzyme. 1-α hydroxylase gets activated by parathyroid hormone (Option C). High calcium and phosphate levels inactivate 1-α hydroxylase activity . (Options A and B ruled out) 1,25-DH vit D increases calcium levels, which in turn inactivates 1-α hydroxylase (Option D ruled out)



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 538, 539

↑ *Back to this question in Part A*

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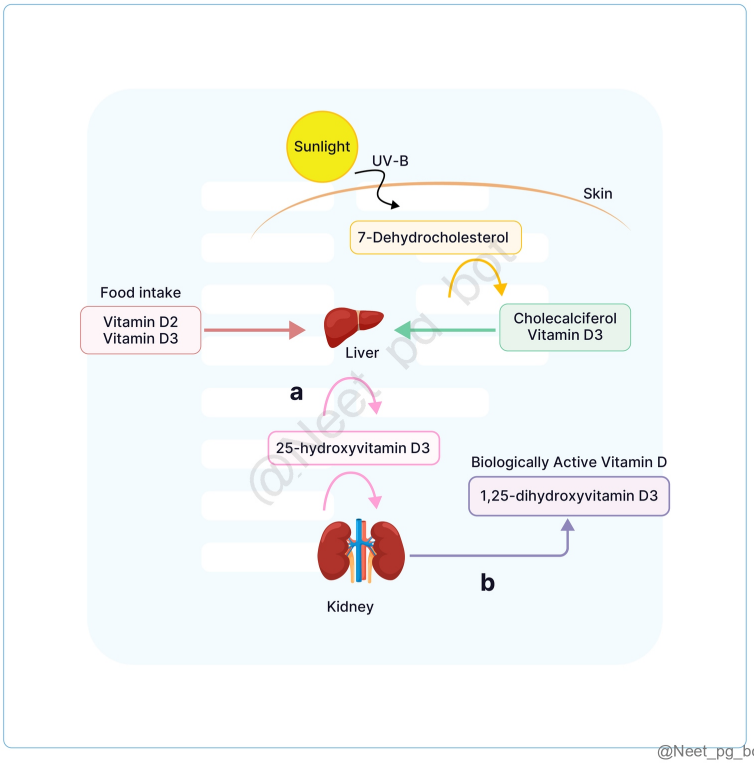
Q10: Identify the enzymes marked as a and b respectively in the flow chart, as related to vitamin D synthesis: (Q844941)

A. α-25 alpha hydroxylase, liver; β-1 alpha hydroxylase, kidney

B. α-1 alpha hydroxylase, kidney; β-25 alpha hydroxylase, liver

C. α-1 alpha hydroxylase, liver; β-25 alpha hydroxylase, kidney

D. α-25 alpha hydroxylase, kidney; β-1 alpha-hydroxylase, liver

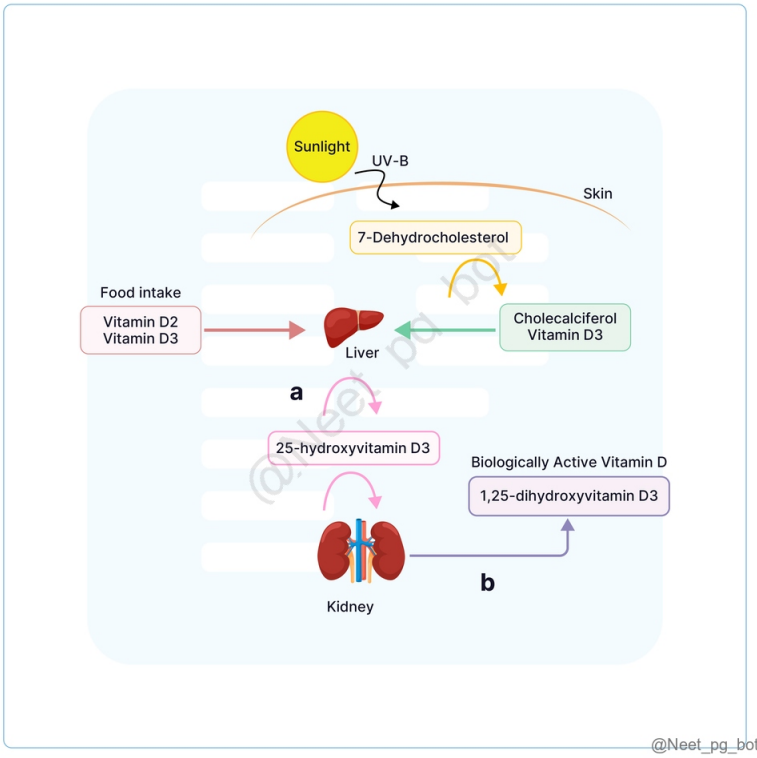


Correct Answer: A. α-25 alpha hydroxylase, liver; β-1 alpha hydroxylase, kidney

■ *Listen to Audio 1*

Explanation: Vitamin D synthesis: (a: α- 25 alpha hydroxylase; b: β-1 alpha hydroxylase) Skin: Most of the precursors for 1,25(OH)2-D3 synthesis is produced in the malpighian layer of epidermis from 7-dehydrocholesterol via UV light-mediated photolysis reaction. Conversion efficiency is affected by UV exposure and skin pigmentation. Aging reduces 7-dehydrocholesterol levels. Liver: Vitamin D is transported to the liver by vitamin D-binding protein. In the liver , vitamin D is hydroxylated to 25(OH)2-D3 (25 dihydroxy cholecalciferol) by 25 alpha-hydroxylase . This process requires magnesium, NADPH, molecular oxygen, and an uncharacterized cytoplasmic factor. Two enzymes are involved: NADPH-dependent cytochrome P450 reductase and cytochrome P450. 25(OH)2-D3 is the main circulating form of vitamin D and is transported to the kidney. Kidney: 25(OH)D3 is converted to the active form, 1,25(OH)2D3 (1,25 dihydroxy cholecalciferol), in the kidneys by the enzyme 1 alpha-hydroxylase . This conversion involves a three-component monooxygenase system requiring NADPH, magnesium, molecular oxygen, and three enzymes: renal ferredoxin reductase, renal ferredoxin, and cytochrome P450. 1,25(OH)2D3 is the most potent active metabolite of vitamin D. Regulation of Vitamin D Activation: Regulator Effect on 1-α hydroxylase Parathyroid hormone (PTH) Stimulates Low serum phosphate Stimulates Hypocalcemia Stimulates High phosphate Inhibits High calcitriol Negative feedback

FGF-23 Inhibits



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 497-499

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: Which of the following features is NOT associated with a deficiency of Vitamin K? (Q844940)

A. Hemorrhagic disease of the newborn

B. Prolonged prothrombin time

C. Hyperbilirubinemia

D. Mucous membrane hemorrhage

Correct Answer: C. Hyperbilirubinemia

■ *Listen to Audio 1*

Explanation: Hyperbilirubinemia is not associated with vitamin K deficiency. Instead, it is related to vitamin K toxicity , particularly in newborns, leading to conditions like hemolysis and kernicterus. Vitamin K Deficiency Vitamin K Toxicity Causes: Fat malabsorption (e.g., cystic fibrosis) Pancreatic insufficiency Causes: High doses of vitamin K Vitamin E deficiency Manifestations: Bleeding mucous membrane (Option D ruled out) Increased PT and Bleeding time. (Option B ruled out) Hemorrhagic disease of the newborn (Option A ruled out) Manifestations: Hemolytic anemia similar to Vitamin E deficiency Hemolytic anemia ➜ Hemolytic jaundice ➜ Kernicterus and brain damage.

References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 475, 476

↑ *Back to this question in Part A*

@Neet_pg_bot

Q12: Which vitamins are least likely to accumulate in the body's adipose tissue due to its solubility characteristics? (Q844929)

A. Retinol

B. Ergocalciferol

C. Tocopherol

D. Pantothenic acid

Correct Answer: D. Pantothenic acid

■ *Listen to Audio 1*

Explanation: Pantothenic acid is a water-soluble vitamin (B5) and is not stored in significant amounts in adipose tissue. Water-soluble vitamins are generally excreted in the urine and do not accumulate in the body. Vitamin Sources A Retinol Retinal Retinoic acid Beta-carotene Milk, butter, cream, cheese, egg yolk, Beta carotene, carrots, papaya D Ergocalciferol (Vitamin D2) Cholecalciferol (Vitamin D3) Fish liver oil, fish, egg yolk, milk E Tocopherol (specifically, alpha-tocopherol) Vegetable oil, Wheat germ oil, sunflower oil, safflower oil, cottonseed oil K Phylloquinone (Vitamin K1) Menaquinone (Vitamin K2) Menadione (Vitamin K3, a synthetic form) Phylloquinone: Green vegetables Menaquinones: Synthesized by intestinal bacteria

References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 465, 469
https://drive.google.com/file/d/1UH2IVlcKfETK3gV9RaszgjPcHWGW1_mt/view https://www.uptodate.com/contents/overview-of-vitamin-e?sectionName=SOURCES&search=vitamin%20E&topicRef=5368&anchor=H2&source=see_link#H2

↑ *Back to this question in Part A*

@Neet_pg_bot

Q13: Which of the following statements about Vitamin K is incorrect? (Q844938)

A. Vitamin K is crucial for the activation of blood clotting factors.

B. Vitamin K2 is a lipid-soluble form produced by intestinal bacteria.

C. Menadione is a natural water-soluble form of Vitamin K.

D. Vitamin K aids in the carboxylation of bone proteins.

Correct Answer: C. Menadione is a natural water-soluble form of Vitamin K.

■ *Listen to Audio 1*

Explanation: Menadione is a water-soluble form of Vitamin K widely used in clinical practice. It can be metabolised to Phylloquinone, but it's a Synthetic form, not a natural form. Vitamin K: Fat-soluble vitamin that acts as a coenzyme or prosthetic group for gamma-carboxylase. Calcium-dependent proteins that rely on Vitamin K: Clotting factors II, VII, IX, and X. (Option A ruled out) Anticoagulant factors like protein C and S. Osteocalcin (promotes bone mineralization by depositing calcium). Forms of Vitamin K: Phylloquinone (K1): Found in green vegetables, this form has an isoprenoid side chain with 20 carbons. Menaquinones (K2): Synthesized by intestinal bacteria, these have varying side chain lengths and an isoprenoid side chain containing 30 carbons, which is lipid soluble. (Option B ruled out) Menadione (K3): A synthetic form which is water-soluble. Menadione and menadione diacetate can be metabolized into phylloquinone. Vitamin K exists in two forms: Epoxide and Hydroquinone form. Functions : Blood Clotting. Bone Health - Carboxylating glutamate to glutamic acid in proteins like osteocalcin is involved in bone mineralization. (Option D ruled out)

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 540.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q14: All of the following are deficiency manifestations of Vitamin E except: (Q844937)

A. Spinocerebellar ataxia

B. Ophthalmoplegia

C. Hemorrhagic tendency

D. Peripheral neuropathy

Correct Answer: C. Hemorrhagic tendency

■ *Listen to Audio 1*

Explanation: Vitamin E Toxicity: Interference with Vitamin K: Antagonize vitamin K, impairing blood coagulation and increasing bleeding risk. Interaction with Warfarin: Enhance the anticoagulant effect of warfarin, leading to a greater risk of bleeding complications. Effects on Platelet Aggregation: Vitamin E may impair platelet function, potentially increasing the risk of bleeding. Enterocolitis in Infants: Associated with an increased risk of necrotizing enterocolitis in premature infants. Vitamin E Deficiency manifestations 1. Hemolytic anemia Due to the destruction of red blood cells by oxidative stress. Vitamin E acts as an antioxidant that protects cell membranes from damage by free radicals■. 2. Spinocerebellar ataxia (Option A ruled out) Degeneration of the cerebellum and spinal cord leading to imbalance, incoordination and ataxia. 3. Peripheral Neuropathy (Option D ruled out) Insufficient antioxidant protection damages peripheral nerves, leading to symptoms such as numbness, tingling, and muscle weakness■. 4. Eyes Pigment retinopathy Ophthalmoplegia (Option B ruled out) Nystagmus

References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 474, 523. https://www.sciencedirect.com/science/article/abs/pii/S1367593123000662#:~:text=Selenium%20and%20vitamin%20E%20work,by%20selenocysteine%2Dcontaining%20glutathione%20peroxidase

↑ *Back to this question in Part A*

@Neet_pg_bot

Q15: Which of the following is NOT a chain-breaking antioxidant? (Q844936)

A. α-Tocopherol

B. β-Carotene

C. Superoxide dismutase

D. Glutathione Peroxidase

Correct Answer: D. Glutathione Peroxidase

■ *Listen to Audio 1*

Explanation: Glutathione Peroxidase is a preventive antioxidant, not a chain-breaking antioxidant. Antioxidants are substances that help control and reduce lipid peroxidation by neutralizing free radicals, which can cause cellular damage. Classification of Antioxidants: 1. Preventive Antioxidants: Reduce the rate of chain initiation. Examples: Glutathione Peroxidase (Option D) Catalase Selenium 2. Chain-Breaking Antioxidants (Option A, B and C ruled out): Interfere with chain propagation by stabilizing free radicals. Examples: α-Tocopherol (Vitamin E) β-Carotene (Vitamin A) Vitamin C Superoxide Dismutase (SOD)

References:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3614697/

↑ *Back to this question in Part A*

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Q16: A 17-year-old girl presents with severe acne on her face and back. She was previously treated with benzoyl peroxide and oral doxycycline and found no improvement. Physical examination shows comedones, pustules, inflammatory papules, and facial scarring. Which of the following is the next best treatment? (Q844935)

A. All trans-retinoic acid

B. Tocopherol

C. Phylloquinone

D. 13 cis-retinoic acid

Correct Answer: D. 13 cis-retinoic acid

■ *Listen to Audio 1*

Explanation: 13 cis retinoic acid (isotretinoin) is used to treat nodulocystic acne, which is refractory to treatment with topical medication and oral antibiotics. 13 cis retinoic acid (isotretinoin): Mechanism Inhibits sebum production Inhibit keratinocyte proliferation Use Nodulocystic acne Adverse effects Dry skin Headache Alopecia Idiopathic intracranial hypertension Teratogenic Contraindications Pregnancy Liver disease Precautions Negative pregnancy test Two contraception methods All trans-retinoic acid (Option A): Used for Acute promyelocytic leukemia (AML M3). Binds to retinoic acid receptors, inducing cell differentiation. Tocopherol (Option B): Antioxidant (prevents free radical damage). Used in Non-alcoholic steatohepatitis. (NASH) Phylloquinone (Option C): Known as vitamin K1 (Other forms: Menadione, menaquinone (K2)) Cofactor for gamma-carboxylation of coagulation. Deficiency leads to bleeding disorders.

References:

Lippincott Illustrated Reviews, Biochemistry, 7th Edition, Page 1113. Harper’s Illustrated Biochemistry, 32nd Edition, Page 537.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: Which of the following is most likely observed in patients with vitamin A toxicity? (Q844934)

A. Decreased CSF pressure

B. Decreased bone mineral density

C. Thinning of long bones

D. Increased appetite

Correct Answer: B. Decreased bone mineral density

■ *Listen to Audio 1*

Explanation: Vitamin A toxicity increases osteoclast activity, causing endosteal bone resorption → ↓ bone mineral density (BMD). This makes the bones osteoporotic, fragile, and prone to fractures. Chronic hypervitaminosis A also stimulates abnormal periosteal bone formation, resulting in bony exostoses (periosteal outgrowths) and cortical irregularities. Thus, bones may show periosteal thickening or exostoses externally, but internally the bone is weak due to resorption. Net clinical outcome --> weak bones with reduced BMD despite external bony prominences. Decreased CSF pressure (Option A) : Wrong. Hypervitaminosis A causes increased intracranial pressure → pseudotumor cerebri. Thinning of long bones (Option C) : Not accurate. Though bone becomes weaker from inside (endosteal resorption), externally there can be periosteal bone deposition and bony exostoses, not uniform thinning. Increased appetite (Option D) : Opposite effect. Vitamin A toxicity causes loss of appetite, nausea, vomiting.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 538

Lippincott Illustrated Reviews, Biochemistry, 7th Edition, Page 1113

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 469

www.ncbi.nlm.nih.gov/books/NBK532916/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q18: Which of the following statements about Vitamin A deficiency is not correct? (Q844933)

A. Causes rough skin and atrophy of epithelial tissues

B. Prolonged deficiency causes keratomalacia

C. Preventable cause of blindness in children under 5

D. Increased retinol-binding protein synthesis is a cause

Correct Answer: D. Increased retinol-binding protein synthesis is a cause

■ *Listen to Audio 1*

Explanation: Increased retinol-binding protein synthesis is a cause Explantion: Causes for Vitamin A Deficiency : Decreased intake. Obstructive jaundice: Defective absorption. Liver cirrhosis: Reduced synthesis of RBP (retinol binding protein). Severe malnutrition: Lack of amino acids for RBP synthesis. (Option D) Chronic nephrosis: RBP excretion through urine. Vitamin A Deficiency Ocular Manifestations : Vitamin A deficiency is the most significant preventable cause of blindness, especially in children below 5 in developing countries. (Option C ruled out) Early Signs: Loss of sensitivity to green light Difficulty adapting to dim light Night blindness Prolonged Deficiency: Xerophthalmia: Dry, thick, keratinized conjunctiva and cornea. If untreated, it leads to corneal ulceration and ultimately results in blindness due to the formation of opaque scar tissue Bitot's Spots: Grayish-white triangular plaques firmly adherent to the conjunctiva Keratomalacia: When xerophthalmia persists for a longer time, it progresses to keratomalacia (corneal softening) (Option B ruled out) Immune Function: Deficiency increases susceptibility to infections Infection reduces retinol-binding protein synthesis, lowering vitamin A levels and impairing immune responses (Option D) Skin and Mucous Membrane Lesions: Follicular Hyperkeratosis (Phrynoderma): Causes rough skin due to hyperkeratinization of the follicular epithelium Keratinizing Metaplasia: Affects respiratory, gastrointestinal, and genitourinary tracts Epithelium is atrophied. (Option A ruled out) Other General Manifestation : Growth retardation due to defective synthesis of chondroitin sulfate Decreased protein synthesis

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 538

Lippincott Illustrated Reviews, Biochemistry, 7th Edition, Page 1112, 1113

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 467,468

↑ *Back to this question in Part A*

@Neet_pg_bot

Q19: A 35-year-old woman reports difficulty seeing in low light. She has a poor diet, lacking in fruits and vegetables. Dark adaptation is delayed on testing. What is the most likely cause of her symptoms? (Q844932)

A. Impaired retinol storage

B. Reduced 11-cis-retinal regeneration

C. Defective retinoic acid transport

D. Accumulation of all-trans-retinal

Correct Answer: B. Reduced 11-cis-retinal regeneration

■ *Listen to Audio 1*

Explanation: Vitamin A Deficiency - ↓ Recycling of All-trans-Retinal; ↓ Regeneration of 11-cis-Retinal; ↑ Prolonged Dark Adaptation; ↑ Night Blindness Vitamin A deficiency impairs the recycling of all-trans-retinal and slows the regeneration of 11-cis-retinal, leading to prolonged dark adaptation and night blindness. Impact of Vitamin A on the Visual Cycle : Adequate Vitamin A 11-cis-Retinal Availability: With sufficient vitamin A, 11-cis-retinal is readily available to bind with opsin, forming rhodopsin. This is crucial for phototransduction, where light is converted into electrical signals in the retina Efficient Dark Adaptation: The rapid conversion of 11-cis-retinal to rhodopsin supports quick adaptation to low-light conditions, enabling effective dark adaptation ↑Recycling of All-trans-Retinal; ↑ 11-cis-Retinal & Rhodopsin Formation; ↓ Rapid Dark Adaptation; ↓ Night blindness Impaired retinol storage (Option A) could lead to vitamin A deficiency, but this option is vague and doesn't specifically explain the mechanism affecting night vision. The critical issue in night blindness is the regeneration of 11-cis-retinal, which is directly involved in the visual cycle. Defective retinoic acid transport (Option C): Retinoic acid is involved in gene expression and cellular differentiation, not in the visual cycle. A defect in its transport would likely lead to other systemic symptoms, not specifically night blindness or delayed dark adaptation. Accumulation of all-trans-retinal (Option D) could potentially cause oxidative stress, but this is more related to photoreceptor damage rather than the primary issue in night blindness, which is the lack of 11-cis-retinal necessary for vision in low light.

References:

www.ncbi.nlm.nih.gov/pmc/articles/PMC8172435/

academic.oup.com/pnasnexus/article/2/6/pgad167/7173164?login=false

www.ncbi.nlm.nih.gov/pmc/articles/PMC3281612/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q20: Which enzyme is responsible for the isomerization of all-trans retinyl ester to 11-cis retinol in the retinal pigment epithelium (RPE)? (Q844931)

A. Lecithin-retinol acyltransferase

B. All-trans-retinol dehydrogenase

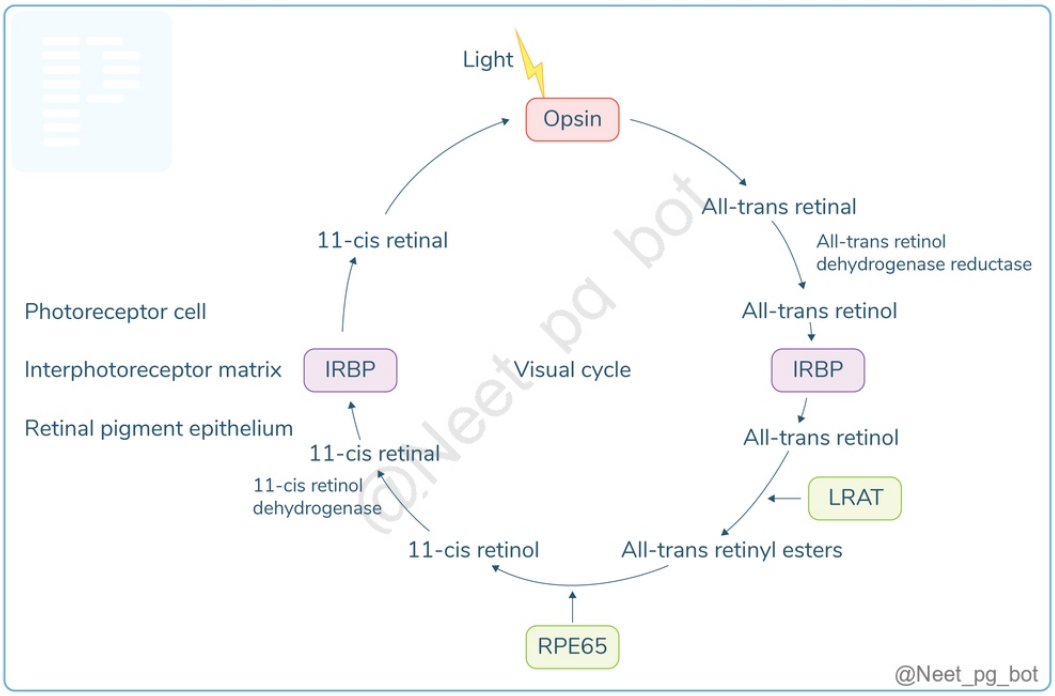
C. Retinoid isomerohydrolase

D. 11-cis-retinol dehydrogenase

Correct Answer: C. Retinoid isomerohydrolase

■ *Listen to Audio 1*

Explanation: Retinoid isomerohydrolase (RPE65) is the enzyme responsible for the isomerization of all-trans retinyl ester to 11-cis retinol, a crucial step in the visual cycle. The visual cycle, also known as Wald's visual cycle, involves the conversion of light into electrical signals in the photoreceptors of the retina and the regeneration of visual pigments to maintain visual sensitivity. Visual Cycle Regeneration - Regenerated 11 cis retinal from All-trans retinal Step Site Reaction Enzyme/Mechanism 1 Photoreceptor outer segment All-trans-Retinal + NADPH → All-trans-Retinol + NADP+ All-trans-Retinol Dehydrogenase (Option B) 2 Retinal pigment epithelium (RPE) cells. Transport to retinal pigment epithelium (RPE) cells. Cellular Retinoid Binding Proteins 3 Esterification : All-trans-Retinol + Fatty Acyl CoA → All-trans-Retinyl Ester + CoA Lecithin-Retinol Acyltransferase (LRAT) (Option A) 4 Isomerization and Hydrolysis: All-trans-Retinyl Ester → 11-cis-Retinol RPE65 (retinoid isomerohydrolase) (Option C) 5 Oxidation: 11-cis-Retinol + NAD+ → 11-cis-Retinal + NADH + H+ 11-cis-Retinol Dehydrogenase (Option D) 6 Photoreceptor outer segment Transport Back to Photoreceptors Cellular Retinoid Binding Proteins 7 11-cis-Retinal + Opsin → Rhodopsin (or Cone Opsin) -



References:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3073571/pdf/nihms254716.pdf Harper's Illustrated Biochemistry, 32nd Edition, Page 537 - 538 Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 465

↑ *Back to this question in Part A*

@Neet_pg_bot

Q21: Which form of vitamin A is stored in the liver and must bind to retinol-binding protein (RBP) for transport to other tissues? (Q844930)

A. Retinoic acid

B. Retinal

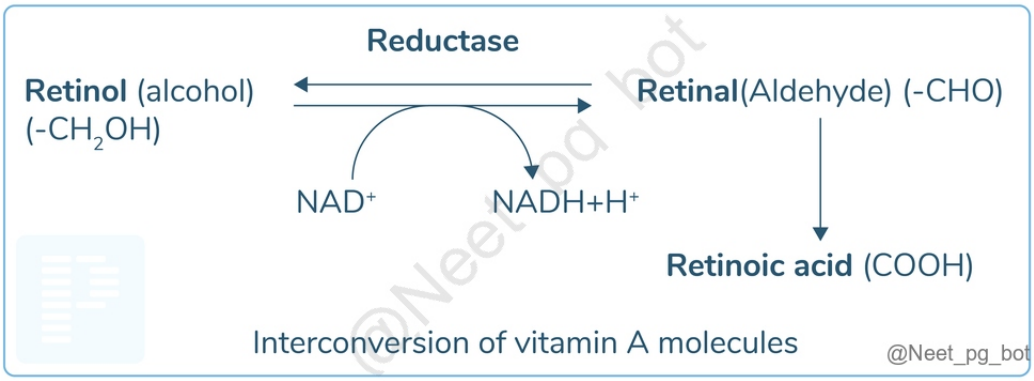
C. Retinol

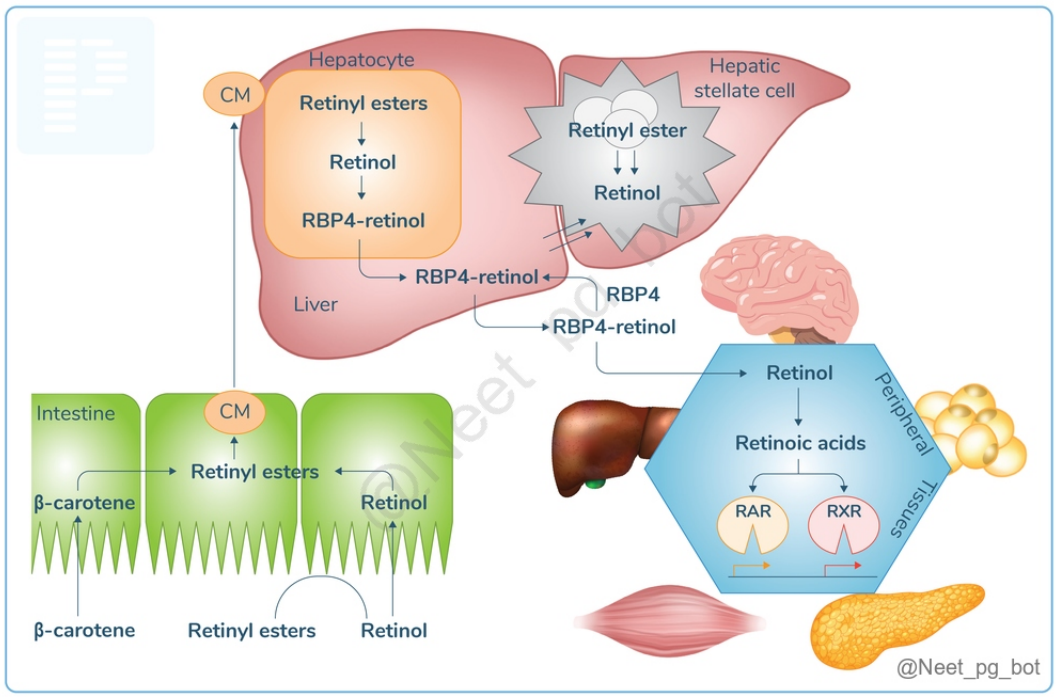
D. β-carotene

Correct Answer: C. Retinol

■ *Listen to Audio 1*

Explanation: Retinol is the form of vitamin A that is stored in the liver as retinyl esters. For transport to other tissues, retinol must bind to retinol-binding protein (RBP). Absorption and Transport of Retinoids: Retinoids Structurally related molecules with vitamin A activity. Functions Vision, reproduction, growth, epithelial tissue maintenance, immune function. Types of Retinoids Retinol (Option C) Primary alcohol containing a ß-ionone ring with an unsaturated side chain Storage form of vitamin A as retinyl ester with long-chain fatty acid. Retinal (Option B) Aldehyde derived from oxidation of retinol. Retinal and retinol can readily be interconnected. Retinoic Acid (Option A) Acid form derived from oxidation of the retinal. Cannot be reduced in body, so can't give rise to retinal or retinol. Binds to retinoic acid receptors (RARs) in target tissue nuclei. Regulates gene expression by modulating RNA synthesis and protein production. 2 Forms: ATRA & Isotretinoin All-trans Retinoic Acid (ATRA) Promotes growth and differentiation of epithelial cells. Mild acne treated with topical tretinoin (all-trans retinoic acid). Treatment in acute promyelocytic leukemia (APL), a subtype of acute myeloid leukemia (AML). 13-cis Retinoic Acid (Isotretinoin) Suppresses keratinization; used to treat resistant acne and Harlequin ichthyosis. Induces apoptosis in sebaceous glands, reducing sebum production. Beta-carotene (Option D): It is found in plant-based foods, β-carotene is a provitamin A. The human body uses an intestinal enzyme to cleave one beta-carotene molecule to produce two retinal molecules, which can then be reduced to retinol.





References:

Lippincott Illustrated Reviews, Biochemistry, 7th Edition, Chapter 28 - XI. VITAMIN A

↑ *Back to this question in Part A*

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• [11_Vitamins] 030 Water-Soluble Vitamins - I

« Back to Folder Directory

[11_Vitamins] 030 Water-Soluble Vitamins - I — Quick Jump

Q1 (p. 1232)	*Q2 (p. 1233)*	*Q3 (p. 1234)*	*Q4 (p. 1235)*	*Q5 (p. 1235)*	*Q6 (p. 1236)*
Q7 (p. 1237)	*Q8 (p. 1238)*	*Q9 (p. 1239)*	*Q10 (p. 1239)*	*Q11 (p. 1240)*	*Q12 (p. 1242)*
Q13 (p. 1243)	*Q14 (p. 1244)*	*Q15 (p. 1245)*	*Q16 (p. 1246)*	*Q17 (p. 1247)*	*Q18 (p. 1248)*
Q19 (p. 1249)	*Q20 (p. 1252)*	*Q21 (p. 1253)*			

Q1: Which of the following statements about vitamin B12 absorption is true? (Q843177)

A. Intrinsic factor is secreted by the pancreas and binds to inactive forms of vitamin B12.

B. Cobalophilin in the saliva binds vitamin B12 initially and is hydrolyzed in the duodenum.

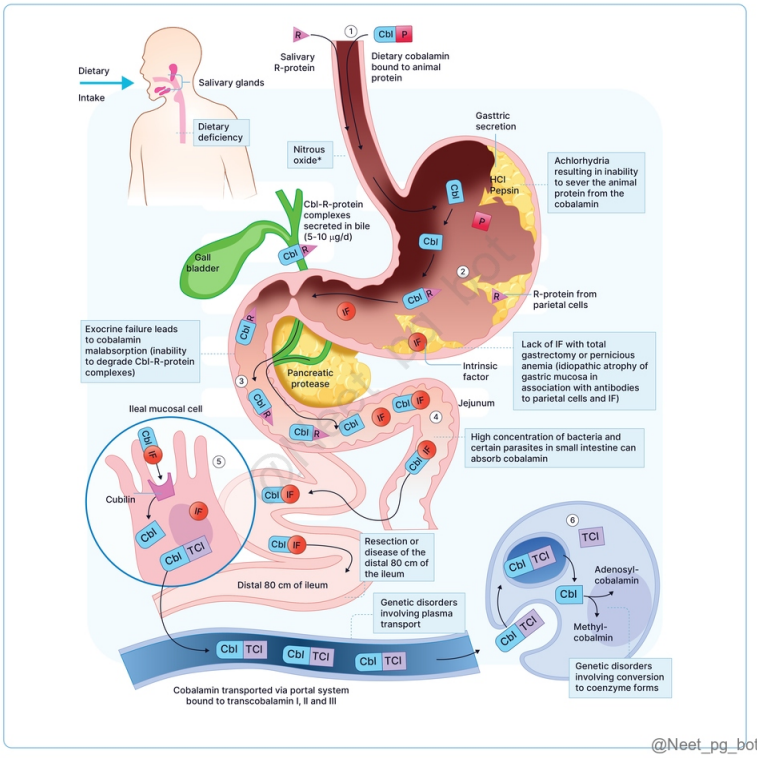
C. Gastric acid and pepsin are not necessary for Vitamin B12 absorption

D. Ileal receptors bind directly to vitamin B12 without the need for intrinsic factors.

Correct Answer: B. Cobalophilin in the saliva binds vitamin B12 initially and is hydrolyzed in the duodenum.

■ *Listen to Audio 1*

Explanation: Cobalophilin (haptocorrin) from saliva binds vitamin B12 in the stomach and protects it from acid. In the duodenum, pancreatic enzymes hydrolyze this complex, releasing B12 to bind intrinsic factor (IF) for absorption in the ileum. Vitamin B12 absorption steps: Release in stomach: Gastric acid and pepsin free B12 from dietary proteins. Binding to cobalophilin: Salivary haptocorrin binds and protects B12. Release in duodenum: Pancreatic enzymes hydrolyze the B12–cobalophilin complex. Binding to intrinsic factor: IF (from parietal cells) binds free B12. Ileal absorption: IF–B12 complex binds to ileal receptors and is absorbed. Transport in blood: Absorbed B12 binds to transcobalamin II for systemic circulation Intrinsic factor is secreted by the pancreas and binds to inactive forms of vitamin B12 (Option A): Incorrect because intrinsic factor (IF) is secreted by parietal cells of the stomach , not the pancreas. It binds active vitamin B12 to enable ileal absorption. Gastric acid and pepsin are not necessary for vitamin B12 absorption (Option C): Incorrect because gastric acid and pepsin are essential to release vitamin B12 from dietary proteins in the stomach. Without them, B12 cannot be freed for absorption. Ileal receptors bind directly to vitamin B12 without the need for intrinsic factor (Option D): Incorrect because ileal receptors recognize the IF–B12 complex , not free B12. Intrinsic factor is required for absorption.



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References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 544

↑ *Back to this question in Part A*

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Q2: A 35-year-old woman presents with fatigue, bleeding gums, easy bruising, and poor wound healing. Laboratory tests confirm scurvy. She reports a diet low in fruits and vegetables for several months. Which nutrient deficiency can mimic these symptoms? (Q845064)

A. Iron Deficiency

B. Zinc Deficiency

C. Folate Deficiency

D. Vitamin B12 Deficiency

Correct Answer: B. Zinc Deficiency

■ *Listen to Audio 1*

Explanation: The patient has scurvy, caused by vitamin C deficiency from a diet low in fruits and vegetables. Clinical features of scurvy include fatigue, bleeding and swollen gums, easy bruising, and poor wound healing. Vitamin C is essential for collagen synthesis, and its deficiency weakens connective tissue, leading to these manifestations. Zinc is a cofactor for metalloproteinases, enzymes crucial for collagen remodeling and tissue repair. Zinc deficiency can therefore mimic scurvy, causing delayed wound healing, skin lesions, and tissue repair defects, especially in patients with poor dietary intake. Acrodermatitis enteropathica is a rare inherited cause of severe zinc deficiency. In patients with restricted diets, both vitamin C and zinc deficiencies should be considered, as they synergistically impair wound healing and collagen integrity. Iron deficiency (Option A): Causes microcytic anemia with fatigue, pallor, and sometimes pica. Does not affect collagen synthesis or wound healing, so it cannot mimic scurvy symptoms like bleeding gums or easy bruising. Folate deficiency (Option C): Leads to megaloblastic anemia due to impaired DNA synthesis. Clinical features include fatigue, pallor, and sometimes glossitis, but does not cause connective tissue defects or poor wound healing. Vitamin B12 deficiency (Option D): Causes megaloblastic anemia and neurological symptoms (e.g., paresthesia, weakness). Does not affect collagen formation or tissue repair, so it cannot mimic scurvy.

References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 497, 522

↑ *Back to this question in Part A*

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Q3: Which of the following statements regarding the avidin-biotin system is not true? (Q845063)

A. Avidin has a higher affinity for biotin than typical antigen-antibody interactions

B. Avidin can bind up to four molecules of biotin, enhancing assay sensitivity

C. Consumption of raw egg whites may lead to biotin deficiency due to avidin-binding biotin

D. Avidin is heat-stable and retains its binding capacity after boiling eggs

Correct Answer: D. Avidin is heat-stable and retains its binding capacity after boiling eggs

■ *Listen to Audio 1*

Explanation: Avidin is a glycoprotein found in raw egg whites that binds biotin with high affinity. Biotin binding by avidin prevents its absorption in the intestine, potentially leading to deficiency. Avidin is heat-labile ; cooking denatures it, rendering it incapable of binding biotin. Since avidin loses its function when exposed to heat, it does not retain its binding capacity after boiling eggs. Avidin-Biotin System: Aspect Details Avidin Glycoprotein found in egg whites; binds biotin with high affinity Biotin (Vitamin H/Vitamin B7) Water-soluble vitamin (B7) is essential for carboxylation reactions in metabolism Affinity (Option A ruled out) Avidin-biotin binding is among the strongest non-covalent interactions (Kd ~10■¹■ M) Binding Sites (Option B ruled out) Each avidin molecule has four binding sites, making it useful in high-sensitivity biochemical assays Biotin Deficiency (Option C ruled

out) Chronic raw egg consumption can lead to deficiency, causing dermatitis, alopecia, and neurological symptoms Heat Stability Avidin is heat-labile and denatured by cooking, preventing it from binding biotin Applications Used in biochemical assays like ELISA, immunohistochemistry, and affinity chromatography

References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 454, 486

↑ *Back to this question in Part A*

@Neet_pg_bot

Q4: Which of the following enzymes is not classified as a biotin-dependent carboxylase? (Q843396)

A. Acetyl CoA carboxylase

B. Pyruvate carboxylase

C. Propionyl CoA carboxylase

D. Malic enzyme

Correct Answer: D. Malic enzyme

■ *Listen to Audio 1*

Explanation: Biotin-dependent carboxylases catalyze carboxylation reactions, where CO■ is added to a substrate. The major biotin-dependent carboxylases include: Acetyl CoA carboxylase → Fatty acid synthesis Pyruvate carboxylase → Gluconeogenesis Propionyl CoA carboxylase → Odd-chain fatty acid metabolism Malic enzyme , on the other hand, is not a carboxylase . It converts malate to pyruvate by decarboxylation , generating NADPH in the process. Biotin-Independent Enzyme Enzyme Reaction Key Point Malic enzyme Malate → Pyruvate + CO■ Decarboxylation reaction , produces NADPH Biotin-Dependent Carboxylases and Their Functions Enzyme Function Pathway Acetyl CoA carboxylase (Option A ruled out) Converts acetyl CoA → malonyl CoA Fatty acid synthesis Pyruvate carboxylase (Option B ruled out) Converts pyruvate → oxaloacetate Gluconeogenesis Propionyl CoA carboxylase (Option C ruled out) Converts propionyl CoA → methylmalonyl CoA Odd-chain fatty acid metabolism

References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 486

↑ *Back to this question in Part A*

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Q5: A 55-year-old man on high-dose niacin for hyperlipidemia develops blurred vision and macular edema. What is the mechanism behind niacin-induced macular edema? (Q843395)

A. Increased prostaglandin synthesis

B. Inhibition of retinal blood flow

C. Direct toxicity to retinal pigment epithelium

D. Enhanced inflammation in the retina

Correct Answer: A. Increased prostaglandin synthesis

■ *Listen to Audio 1*

Explanation: Niacin toxicity increases prostaglandin synthesis , causing vasodilation and retinal vascular leakage, leading to macular edema and flushing. Stopping niacin or using prostaglandin inhibitors (e.g., aspirin, NSAIDs) helps resolve symptoms. Niacin Toxicity and Ocular Complications Flushing: Prostaglandin-mediated vasodilation → redness, warmth, tingling Macular edema: Prostaglandin-induced vascular permeability → blurred or central vision loss Hepatotoxicity: Disrupted lipid metabolism → elevated liver enzymes, jaundice Hyperglycemia: Alcohol-induced insulin resistance → increased blood glucose Hyperuricemia: Decreased renal uric acid excretion → risk of gout Inhibition of retinal blood flow (Option B): Incorrect because niacin increases rather than decreases retinal blood flow due to vasodilation. Direct toxicity to retinal pigment epithelium (Option C): Incorrect because niacin does not cause direct retinal pigment epithelial damage . Enhanced inflammation in the retina (Option D): Incorrect because macular edema in niacin toxicity is not due to inflammatory cell infiltration , but rather increased prostaglandin-induced vascular leakage .

References:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4458328/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: A 28 year old pregnant woman presents with complaints of fatigue and weakness. Her dietary history reveals she follows a strict vegetarian diet. Blood tests reveal megaloblastic anaemia. Which of the following complications is most likely associated with her condition if left untreated? (Q843393)

A. Increased risk of gestational diabetes

B. Neurological problems in the mother and cognitive impairments in the foetus

C. Higher incidence of hypertension during pregnancy

D. Increased risk of maternal infections

Correct Answer: B. Neurological problems in the mother and cognitive impairments in the foetus

■ *Listen to Audio 1*

Explanation: The patient's fatigue, weakness, pallor, mild jaundice, and megaloblastic anemia suggest vitamin B12 deficiency , which is common in strict vegetarians as B12 is found primarily in animal-based foods . Vitamin B12 is essential for DNA synthesis, myelin formation, and neurological function , making its deficiency particularly dangerous during pregnancy. Complications of Vitamin B12 Deficiency in Pregnancy: Maternal: Peripheral neuropathy, memory loss, depression, cognitive decline Fetal: Poor brain development, neural tube defects, developmental delay Hematological: Megaloblastic anemia, risk of preterm birth Increased risk of gestational diabetes (Option A): Incorrect . Vitamin B12 deficiency is not directly linked to gestational diabetes. Higher incidence of hypertension during pregnancy (Option C): Incorrect. No strong evidence links vitamin B12 deficiency to gestational hypertension or preeclampsia. Increased risk of maternal infections (Option D): Incorrect. While B12 deficiency weakens the immune system, it does not significantly increase maternal infections.

References:

https://www.ncbi.nlm.nih.gov/books/NBK441923/

↑ *Back to this question in Part A*

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Q7: After consuming illicit homemade alcohol at a local event, numerous individuals were hospitalized with severe abdominal pain, vomiting, malaise and vision changes. Which of the following cannot be used as an antidote for methanol poisoning? (Q843186)

A. Ethanol

B. Fomepizole

C. Folinic acid

D. Formic acid

Correct Answer: D. Formic acid

■ *Listen to Audio 1*

Explanation: Methanol poisoning occurs due to the metabolism of methanol into formic acid and formate , which are highly toxic and lead to: Metabolic acidosis (high anion gap) Optic nerve toxicity → Vision changes ("snowfield vision") Central nervous system depression Severe abdominal symptoms Antidotes work by preventing methanol metabolism or enhancing its detoxification. Antidotes for Methanol Poisoning: Antidote

Mechanism of Action Ethanol (Option A ruled out) Competitive inhibition of alcohol dehydrogenase (prevents methanol metabolism). Fomepizole (Option B ruled out) Direct inhibitor of alcohol dehydrogenase (preferred treatment). Folinic acid (Leucovorin) (Option D ruled out) Enhances formate metabolism into non-toxic byproducts. Hemodialysis Used in severe cases to remove methanol and formate.

References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page553
https://www.researchgate.net/publication/292237562_Hooch_tragedies_in_India_A_review
https://pubmed.ncbi.nlm.nih.gov/12216995/

↑ *Back to this question in Part A*

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Q8: A 50-year-old chronic alcoholic develops agitation, tremors, hallucinations, and autonomic instability three days after stopping alcohol. What is the most appropriate management? (Q843185)

A. Initiate antipsychotic medication

B. Prescribe selective serotonin reuptake inhibitors (SSRIs)

C. Administer benzodiazepines for sedation

D. Begin opioid replacement therapy

Correct Answer: C. Administer benzodiazepines for sedation

■ *Listen to Audio 1*

Explanation: The patient has delirium tremens (DTs), a severe alcohol withdrawal state with agitation, tremors, hallucinations, and autonomic instability occurring 48–96 hours after cessation. Benzodiazepines (diazepam, lorazepam, chlordiazepoxide) are first-line as they enhance GABA activity, control symptoms, and prevent seizures and cardiovascular complications. Timeline of Alcohol Withdrawal Time after last drink Clinical Manifestations 6–12 hours Tremors, anxiety, insomnia, headache, nausea, vomiting 12–24 hours Alcoholic hallucinosis (visual, auditory hallucinations) 24–48 hours Withdrawal seizures (generalized tonic-clonic) 48–96 hours Delirium tremens → confusion, agitation, hallucinations, autonomic instability Management of Alcohol Withdrawal Syndrome Severity Symptoms Treatment Mild Anxiety, insomnia, tremors Supportive care, thiamine, hydration Moderate Tachycardia, hypertension, diaphoresis, hallucinations Benzodiazepines (e.g., lorazepam, diazepam) Severe (Delirium Tremens) Confusion, hallucinations, seizures, autonomic instability High-dose benzodiazepines, ICU monitoring Initiate antipsychotic medication (Option A): Antipsychotics (e.g., haloperidol) can worsen seizures and prolong withdrawal . They are used only as an adjunct for persistent agitation after benzodiazepine administration. Prescribe SSRIs (Option B): SSRIs have no role in managing acute alcohol withdrawal or DTs. Mood stabilization is considered after withdrawal resolution . Begin opioid replacement therapy (Option D): Opioid therapy is for opioid withdrawal, not alcohol withdrawal . Alcohol withdrawal is managed with benzodiazepines .

References:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6286444/

↑ *Back to this question in Part A*

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Q9: A 45-year-old man with a history of heavy alcohol consumption for the past 10 years, presents with fatigue, abdominal discomfort, weight gain, and hepatomegaly. Laboratory tests show elevated liver enzymes, and a liver ultrasound reveals fatty liver. What commonly causes fatty liver in chronic alcohol consumption? (Q843183)

A. Decreased acetyl CoA levels

B. Increased pyruvate oxidation

C. Accumulation of acetaldehyde

D. Increased fatty acid oxidation

Correct Answer: C. Accumulation of acetaldehyde

■ *Listen to Audio 1*

Explanation: Chronic alcohol consumption leads to excessive production of acetaldehyde , a toxic intermediate of alcohol metabolism Acetaldehyde disrupts mitochondrial function and promotes fatty liver (hepatic steatosis) by: Impairing beta-oxidation of fatty acids Increasing triglyceride synthesis and storage in the liver Causing mitochondrial damage and oxidative stress This results in hepatomegaly, abnormal liver function tests, and progression to alcoholic liver disease Pathophysiology of Alcohol-Induced Fatty Liver Effect Mechanism Increased Acetaldehyde (Option A ruled out) Alcohol metabolism (via alcohol dehydrogenase) generates toxic acetaldehyde, which disrupts liver function. Inhibition of Fatty Acid Oxidation (Option D ruled out) High NADH/NAD+ ratio inhibits beta-oxidation, leading to fat accumulation in hepatocytes. Increased Lipogenesis Excess NADH promotes triglyceride synthesis, worsening hepatic steatosis. Mitochondrial Dysfunction Acetaldehyde causes oxidative stress and mitochondrial damage, impairing lipid metabolism. Increased pyruvate oxidation (Option B): Incorrect. Pyruvate is converted to lactate (not oxidized) due to excess NADH, leading to lactic acidosis, not fatty liver.

References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 141

↑ *Back to this question in Part A*

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Q10: A 45-year-old chronic alcoholic presents with confusion, tremors, diaphoresis, and hypoglycemia (50 mg/dL). He has been drinking heavily with poor nutrition. Which of the following statements about the effects of chronic alcoholism on nutritional deficiencies and glucose metabolism is false? (Q843181)

A. Chronic alcohol use can cause vitamin B12 deficiency despite normal blood levels

B. Folate deficiency in alcoholics leads to megaloblastic anemia

C. Chronic alcohol use causes hypoglycemia by increasing the NADH/NAD+ ratio

D. Chronic alcohol use causes hypoglycemia by decreasing the NADH/NAD+ ratio

Correct Answer: D. Chronic alcohol use causes hypoglycemia by decreasing the NADH/NAD+ ratio

■ *Listen to Audio 1*

Explanation: Chronic alcohol metabolism (via alcohol dehydrogenase and aldehyde dehydrogenase) generates excess NADH , leading to an increased NADH/NAD■ ratio , not decreased. This shift inhibits key gluconeogenic reactions: Pyruvate → Lactate Oxaloacetate → Malate As a result, gluconeogenesis is suppressed , causing hypoglycemia , especially in fasting or malnourished individuals. Chronic alcohol use can cause vitamin B12 deficiency despite normal blood levels (Option A): True – malabsorption and hepatic dysfunction impair B12 utilization. Folate deficiency in alcoholics leads to megaloblastic anemia (Option B): True – alcohol inhibits folate absorption and metabolism. Chronic alcohol use causes hypoglycemia by increasing the NADH/NAD■ ratio (Option C): True – elevated NADH inhibits gluconeogenesis.

References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page140

https://pubmed.ncbi.nlm.nih.gov/20206879/

https://www.psychiatryinvestigation.org/m/journal/view.php?number=744

https://www.medicinenet.com/alcohol_and_nutrition/article.htm

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: A 50-year-old patient presents with symptoms of fatigue and pallor. The physician suspects a vitamin B12 absorption issue and decides to perform the Schilling test. Which of the following statements about the Schilling test is correct? (Q843179)

A. The Schilling test involves administering one dose of vitamin B12 to assess absorption.

B. An abnormal result in stage 1 and a normal result in Stage 2 of the Schilling test typically suggests a lack of intrinsic factor.

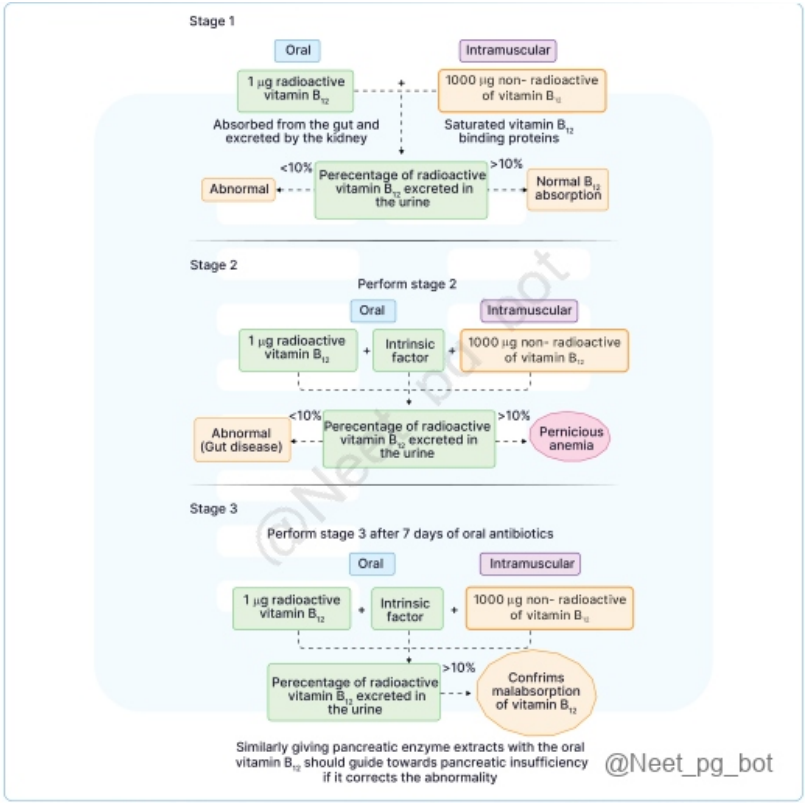
C. Radioactive vitamin B12 injection is given to saturate body stores.

D. Abnormal Stage 2 suggests intrinsic factor deficiency.

Correct Answer: B. An abnormal result in stage 1 and a normal result in Stage 2 of the Schilling test typically suggests a lack of intrinsic factor.

■ *Listen to Audio 1*

Explanation: The Schilling test evaluates vitamin B12 absorption by measuring urinary excretion of radioactive B12 across four stages to identify the cause of malabsorption. If Stage 1 is abnormal but Stage 2 normal , it indicates intrinsic factor deficiency (pernicious anemia) The Schilling test has four stages to identify the cause of vitamin B12 malabsorption. In Stage 1 , oral radioactive vitamin B12 is given; urinary excretion ≥8% indicates normal absorption, while <8% suggests general malabsorption. In Stage 2 , vitamin B12 is administered with intrinsic factor; if excretion normalizes, it indicates intrinsic factor deficiency (pernicious anemia), but if still low, another cause is likely. In Stage 3 , after giving antibiotics, normalization suggests bacterial overgrowth as the cause; persistent low levels indicate otherwise. In Stage 4 , after pancreatic enzymes, normalization confirms pancreatic insufficiency as the underlying problem. The Schilling test involves administering one dose of vitamin B12 to assess absorption (Option A): The test has multiple stages , not a single dose. Radioactive vitamin B12 injection is given to saturate body stores (Option C): IM non-radioactive B12 dose prevents liver uptake, not for saturation. Abnormal Stage 2 suggests intrinsic factor deficiency (Option D): A normal Stage 2 (not abnormal) indicates intrinsic factor deficiency ; persistent abnormality suggests other causes like bacterial overgrowth or pancreatic insufficiency .



References:

https://www.healthline.com/health/schilling-test

↑ *Back to this question in Part A*

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Q12: A 35-year-old woman with a white rice–based diet presents with breathlessness, palpitations, and edema. Examination shows distended neck veins and elevated lactate and pyruvate levels, suggesting thiamine deficiency. What is the likely diagnosis, and which enzyme activity confirms it? (Q843152)

A. Dry beriberi; pyruvate dehydrogenase

B. Maple Syrup Urine Disease (MSUD); branched-chain ketoacid dehydrogenase

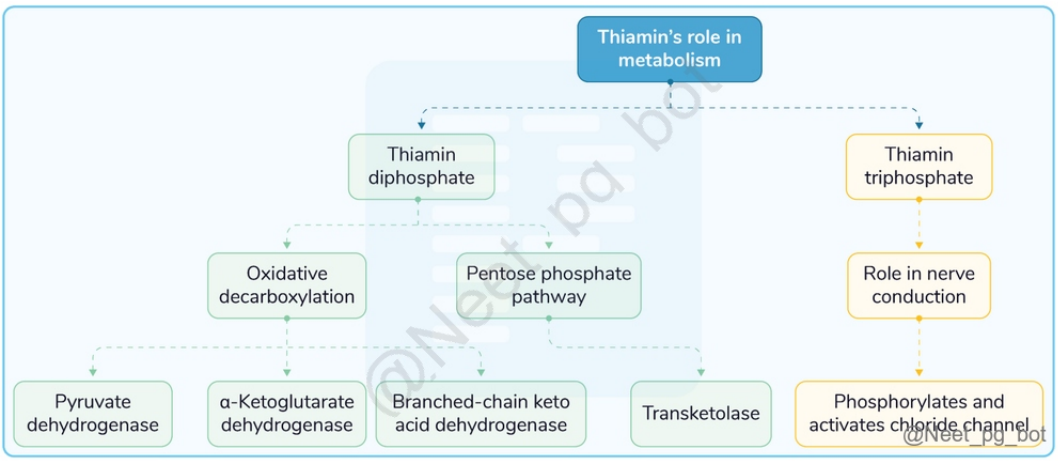
C. Wet beriberi; transketolase

D. Lactic acidosis; lactate dehydrogenase

Correct Answer: C. Wet beriberi; transketolase

■ *Listen to Audio 1*

Explanation: The woman’s symptoms— breathlessness, palpitations, leg and facial swelling, distended neck veins, and elevated lactate and pyruvate —indicate cardiovascular involvement due to thiamine (vitamin B■) deficiency . Her diet of polished white rice lacks thiamine because the aleurone layer (rich in vitamin B■) is removed during milling. Confirmatory Test: Reduced Erythrocyte transketolase activity — the earliest and most specific marker of thiamine deficiency. Thiamine acts as a coenzyme for pyruvate dehydrogenase , α-ketoglutarate dehydrogenase , and transketolase (in the pentose phosphate pathway). Deficiency impairs energy metabolism, causing lactic acidosis and cardiac dysfunction. Wet Beriberi: Cardiovascular type— breathlessness, palpitations, edema, distended neck veins, and high-output cardiac failure . Dry Beriberi : Mainly neurological— peripheral neuritis, weakness, sensory loss . (Option A ruled out) Maple Syrup Urine Disease (Option B ruled out): B1 acts as cofactor for decarboxylation of branched-chain keto acids, accumulation of branched-chain keto acids causes convulsions, mental retardation, vomiting, acidosis, coma, and death Lactic Acidosis (Option D ruled out): B1 acts as cofactor for pyruvate dehydrogenase, which converts pyruvate to acetyl-CoA. Impaired pyruvate metabolism, leading to increased lactate and pyruvate, causing lactic acidosis.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 541, 542 Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Pages 230, 477–479

↑ *Back to this question in Part A*

@Neet_pg_bot

Q13: Match the following vitamins with their respective functional assessment tests: 1. Vitamin B1 (Thiamine) a) Measure urinary methylmalonic acid 2. Vitamin B2 (Riboflavin) b) Measure transketolase activity in red blood cells 3. Vitamin B6 (Pyridoxine) c) Measure glutathione reductase activity in red blood cells 4. Vitamin B12 (Cobalamin) d) Measure activation of red blood cell transaminases with pyridoxal phosphate (Q843176)

A. 1-b, 2-c, 3-d, 4-a

B. 1-a, 2-b, 3-c, 4-d

C. 1-b, 2-d, 3-c, 4-a

D. 1-a, 2-c, 3-b, 4-d

Correct Answer: A. 1-b, 2-c, 3-d, 4-a

■ *Listen to Audio 1*

Explanation: Vitamin Functional Test Principle Vitamin B1 (Thiamine) RBC transketolase activity (b) Thiamin acts as a coenzyme for transketolase in the pentose phosphate pathway Measuring transketolase activity in red blood cells after adding thiamin pyrophosphate helps assess thiamin status Vitamin B2 (Riboflavin) RBC glutathione reductase activity (c) Riboflavin functions as a coenzyme for glutathione reductase (FAD-dependent).

Measuring glutathione reductase activity in RBCs helps assess riboflavin status. Vitamin B6 (Pyridoxine) Activation of RBC transaminases with pyridoxal phosphate (d) Pyridoxal phosphate (PLP) is a coenzyme for transaminases in amino acid metabolism. Measuring activation of RBC transaminases after PLP supplementation assesses B6 status. Vitamin B12 (Cobalamin) Urinary methylmalonic acid level (a) B12 is required for methylmalonyl-CoA mutase , which converts methylmalonyl-CoA to succinyl-CoA. B12 deficiency leads to increased urinary methylmalonic acid , making it a reliable marker.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 542–544, 546, 573

Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Pages 243, 483

↑ *Back to this question in Part A*

@Neet_pg_bot

Q14: Match the following deficiencies with their respective impacts: 1) Adenosylcobalamin Deficiency a) Elevated homocysteine levels 2) Methylcobalamin Deficiency b) Impaired energy production and chronic fatigue 3) Methyl Malonyl-CoA Mutase Deficiency c) Accumulation of methylmalonyl-CoA 4) Methionine Synthase Deficiency d) Pernicious anaemia with immature red blood cells (Q843173)

A. 1-b, 2-a, 3-c, 4-d

B. 1-a, 2-d, 3-b, 4-c

C. 1-b, 2-d, 3-c, 4-a

D. 1-c, 2-b, 3-d, 4-a

Correct Answer: C. 1-b, 2-d, 3-c, 4-a

Explanation: Deficiency Pathway / Enzyme Affected Mechanism & Effect Adenosylcobalamin deficiency Cofactor for Methylmalonyl-CoA mutase Impairs conversion of methylmalonyl-CoA → succinyl-CoA (TCA cycle). Leads to energy metabolism defects , causing fatigue and neurological issues . Methylcobalamin deficiency Cofactor for Methionine synthase Impairs conversion of homocysteine → methionine , leading to folate trap , defective DNA synthesis, and megaloblastic (pernicious) anemia . Methylmalonyl-CoA mutase deficiency Enzyme itself defective (even with normal adenosylcobalamin) Causes accumulation of methylmalonyl-CoA , resulting in metabolic acidosis and neurologic dysfunction . Methionine synthase deficiency Impairs homocysteine remethylation Causes elevated homocysteine levels (homocystinuria) and decreased methionine, affecting methylation reactions.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 544

https://www.b12-vitamin.com/adenosylcobalamin/

https://www.b12-vitamin.com/methylcobalamin/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q15: Which of the following consequences of vitamin B6 deficiency directly contributes to an increased risk of seizures in a neonate with refractory seizures? (Q843171)

A. Decreased GABA synthesis

B. Elevated glutamate levels

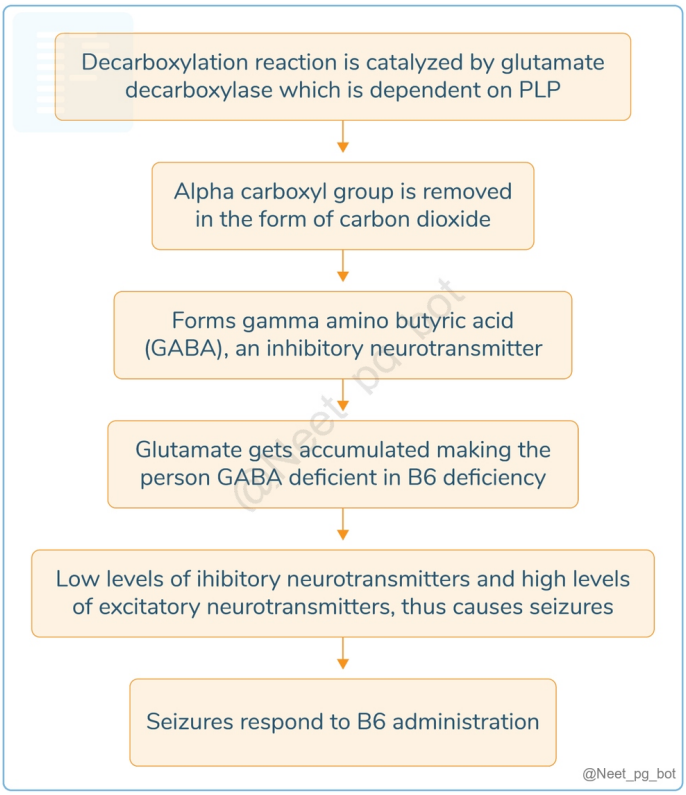
C. Increased serotonin production

D. Enhanced myelin formation

Correct Answer: A. Decreased GABA synthesis

■ *Listen to Audio 1*

Explanation: Vitamin B6 (Pyridoxal Phosphate, PLP) is a coenzyme for glutamate decarboxylase , which converts glutamate to GABA (an inhibitory neurotransmitter). Deficiency of vitamin B6 impairs GABA synthesis , leading to an imbalance between excitatory (glutamate) and inhibitory (GABA) signals, resulting in seizures, particularly in neonates with refractory seizures . Pyridoxine-dependent epilepsy (PDE) is a rare condition where B6 supplementation is required to control seizures. Elevated glutamate levels (Option B): Incorrect because while B6 deficiency can lead to increased glutamate, the primary cause of seizures is GABA deficiency, not glutamate excess . Increased serotonin production (Option C): Incorrect because vitamin B6 deficiency reduces serotonin synthesis due to impaired decarboxylation of 5-hydroxytryptophan. Enhanced myelin formation (Option D): Incorrect because vitamin B6 deficiency impairs , rather than enhances, myelin synthesis by disrupting sphingolipid metabolism.



References:

Textbook of Biochemistry, DM Vasudevan, 7th Edition, Page 483 Harrison's Principles of Internal Medicine, 21st Edition, Page 3309

↑ *Back to this question in Part A*

@Neet_pg_bot

Q16: Which of the following metabolic processes is not directly supported by vitamin B6 in its active form, pyridoxal 5'-phosphate (PLP)? (Q843169)

A. Transamination of amino acids

B. Decarboxylation of amino acids

C. Breakdown of glycogen to glucose-1-phosphate

D. Conversion of glucose to glycogen

Correct Answer: D. Conversion of glucose to glycogen

■ *Listen to Audio 1*

Explanation: Vitamin B6 (Pyridoxal phosphate, PLP) is a coenzyme involved in amino acid metabolism and glycogen breakdown , but not in glycogen synthesis . PLP supports: Transamination (e.g., ALT, AST) Decarboxylation (e.g., neurotransmitter synthesis) Glycogenolysis (via glycogen phosphorylase) Glycogen synthesis (glycogenesis) is catalyzed by glycogen synthase, which does not require PLP. Vitamin B6- Coenzyme Roles Metabolic Process Enzyme Involved Role of Vitamin B6 (PLP) Transamination (Option A ruled out) ALT, AST Transfers amino groups between amino acids and keto acids Decarboxylation (Option B ruled out) Glutamate decarboxylase Produces neurotransmitters (e.g., GABA, serotonin, dopamine) Glycogenolysis (Option C ruled out) Glycogen phosphorylase PLP is a coenzyme for glycogen phosphorylase, aiding glycogen breakdown Heme synthesis δ-aminolevulinate synthase Required for the first step in heme production Niacin synthesis Kynureninase Converts tryptophan to niacin Sphingolipid synthesis Serine palmitoyltransferase Involved in sphingomyelin metabolism Homocysteine metabolism Cystathionine β-synthase Helps convert homocysteine to cystathionine

References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 543, 299 Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Pages 482, 483

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: A 15 year old child presents with skin rashes, gastrointestinal issues, aminoaciduria and cognitive disturbances. Which of the following mechanisms explains the symptoms observed? (Q843166)

A. Hartnup disease causes a direct deficiency in niacin, leading to pellagra symptoms.

B. Impaired tryptophan absorption → ↓production of niacin → pellagra-like symptoms.

C. Tryptophan excess → disrupts niacin metabolism → pellagra-like symptoms

D. Increases the metabolism of tryptophan to serotonin, depleting niacin stores

Correct Answer: B. Impaired tryptophan absorption → ↓production of niacin → pellagra-like symptoms.

■ *Listen to Audio 1*

Explanation: Hartnup's disease is a defect in tryptophan absorption due to a mutation in the SLC6A19 gene , affecting neutral amino acid transport in the intestine and kidneys. ↓ Tryptophan → ↓ Niacin synthesis → Pellagra-like symptoms (dermatitis, diarrhea, dementia) Other features: Aminoaciduria, ataxia, mood changes Diagnosis: ↑ Neutral amino acids in urine, ↓ Tryptophan, and niacin Obermeyer test : Used to detect indole compounds derived from tryptophan metabolism in urine ■■■■■■■ Principle: Tryptophan is converted by intestinal bacteria to indole Reaction: Indole reacts with Obermeyer reagent (FeCl■ in HCl) Result: Blue/violet color indicates increased indole compounds Seen in Hartnup disease due to excess tryptophan degradation In carcinoid syndrome , tryptophan metabolism is diverted toward serotonin production . Approximately 70% of

tryptophan may be used for serotonin synthesis in carcinoid tumors Treatment: Niacin supplements and a high-protein diet Hartnup's disease causes a direct deficiency in niacin, leading to pellagra symptoms (Option A): Incorrect because Hartnup's disease primarily affects tryptophan absorption , not niacin itself. Niacin deficiency is secondary to tryptophan deficiency. Tryptophan excess → disrupts niacin metabolism → pellagra-like symptoms (Option C): Incorrect because excess tryptophan does not disrupt niacin metabolism ; instead, it increases niacin synthesis. Increases the metabolism of tryptophan to serotonin, depleting niacin stores (Option D): Incorrect because this mechanism occurs in carcinoid syndrome , where tryptophan is preferentially converted to serotonin, leading to niacin deficiency . However, this is not the mechanism in Hartnup's disease .

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 543

Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Page 482

↑ *Back to this question in Part A*

@Neet_pg_bot

Q18: A 45-year-old woman consuming mainly maize and sorghum develops bright red rash on her feet, ankles, and face that worsens with sun exposure. She also reports frequent diarrhea and recent episodes of irritability and difficulty concentrating. What is the likely diagnosis? (Q843163)

A. Vitamin B12 deficiency

B. Pellagra

C. Rickets

D. Iron deficiency anemia

Correct Answer: B. Pellagra

■ *Listen to Audio 1*

Explanation: Pellagra results from niacin (Vitamin B■) deficiency , characterized by the classic triad of symptoms: Dermatitis Bright red erythema- in sun-exposed areas (face, ankles, feet) Casal's necklace - ↑pigmentation around the neck Diarrhoea Dementia Dietary cause: Maize is low in tryptophan, and sorghum's high leucine inhibits niacin synthesis (via QPRTase). Other causes: Vitamin B■ deficiency, isoniazid use, estrogen, Hartnup disease, carcinoid syndrome, and alcoholism. Vitamin B12 deficiency (Option A) causes fatigue, neurological issues, and anaemia but is not typically associated with the combination of dermatitis, diarrhoea, and cognitive disturbances seen in this case. Rickets (Option C) resulting from a deficiency in vitamin D, calcium, or phosphate, present with bone deformities and growth retardation. It is not associated with dermatitis, diarrhoea, and neurological symptoms characteristic of pellagra. Iron deficiency anaemia (Option D) can cause fatigue, pallor, and shortness of breath but does not typically present with skin rash, gastrointestinal symptoms, or cognitive issues associated with pellagra.



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 542, 543 Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Pages 481, 482

↑ *Back to this question in Part A*

@Neet_pg_bot

Q19: Which of the following is the earliest sign of riboflavin (Vitamin B2) deficiency? (Q843158)

A. Cheilosis

B. Glossitis

C. Seborrheic dermatitis

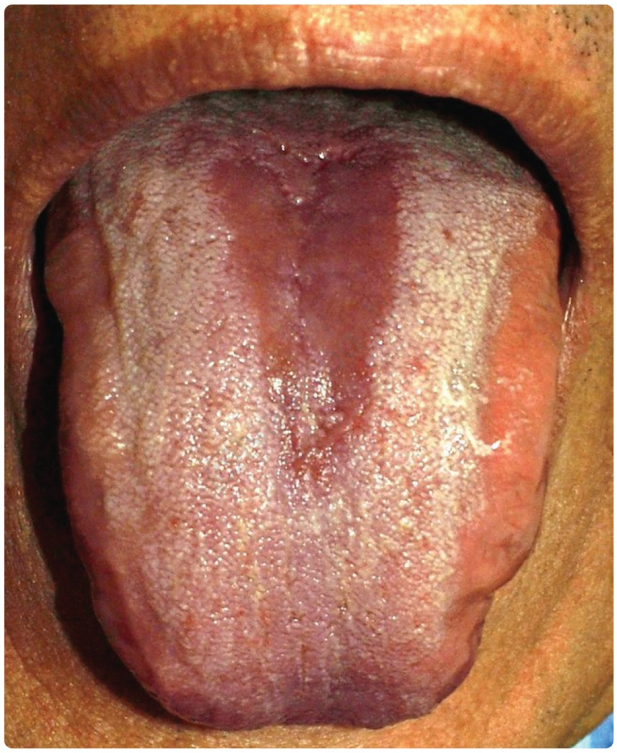
D. Proliferation of the bulbar conjunctival capillaries

Correct Answer: D. Proliferation of the bulbar conjunctival capillaries

■ *Listen to Audio 1*

Explanation: The proliferation of bulbar conjunctival capillaries, which refers to the visible dilation of blood vessels in the eyes, is often the earliest clinical sign of riboflavin deficiency. This sign appears before more obvious symptoms like cheilosis or glossitis develop. Riboflavin Deficiency Symptom Feature Image Cheilosis Cracks and inflammation at the corners of the mouth (Option A ruled out) Glossitis Inflammation and changes in the appearance of the tongue. It becomes smooth and glossy It may appear purplish red (Option B ruled out) Seborrheic dermatitis Skin condition with scaly patches, redness and dandruff (Option C ruled out) Proliferation of bulbar conjunctival capillaries Corneal Neovascularization Earliest sign It is the visible dilation of blood vessels in the eyes.

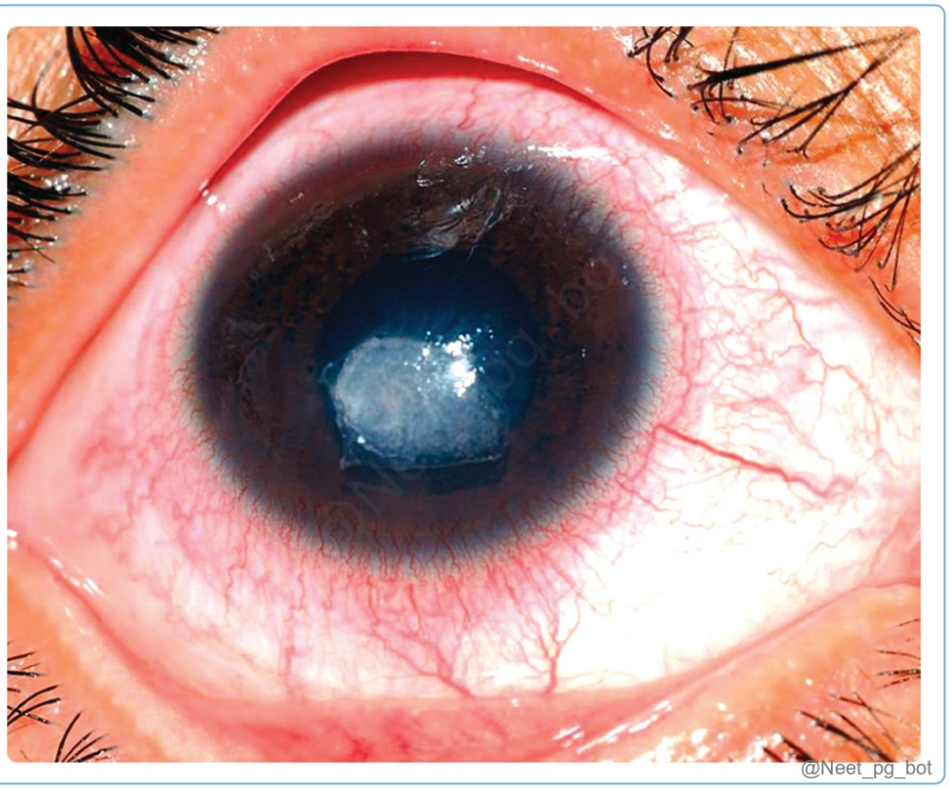




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References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 542

Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Page 480

↑ *Back to this question in Part A*

@Neet_pg_bot

Q20: Which of the following enzymes does not utilize FAD as a coenzyme? (Q843156)

A. Succinate Dehydrogenase

B. NADH Dehydrogenase

C. Xanthine Oxidase

D. Pyruvate Dehydrogenase

Correct Answer: B. NADH Dehydrogenase

■ *Listen to Audio 1*

Explanation: NADH Dehydrogenase (Complex I of the Electron Transport Chain) does not use FAD as a coenzyme ; instead, it primarily relies on FMN (Flavin Mononucleotide) as its prosthetic group. It transfers electrons from NADH to Coenzyme Q (Ubiquinone) through a series of iron-sulfur clusters. Unlike FAD-containing enzymes, Complex I utilizes FMN for its redox reactions. FMN-Dependent vs. FAD-Dependent Enzymes Category FMN-Dependent Enzymes FAD-Dependent Enzymes Derived from Riboflavin Yes Yes Coenzyme Form Flavin Mononucleotide (FMN) Flavin Adenine Dinucleotide (FAD) Electron Transport Accepts two electrons and donates them one at a time Accepts and donates two electrons and two protons Role in Metabolism Electron transport in oxidative phosphorylation Redox reactions in various metabolic pathways Location Mitochondrial ETC (Complex I) Mitochondrial matrix, peroxisomes, cytoplasm Examples NADH Dehydrogenase (Complex I) L-Amino Acid Oxidase Succinate Dehydrogenase Acyl CoA Dehydrogenase Xanthine Oxidase Pyruvate Dehydrogenase Alpha-Ketoglutarate Dehydrogenase Mitochondrial Glycerol-3-Phosphate Dehydrogenase

References:

Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Page 479

↑ *Back to this question in Part A*

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Q21: A 52-year-old alcoholic with poor nutrition presents with confusion, ataxia, and eye movement abnormalities. What is the most likely diagnosis? (Q843155)

A. Wernicke encephalopathy

B. Korsakoff psychosis

C. Lactic acidosis

D. Dry beri-beri

Correct Answer: A. Wernicke encephalopathy

■ *Listen to Audio 1*

Explanation: The triad of confusion, ataxia, and nystagmus/ophthalmoplegia in a chronic alcoholic strongly indicates Wernicke encephalopathy , a neurological emergency caused by thiamine (vitamin B■) deficiency . Chronic alcohol intake leads to poor nutrition, reduced intestinal absorption, and increased thiamine demand , resulting in impaired pyruvate metabolism and lactic acid accumulation . MRI findings: Bilateral hyperintensities in the mammillary bodies and periaqueductal gray matter support the diagnosis. If untreated, Wernicke encephalopathy can progress to Korsakoff psychosis which presents with anterograde and retrograde amnesia, sensory agnosia, and confabulation. (Option B ruled out) Lactic acidosis (Option C): While thiamine deficiency can lead to lactic acidosis but is not the primary diagnosis based on the symptoms described. Dry beriberi (Option D): Primarily affects the peripheral nervous system, causing peripheral neuritis, paresthesia, foot drop, muscle weakness, and distal sensory loss.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 542 Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Page 478 Wernicke-Korsakoff Syndrome | National Institute of Neurological Disorders and Stroke (nih.gov) Wernicke-Korsakoff Syndrome: Practice Essentials, Background, Etiology (medscape.com)

↑ *Back to this question in Part A*

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• [11_Vitamins] 031 Previous Year Questions

« Back to Folder Directory

[11_Vitamins] 031 Previous Year Questions — Quick Jump

Q1 (p. 1255)	*Q2 (p. 1256)*	*Q3 (p. 1258)*	*Q4 (p. 1258)*	*Q5 (p. 1259)*	*Q6 (p. 1261)*
Q7 (p. 1262)	*Q8 (p. 1264)*	*Q9 (p. 1265)*	*Q10 (p. 1266)*	*Q11 (p. 1268)*	*Q12 (p. 1268)*
Q13 (p. 1269)	*Q14 (p. 1270)*	*Q15 (p. 1271)*	*Q16 (p. 1273)*	*Q17 (p. 1274)*	*Q18 (p. 1275)*
Q19 (p. 1276)	*Q20 (p. 1277)*	*Q21 (p. 1278)*	*Q22 (p. 1279)*	*Q23 (p. 1281)*	*Q24 (p. 1282)*
Q25 (p. 1283)	*Q26 (p. 1284)*	*Q27 (p. 1286)*	*Q28 (p. 1287)*	*Q29 (p. 1288)*	*Q30 (p. 1288)*
Q31 (p. 1289)	*Q32 (p. 1290)*	*Q33 (p. 1290)*	*Q34 (p. 1291)*	*Q35 (p. 1292)*	*Q36 (p. 1293)*
Q37 (p. 1294)	*Q38 (p. 1295)*	*Q39 (p. 1295)*	*Q40 (p. 1296)*	*Q41 (p. 1296)*	*Q42 (p. 1297)*
Q43 (p. 1298)	*Q44 (p. 1299)*	*Q45 (p. 1301)*	*Q46 (p. 1303)*	*Q47 (p. 1303)*	*Q48 (p. 1304)*
Q49 (p. 1305)	*Q50 (p. 1306)*	*Q51 (p. 1307)*	*Q52 (p. 1307)*	*Q53 (p. 1308)*	*Q54 (p. 1310)*
Q55 (p. 1311)	*Q56 (p. 1312)*	*Q57 (p. 1313)*	*Q58 (p. 1314)*	*Q59 (p. 1315)*	*Q60 (p. 1316)*
Q61 (p. 1317)	*Q62 (p. 1318)*	*Q63 (p. 1319)*	*Q64 (p. 1320)*	*Q65 (p. 1320)*	*Q66 (p. 1322)*

Q1: To evaluate a deficiency of vitamin B2 (riboflavin), one can assess the activity of: (Q857548)

A. Transketolase

B. Pyruvate dehydrogenase

C. Glutathione reductase

D. Isocitrate dehydrogenase

Correct Answer: C. Glutathione reductase

Explanation: Laboratory Tests for Vitamin Deficiencies: Vitamin B1 (Thiamin): Test : Measure transketolase activity in red blood cells with and without thiamin diphosphate. Indicator : Increased activity with thiamin diphosphate indicates deficiency. Vitamin B2 (Riboflavin): Test : Measure glutathione reductase activity in red blood cells with and without flavin adenine dinucleotide (FAD). Indicator : Increased activity with FAD indicates deficiency. Vitamin B6 (Pyridoxine): Test : Measure activation of red blood cell transaminases with pyridoxal phosphate. Xanthurenic acid in urine Indicator: Increased activity indicates deficiency. The metabolic disorders that respond to vitamin B6 therapy include xanthurenic aciduria and homocystinuria. Vitamin B12 (Cobalamin): Tests : Urinary methylmalonic acid: Elevated levels indicate impaired conversion of methylmalonyl-CoA to succinyl-CoA, suggesting deficiency. Megaloblastic anaemia assessment: Detects anaemia caused by disrupted folate metabolism due to B12 deficiency. Folate : Test : Assess for megaloblastic anaemia. Indicator : The presence of this type of anaemia suggests folate deficiency. When folic acid is deficient, FIGLU is excreted in

urine. Histidine can be transaminated with pyruvate to form imidazole pyruvate and alanine.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg. 542, 543, 544, 546, 573

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg. 243, 483

↑ *Back to this question in Part A*

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Q2: A 34-year-old male was on maize as a staple diet for a prolonged period. He presented with diarrhea and lesion over his neck as shown in the image below. The condition occurs due to deficiency of: (Q854888)

A. Niacin

B. Vitamin B12

C. Vitamin D

D. Iron



Correct Answer: A. Niacin

Explanation: Pellagra is characterized by the classic triad of symptoms: Dermatitis: Bright red erythema- in sun-exposed areas (face, ankles, feet). Casal's necklace - ↑pigmentation around the neck Diarrhoea Dementia . Causes of pellagra : Dietary Deficiency- Maize and Sorghum based diet Factors Affecting Tryptophan to Niacin Conversion: Vitamin B6 Deficiency Medications: Isoniazid (INH), OCPs Hormonal Influences: Estrogen metabolites hinder tryptophan metabolism. Genetic Disorders: Inherited conditions like Hartnup disease can impede tryptophan absorption in the intestines and reabsorption in the kidneys Carcinoid Syndrome Other Contributing Factors: Alcoholism : Interferes with nutrient absorption, including niacin and tryptophan. Malabsorption Syndromes {{caption_text}} Incorrect Options: Vitamin B12 deficiency (Option B) can lead to symptoms such as fatigue, neurological issues, and anaemia. However, it is not typically associated with the specific combination of dermatitis, diarrhoea, and cognitive disturbances seen in this case. Rickets (Option C) resulting from a deficiency in vitamin D, calcium, or phosphate, present with bone deformities and growth retardation. It is not associated with dermatitis, diarrhoea, and neurological symptoms characteristic of pellagra. Iron deficiency anaemia (Option D) can cause fatigue, pallor, and shortness of breath but does not typically present with skin rash, gastrointestinal symptoms, or cognitive issues associated with pellagra.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Pg.542,543

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg. 481,482

↑ *Back to this question in Part A*

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Q3: Which vitamin, when infused in supraphysiological doses, causes macular edema and macular cysts? (Q854868)

A. Vitamin A

B. Vitamin D

C. Vitamin E

D. Vitamin B3

Correct Answer: D. Vitamin B3

Explanation: Niacin toxicity (Vit B3) can lead to macular edema and cysts through increased prostaglandin synthesis, resulting in fluid accumulation in the retina. Mechanism of Macular Edema and cysts: Niacin can raise levels of prostaglandins, particularly prostaglandin D2 (PGD2). Elevated prostaglandin levels lead to increased vascular permeability in the retina. Increased vascular permeability causes fluid and proteins to leak into the retinal layers, resulting in macular edema. Patients may experience blurred vision and distortion in their visual field. Management: Reducing or stopping niacin can resolve the symptoms. Patients on high doses should be regularly monitored for ocular side effects. Other effects of niacin toxicity includes: Flushing: Caused by the release of prostaglandins leading to vasodilation. Gastrointestinal Distress: Nausea, vomiting, diarrhea, and abdominal discomfort . Due to increased gastric acid secretion and irritation of the gastrointestinal lining. Hepatotoxicity Hyperglycemia Hyperurecemia

References:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4458328/

↑ *Back to this question in Part A*

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Q4: Dicoumarol inhibits the action of vitamin K by? (Q856506)

A. Suicide inhibition

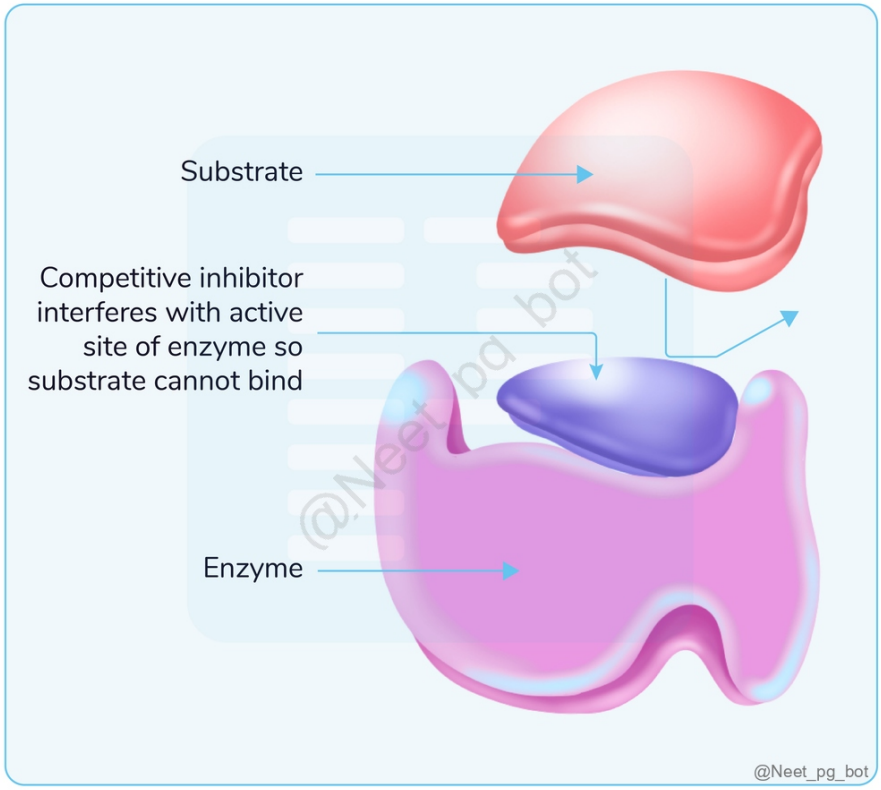
B. Competitive inhibition

C. Non-competitive inhibition

D. Uncompetitive inhibition

Correct Answer: B. Competitive inhibition

Explanation: icoumarol is a structural analog of vitamin K. It acts as a competitive inhibitor of the enzyme vitamin K epoxide reductase , which is essential for recycling vitamin K to its active form. Competitive Inhibition Inhibitor binds to the active site of an enzyme, competing directly with the substrate. The inhibitor's presence prevents the substrate from binding, thereby reducing the enzyme's activity. However, increasing the substrate concentration can overcome this inhibition. Key Features: Binds at the active site. Reversible : Substrate can displace the inhibitor if present in excess. Km increases: Higher substrate concentration is needed to reach half-maximal velocity (). V max remains unchanged: At high substrate concentrations, the enzyme can still achieve its maximum velocity. Examples : Dicoumarol : Inhibits vitamin K epoxide reductase, blocking vitamin K recycling. Sulfonamides : Compete with para-aminobenzoic acid (PABA) for the active site of dihydropteroate synthase, inhibiting bacterial folate synthesis. Statins : Compete with HMG-CoA for HMG-CoA reductase, reducing cholesterol synthesis. Methotrexate : Competes with dihydrofolate for the active site of dihydrofolate reductase, inhibiting nucleotide synthesis. Ethanol : Competes with methanol or ethylene glycol for alcohol dehydrogenase, used in cases of poisoning.



References:

Harper biochemistry ed 32,Pg 79 & 80

Vasudevan ed 7,pg 59&60

↑ *Back to this question in Part A*

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Q5: The sequence of vitamin D synthesis is? (Q856503)

A. Liver-skin-kidney

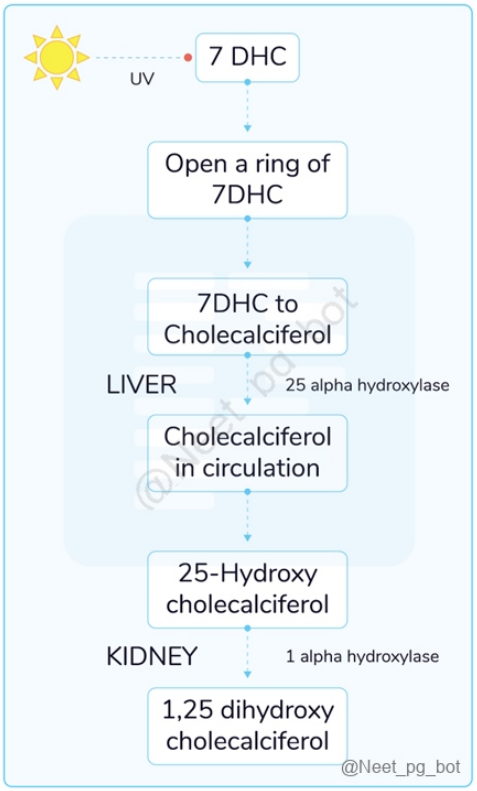
B. Skin-liver-kidney

C. Skin-kidney-liver

D. Liver-kidney-skin

Correct Answer: B. Skin-liver-kidney

Explanation: - Skin-liver-kidney: The synthesis of vitamin D involves three key steps: Skin : UVB radiation converts 7-dehydrocholesterol into cholecalciferol (vitamin D3) in the skin. Liver : Cholecalciferol is hydroxylated to 25-hydroxyvitamin D (calcidiol) by the enzyme 25-hydroxylase. Kidney : Calcidiol is further hydroxylated to the active form, 1,25-dihydroxyvitamin D (calcitriol), by 1α-hydroxylase. This sequence ensures the production of bioactive vitamin D necessary for calcium and phosphate homeostasis. Calcitriol plays a crucial role in calcium and phosphorus metabolism and responsible for regulating their blood levels.



References:

Vit D synthesis

↑ *Back to this question in Part A*

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Q6: Which of the following are vitamin B12-dependent enzymes? Methyl malonyl CoA mutase Homocysteine synthase Folate reductase Methionine synthase (Q856920)

A. 1,2,3 and 4

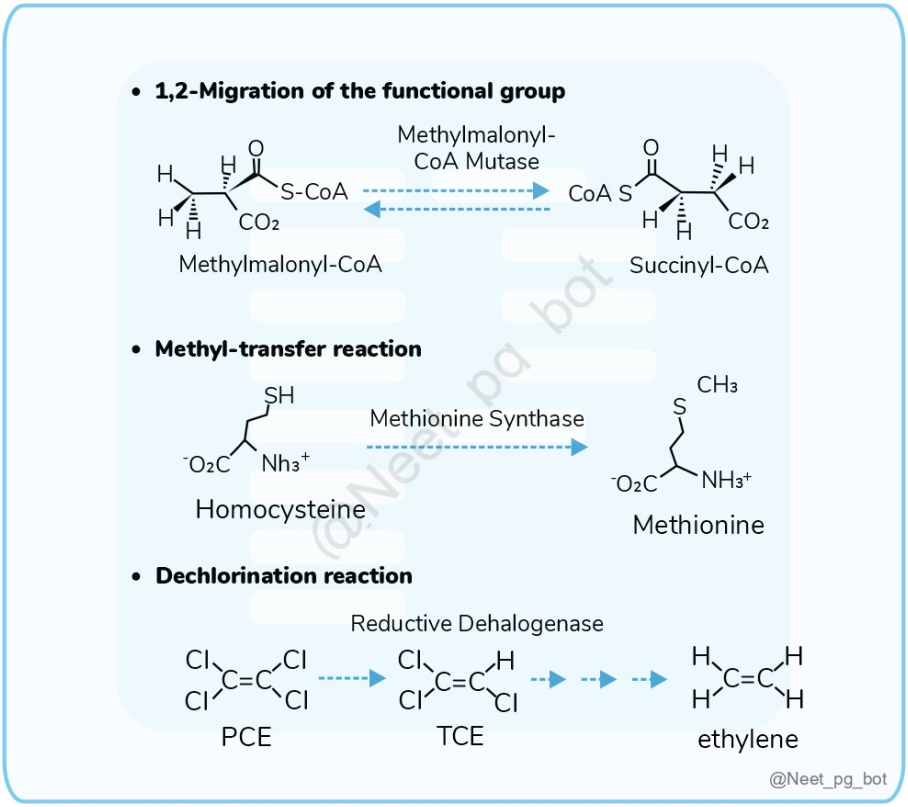
B. 1 and 4 only

C. 1 and 3 only

D. 1,2 and 3

Correct Answer: B. 1 and 4 only

Explanation: Vitamin B12-dependent enzymes: Methyl malonyl CoA mutase Methionine synthase Enzyme Cofactor form of B■■ Reaction catalyzed Pathway significance Methionine synthase (Homocysteine → Methionine) Methylcobalamin Transfers a methyl group from 5-methyl-tetrahydrofolate (THF) to homocysteine , forming methionine Links folate cycle and methionine cycle Methylmalonyl-CoA mutase Adenosylcobalamin Converts methylmalonyl-CoA → succinyl-CoA Important in odd-chain fatty acid and branched-chain amino acid metabolism Homocysteine synthase: This enzyme is not directly dependent on vitamin B12. It utilizes methylcobalamin, a form of vitamin B12, as a cofactor to catalyze the conversion of homocysteine to methionine. However, it is not strictly considered a vitamin B12-dependent enzyme. Folate reductase: This enzyme is not directly dependent on vitamin B12. It is involved in the metabolism of folate (vitamin B9) and plays a role in the synthesis of DNA and other important cellular processes.



References:

Vitamin B12

↑ *Back to this question in Part A*

@Neet_pg_bot

Q7: An alcoholic patient presented with itchy lesions as shown in the image. He also has a red-colored beefy tongue. Which of the following histories would you like to ask/elicit in this patient additionally? (Q855973)

A. Ask for diarrhea and a history of memory loss

B. Ask for diarrhea, thiamine deficiency features, and detailed dietary history

C. History of allergy

D. Ask for diarrhea and conjunctivitis



Correct Answer: A. Ask for diarrhea and a history of memory loss

Explanation: The clinical scenario of alcoholic, red beefy tongue and Casal's necklace (dermatitis) in image indicates niacin deficiency Pellagra is characterized by the classic triad of symptoms: Dermatitis Bright red erythema- in sun-exposed areas (face, ankles, feet) Casal's necklace- ↑pigmentation around the neck Diarrhoea Dementia {{caption_text}} Ask for diarrhea, thiamine deficiency features, and detailed dietary history (OptionA): While thiamine deficiency is a potential concern in alcoholics,it features (muscle weakness, fatigue, cardiovascular symptoms). History of allergy (Option C): Allergies can cause skin manifestations, including itching, the specific symptoms mentioned in this case are indicative of a condition associated with alcohol abuse and thiamine deficiency. Ask for diarrhea and conjunctivitis (Option D): Conjunctivitis (pink eye) and diarrhea are symptoms that can be associated with certain infections.



↑ *Back to this question in Part A*

@Neet_pg_bot

Q8: A farmer presented with loose stools and rashes over the sun-exposed areas. Careful diet history reveals a diet rich in maize. Which of the following deficiencies would you suspect? (Q855968)

A. Niacin

B. Pyridoxine

C. Thiamine

D. Biotin

Correct Answer: A. Niacin

Explanation: The Clinical scenario of Diarrhoea, rashes over sun exposed areas (Dermatitis) and a history of maize based diet indicates Pellagra, seen in niacin deficiency Pellagra is characterized by the classic triad of symptoms: Dermatitis Bright red erythema- in sun-exposed areas (face, ankles, feet) Casal's necklace- ↑pigmentation around the neck (image) {{caption_text}} Diarrhoea Dementia The patient's symptoms, combined with a diet high in maize and sorghum, both known to contribute to niacin and tryptophan deficiencies suggest pellagra as the most

likely diagnosis. Sorghum has high leucine content which inhibits the QPRTase enzyme required for synthesis of Niacin from tryptophan. Additionally, Maize is low in tryptophan. Incorrect Options: Option B) Pyridoxine (Vitamin B6) deficiency can lead to various neurological symptoms such as peripheral neuropathy, but it is not associated with specific skin rashes or gastrointestinal symptoms like loose stools. Option C) Thiamine (Vitamin B1) deficiency typically presents with symptoms related to the nervous system, such as peripheral neuropathy, cardiovascular abnormalities, and Wernicke-Korsakoff syndrome. Option D) Biotin deficiency is rare and usually occurs in individuals with a genetic disorder or those consuming large quantities of raw egg whites, which contain a protein called avidin that binds to biotin and impairs its absorption. Biotin deficiency can lead to symptoms such as hair loss, skin rash, and neurological abnormalities.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Pg.542,543

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg. 481,482

↑ *Back to this question in Part A*

@Neet_pg_bot

Q9: A patient presented with symptoms of delayed adaptation to vision in dark places, gritty sensation in the eyes, dry eyes, and softening of the cornea. What is the likely deficiency in this case? (Q855948)

A. Retinoic acid

B. Riboflavin

C. Niacin

D. Pantothenic acid

Correct Answer: A. Retinoic acid

Explanation: Vitamin A Deficiency Manifestations : 1. Ocular manifestations : Vitamin A deficiency is the most significant preventable cause of blindness, especially in children below 5 in developing countries. Early Signs: Loss of sensitivity to green light. Difficulty adapting to dark places . Night blindness . Prolonged Deficiency: Xerophthalmia: Dry, thick, keratinized conjunctiva and cornea. If untreated, it leads to corneal ulceration and ultimately results in blindness due to the formation of opaque scar tissue. WHO grading of xerophthalmia: XN Night blindness. X1 X1A: Conjunctival xerosis. X1B: Bitot spots. X2 Corneal xerosis. X3 X3A: Corneal ulceration, less than one-third. X3B: Corneal ulceration, more than one-third. XS Corneal scar. XF Xerophthalmic fundus. Bitot's Spots: Grayish-white triangular plaques firmly adherent to the conjunctiva. Keratomalacia: When xerophthalmia persists for a longer time, it progresses to keratomalacia (corneal softening). 2. Immune Function: Deficiency increases susceptibility to infections. Infection reduces retinol-binding protein synthesis, lowering vitamin A levels and impairing immune responses. 3. Skin and Mucous Membrane Lesions: Follicular Hyperkeratosis (Phrynoderma): Causes rough skin due to hyperkeratinization of the follicular epithelium. Keratinizing Metaplasia: Affects respiratory, gastrointestinal, and genitourinary tracts. Epithelium is atrophied. 4. Other General Manifestation : Growth retardation due to defective synthesis of chondroitin sulfate. Decreased protein synthesis. Incorrect Options: Option B) Riboflavin: Riboflavin deficiency typically manifests as oral and ocular symptoms, such as cracked lips, inflamed tongue, and sensitivity to light (photophobia). Option C) Niacin: Niacin deficiency, also known as pellagra, primarily presents with dermatitis, diarrhea, and dementia. Option D) Pantothenic acid: Pantothenic acid, or vitamin B5, deficiency is rare and not commonly associated with eye-related symptoms. It is involved in energy production and plays a role in various physiological processes.

References:

Harper Edition 32, Page no: 538

Lippincott Edition 7, page no:1112,1113

Vasudevan Edition 7, page no: 467,468

↑ *Back to this question in Part A*

@Neet_pg_bot

Q10: What is the most likely cause of the worsening neurological symptoms in a 30-year-old woman who presented with complaints of fatigue, difficulty breathing during exertion, weight loss, and frequent falls, and was found to have bilateral decrease in vibration sense, a hemoglobin level of 8.2 g% with high MCV, and was

initially treated with folate which improved her anemia? (Q854890)

A. Folate is not well absorbed

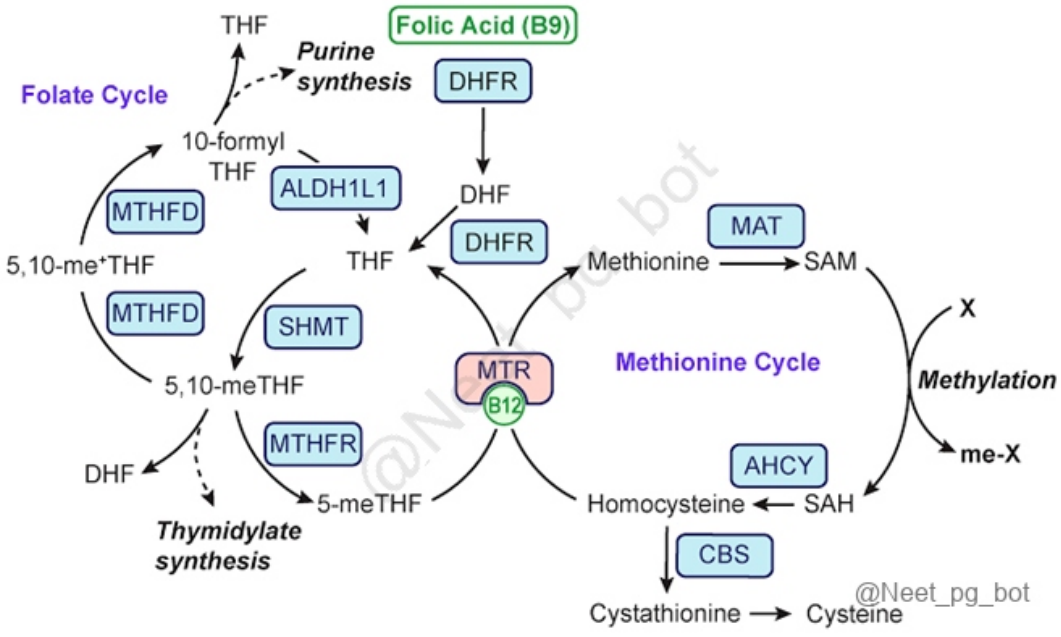
B. Folic acid therapy unmasked pyridoxine deficiency.

C. Deficiency of folate reductase in CNS.

D. Folate therapy caused rapid use of vitamin B12 stores aggravating symptoms.

Correct Answer: D. Folate therapy caused rapid use of vitamin B12 stores aggravating symptoms.

Explanation: These are classic symptoms of vitamin B12 deficiency, which leads to impaired DNA synthesis (macrocytic anemia) and demyelination (peripheral neuropathy). Folate supplementation in isolation: Folate can improve the hematologic symptoms (macrocytic anemia) by bypassing the impaired DNA synthesis caused by B12 deficiency. However, folate does not address the neurologic symptoms caused by the lack of B12, which are due to accumulation of methylmalonic acid and demyelination in the central and peripheral nervous systems. Mechanism of worsening symptoms: When folate is supplemented without correcting B12 deficiency, it increases the demand for B12 in the methylation cycle (conversion of homocysteine to methionine). This depletes the already low B12 stores, worsening the neurologic symptoms, despite initial improvement in anemia. Folate is not well absorbed (Option A): Absorption issue would not cause neurological worsening. Pyridoxine deficiency (Option B): Does not explain macrocytosis or posterior column loss. Folate reductase deficiency (Option C): Rare and does not fit this clinical scenario.



References:

Vit B12

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: Which antioxidant deficiency can cause the symptoms of iron-deficiency anemia, painful and tender joints, and bleeding manifestations in a 34-year-old woman who primarily consumes packaged and reheated meals? (Q857546)

A. Glutathione

B. ß-carotene

C. Vitamin C

D. Zinc

Correct Answer: C. Vitamin C

Explanation: Vitamin C deficiency develops after weeks to months of vitamin C depletion. Vitamin C (ascorbic acid) deficiency, also known as scurvy, can cause: Iron-deficiency anemia: Impaired non-heme iron absorption. Painful, tender joints: Due to defective collagen synthesis affecting cartilage. Bleeding manifestations: Easy bruising, gum bleeding, and perifollicular hemorrhages due to weakened capillary walls from defective collagen. Scorbutic rosary Risk factor: Diet lacking fresh fruits and vegetables, relying on packaged and reheated meals , as heat destroys vitamin C. Glutathione (Option A): Involved in oxidative stress protection, no direct link to these symptoms. ß-carotene (Option B): Precursor of vitamin A, deficiency affects vision and skin, not bleeding or anemia. Zinc (Option D): Deficiency causes delayed wound healing and dermatitis, not anemia or bleeding.

References:

Vitamin C

↑ *Back to this question in Part A*

@Neet_pg_bot

Q12: What is the underlying cause of the presenting symptoms in a 21-year-old individual with chronic alcoholism who is admitted to the emergency department with confusion, tremors, ataxia, and ophthalmoplegia? (Q857545)

A. Vitamin B12

B. Vitamin B9

C. Vitamin B1

D. Vitamin B3

Correct Answer: C. Vitamin B1

Explanation: Thiamine deficiency is the first seen micronutrient deficiency in chronic alcoholism, as alcohol interferes with thiamine absorption. The patient's symptoms of confusion, difficulty walking (cerebellar ataxia), and abnormal eye movements (nystagmus) are classic signs of Wernicke encephalopathy , which is caused by thiamine deficiency often seen in chronic alcoholics. WERNICKE-KORSAKOFF SYNDROME (CEREBRAL BERI-BERI) Clinical Manifestations Wernicke Encephalopathy: Symptoms: Ophthalmoplegia, nystagmus, cerebellar ataxia, global confusion. Korsakoff Psychosis: Symptoms: Anterograde and retrograde amnesia, sensory agnosia, and confabulation. Causes of Thiamine Deficiency in Alcoholics Increased thiamine requirements due to alcohol consumption. Alcohol is a source of empty calories. Inhibition of intestinal thiamine absorption by alcohol. Metabolic Implications Impaired conversion of pyruvate to acetyl-CoA. Accumulation of pyruvate and lactate in the blood. Potential for life-threatening lactic acidosis. Progression and Risk Factors Progression from Wernicke encephalopathy to Korsakoff psychosis if untreated. Associated with severely compromised nutritional status, often due to chronic alcoholism. MRI findings Symmetrical T2/FLAIR signal increase in the mammillary bodies, dorsomedial thalami, tectal plate, periaqueductal area, and around the third ventricle.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg.542

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg.478

Wernicke-Korsakoff Syndrome | National Institute of Neurological Disorders and Stroke (nih.gov)

Wernicke-Korsakoff Syndrome: Practice Essentials, Background, Etiology (medscape.com)

https://radiopaedia.org/articles/wernicke-encephalopathy#:~:text=On%20imaging%2C%20it%20is%20commonly,or%20around%20the%20third%20ventricle

↑ *Back to this question in Part A*

@Neet_pg_bot

Q13: A young boy presented with fissured tongue, lips, and lesions in the angle of the mouth.On investigation, he had low RBC glutathione reductase activity. Which vitamin is deficient here? (Q857487)

A. Vitamin B12

B. Vitamin B3

C. Biotin

D. Riboflavin

Correct Answer: D. Riboflavin

Explanation: Riboflavin or Vitamin B2 acts as the coenzyme flavin mononucleotide (FMN) and flavin adenine dinucleotide (FAD) in oxidation and reduction reactions. Lesions of the corner of mouth, lips, and prosthetic group of flavoproteins. The main dietary sources of riboflavin are milk and dairy products. In addition, because of its intense yellow color, riboflavin is widely used as a food additive. Riboflavin deficiency leads to following signs and symptoms: Cheilosis : It is pallor in the angle of mouth and fissuring of lips. Glossitis : Loss of papilla and increased smoothness of tongue Eyes : Keratitis, conjunctivitis, subcorneal congestion(ist sign) • Seborrheic dermatitis Anemia Vitamin B12 (Option A): Causes neuropathy and megaloblastic anemia, not mouth fissures. Vitamin B3 (Option B): Causes dermatitis, diarrhea, and dementia (pellagra), not low GR activity. Biotin (Option C): Causes dermatitis and alopecia, unrelated to glutathione reductase.

References:

https://www.ncbi.nlm.nih.gov/books/NBK470460/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q14: Which of the following substance, facilitate the absorption of dietary iron? (Q857479)

A. Calcium

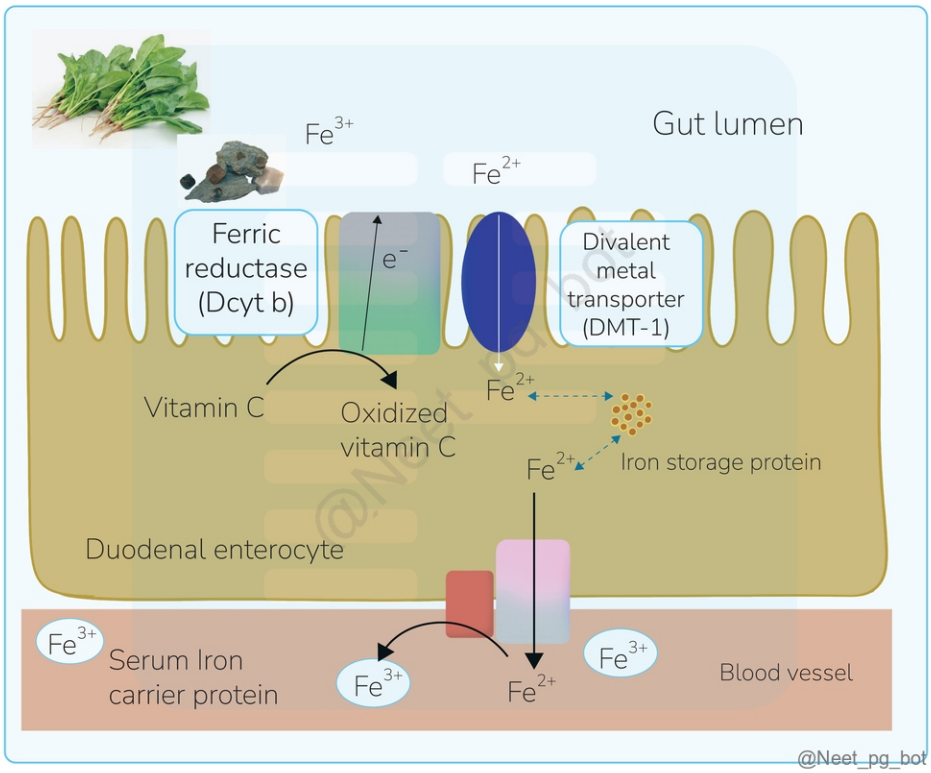
B. Vitamin C

C. Vitamin D

D. Vitamin K

Correct Answer: B. Vitamin C

Explanation: Vitamin C enhances dietary iron absorption by: Reducing Ferric to Ferrous Iron: Converts ferric iron (Fe^{3}■) to the more absorbable ferrous form (Fe^{2}■) in the acidic stomach environment. Chelation : Forms a soluble complex with iron, preventing it from binding to inhibitors like phytates and tannins. Enhanced Transport: Improves iron uptake by intestinal mucosal cells. {{caption_text}}



References:

https://pubmed.ncbi.nlm.nih.gov/6940487/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q15: Which of the subsequent stages in the vitamin D metabolism necessitates exposure to sunlight? (Q857003)

A. 1,25 dihydroxy cholecalciferol to 7-dehydrocholesterol

B. Cholecalciferol to 25-hydroxycholecalciferol

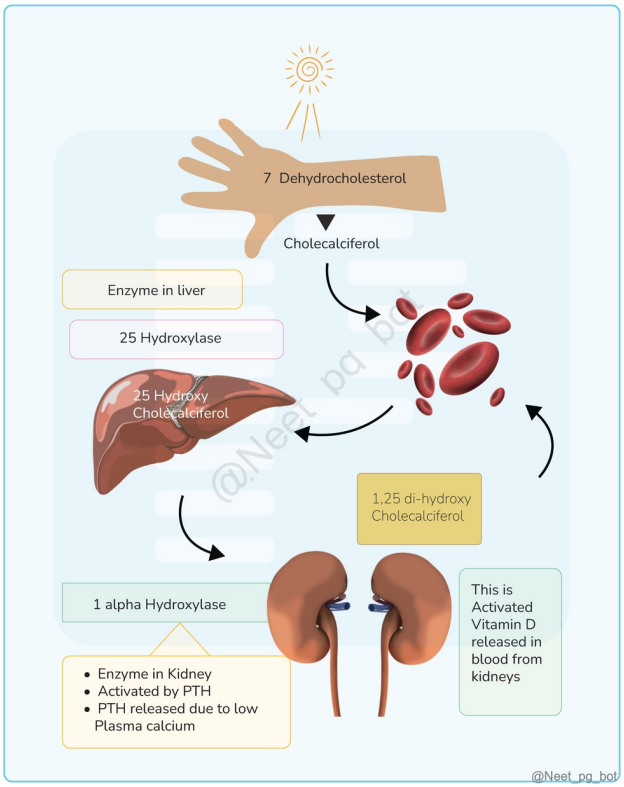
C. 7-dehydrocholesterol to cholecalciferol

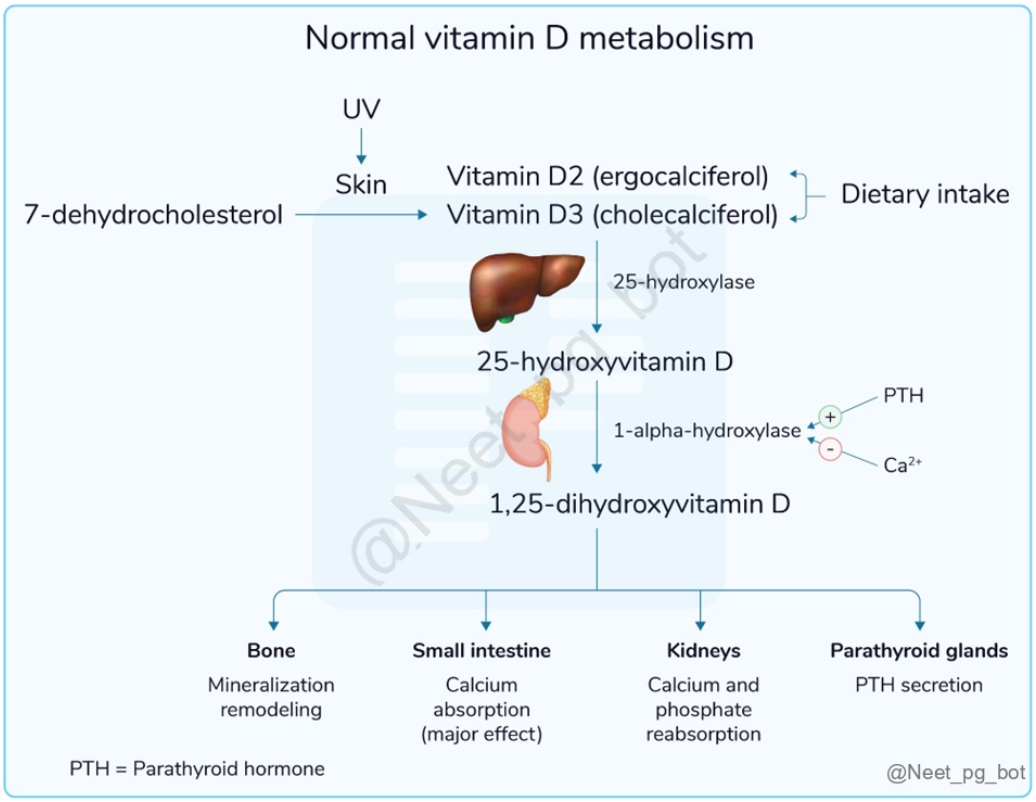
D. 25-hydroxycholecalciferol to 1,25 dihydroxycholecalciferol

Correct Answer: C. 7-dehydrocholesterol to cholecalciferol

Explanation: The conversion of 7-dehydrocholesterol to cholecalciferol (vitamin D3) is the stage in vitamin D metabolism that requires exposure to sunlight. This reaction occurs in the skin under the influence of UVB

radiation from sunlight. 7-dehydrocholesterol is a compound found in the skin, and when exposed to UVB light, it is converted to cholecalciferol (vitamin D3). Vitamin D Metabolism: Skin (sunlight exposure): 7-dehydrocholesterol is converted to cholecalciferol (vitamin D3) under UVB light. Liver: Cholecalciferol is then converted to 25-hydroxycholecalciferol (calcidiol), the major circulating form of vitamin D. Kidneys: 25-hydroxycholecalciferol is further hydroxylated to form the active metabolite, 1,25-dihydroxycholecalciferol (calcitriol).





References:

Vit D

↑ *Back to this question in Part A*

@Neet_pg_bot

Q16: What could be the potential diagnosis for a 34-year-old male who has been consuming maize as his main diet for an extended period and is now experiencing symptoms such as abdominal pain, decreased appetite, and diarrhea? Additionally, upon examination, glossitis (inflammation of the tongue) and a pigmented rash in sun-exposed areas were observed, along with a lesion on his neck. (Q856983)

A. Pellagra

B. Scurvy

C. Megaloblastic anemia

D. Beriberi

Correct Answer: A. Pellagra

Explanation: Pellagra is characterized by the classic triad of symptoms: Dermatitis : Bright red erythema - in sun-exposed areas (face, ankles, feet) Casal’s necklace - ↑pigmentation around the neck Diarrhoea Dementia . The patient's symptoms, combined with a diet high in maize and sorghum, both known to contribute to niacin and tryptophan deficiencies suggest pellagra as the most likely diagnosis. Sorghum has high leucine content which inhibits the QPRTase enzyme required for synthesis of Niacin from tryptophan. Additionally, Maize is low in tryptophan. {{caption_text}} Causes of pellagra : Dietary Deficiency- Maize and Sorghum based diet Impaired Tryptophan Metabolism Factors Affecting Tryptophan to Niacin Conversion: Vitamin B6 Deficiency Medications: Isoniazid (INH), OCPs Hormonal Influences: Estrogen metabolites hinder tryptophan metabolism. Genetic Disorders: Inherited conditions like Hartnup disease can impede tryptophan absorption in the intestines and reabsorption in the kidneys Carcinoid Syndrome Other Contributing Factors: Alcoholism: Interferes with nutrient absorption, including niacin and tryptophan. Malabsorption Syndromes



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg.542,543

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg. 481,482

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: Which of the following vitamins is incorrectly paired with their respective enzymes? (Q856939)

A. Vitamin B1 (Thiamine): RBC Trans-ketolase

B. Vitamin B2 (Riboflavin): RBC glutathione reductase

C. Vitamin B9 (Folic acid): FIGLU excretion/Histidine load test

D. Vitamin C: Serum methylmalonate level

Correct Answer: D. Vitamin C: Serum methylmalonate level

Explanation: Vitamin B12 is associated with Methyl malonic acid. Laboratory Tests for Vitamin Deficiencies: Vitamin B1 (Thiamin): Test : Measure transketolase activity in red blood cells with and without thiamin diphosphate. Indicator : Increased activity with thiamin diphosphate indicates deficiency. Vitamin B2 (Riboflavin): Test : Measure glutathione reductase activity in red blood cells with and without flavin adenine dinucleotide (FAD). Indicator : Increased activity with FAD indicates deficiency. Vitamin B6 (Pyridoxine): Test : Measure activation of red blood cell transaminases with pyridoxal phosphate. Xanthurenic acid in urine Indicator: Increased activity indicates deficiency. The metabolic disorders that respond to vitamin B6 therapy include xanthurenic aciduria and homocystinuria. Vitamin B12 (Cobalamin): Tests : Urinary methylmalonic acid: Elevated levels indicate impaired conversion of methylmalonyl-CoA to succinyl-CoA, suggesting deficiency. Megaloblastic anaemia assessment: Detects anaemia caused by disrupted folate metabolism due to B12 deficiency. Folate : Test : Assess for megaloblastic anaemia. Indicator : The presence of this type of anaemia suggests folate deficiency. When folic acid is deficient, FIGLU is excreted in urine. Histidine can be transaminated with pyruvate to form imidazole pyruvate and alanine. Vitamin C: Function as cofactor for prolyl-3-hydroxylases, prolyl-4-hydroxylases.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg. 542, 543, 544, 546, 573

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg. 243, 483

↑ *Back to this question in Part A*

@Neet_pg_bot

Q18: A family consumes a diet predominantly of rice and pulses developed signs of muscle weakness and movement. Which of the following should be given for prophylaxis? (Q866669)

A. Calcium

B. Thiamine

C. Vitamin D

D. Iron

Correct Answer: B. Thiamine

Explanation: The given case scenario of family that consumes a diet predominantly consisting of rice and pulses causing muscle weakness points to Dry beri-beri caused by deficiency of Vitamin B1 (Thiamine) . Beriberi is caused by thiamine (vitamin B1) deficiency, which is common in individuals with chronic alcoholism and diets lacking in unpolished grains. The Aleurone layer of cereals (food grains) is a rich source of thiamine. Therefore, whole wheat flour and unpolished hand-pound rice have better nutritive value than completely polished refined foods. When the grains are polished, the aleurone layer is usually removed. Deficiency: Dry beriberi: CNS manifestations are more common Pain, paresthesia and loss of reflexes Peripheral neuritis with sensory disturbances Wet beriberi: Wet beriberi is characterized by cardiovascular symptoms such as breathlessness, palpitations, and peripheral oedema. Distended neck veins observed High output cardiac failure Biochemical parameters: Thiamine deficiency affects the enzyme transketolase, which is involved in the pentose phosphate pathway. Measuring transketolase activity in red blood cells is a specific test for thiamine deficiency and can confirm the diagnosis. Erythrocyte transketolase activity is reduced; this is the earliest manifestation seen even before clinical disturbances. Recommended Daily Allowance of Thiamine is 1–1.5 mg/day. Treatment: In acute thiamine deficiency with either cardiovascular or neurologic signs, 200 mg of thiamine three times daily should be given intravenously until there is no further improvement in acute symptoms; oral thiamine (10 mg/d) should subsequently be given until recovery is complete. Incorrect Options: Calcium (Option A): Needed for bone health, muscle contraction, and nerve conduction. Deficiency causes osteoporosis, rickets (in children), tetany, but not the neuropathy seen in dry beriberi. Rice-based diets may be low in calcium, but the described neurological weakness is more consistent with B1 deficiency. Vitamin D (Option C): Regulates calcium/phosphate metabolism. Deficiency → rickets in children, osteomalacia in adults. Presents with bone pain, deformities, and muscle weakness due to hypocalcemia, but not peripheral neuropathy . Iron (Option D): Deficiency → microcytic, hypochromic anaemia (fatigue, pallor, pica). No direct link to the described nerve-related muscle weakness pattern.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q19: Which of the following contains the maximum amount of vitamin B12? (Q858455)

A. Animal products

B. Green leafy vegetables

C. Roots and tubers

D. Vegetarian diet

Correct Answer: A. Animal products

Explanation: Vitamin B12 is primarily found in animal products such as meat, fish, eggs, and dairy. Plant-based sources like green leafy vegetables, roots, tubers, and vegetarian diets generally lack significant amounts of vitamin B12 unless fortified or supplemented. So Vit B12 deficiency is significantly seen among plant sources. Symptoms: Hematologic : Macrocytic anemia, fatigue, pallor. Neurological : Paresthesias, ataxia, memory loss, peripheral neuropathy. Psychiatric : Depression, irritability, cognitive decline. Glossitis : Sore, red, swollen tongue. Diagnosis: Low serum vitamin B12 levels. Elevated methylmalonic acid (MMA) and homocysteine levels. Peripheral blood smear: Macrocytes, hypersegmented neutrophils.

References:

Vit B12 deficiency

↑ *Back to this question in Part A*

@Neet_pg_bot

Q20: What is the probable reason for the worsening of neurologic symptoms in a patient who initially showed improvement in blood picture after receiving a dose of 5mg folate for peripheral neuropathy and macrocytic anemia? (Q858458)

A. Malabsorption of folate

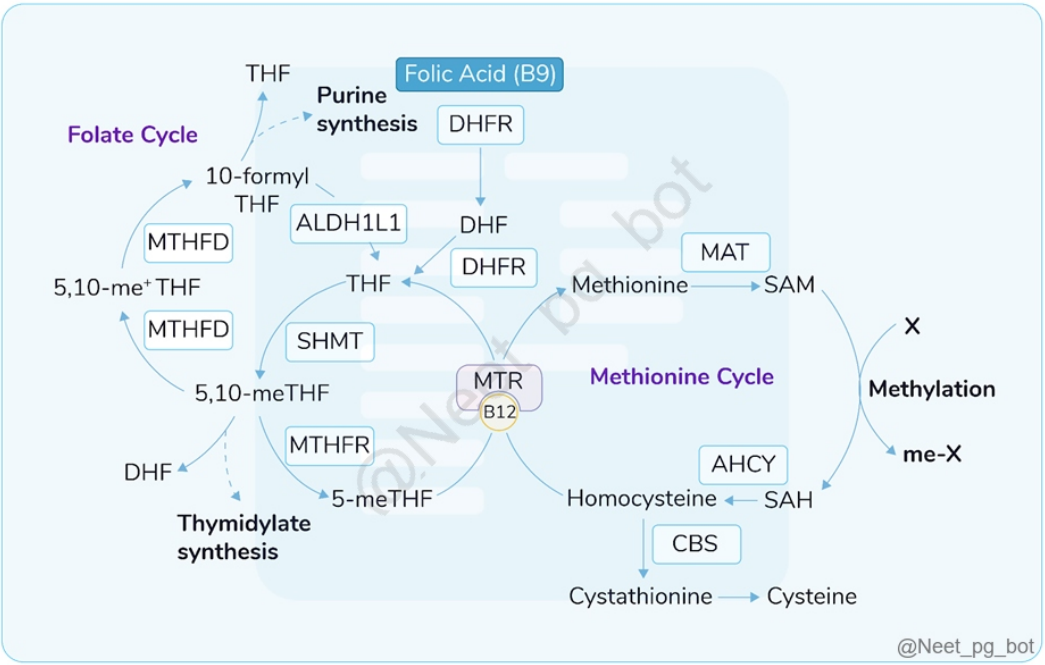
B. Treatment with folate unmasking pyridoxine deficiency

C. Deficiency of folate reductase in CNS

D. Folate therapy using the B12 stores

Correct Answer: D. Folate therapy using the B12 stores

Explanation: These are classic symptoms of vitamin B12 deficiency, which leads to impaired DNA synthesis (macrocytic anemia) and demyelination (peripheral neuropathy). Folate supplementation in isolation: Folate can improve the hematologic symptoms (macrocytic anemia) by bypassing the impaired DNA synthesis caused by B12 deficiency. However, folate does not address the neurologic symptoms caused by the lack of B12, which are due to accumulation of methylmalonic acid and demyelination in the central and peripheral nervous systems. Mechanism of worsening symptoms: When folate is supplemented without correcting B12 deficiency, it increases the demand for B12 in the methylation cycle (conversion of homocysteine to methionine). This depletes the already low B12 stores, worsening the neurologic symptoms, despite initial improvement in anemia.
{{caption_text}}



References:

https://pmc.ncbi.nlm.nih.gov/articles/PMC1828842/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q21: What is the recommended treatment for a 45-year-old male patient with a history of chronic alcoholism, who is currently experiencing symptoms of confusion, ataxia, and diplopia? (Q859571)

A. Vitamin B1 injection followed by glucose infusion

B. Glucose infusion followed thiamine injection

C. Glucose infusion alone

D. Thiamine infusion alone

Correct Answer: A. Vitamin B1 injection followed by glucose infusion

Explanation: This patient is presenting with symptoms of Wernicke's encephalopathy , commonly seen in patients with chronic alcoholism. The treatment for Wernicke's encephalopathy is to administer thiamine (vitamin B1) before or simultaneously with glucose to prevent exacerbating the condition. WERNICKE-KORSAKOFF SYNDROME (CEREBRAL BERI-BERI) Clinical Manifestations Wernicke Encephalopathy: Symptoms:

Ophthalmoplegia, nystagmus, cerebellar ataxia, global confusion. Korsakoff Psychosis: Symptoms: Anterograde and retrograde amnesia, sensory agnosia, and confabulation. (Option B) Causes of Thiamine Deficiency in Alcoholics Increased thiamine requirements due to alcohol consumption. Alcohol is a source of empty calories. Inhibition of intestinal thiamine absorption by alcohol. Metabolic Implications Impaired conversion of pyruvate to acetyl-CoA. Accumulation of pyruvate and lactate in the blood. Potential for life-threatening lactic acidosis. Progression and Risk Factors Progression from Wernicke encephalopathy to Korsakoff psychosis if untreated. Associated with severely compromised nutritional status, often due to chronic alcoholism. MRI findings Symmetrical T2/FLAIR signal increase in the mammillary bodies, dorsomedial thalami, tectal plate, periaqueductal area, and around the third ventricle. Treatment Acute Phase (Wernicke Encephalopathy) : Intravenous thiamine (100 mg) for 3-7 days is standard; however, higher doses (500 mg TID) may be required if symptoms persist. Glucose should be administered after thiamine to prevent exacerbating symptoms. Chronic Phase (Korsakoff's Psychosis) : Continue oral thiamine indefinitely, especially in alcohol-dependent patients. Address co-occurring deficiencies , provide nutritional support , and consider rehabilitation or residential care if needed. Glucose infusion followed by thiamine injection (Option B): Giving glucose before thiamine can exacerbate Wernicke's encephalopathy, as it can increase the metabolic demand for thiamine, worsening the deficiency. Glucose infusion alone (Option C): Glucose infusion without thiamine can precipitate or worsen Wernicke's encephalopathy in patients with thiamine deficiency (such as in chronic alcoholics). Thiamine infusion alone (Option D): While thiamine is essential, glucose infusion is also necessary if the patient is hypoglycemic. Thiamine alone is insufficient for immediate management if glucose is needed for the patient's energy requirements.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Pg.542

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg.478

Wernicke-Korsakoff Syndrome | National Institute of Neurological Disorders and Stroke (nih.gov)

Wernicke-Korsakoff Syndrome: Practice Essentials, Background, Etiology (medscape.com)

↑ *Back to this question in Part A*

@Neet_pg_bot

Q22: A patient has come in with bleeding gums and the examination results are as follows. This particular condition can be observed in the case of a deficiency in (Q859575)

A. Vitamin A

B. Vitamin B12

C. Vitamin C

D. Vitamin D



Correct Answer: C. Vitamin C

Explanation: Vitamin C is essential for collagen production. Its deficiency causes scurvy, leading to symptoms like bleeding gums, swollen gums, and delayed wound healing. Vitamin C Dietary Sources : Amla, guava, lime, lemon, and green leafy vegetables. Requirements : The daily recommended intake is 75 mg/day, increased to 100 mg/day during pregnancy, lactation, for the elderly, smokers, and those on oral contraceptives. Scurvy : Vitamin C deficiency leads to scurvy, which can be severe in infants between 6–12 months (infantile scurvy or Barlow's disease). Hemorrhagic Tendency : Collagen is abnormal in vitamin C deficiency, causing fragile capillaries that lead to bleeding under pressure, seen as petechiae or ecchymoses. Internal Hemorrhage : Severe deficiency may cause hemorrhages in the conjunctiva, retina, nose (epistaxis), urine (hematuria), and stools (melena). Oral Cavity : Severe scurvy causes swollen, painful gums, and tooth loss due to separation of pulp from dentine. Wound healing is delayed. Bones : Vitamin C deficiency weakens bones, leading to fractures, joint hemorrhage, and pain due to failure of osteoblasts to form osteoid. Anemia : Vitamin C deficiency causes microcytic, hypochromic anemia, with poikilocytosis and anisocytosis. Causes include blood loss, decreased iron absorption, and decreased tetrahydrofolic acid. Vitamin A (Option A) : Deficiency in vitamin A is associated with night blindness, dry skin, and impaired immune function, not bleeding gums. Vitamin B12 (Option B) : Deficiency in vitamin B12 leads to megaloblastic anemia, neurological issues, and glossitis, but not specifically bleeding gums. Vitamin D (Option D) : Deficiency in vitamin D can lead to bone issues like rickets in children and osteomalacia in adults, but it does not directly cause bleeding gums.

References:

Vasudevan 7th edition Pg no. 497, 498

↑ *Back to this question in Part A*

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Q23: What is the probable vitamin deficiency in a patient who displays symptoms of diarrhea, memory impairment, and a distinctive rash as described? (Q859712)

A. Thiamine

B. Niacin

C. Cyanocobalamin

D. Ascorbic acid



Correct Answer: B. Niacin

Explanation: Pellagra is characterized by the classic triad of symptoms: Dermatitis Bright red erythema- in sun-exposed areas (face, ankles, feet) Casal's necklace - ↑pigmentation around the neck Diarrhoea, and Dementia. The patient's symptoms, combined with a diet high in maize and sorghum, both known to contribute to niacin and tryptophan deficiencies suggest pellagra as the most likely diagnosis. Sorghum has high leucine content which inhibits the QPRTase enzyme required for synthesis of Niacin from tryptophan. Additionally, Maize is low in tryptophan. {{caption_text}} Causes of pellagra: Dietary Deficiency- Maize and Sorghum based diet Impaired Tryptophan Metabolism Factors Affecting Tryptophan to Niacin Conversion: Vitamin B6 Deficiency Medications: Isoniazid (INH), OCPs Hormonal Influences: Estrogen metabolites hinder tryptophan metabolism. Genetic Disorders: Inherited conditions like Hartnup disease can impede tryptophan absorption in the intestines and

reabsorption in the kidneys Carcinoid Syndrome Other Contributing Factors: Alcoholism: Interferes with nutrient absorption, including niacin and tryptophan. Malabsorption Syndromes Incorrect Options: Thiamine (Option A) deficiency causes beriberi or Wernicke-Korsakoff syndrome , with symptoms like confusion, ataxia, and neuropathy, but not diarrhea or rash . Memory impairment in thiamine deficiency is usually associated with neurological issues, not the dermatitis seen in pellagra. Cyanocobalamin (Option C) is vitamin B12 , and its deficiency typically leads to pernicious anemia , neuropathy , and cognitive dysfunction . It does not present with diarrhea or a characteristic rash. Ascorbic acid (Option D) is vitamin C , and its deficiency leads to scurvy , characterized by gum bleeding , easy bruising , and poor wound healing , but not typically diarrhea or a rash like niacin deficiency.



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg.542,543

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg. 481,482

↑ *Back to this question in Part A*

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Q24: Which of the following enzyme activities can be estimated in red blood cells to diagnose vitamin B2 deficiency? (Q855541)

A. Transketolase

B. Glutathione reductase

C. Kynureninase

D. Pyruvate dehydrogenase

Correct Answer: B. Glutathione reductase

Explanation: In order to measure vitamin B2 levels, glutathione reductase activity in red blood cells can be monitored. Flavin adenine dinucleotide (FAD), a coenzyme generated from vitamin B2, is needed as a cofactor for the enzyme glutathione reductase inorder for it to work properly. Low FAD levels are caused by a vitamin B2 deficiency, and this lowers glutathione reductase activity. Vitamin deficiencies estimation by enzymes in RBC: Transketolase - Vit B1 Kynureninase - Vit B6 Pyurvate dehydrogenase - Vit C Incorrect options A) Transketolase: Used to assess vitamin B1 (thiamine) status, as it requires thiamine pyrophosphate (TPP) as a cofactor. C) Kynureninase: Requires vitamin B6 (pyridoxine) , not B2. D) Pyruvate dehydrogenase: Requires multiple cofactors — thiamine (B1), FAD (B2), NAD (B3), CoA (B5), and lipoic acid — but not specific for B2 deficiency testing.

References:

Vitamin B2

↑ *Back to this question in Part A*

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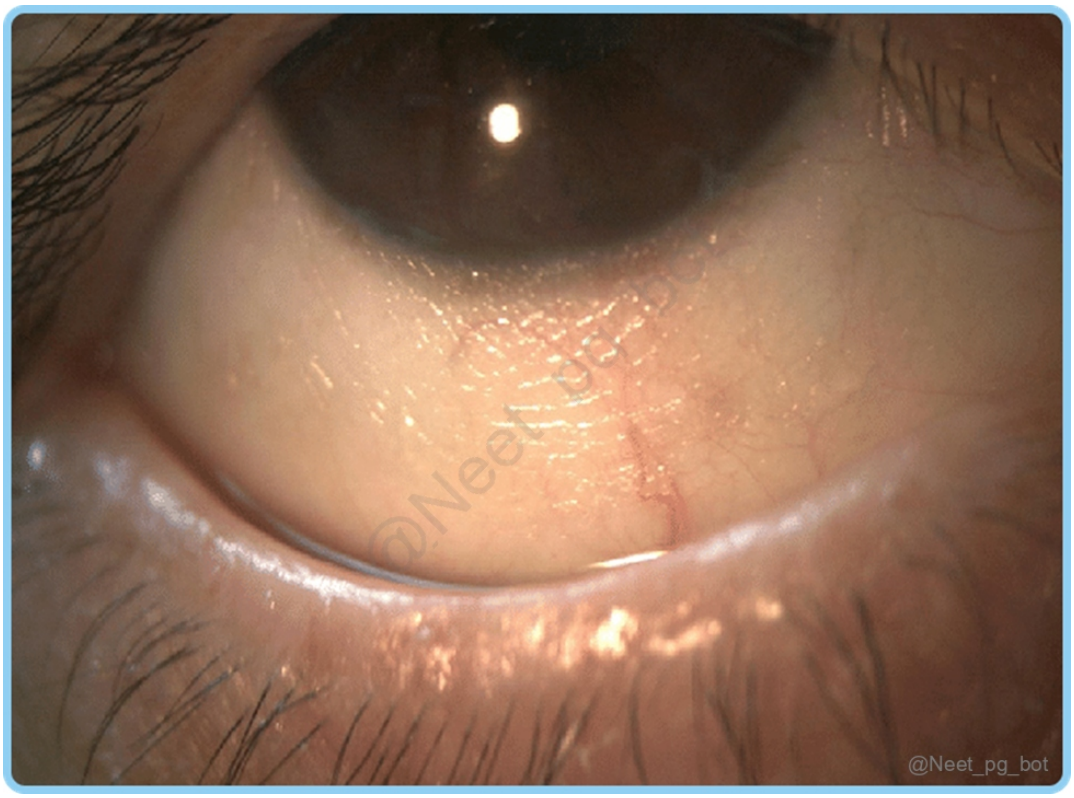
Q25: What is the cause of following condition due to? (Q855542)

A. Vitamin C

B. Vitamin A

C. Vitamin B

D. Vitamin E



Correct Answer: B. Vitamin A

Explanation: This disorder is known as Conjunctival xerosis caused by vitamin A deficiency where the conjunctiva becomes dry, rough and irritating XN Night blindness. X1 X1A: Conjunctival xerosis. X1B: Bitot spots. X2 Corneal xerosis. X3 X3A: Corneal ulceration, less than one-third. X3B: Corneal ulceration, more than one-third. XS Corneal scar. XF Xerophthalmic fundus. Skin manifestations of vit A deficiency : Phrynoderma: Follicular hyperkeratosis

References:

Harper Edition 32, Page no: 538

Lippincott Edition 7, page no:1112,1113

Vasudevan Edition 7, page no: 467,468

Image reference: Kanski's Ophthalmology Edition 9, page no:247

↑ *Back to this question in Part A*

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Q26: A 15-year-old boy presents with tremors, difficulty in speech, and behavioral changes. On examination, he has hepatomegaly and a golden-brown ring at the limbus of the cornea (Kayser-Fleischer ring). Wilson's disease is suspected. Which of the

following is the best investigation to support the diagnosis? (Q866667)

A. Serum ceruloplasmin

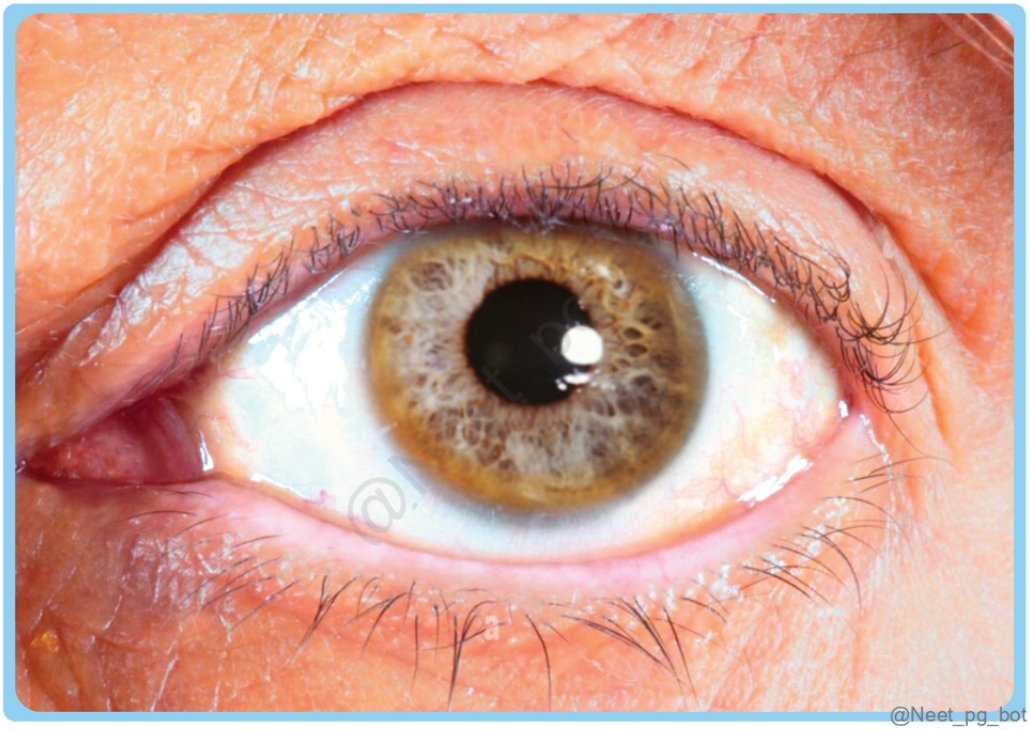
B. Liver biopsy for copper estimation

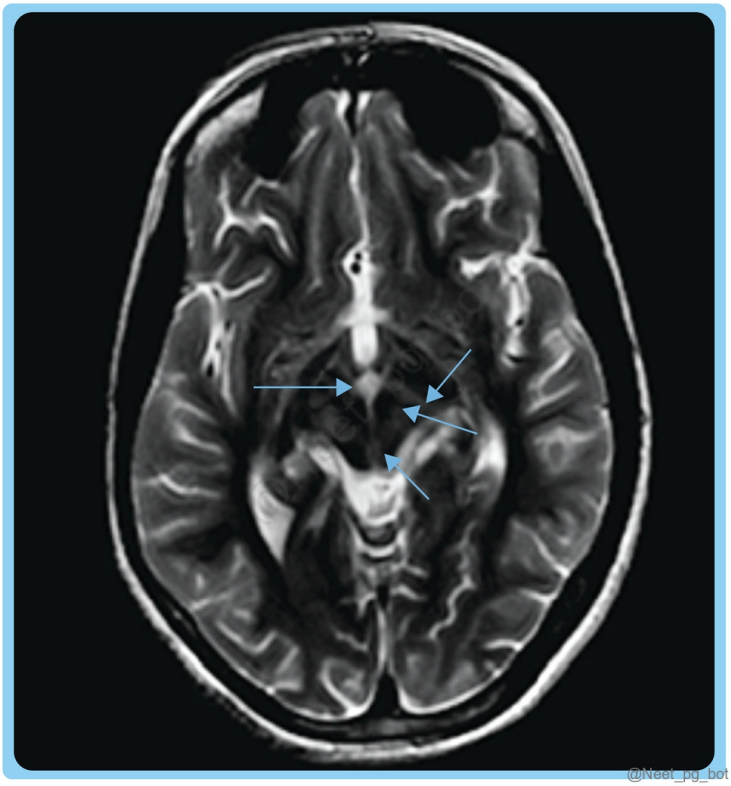
C. Serum copper

D. Copper excretion in urine

Correct Answer: B. Liver biopsy for copper estimation

Explanation: Wilson’s Disease is characterized by excessive copper accumulation, particularly in the liver and brain, and the gold standard for diagnosis is a liver biopsy with quantitative copper estimation. Inheritance Autosomal recessive Gene Mutation in ATP7B gene (chromosome 13) Defect Impaired copper excretion into bile → copper accumulation in liver , brain , cornea Clinical features Hepatic: Hepatitis, hepatomegaly, cirrhosis Neurological: Tremors, dysarthria, dystonia, parkinsonism Psychiatric: Behavioural changes, personality disorders Ocular: Kayser-Fleischer rings (golden-brown ring at corneal limbus) Diagnosis ↓ Serum ceruloplasmin (Option A ruled out) ↑ 24-hour urinary copper excretion (Option D ruled out) ↑ Hepatic copper on liver biopsy (gold standard) (Option B) MRI brain: Panda sign (in severe cases) Management Chelation therapy: Penicillamine or Trientine Zinc supplements: Inhibit copper absorption Liver transplant: In fulminant hepatic failure Serum copper (Option C) is usually low, since most copper is ceruloplasmin-bound.





References:

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↑ *Back to this question in Part A*

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Q27: A child presents with erythematous scaly patches in the perioral region, mucosal ulcers, and impaired epithelial wound healing. The most likely mineral deficiency associated with this condition is (Q855233)

A. Iron deficiency

B. Zinc deficiency

C. Calcium deficiency

D. Copper deficiency

Correct Answer: B. Zinc deficiency

Explanation: The above scenario is suggestive of Zinc deficiency. Zinc Deficiency Manifestations: Poor Wound Healing Skin Lesions: Rashes, dermatitis, and hyperkeratosis. Alopecia. Impaired Spermatogenesis. Immune Dysfunction: Decreased T and B lymphocyte counts ; impaired macrophage function. Neurological Effects: Depression, dementia, and other psychiatric disorders. Zinc also forms plaques with amyloid in Alzheimer's disease . Acrodermatitis Enteropathica: A genetic condition characterized by: Inflammation around the mouth, nose, and fingers. Diarrhea. Hair loss. Ophthalmoplegia and hypogonadism. Iron deficiency (Option A): Iron deficiency typically causes symptoms like pallor, fatigue, and glossitis but does not result in scaly patches or impaired wound healing. Calcium deficiency (Option C): Calcium deficiency primarily affects bone health, leading to rickets in children or osteomalacia in adults. Skin or mucosal symptoms are not characteristic. Copper deficiency (Option D): Copper deficiency can cause anemia, neutropenia, and neurological deficits. It is associated with poor connective tissue integrity (e.g., kinky hair in Menkes disease) but does not cause scaly patches or mucosal ulcers.

References:

Textbook of Biochemistry- Vasudevan, 7th Edition, Page 497, 522

↑ *Back to this question in Part A*

@Neet_pg_bot

Q28: Which of the following minerals is best known for insulin secretion stabilisation? (Q857866)

A. Cadmium

B. Copper

C. Iron

D. Zinc

Correct Answer: D. Zinc

Explanation: Zinc is an essential trace mineral that plays a crucial role in numerous biological processes, including insulin secretion and stabilization. It is involved in the synthesis, storage, and release of insulin from pancreatic beta cells. Zinc also helps in maintaining the structure and stability of insulin molecules. Zinc : Dietary Sources Grains, beans, nuts, cheese, meat, shellfish. Therapeutic Use Zinc reduces copper absorption in Wilson’s disease. Zinc-Dependent Enzymes Carboxypeptidase Carbonic anhydrase Alkaline phosphatase Lactate dehydrogenase Ethanol dehydrogenase Glutamate dehydrogenase RNA polymerase (required for protein biosynthesis) Functions Antioxidant RoleExtracellular superoxide dismutase is zinc-dependent, contributing to zinc’s antioxidant properties. Insulin and ZincZinc stabilizes insulin in beta cells of the pancreas but is not present in released insulin. Taste Sensation Gusten, a zinc-containing protein in saliva, is important for taste sensation. Deficiency Manifestations Poor wound healing Skin lesions, hyperkeratosis, dermatitis, alopecia Impaired spermatogenesis Reduced T and B lymphocyte numbers, impaired macrophage function Psychiatric disorders: depression, dementia, and cognitive decline (e.g., Alzheimer's) Acrodermatitis Enteropathica Acrodermatitis (inflammation

around mouth, nose, fingers, etc.) Diarrhea, alopecia, ophthalmoplegia, hypogonadism Toxicity Manifestations Chronic Toxicity: Gastric ulcers, pancreatitis, anemia, nausea, vomiting, pulmonary fibrosis Acute Toxicity: Fever, excessive salivation, headache, anemia Cadmium (Option A) is a toxic heavy metal that can have detrimental effects on health, including disrupting various physiological processes. Copper (Option B) is an essential trace mineral that plays a role in various bodily functions, including the formation of red blood cells and maintaining connective tissues. While it is involved in several enzymatic reactions. Iron (Option C) is vital for oxygen transport in the blood and is a key component of hemoglobin.

References:

Vasudevan Edition 7 page no:522

↑ *Back to this question in Part A*

@Neet_pg_bot

Q29: Menke's disease is due to a deficiency of: (Q857490)

A. Zinc

B. Copper

C. Iron

D. Magnesium

Correct Answer: B. Copper

Explanation: Menke's disease occurs due to a deficiency of copper due to a defect in the absorption of copper from the intestine. Menkes disease (also called kinky hair disease) is a rare, X-linked genetic disorder caused by a deficiency in copper absorption due to mutations in the ATP7A gene. Key features: Brittle, kinky hair Growth failure Neurodegeneration (seizures, developmental delay) Pathophysiology: Copper is essential for enzymes like lysyl oxidase (collagen cross-linking) and cytochrome c oxidase (cellular respiration). Impaired copper transport leads to systemic deficiency, affecting enzyme functions. The copper gets trapped in the metallothionine present in the intestinal cells. This prevents the copper from being absorbed into the bloodstream. The symptoms of Menke's disease include reduced copper in plasma and urine, anemia and depigmentation of hair. Can be treated by copper replacement therapy.

References:

Menke disease

↑ *Back to this question in Part A*

@Neet_pg_bot

Q30: The co-factor for glutathione peroxidase is: (Q857493)

A. Manganese

B. Selenium

C. Copper

D. Magnesium

Correct Answer: B. Selenium

Explanation: Selenium (Se) is an essential micronutrient for an antioxidant defence that integrates an important part of selenoproteins. The most well-known selenoproteins are five glutathione peroxidases (GPx), which protect cells from damage caused by free radicals such as reactive oxygen species (ROS). Reduce lipid hydroperoxides to their corresponding alcohols and to reduce free hydrogen peroxide to water. Manganese (Option A): Not the cofactor for glutathione peroxidase.It is a cofactor for manganese superoxide dismutase, arginase, and pyruvate carboxylase. Copper (Option C): Cofactor and regulator of copper, zinc superoxide dismutase. Magnesium (Option D): Cofactor in ATP-generating reactions.

References:

Glutathione peroxidase

↑ *Back to this question in Part A*

@Neet_pg_bot

Q31: Which one of the following is not classified as a micromineral? (Q859713)

A. Calcium

B. Selenium

C. lodine

D. Copper

Correct Answer: A. Calcium

Explanation: Minerals are crucial for normal growth and body maintenance. Those required in amounts greater than 100 mg/day are called macrominerals or major elements , while those needed in smaller amounts (less than 100 mg/day) are known as microminerals , trace elements , or minor elements . Calcium is classified as a macromineral (major element), not a micromineral. It is required by the body in large amounts (greater than 100 mg/day) for functions like bone formation, muscle contraction, and nerve transmission. Macrominerals Microminerals (Trace elements) Calcium (Option A) Magnesium Phosphorus Sodium Potassium Chloride Sulfur

Iron Iodine (Option C ruled out) Copper (Option D ruled out) Manganese Zinc Molybdenum Selenium (Option B ruled out) Fluoride

References:

Vasudevan 7th edition Pg no. 502, 503

↑ *Back to this question in Part A*

@Neet_pg_bot

Q32: A patient with megaloblastic anemia is mistakenly treated with folate alone, after which the neurological symptoms worsen rather than improve. Which of the following best explains this phenomenon? (Q879002)

A. Folate trap

B. Homocysteine accumulation

C. Masking of vitamin B12 deficiency by folate therapy

D. Pemicious anemia due to intrinsic factor deficiency

Correct Answer: A. Folate trap

Explanation: In vitamin B12 deficiency , 5-methyl-THF cannot be converted back to THF due to lack of methylcobalamin (B12). This results in: “Folate trap” → folate becomes trapped as 5-methyl-THF and cannot be used for DNA synthesis. Giving folate alone bypasses the hematologic block and improves anemia, but Neurological symptoms worsen because B12-dependent pathways (especially methylmalonyl-CoA → succinyl-CoA) remain impaired, leading to myelin damage. Thus, the worsening neurological symptoms occur due to the folate trap in untreated B12 deficiency . Incorrect Options: Homocysteine accumulation (Option B): Occurs but does not explain neurologic worsening after folate therapy. Masking of vitamin B12 deficiency (Option C): True hematologically, but the mechanism is still the folate trap. Pernicious anaemia (Option D): A cause of B12 deficiency, not the mechanism of neurological worsening.

References:

Textbook of Biochemistry - DM Vasudevan, 7th Edition, Page 490, 491

↑ *Back to this question in Part A*

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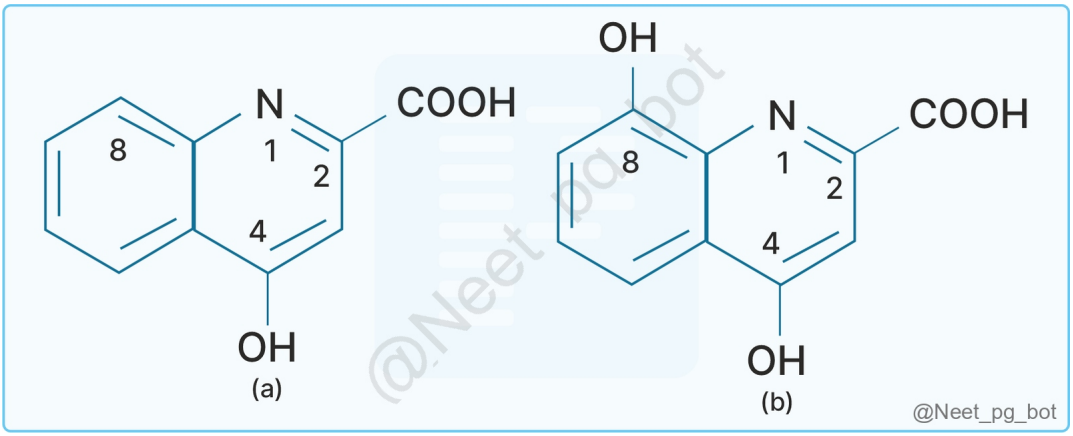
Q33: The vitamin deficiency that impairs the conversion of 3-hydroxykynurenine to xanthurenic acid is (Q879284)

A. Pyridoxal phosphate (PLP)

B. Biotin

C. Thiamine

D. B12



Correct Answer: A. Pyridoxal phosphate (PLP)

Explanation: Pyridoxal phosphate (PLP), the active form of vitamin B6 , is required for the conversion of 3-hydroxykynurenine to xanthurenic acid. PLP deficiency reduces the activity of kynureninase and kynurenine aminotransferase, leading to the accumulation of 3-hydroxykynurenine and decreased xanthurenic acid production. Incorrect Options: Biotin (Option B): Involved in carboxylation reactions, not tryptophan metabolism. Thiamine (Option C): Functions as a coenzyme in the decarboxylation of keto acids, unrelated to 3-hydroxykynurenine conversion. ■■■■■■■ Vitamin B12 (Option D): Required for methylation reactions and red blood cell formation, not for this conversion.

References:

DM Vasudevan's Textbook of Biochemistry, 7th Edition, Page 240, 482-483.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q34: A patient presents with gum bleeding, petechiae, poor wound healing, and other related symptoms. Which of the following vitamin deficiencies is most likely responsible for this presentation? (Q852545)

A. Vitamin A

B. Vitamin B6

C. Vitamin B3

D. Vitamin C

Correct Answer: D. Vitamin C

Explanation: Gum bleeding & petechiae: Classic signs of scurvy , caused by vitamin C deficiency, due to impaired collagen synthesis leading to fragile capillaries. Poor wound healing: Vitamin C is crucial for collagen formation and connective tissue repair , and its deficiency results in defective wound healing. Other symptoms of scurvy: Fatigue, corkscrew hairs, anemia, and subperiosteal hemorrhages. Vitamin C Deficiency (Scurvy) Feature Vitamin C Deficiency (Scurvy) Cause Poor dietary intake (malnutrition, alcoholism, elderly, infants on cow's milk) Key Symptoms Bleeding gums, petechiae, bruising, corkscrew hairs, poor wound healing Other Signs Subperiosteal hemorrhage, joint pain, anemia, weakness Diagnosis Clinical features, low plasma vitamin C levels Treatment Vitamin C supplementation Vitamin A Deficiency (Option A): Incorrect because it causes night blindness, dry skin, and keratomalacia, not bleeding gums or petechiae. Vitamin B6 Deficiency (Option B): Incorrect because it presents with irritability, glossitis, peripheral neuropathy, and seizures, not connective tissue defects. Vitamin B3 (Niacin) Deficiency (Option C): Incorrect because it causes pellagra (3 Ds- diarrhea, dermatitis, dementia) with photosensitive rash, not gum bleeding.

References:

Nelson textbook of pediatrics, 21st edition, Pages 373,374, 375.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7607440/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q35: Which vitamin acts as a coenzyme required for Glucose-6-Phosphate Dehydrogenase (G6PD)? (Q860364)

A. Riboflavin

B. Thiamine

C. Niacin

D. Biotin

Correct Answer: C. Niacin

Explanation: Niacin, in the form of nicotinamide adenine dinucleotide phosphate (NADP+), is required as a coenzyme for G6PD. This enzyme catalyzes the first step of the pentose phosphate pathway, producing NADPH, which is essential for reductive biosynthesis and maintaining reduced glutathione in cells. Niacin (Vitamin B3) Chemical Structure Niacin : Pyridine-3-carboxylic acid. Niacinamide : Acid amide derivative. Active tissue form: Niacinamide Coenzyme Forms and Functions NAD+ : Generates 2.5 ATPs in oxidative phosphorylation. NADPH : Used in reductive biosynthesis (e.g., fatty acid synthesis). Provides ADP-ribose for protein modification (ADP-ribosylation). Synthesis and Metabolism 60 mg of tryptophan = 1 mg niacin (requires pyridoxal phosphate-dependent kynureninase). Niacin attaches to ribose phosphate, forming a mononucleotide, then combines with AMP to form dinucleotides. Deficiency Causes Dietary Deficiency: Staple diets like maize (bound niacin) or sorghum (leucine inhibits niacin conversion). Enzyme Defects: Pyridoxal deficiency inhibits kynureninase activity. Drugs: Isoniazid inhibits pyridoxal phosphate formation. Congenital Disorders: Hartnup disease (impaired tryptophan absorption and increased urinary excretion). Carcinoid Syndrome : Excess tryptophan used for serotonin synthesis. Clinical Condition Pellagra : Niacin or tryptophan deficiency causing "rough skin". Symptoms (3 Ds): Dermatitis (Casal’s necklace), Diarrhea (may involve blood/mucus), Dementia (irritability, poor memory, delirium). Sources Richest: Dried yeast, rice , liver, peanuts, cereals, legumes, meat, fish. 50% requirement met by tryptophan conversion. RDA Adults : 20 mg/day ; Additional 5 mg during lactation. Therapeutic Uses Lowers serum cholesterol and Lp(a) by reducing free fatty acid flux from adipose tissue. Toxicity Excess (>50 mg/day): Vasodilation, itching, liver damage. Riboflavin (Option A): Riboflavin is a precursor for flavin adenine dinucleotide (FAD) and flavin mononucleotide (FMN), which are coenzymes in redox reactions. However, these are not directly involved with G6PD activity. Thiamine (Option B): Thiamine, as thiamine pyrophosphate (TPP), is a coenzyme for enzymes involved in carbohydrate metabolism, such as pyruvate dehydrogenase and transketolase, but not for G6PD. Biotin (Option D): Biotin serves as a coenzyme for carboxylase enzymes involved in carboxylation reactions, such as pyruvate carboxylase, but not for G6PD.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg.542, 543

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg.480-482

↑ *Back to this question in Part A*

@Neet_pg_bot

Q36: Which of the following are metabolites of methanol poisoning? (Q844278)

A. Formic acid + Oxalic acid

B. Formic acid + Lactic acid

C. Glycolic acid + Oxalic acid

D. Lactic acid + Glycolic acid

Correct Answer: B. Formic acid + Lactic acid

Explanation: In methanol poisoning, metabolites like formic acid and lactic acid accumulate and cause toxic effects like histotoxic hypoxia and optic neuritis. Methanol poisoning causes formation of Formate ions . Acids formed lead to defective mitochondria and lactic acidosis, which increase AG. Increase in plasma osmolality. Osmolal gap is also seen Methanol, also known as wood alcohol, is a CNS depressant often found in illicit liquors. It is highly toxic. Methanol toxicity is infamous for causing metabolic acidosis and optic nerve damage, leading to snowfield vision and potential blindness. Methanol Poisoning: Metabolism and Pathophysiology: Methanol → Formaldehyde (via alcohol dehydrogenase). Formaldehyde → Formic acid (via aldehyde dehydrogenase). Formic acid leads to: Metabolic acidosis. Retinal damage (optic neuritis). Symptoms: Early: Severe abdominal cramps. Neurological: Snowfield vision, vision loss, dilated and fixed pupils, muscular weakness . Systemic: CNS and cardiac depression. Treatment: Stabilization: Immediate supportive care. Hemodialysis: To remove methanol and formic acid. Antidotes: Ethanol: Competes with methanol for alcohol dehydrogenase. Fomepizole: Inhibits alcohol dehydrogenase. Other Therapies: Folinic acid: Enhances formic acid metabolism. Sodium bicarbonate: Treats acidosis. Glycolic acid and oxalic acid (Option A,C,D) is mainly formed in ethylene glycol poisoning Postmortem Findings: Putamen hemorrhagic necrosis (specific finding). Absence of postmortem clotting.

References:

.Knight's Forensic Pathology, 4th Edition, Page 586 Textbook of Biochemistry by DM Vasudevan, 7th edition, Page 400

↑ *Back to this question in Part A*

@Neet_pg_bot

Q37: A child presents with deformities, normal serum calcium, normal PTH, low phosphate, and elevated ALP. Which is the most likely diagnosis? (Q844271)

A. Hypophosphatemic rickets

B. Vitamin D Dependent Rickets Type 1 (VDDR type 1)

C. Vitamin D Dependent Rickets Type 2 (VDDR type 2)

D. Renal osteodystrophy

Correct Answer: A. Hypophosphatemic rickets

Explanation: Correct Option A - Hypophosphatemic rickets: Hypophosphatemic rickets is characterized by low phosphate levels, normal calcium and PTH, and elevated ALP, leading to bone deformities. Incorrect Options: Option B and C - VDDR type 1 & 2 present with low calcium and elevated PTH. Option D - Renal osteodystrophy will present with underlying renal disease.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q38: A patient presents with anemia, neutropenia, and hypopigmentation of skin and hair. Which micronutrient deficiency is most likely? (Q844267)

A. Zinc

B. Fluoride

C. Copper

D. Selenium

Correct Answer: C. Copper

Explanation: Correct Option C – Copper: Copper deficiency leads to a triad of: Anemia (due to impaired iron mobilization from tissues to plasma — copper is required for ceruloplasmin and ferroxidase activity), Neutropenia , and Hypopigmentation of skin and hair (due to defective activity of tyrosinase, a copper-dependent enzyme). Copper deficiency is often seen in: Malabsorption , Post–gastric bypass surgery , Excessive zinc supplementation (which induces metallothionein that traps copper in enterocytes). Incorrect Options: A) Zinc: Deficiency causes periorificial dermatitis, alopecia, and poor wound healing — not anemia or neutropenia. B) Fluoride: Deficiency affects dental enamel formation but does not cause hematologic or pigmentation abnormalities. D) Selenium: Deficiency causes cardiomyopathy (Keshan disease) and muscle weakness, not anemia or pigmentation changes.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q39: A patient presents with follicular hyperkeratosis on the extensor aspect of the forearm and has a dietary history of Vitamin A and C deficiency. What is the diagnosis? (Q844247)

A. Phrynoderma

B. Scurvy

C. Pellagra

D. Keratosis pilaris

Correct Answer: A. Phrynoderma

Explanation: Correct Option A – Phrynoderma: Phrynoderma: Also known as follicular hyperkeratosis, this condition is characterized by small, rough papules, typically on the extensor surfaces of limbs, due to Vitamin A deficiency. Incorrect Options: Option B - Scurvy is caused by Vitamin C deficiency; it presents with symptoms like bleeding gums, bruising, and joint pain, not follicular hyperkeratosis. Option C - Pellagra is caused by niacin (Vitamin B3) deficiency, presenting with the classic triad of dermatitis, diarrhea, and dementia. Option D - Keratosis pilaris is not associated with Vitamin A deficiency.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q40: Which micronutrient deficiency causes anemia, bleeding gums, and Frenkel's white line on X-ray? (Q844253)

A. Vitamin K

B. Vitamin A

C. Vitamin C

D. Vitamin E

Correct Answer: C. Vitamin C

Explanation: Correct Option C - Vitamin C: Vitamin C (ascorbic acid) deficiency leads to scurvy, which manifests with: Bleeding gums, petechiae, and poor wound healing due to defective collagen synthesis, Anemia (from chronic bleeding and impaired iron absorption), and Skeletal changes in children such as Frenkel's white line on X-ray — a dense metaphyseal line representing defective osteoid formation and disordered bone growth. Vitamin C is required for hydroxylation of proline and lysine residues in collagen and for iron absorption in the intestine. Incorrect Options: Options A, B, and D do not present with the given clinical condition.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q41: A 45-year-old patient with a known case of homocystinuria presents with joint pain and weakness. Which vitamin is required for the treatment? (Q844246)

A. Vitamin B6

B. Vitamin B12

C. Vitamin B1

D. Vitamin B7

Correct Answer: A. Vitamin B6

Explanation: Correct Option A - Vitamin B6: Homocystinuria is most commonly caused by a deficiency of the enzyme cystathionine β-synthase , which requires pyridoxal phosphate (Vitamin B6) as a coenzyme. Deficiency of this enzyme leads to accumulation of homocysteine and methionine , resulting in: Skeletal abnormalities (joint pain, osteoporosis, marfanoid habitus) Weakness and developmental delay Thromboembolic episodes (due to endothelial damage) Pyridoxine (Vitamin B6) supplementation can significantly reduce homocysteine levels in B6-responsive forms of homocystinuria. Incorrect Options: B) Vitamin B12: Required for methionine synthase, which converts homocysteine → methionine, but B6 deficiency is the classic cause of homocystinuria type I. C) Vitamin B1 (Thiamine): Coenzyme in oxidative decarboxylation reactions, unrelated to sulfur amino acid metabolism. D) Vitamin B7 (Biotin): Involved in carboxylation reactions, not homocysteine metabolism.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q42: Which deficiency is most likely in a patient with delayed wound healing and alopecia? (Q852568)

A. Zinc

B. Copper

C. Calcium

D. Vitamin D

Correct Answer: A. Zinc

Explanation: Zinc plays a crucial role in wound healing, protein synthesis, and maintenance of skin integrity, including hair follicle health. The combination of delayed wound healing and alopecia is particularly characteristic of zinc deficiency , as zinc is essential for both collagen synthesis and cell division necessary for tissue repair and hair growth. Zinc Function Component of metalloenzymes Involved in protein, DNA, and RNA synthesis Necessary for steroid hormone receptors Essential for spermatogenesis and fetal growth Clinical Features of Zinc Deficiency Diarrhea Alopecia Muscle wasting Depression Irritability Rash Delayed wound healing Scaly dermatitis Diagnostic Level of Zinc Deficiency Serum zinc level <12 µmol/L (70 µg/dL) Copper (Option B): While copper is important for collagen cross-linking and wound healing, it is not typically associated with alopecia

as a primary manifestation. The primary features of copper deficiency include anemia and neutropenia. Copper deficiency more commonly presents with neurological symptoms and hematological abnormalities. Calcium (Option C): Calcium deficiency primarily affects bone metabolism and neuromuscular function. It does not typically cause delayed wound healing or alopecia. The classic manifestations of calcium deficiency include tetany, muscle cramps, and bone abnormalities. Vitamin D (Option D): Vitamin D deficiency primarily affects calcium homeostasis and bone metabolism. While it can have wide-ranging effects, delayed wound healing and alopecia are not characteristic features. The primary manifestations of vitamin D deficiency include bone pain, muscle weakness, and increased risk of fractures.

References:

Harrison's Principles of Internal Medicine, 21st Edition, Page 2532

↑ *Back to this question in Part A*

@Neet_pg_bot

Q43: Which of the following is a mitochondrial matrix enzyme? (Q860380)

A. Citrate synthase

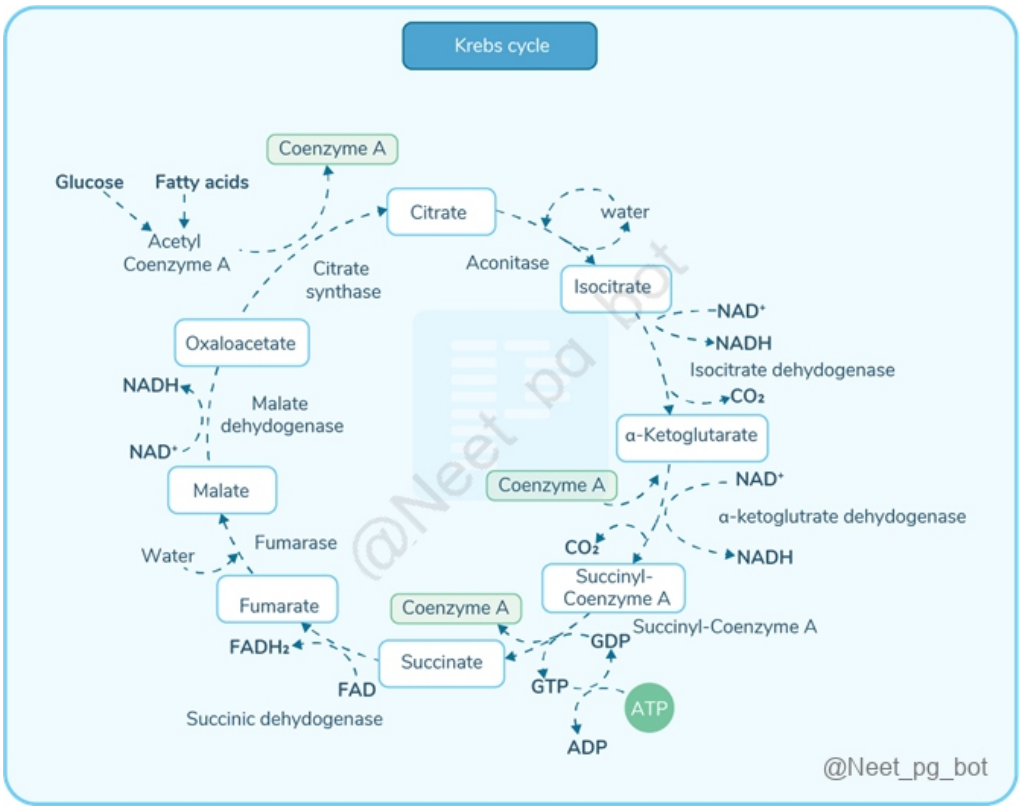
B. Monoamine oxidase

C. ATP synthase

D. Catalase

Correct Answer: A. Citrate synthase

Explanation: Citrate synthase is a key enzyme of the tricarboxylic acid (TCA) cycle and is located in the mitochondrial matrix. It catalyzes the condensation of oxaloacetate and acetyl-CoA to form citrate. Monoamine oxidase (Option B): Monoamine oxidase is an enzyme located in the outer mitochondrial membrane and is involved in the oxidative deamination of monoamines, such as serotonin and dopamine. ATP synthase (Option C): ATP synthase is embedded in the inner mitochondrial membrane and extends into the matrix. While it has a role in the matrix during ATP production, it is not classified as a matrix enzyme. Catalase (Option D): Catalase is an enzyme located in peroxisomes , not mitochondria. It catalyzes the decomposition of hydrogen peroxide into water and oxygen.



References:

Harper’s Illustrated Biochemistry 32nd Edition Pg - 158,159

Monoamine Oxidase B Prompts Mitochondrial and Cardiac Dysfunction in Pressure Overloaded Hearts - PMC

Understanding structure, function, and mutations in the mitochondrial ATP synthase - PMC).

Role of Catalase in Oxidative Stress- and Age-Associated Degenerative Diseases - PMC.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q44: A baby presents with the clinical features shown in the image, including characteristic perioral and perianal rash along with a history of diarrhea. Which of the following is the most likely deficiency responsible for this condition? (Q852463)

A. Zinc Deficiency

B. Niacin Deficiency

C. Selenium Deficiency

D. Magnesium Deficiency



Correct Answer: A. Zinc Deficiency

Explanation: Characteristic rash: The image likely shows acrodermatitis enteropathica , which presents with perioral, perianal, and acral dermatitis —a hallmark of zinc deficiency . History of diarrhea: Zinc plays a crucial role in intestinal function and immunity ; its deficiency leads to chronic diarrhea. Other symptoms of zinc deficiency: Growth retardation, poor wound healing, alopecia, and immune dysfunction. Zinc Deficiency & Acrodermatitis Enteropathica Feature Zinc Deficiency Cause Congenital (SLC39A4 mutation) or acquired (malnutrition, chronic diarrhea) Key Skin Features Perioral, perianal, and acral dermatitis with erythematous, scaly plaques Other Symptoms Growth retardation, diarrhea, alopecia, immune dysfunction Diagnosis Low serum zinc levels Treatment Zinc supplementation Niacin Deficiency (Option B) is incorrect because niacin deficiency leads to pellagra , characterized by the "3 Ds"— dermatitis, diarrhoea, and dementia —but the rash is photosensitive and occurs in sun-exposed areas, not perioral/perianal. Selenium Deficiency (Option C) is incorrect because selenium deficiency primarily affects the cardiovascular system (e.g., Keshan disease , causing cardiomyopathy) and does not cause a perioral/perianal rash. Magnesium Deficiency (Option D) is incorrect because magnesium deficiency presents with neuromuscular symptoms (tetany, muscle cramps, seizures) rather than a characteristic rash.



References:

Vasudevan, 7th edition, Page 497, 522

↑ *Back to this question in Part A*

@Neet_pg_bot

Q45: In a patient who presents with Phrynoderma as shown. What is the next best step in management of this patient? (Q866335)

A. Ocular examination

B. Check vitamin D levels

C. Check vitamin B12 levels

D. Check for DTR



Correct Answer: A. Ocular examination

Explanation: Phrynoderma, as illustrated and commonly known as "toad skin," presents with follicular hyperkeratotic papules, and since it is strongly linked to vitamin A deficiency, which can lead to ocular complications, the appropriate next step is an ocular examination to check for signs like xerosis, Bitot’s spots, or other early eye involvement. Vitamin A Deficiency Manifestations Ocular manifestations Vitamin A deficiency is the most significant preventable cause of blindness, especially in children below 5 in developing countries. Early Signs: Loss of sensitivity to green light. Difficulty adapting to dim light. Night blindness. Prolonged Deficiency: Xerophthalmia: Dry, thick, keratinized conjunctiva and cornea. If untreated, it leads to corneal ulceration and ultimately results in blindness due to the formation of opaque scar tissue. Bitot's Spots: Grayish-white triangular plaques firmly adherent to the conjunctiva. Keratomalacia: When xerophthalmia persists for a longer time, it progresses to keratomalacia (corneal softening). Immune Function Deficiency increases susceptibility to infections. Infection reduces retinol-binding protein synthesis, lowering vitamin A levels and impairing immune responses. Skin and Mucous Membrane Lesions Follicular Hyperkeratosis (Phrynoderma): Causes rough skin due to hyperkeratinization of the follicular epithelium. (Option A) Keratinizing Metaplasia: Affects respiratory, gastrointestinal, and genitourinary tracts. Epithelium is atrophied. Other General Manifestation Growth retardation due to defective synthesis of chondroitin sulfate. Decreased protein synthesis. WHO grading of xerophthalmia: XN Night blindness. X1 X1A: Conjunctival xerosis. X1B: Bitot spots. X2 Corneal xerosis. X3 X3A: Corneal ulceration, less than one-third. X3B: Corneal ulceration, more than one-third. XS Corneal scar. XF Xerophthalmic fundus. Check vitamin D levels (Option B): Vitamin D is not primarily associated with phrynoderma. Check vitamin B12 levels (Option C): B12 deficiency presents with neurologic symptoms, not follicular keratosis. Check for DTR (Option D): Deep tendon reflexes are not relevant to the cutaneous signs of vitamin A deficiency.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q46: A patient on long-term hydrochlorothiazide therapy presents with features of neuropathy, heart failure, and symmetrical tingling sensations. Which of the following nutrient deficiencies is most likely responsible? (Q866326)

A. Selenium

B. Thiamine

C. Vitamin B12

D. Zinc

Correct Answer: B. Thiamine

Explanation: Long-term diuretic use like hydrochlorothiazide can increase Thiamine excretion and cause deficiency. Thiamine (Vitamin B1) deficiency Classic cause of beriberi , which can be: Dry beriberi : Symmetrical peripheral neuropathy (tingling, numbness). Wet beriberi : High-output heart failure and edema. Long-term diuretic use like hydrochlorothiazide can increase thiamine excretion and cause deficiency. (Option B) Fits neuropathy + heart failure + tingling symptoms very well. Selenium (Option A): Deficiency mainly causes cardiomyopathy but not neuropathy or tingling sensations. Vitamin B12 (Option C): Causes neuropathy but usually with anemia and cognitive symptoms, not heart failure. Zinc (Option D): Deficiency leads to skin problems and immune dysfunction, not neuropathy or heart failure.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q47: A patient presents with anemia, positive Romberg sign, and other neurological findings suggestive of vitamin B12 deficiency. Laboratory findings show elevated homocysteine levels. Which amino acid is likely to be deficient in this patient? (Q866321)

A. Cysteine

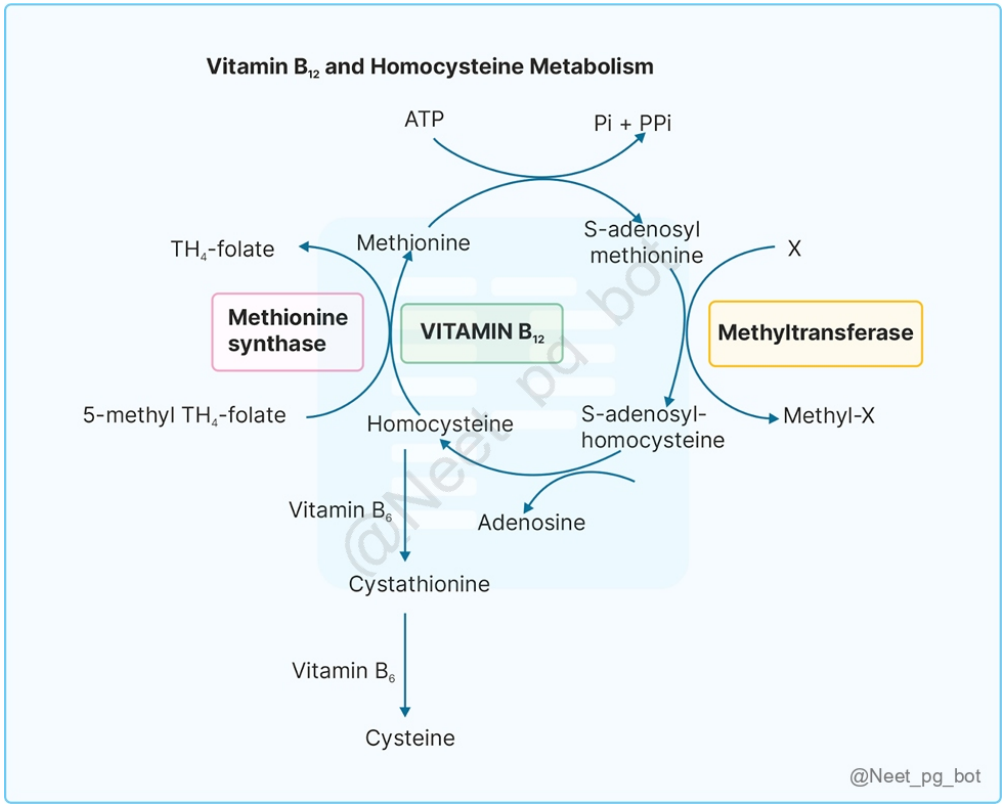
B. Methionine

C. Tyrosine

D. Glutamate

Correct Answer: B. Methionine

Explanation: The patient's anemia, positive Romberg sign, neurological symptoms, and elevated homocysteine indicate vitamin B12 deficiency, which impairs methionine synthase activity and leads to methionine deficiency despite high homocysteine levels. Vitamin B12-dependent Enzymes: Methyl-malonyl-CoA Mutase: Function: Converts methylmalonyl-CoA to succinyl-CoA. Methyl-malonyl CoA is an intermediate in the breakdown of valine, cholesterol, odd-chain fatty acids, propionate, and carboxylation of propionyl-CoA. Deficiency Impact: Leads to accumulation of methylmalonyl-CoA and increased urinary methylmalonic acid, indicating B12 deficiency status. Methylmalonic acid will be normal in megaloblastic anaemia caused by folate deficiency. Methionine Synthase: Function: Converts homocysteine to methionine. Role: Impairment affects folic acid metabolism, leading to functional folate deficiency. Deficiency Impact: Causes pernicious anaemia and spinal cord degeneration due to failed methylation of arginine in myelin basic protein. This is a result of methionine deficiency in the CNS. Cysteine (Option A): Synthesized from homocysteine via the transsulfuration pathway (requires vitamin B6), not directly affected in B12 deficiency. Tyrosine (Option C): Formed from phenylalanine, unrelated to B12 metabolism. Glutamate (Option D): Involved in nitrogen metabolism and neurotransmission; not affected by B12 or homocysteine levels.



References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q48: A person with jaundice presented with a history of bleeding, and symptoms normalised after giving a Vitamin K injection. The probable cause is? (Q866316)

A. Biliary obstruction

B. Alpha antitrypsin deficiency

C. Autoimmune hepatitis

D. Factor XI deficiency

Correct Answer: A. Biliary obstruction

Explanation: The patient's jaundice and bleeding tendency that resolved with vitamin K suggest a vitamin K deficiency due to biliary obstruction, which impairs bile flow and subsequently the absorption of fat-soluble vitamins like Vitamin K , leading to defective clotting. Fat-soluble vitamin that acts as a coenzyme or prosthetic group for gamma-carboxylase. Calcium-dependent proteins that rely on Vitamin K: Clotting factors II, VII, IX, and X. Anticoagulant factors like protein C and S. Osteocalcin (promotes bone mineralization by depositing calcium). Forms of Vitamin K: Phylloquinone (K1): Found in green vegetables, this form has an isoprenoid side chain with 20 carbons. Menaquinones (K2): Synthesized by intestinal bacteria, these have varying side chain lengths and an isoprenoid side chain containing 30 carbons, which is lipid soluble. Menadione (K3): A synthetic form which is water-soluble. Menadione and menadione diacetate can be metabolized into phylloquinone. Vitamin K exists in two forms: Epoxide and Hydroquinone form. Functions : Blood Clotting. Bone Health - Carboxylating glutamate to glutamic acid in proteins like osteocalcin is involved in bone mineralization. Alpha antitrypsin deficiency (Option B) can cause liver disease and jaundice, but does not cause vitamin K–responsive bleeding. Autoimmune hepatitis (Option C) causes liver inflammation, but bleeding due to this is typically from synthetic liver dysfunction, not corrected by vitamin K. Factor XI deficiency (Option D) is a hereditary bleeding disorder, not related to vitamin K or jaundice, and not corrected by vitamin K injection.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q49: A child presents with a history of fractures, multiple petechiae, perifollicular hemorrhages, and gum bleeding. Which of the following enzyme defects is involved? (Q866315)

A. Lysyl oxidase

B. Prolyl hydroxylase

C. Tyrosinase

D. Alkaline phosphatase

Correct Answer: B. Prolyl hydroxylase

Explanation: The child shows classic signs of Scurvy , caused by vitamin C deficiency, where vitamin C acts as a cofactor for prolyl hydroxylase—an enzyme crucial for hydroxylating proline and lysine residues, thereby stabilizing and cross-linking the collagen triple helix. Cause: Vitamin C deficiency Enzyme Affected: Prolyl hydroxylase (also lysyl hydroxylase) → Requires vitamin C as a cofactor (Option B) Hydroxylates proline and lysine residues in collagen → Essential for collagen stability and cross-linking. Clinical Features: Bone fractures (due to defective bone matrix) Petechiae, perifollicular hemorrhages Bleeding gums, poor wound healing Pathophysiology: Defective collagen → Weak blood vessels and connective tissue Lysyl oxidase (Option A) is a copper-dependent enzyme involved in cross-linking collagen and elastin. A defect in this enzyme causes conditions like Ehlers-Danlos syndrome or Menkes disease, but not scurvy. Tyrosinase (Option C) plays a role in melanin synthesis from tyrosine. Its deficiency leads to albinism, which affects pigmentation rather than connective tissue. Alkaline phosphatase (Option D) is important for bone mineralisation. Deficiency of this enzyme results in hypophosphatasia, causing rickets-like symptoms, but it does not cause bleeding or petechiae.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q50: Which vitamin deficiency can lead to lactic acidosis? (Q866652)

A. Thiamine

B. Riboflavin

C. Niacin

D. Biotin

Correct Answer: A. Thiamine

Explanation: Thiamine (Vitamin B1) is a cofactor for pyruvate dehydrogenase; its deficiency blocks pyruvate conversion to acetyl-CoA, causing excess pyruvate to convert into lactate and resulting in lactic acidosis, as seen in beri-beri and Wernicke’s encephalopathy. Source The Aleurone layer of cereals (food grains) is a rich source of thiamine. Therefore, whole wheat flour and unpolished hand-pound rice have better nutritive value than completely polished refined foods. When the grains are polished, the aleurone layer is usually removed. Deficiency Dry beriberi: CNS manifestations are more common Pain, paresthesia and loss of reflexes Peripheral neuritis with sensory disturbances Wet beriberi: Wet beriberi is characterized by cardiovascular symptoms such as breathlessness, palpitations, and peripheral oedema. Distended neck veins observed High output cardiac failure Biochemical parameters Thiamine deficiency affects the enzyme transketolase, which is involved in the pentose phosphate pathway. Measuring transketolase activity in red blood cells is a specific test for thiamine deficiency and can confirm the diagnosis. Erythrocyte transketolase activity is reduced; this is the earliest manifestation seen even before clinical disturbances. RDA Recommended Daily Allowance of Thiamine is 1–1.5 mg/day. Incorrect Options: Riboflavin (vitamin B2) (Option B) is involved in electron transport chain but not directly linked to lactic acidosis. Niacin (vitamin B3) (Option C) is precursor of NAD + /NADP + , deficiency causes pellagra. Biotin (Option D) is cofactor for carboxylases, deficiency causes multiple carboxylase deficiency, but not typically lactic

acidosis.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q51: Features of Korsakoff Psychosis are all except Amnesia Confabulation Opthalmoplegia Polyneuropathy (Q839944)

A. 1, 2,3

B. 2,3,4

C. 3

D. 3,4

Correct Answer: C. 3

Explanation: Korsakoff psychosis is a chronic neuropsychiatric complication of thiamine (vitamin B■) deficiency , often following Wernicke's encephalopathy in chronic alcoholics. It is characterized by: Anterograde and retrograde amnesia Confabulation (filling memory gaps with fabricated stories) Apathy and lack of insight However, ophthalmoplegia (along with ataxia and confusion) is a feature of Wernicke's encephalopathy , not of Korsakoff psychosis. Polyneuropathy may coexist in chronic alcoholics with thiamine deficiency but can be seen in both Wernicke and Korsakoff stages.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q52: Which of the following statements is not true regarding folate deficiency? (Q839953)

A. Folate supplementation in pregnancy prevents NTD

B. High Homocysteine and Normal Methylmalonic acid

C. Hemolytic anemia is a feature

D. Treat coexisting B12 deficiency before initiating folate therapy

Correct Answer: C. Hemolytic anemia is a feature

Explanation: Correct Option C - Hemolytic anemia is a feature: Hemolytic anemia is not a feature of folate deficiency. Folate deficiency typically causes megaloblastic anemia, characterized by the presence of large, immature red blood cells. Hemolytic anemia involves the destruction of red blood cells and is not directly related to folate deficiency. Incorrect Options: Option B - High Homocysteine and Normal Methylmalonic Acid: In folate deficiency, homocysteine levels are elevated because folate is necessary for its conversion to methionine. However, methylmalonic acid levels remain normal because MMA accumulation is specifically associated with vitamin B12 deficiency. Option A - Folate supplementation in pregnancy prevents NTD : Folate, also known as folic acid when it's in its synthetic form, is crucial during pregnancy for preventing neural tube defects (NTDs) in the developing fetus. Option D - Treat coexisting B12 deficiency before initiating folate therapy : B12 deficiency indirectly causes folate deficiency. It's important to treat coexisting B12 deficiency before initiating folate therapy. Folate supplementation can improve the production of red blood cells, which may mask the anemia symptoms of B12 deficiency. This can lead to a delay in diagnosing and treating the underlying B12 deficiency, which, if left untreated, can cause irreversible nerve damage.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q53: The image illustrates which vitamin deficiency? (Q860071)

A. Vitamin A

B. Vitamin E

C. Vitamin C

D. Niacin (Vitamin B3) Deficiency



Correct Answer: D. Niacin (Vitamin B3) Deficiency

Explanation: The patient’s presentation with diarrhea, dermatitis (lesions on the neck resembling Casal's necklace), and likely dementia indicates pellagra, a condition caused by niacin (vitamin B3) deficiency. Maize lacks adequate niacin and tryptophan (a precursor of niacin), predisposing individuals to pellagra when consumed as a staple without proper processing or supplementation. Niacin (Vitamin B3) Chemical Structure Niacin : Pyridine-3-carboxylic acid. Niacinamide : Acid amide derivative. Active tissue form: Niacinamide Coenzyme Forms and Functions NAD+ : Generates 2.5 ATPs in oxidative phosphorylation. NADPH : Used in reductive biosynthesis (e.g., fatty acid synthesis). Provides ADP-ribose for protein modification (ADP-ribosylation). Synthesis and Metabolism 60 mg of tryptophan = 1 mg niacin (requires pyridoxal phosphate-dependent kynureninase). Niacin attaches to ribose phosphate, forming a mononucleotide, then combines with AMP to form dinucleotides. Deficiency Causes Dietary Deficiency: Staple diets like maize (bound niacin) or sorghum (leucine inhibits niacin conversion). Enzyme Defects: Pyridoxal deficiency inhibits kynureninase activity. Drugs: Isoniazid inhibits pyridoxal phosphate formation. Congenital Disorders: Hartnup disease (impaired tryptophan absorption and increased urinary excretion). Carcinoid Syndrome : Excess tryptophan used for serotonin synthesis. Clinical Condition Pellagra : Niacin or tryptophan deficiency causing "rough skin". Symptoms (3 Ds): Dermatitis (Casal’s necklace), Diarrhea (may involve blood/mucus), Dementia (irritability, poor memory, delirium). Sources Richest: Dried yeast, rice polishing, liver, peanuts, cereals, legumes, meat, fish. 50% requirement met by tryptophan conversion. RDA Adults : 20 mg/day ; Additional 5 mg during lactation. Therapeutic Uses Lowers serum cholesterol and Lp(a) by reducing free fatty acid flux from adipose tissue. Toxicity Excess (>50 mg/day): Vasodilation, itching, liver damage. Vitamin A Deficiency (Option A): This typically leads to issues such as night blindness and xerophthalmia (dry eyes), but it does not cause the symptoms shown in the image. Vitamin E Deficiency (Option B): Vitamin E deficiency can result in neurological problems and muscle weakness, but it does not cause the symptoms depicted in the image. Vitamin C Deficiency (Option C): This leads to scurvy, which is characterized by bleeding gums, joint pain, and skin issues, but is not associated with the symptoms shown in the

image.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg.542, 543

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg.480-482

↑ *Back to this question in Part A*

@Neet_pg_bot

Q54: A chronic alcoholic patient presents with agitation, confusion, and gait abnormalities. What vitamin deficiency is most likely responsible for these symptoms? (Q860084)

A. Nicotinamide (Vitamin B3) deficiency

B. Folate (Vitamin B9) deficiency

C. Methylcobalamin (Vitamin B12) deficiency

D. Tyrosinase

Correct Answer: C. Methylcobalamin (Vitamin B12) deficiency

Explanation: Vitamin B12 deficiency can lead to neurological symptoms such as gait abnormalities, confusion, and agitation. It often causes a condition known as Subacute Combined Degeneration (SACD) of the spinal cord. This condition includes: Sensory neuropathy: Tingling and numbness in the extremities. Motor neuropathy: Weakness and gait disturbances. Cranial nerve involvement: Potentially affecting cognitive functions and causing confusion. Features of Chronic Alcoholism Vit. B12 Deficiency (Option C) Chronic alcohol use can lead to functional vitamin B12 deficiency, even if blood tests show normal levels. Alcohol interferes with vitamin B12 absorption and metabolism. Vitamin B12 is essential for DNA synthesis and maintaining healthy nerves and red blood cells. Symptoms of B12 deficiency include fatigue, weakness, numbness, and balance problems. Alcohol can damage the liver, which might cause falsely high B12 levels in blood tests, making diagnosis challenging. Folate Deficiency (Option B) Folate (vitamin B9) deficiency is common in chronic alcoholics, affecting 6-80% of them. Alcohol disrupts folate by impairing its absorption, transport, and liver storage. Poor diet in alcoholics worsens folate deficiency. Folate is crucial for making red blood cells and DNA. Deficiency can cause megaloblastic anaemia and neurological issues like peripheral neuropathy and cognitive problems. Hypoglycemia Chronic alcohol consumption can cause low blood sugar (hypoglycemia), especially if not eating enough. Alcohol produces a high NADH/NAD+ ratio in the cytoplasm, the high NADH level favours the conversion of pyruvate to lactate, leading to lactic acidosis. Deficiency of pyruvate leads to inadequate formation of oxaloacetate which blocks gluconeogenesis This results in low blood sugar levels. Symptoms of alcohol-induced hypoglycemia include confusion, irritability, and, in severe cases, loss of consciousness. Nicotinamide (Vitamin B3) Deficiency (Option A): Deficiency in Vitamin B3 can lead to pellagra, characterized by the "three D's": dermatitis, diarrhea, and dementia. Tyrosinase (Option D): Tyrosinase is an enzyme involved in melanin production, not a vitamin. It is

not related to the symptoms described.

References:

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg. 140

Vitamin B12 Deficiency in alcoholics

Nutritional deficiency in alcoholism

Alcohol and Nutrition: B12 deficiencies

https://www.ncbi.nlm.nih.gov/books/NBK559316/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q55: Which of the following manifestations is associated with Vitamin A deficiency? (Q860087)

A. Vogt's striae

B. Bitot's spots

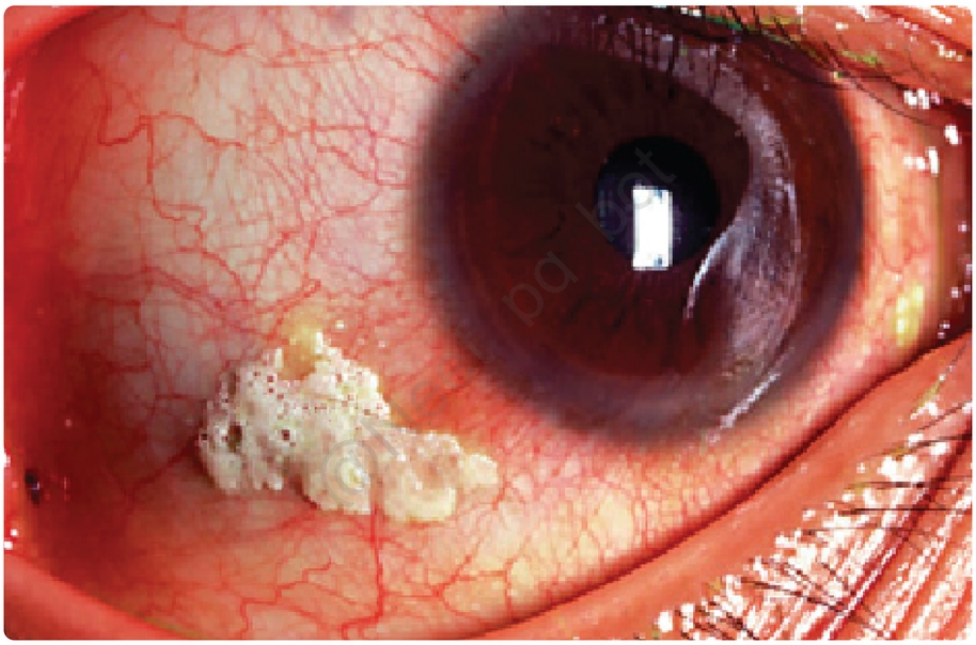
C. Arlt's line

D. Horner's dots

Correct Answer: B. Bitot's spots

Explanation: Bitot's spots are characteristic of Vitamin A deficiency. They appear as triangular, foamy, white or yellowish spots on the conjunctiva, typically found on the lateral side of the eye and are a result of keratinization of the conjunctiva. {{caption_text}} Bitot's spots Causes for Vitamin A Deficiency : Decreased intake. Obstructive jaundice: Defective absorption. Liver cirrhosis: Reduce synthesis of RBP (retinol binding protein). Severe malnutrition: Lack of amino acids for RBP synthesis. Chronic nephrosis: RBP excretion through urine. Vitamin A Deficiency Manifestations : 1) Ocular manifestations : Vitamin A deficiency is the most significant preventable cause of blindness, especially in children below 5 in developing countries. Early Signs: Loss of sensitivity to green light. Difficulty adapting to dim light. Night blindness. Prolonged Deficiency: Xerophthalmia: Dry, thick, keratinized conjunctiva and cornea. If untreated, it leads to corneal ulceration and ultimately results in blindness due to the formation of opaque scar tissue. WHO grading of xerophthalmia: XN Night blindness. X1 X1A: Conjunctival xerosis. X1B: Bitot spots. X2 Corneal xerosis. X3 X3A: Corneal ulceration, less than one-third. X3B: Corneal ulceration, more than one-third. XS Corneal scar. XF Xerophthalmic fundus. Bitot's Spots Keratomalacia: When xerophthalmia persists for a longer time, it progresses to keratomalacia (corneal softening) 2) Immune Function: Deficiency increases susceptibility to infections. Infection reduces retinol-binding protein synthesis, lowering vitamin A levels and impairing immune responses. 3) Skin and Mucous Membrane Lesions: Follicular Hyperkeratosis (Phrynoderma): Causes rough skin due to hyperkeratinization of the follicular

epithelium. Keratinizing Metaplasia: Affects respiratory, gastrointestinal, and genitourinary tracts. Epithelium is atrophied. 4) Other General Manifestation : Growth retardation due to defective synthesis of chondroitin sulfate. Decreased protein synthesis. Vogt's striae (Option A): These are associated with keratoconus, not Vitamin A deficiency. Vogt's striae are streaks in the cornea seen in this condition. Arlt's line (Option C): This is associated with trachoma, an infectious disease caused by Chlamydia trachomatis, not Vitamin A deficiency. Arlt's line is a horizontal line found on the inner surface of the eyelid. Horner's dots (Option D): These are associated with vernal keratoconjunctivitis, not Vitamin A deficiency. Horner's dots are small, discrete, grayish-white spots found on the conjunctiva.



References:

Harper Edition 32, Page no: 538

Lippincott Edition 7, page no:1112,1113

Vasudevan Edition 7, page no: 467,468

↑ *Back to this question in Part A*

@Neet_pg_bot

Q56: A 50-year-old man with a history of chronic alcoholism presents with skin lesions. His diet primarily consists of polished rice. Based on his symptoms and dietary history, what is the most likely diagnosis? (Q860102)

A. Phrynoderma

B. Beriberi

C. Psoriasis

D. Eczema

Correct Answer: B. Beriberi

Explanation: Beriberi is caused by thiamine (vitamin B1) deficiency, which is common in individuals with chronic alcoholism and diets lacking in unpolished grains. The Aleurone layer of cereals (food grains) is a rich source of thiamine. Therefore, whole wheat flour and unpolished hand-pound rice have better nutritive value than completely polished refined foods. When the grains are polished, the aleurone layer is usually removed. Deficiency: Dry beriberi: CNS manifestations are more common Pain, paresthesia and loss of reflexes Peripheral neuritis with sensory disturbances Wet beriberi: Wet beriberi is characterized by cardiovascular symptoms such as breathlessness, palpitations, and peripheral oedema. Distended neck veins observed High output cardiac failure (Option C) Biochemical parameters: Thiamine deficiency affects the enzyme transketolase, which is involved in the pentose phosphate pathway. Measuring transketolase activity in red blood cells is a specific test for thiamine deficiency and can confirm the diagnosis. (Option C) Erythrocyte transketolase activity is reduced; this is the earliest manifestation seen even before clinical disturbances. Recommended Daily Allowance of Thiamine is 1–1.5 mg/day. Phrynoderma (Option A): Phrynoderma, also known as follicular hyperkeratosis, is associated with vitamin A deficiency. While it can present with skin changes, it is not typically linked to chronic alcoholism or a diet primarily consisting of polished rice. Beriberi (Option B): Polished rice lacks thiamine, leading to symptoms such as skin lesions, neuropathy, and cardiac issues in severe cases. Psoriasis (Option C): Psoriasis is a chronic autoimmune skin condition characterized by well-demarcated, erythematous plaques with silvery scales. It is not related to diet or chronic alcoholism. Eczema (Option D): Eczema, or atopic dermatitis, is a hypersensitivity reaction presenting with pruritic, inflamed skin. It is unrelated to nutritional deficiencies or chronic alcoholism.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg.541, 542

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg. 477,478,479

↑ *Back to this question in Part A*

@Neet_pg_bot

Q57: Which of the following zymogens requires gamma-glutamyl carboxylase? (Q855186)

A. Factors II, VIII, IX, X

B. Factors II, VII, IX, and X

C. Factors II, VII, IX, XI

D. Factors II, VIII, X, XI

Correct Answer: B. Factors II, VII, IX, and X

Explanation: Gamma-glutamyl carboxylase is an enzyme located in the rough endoplasmic reticulum that modifies glutamate residues in vitamin K-dependent proteins, converting them to gamma-carboxyglutamate. This modification is essential for their biological activity. Vitamin K acts as a co-substrate along with carbon dioxide and oxygen. Factors II (Prothrombin), VII (Stable factor), IX (Christmas factor), X (Stuart-Prower factor), and Protein C depend on vitamin K for synthesis in the liver. Proteins modified by this enzyme are involved in blood coagulation, inflammation, and vascular health. Factors VIII, XI are not Vit K dependent. (Option A, C, D ruled out)

References:

Robbin's Pathology, 10th edition, pg 122

https://www.ncbi.nlm.nih.gov/gene/2677

↑ *Back to this question in Part A*

@Neet_pg_bot

Q58: A patient presented with diarrhea, dermatitis, and dementia. Which of the following vitamin deficiencies would you suspect in this patient? (Q855190)

A. Vitamin B3 deficiency

B. Vitamin B1 deficiency

C. Vitamin B6 deficiency

D. Vitamin B12 deficiency

Correct Answer: A. Vitamin B3 deficiency

Explanation: The above scenario is a case of Pellagra due to Vitamin B3 deficiency. Pellagra is characterized by the classic triad of symptoms: Dermatitis Bright red erythema- in sun-exposed areas (face, ankles, feet) Casal's necklace - ↑pigmentation around the neck Diarrhoea, and Dementia. {{caption_text}} Causes of pellagra : Dietary Deficiency- Maize and Sorghum based diet Impaired Tryptophan Metabolism Factors Affecting Tryptophan to Niacin Conversion: Vitamin B6 Deficiency Medications: Isoniazid (INH), OCPs Hormonal Influences: Estrogen metabolites hinder tryptophan metabolism. Genetic Disorders: Inherited conditions like Hartnup disease can impede tryptophan absorption in the intestines and reabsorption in the kidneys Carcinoid Syndrome Other Contributing Factors: Alcoholism: Interferes with nutrient absorption, including niacin and tryptophan. Malabsorption Syndromes Vitamin B1 deficiency (Option B): Thiamine (vitamin B1) deficiency causes Wet beriberi- pedal edema and cardiomegaly; Dry Beriberi- CNS manifestations such as peripheral neuritis, sensory disturbances, difficulty walking, and paralysis due to CNS manifestations; Lactic acidosis Maple Syrup urine

disease - Accumulation of branched-chain keto acids l eads to convulsions, severe mental retardation, vomiting, acidosis, coma, and death. Vitamin B6 deficiency (Option C): Pyridoxine (vitamin B6) deficiency can cause cheilosis and glossitis but is not associated with specific combination of dermatitis, diarrhoea, and cognitive disturbances seen in this case. Vitamin B12 deficiency (Option D) can lead to symptoms such as fatigue, neurological issues, and anemia. However, it is not typically associated with the specific combination of dermatitis, diarrhoea, and cognitive disturbances seen in this case.



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg.542,543

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg. 481,482

↑ *Back to this question in Part A*

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Q59: A family consumes only polished rice. Which of the following combinations of vitamin deficiency and enzymatic defect will be present in this family? (Q855197)

A. Riboflavin-glutathione reductase

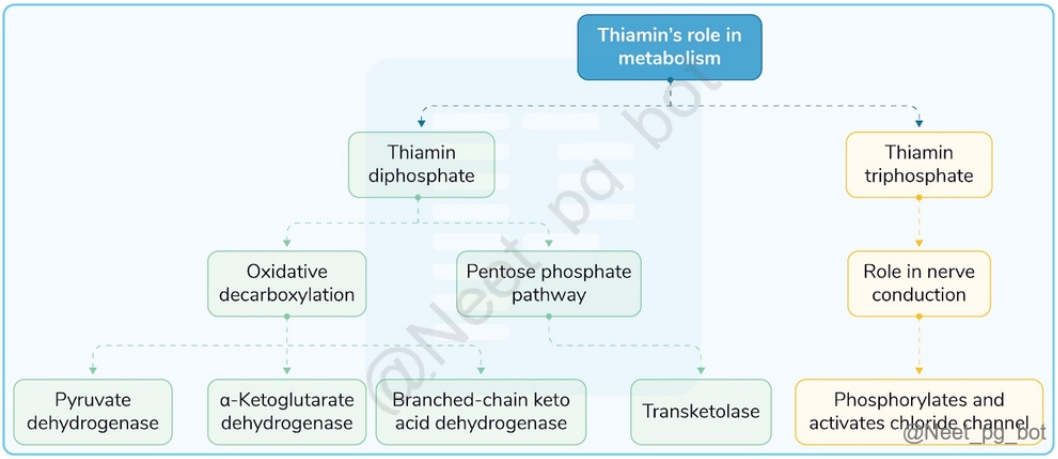
B. Thiamine-transketolase

C. Thiamine-transaminase

D. Riboflavin-transketolase

Correct Answer: B. Thiamine-transketolase

Explanation: The Aleurone layer of cereals (food grains) is a rich source of thiamine . Therefore, whole wheat flour and unpolished hand-pound rice have better nutritive value than completely polished refined foods. When the grains are polished, the aleurone layer is usually removed. Biochemical parameters: Thiamine deficiency affects the enzyme transketolase , which is involved in the pentose phosphate pathway. Measuring transketolase activity in red blood cells is a specific test for thiamine deficiency and can confirm the diagnosis. Erythrocyte transketolase activity is reduced; this is the earliest manifestation seen even before clinical disturbances. Recommended Daily Allowance of Thiamine is 1–1.5 mg/day. {{caption_text}} Riboflavin-glutathione reductase (Option A) Riboflavin (vitamin B2) deficiency affects glutathione reductase, an enzyme involved in maintaining cellular redox balance by regenerating reduced glutathione. Polished rice is not specifically deficient in riboflavin, so this combination is unlikely. Thiamine-transaminase (Option C) Transaminases (e.g., ALT, AST) are not thiamine-dependent enzymes; they require pyridoxal phosphate (vitamin B6) as a cofactor. Riboflavin-transketolase (Option D) Transketolase is dependent on thiamine, not riboflavin. Riboflavin deficiency primarily affects flavoprotein enzymes such as glutathione reductase or succinate dehydrogenase.



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg.541, 542, 543

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg. 477, 478, 479, 482, 483

↑ *Back to this question in Part A*

@Neet_pg_bot

Q60: A child was brought with pedal edema and cheilosis.Cardiomegaly was present. What is the vitamin deficiency associated with this clinical presentation? (Q855227)

A. Riboflavin

B. Thiamine

C. Pyridoxine

D. Niacin

Correct Answer: B. Thiamine

Explanation: Pedal edema and cardiomegaly : Indicate heart failure, commonly associated with wet beriberi , caused by thiamine (vitamin B1) deficiency . Cheilosis (cracking at the corners of the mouth) : Can occur in various nutritional deficiencies, but in this case, the systemic symptoms point toward thiamine deficiency. Conditions Biochemical Role of Thiamine Deficiency Manifestations Dry Beriberi Essential for carbohydrate metabolism and nerve function CNS manifestations such as peripheral neuritis, sensory disturbances, difficulty walking, and paralysis due to CNS manifestations. Wet Beriberi Thiamine is necessary for the PDH complex and low activity causes Lactic acidosis. Lactic acidosis causes vasodilatation and hence cardiovascular manifestations. Cardiovascular manifestations such as oedema, palpitations, breathlessness, and heart failure. Maple Syrup Urine Disease Cofactor for decarboxylation of branched-chain keto acids Accumulation of branched-chain keto acids leads to convulsions, severe mental retardation, vomiting, acidosis, coma, and death. Lactic Acidosis Cofactor for pyruvate dehydrogenase, converting pyruvate to acetyl-CoA Impaired conversion leads to increased lactate and pyruvate levels, causing lactic acidosis. Riboflavin (Option A): Riboflavin (vitamin B2) deficiency causes cheilosis, glossitis, and angular stomatitis but does not lead to cardiomegaly or edema. Pyridoxine (Option C): Pyridoxine (vitamin B6) deficiency can cause cheilosis and glossitis but is not associated with cardiac manifestations like cardiomegaly or edema. Niacin (Option D): Niacin (vitamin B3) deficiency causes pellagra, characterized by the "3 Ds"—dermatitis, diarrhea, and dementia. Cardiomegaly and pedal edema are not features.

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg.541, 542

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg. 477,478,479

↑ *Back to this question in Part A*

@Neet_pg_bot

Q61: A patient presenting with bleeding gums and easy bruisability was diagnosed with scurvy. This is due to (Q855238)

A. Low calcium

B. Defective collagen formation

C. Inhibition of clotting factors

D. Increased keratinization of epithelium

Correct Answer: B. Defective collagen formation

Explanation: Scurvy is caused by a deficiency of vitamin C, which is essential for the synthesis of collagen. Collagen is crucial for maintaining the structural integrity of blood vessels, skin, and connective tissues. Without adequate collagen, patients develop symptoms like bleeding gums, easy bruising, and poor wound healing. Low calcium (Option A): Calcium is important for bone health and muscle function, but it does not directly cause the bleeding and bruising seen in scurvy. Inhibition of clotting factors (Option C): While vitamin K deficiency can lead to clotting issues, scurvy is not related to clotting factor inhibition but rather to impaired collagen synthesis, affecting vascular integrity. Increased keratinization of epithelium (Option D): Keratinization of epithelium is not a feature of scurvy.

References:

Vasudevan Textbook of Biochemistry (7th Edition), Page 497

https://pubmed.ncbi.nlm.nih.gov/1720597/

↑ *Back to this question in Part A*

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Q62: A child presents with thinning of hair and scaling of the scalp. On taking further history, it is found that she consumes raw eggs in her diet regularly. The given manifestations are due to the deficiency of which of the following vitamins? (Q855283)

A. Niacin

B. Pyridoxine

C. Biotin

D. Thiamine

Correct Answer: C. Biotin

Explanation: Biotin (vitamin B7) is a water-soluble vitamin essential for carboxylation reactions in metabolism, including fatty acid synthesis and gluconeogenesis. It is crucial for maintaining healthy hair, skin, and nails. Effect of Raw Egg Consumption: Raw egg whites contain avidin, a protein that binds to biotin with high affinity, preventing its absorption in the gut. Chronic consumption of raw eggs can lead to biotin deficiency, causing: Thinning of hair (alopecia). Scaling of the scalp and other skin lesions. Neurological symptoms like depression, lethargy, and paresthesia (in severe cases). Niacin (Option A): Niacin (vitamin B3) deficiency causes pellagra , characterized by the "3 Ds": dermatitis, diarrhea, and dementia . Hair thinning and scalp scaling are not typical features. Pyridoxine (Option B): Pyridoxine (vitamin B6) deficiency can cause seborrheic dermatitis, irritability, and peripheral neuropathy, but it is not specifically linked to raw egg consumption. Thiamine (Option D): Thiamine (vitamin B1) deficiency leads to beriberi or Wernicke-Korsakoff syndrome , which primarily affects the cardiovascular and nervous systems, not hair or scalp health.

References:

Vasudevan Textbook of Biochemistry (7th Edition), Page 454, 486

↑ *Back to this question in Part A*

@Neet_pg_bot

Q63: What are the consequences of a deficiency in Vitamin A? (Q857843)

A. Beriberi

B. Pellagra

C. Xerophthalmia

D. Neuropathy

Correct Answer: C. Xerophthalmia

Explanation: Correct Option: C) Xerophthalmia Xerophthalmia is a condition associated with severe vitamin A deficiency . Vitamin A is crucial for maintaining healthy eyes and vision. Vitamin A Deficiency Manifestations : 1. Ocular manifestations : Vitamin A deficiency is the most significant preventable cause of blindness, especially in children below 5 in developing countries. Early Signs: Loss of sensitivity to green light. Difficulty adapting to dim light. Night blindness. Prolonged Deficiency: Xerophthalmia: Dry, thick, keratinized conjunctiva and cornea. If untreated, it leads to corneal ulceration and ultimately results in blindness due to the formation of opaque scar tissue. WHO grading of xerophthalmia: XN Night blindness. X1 X1A: Conjunctival xerosis. X1B: Bitot spots. X2 Corneal xerosis. X3 X3A: Corneal ulceration, less than one-third. X3B: Corneal ulceration, more than one-third. XS Corneal scar. XF Xerophthalmic fundus. Bitot's Spots: Grayish-white triangular plaques firmly adherent to the conjunctiva. Keratomalacia: When xerophthalmia persists for a longer time, it progresses to keratomalacia (corneal softening) 2) Immune Function: Deficiency increases susceptibility to infections. Infection reduces retinol-binding protein synthesis, lowering vitamin A levels and impairing immune responses. 3) Skin and Mucous Membrane Lesions: Follicular Hyperkeratosis (Phrynoderma): Causes rough skin due to hyperkeratinization of the follicular epithelium. Keratinizing Metaplasia: Affects respiratory, gastrointestinal, and genitourinary tracts. Epithelium is atrophied. 4) Other General Manifestation : Growth retardation due to defective synthesis of chondroitin sulfate. Decreased protein synthesis. Beri-beri (Option A) is a condition caused by thiamine (vitamin B1) deficiency lead to symptoms such as muscle weakness, peripheral neuropathy, cardiovascular problems, and impaired mental function. Pellagra (Option B) is a condition caused by niacin (vitamin B3) deficiency causes Pellagra is characterized by the "4 Ds" - dermatitis (skin inflammation), diarrhea, dementia (mental confusion), and death if left untreated. Neuropathy (Option D) refers to nerve damage, which can have various causes, such as diabetes, alcohol abuse, certain medications, and vitamin deficiencies. Vitamin A deficiency is not typically associated with neuropathy.

References:

Harper Edition 32, Page no: 538

↑ *Back to this question in Part A*

@Neet_pg_bot

Q64: A 42-year-old female came to the emergency department with a bleeding condition and dental caries. Which vitamin supplementation from the list below would be best for treating this condition? (Q857896)

A. Vitamin B1

B. Vitamin B9

C. Vitamin D

D. Vitamin K

Correct Answer: D. Vitamin K

Explanation: Vitamin K is essential for blood clotting and proper wound healing. Vitamin K: Fat-soluble vitamin that acts as a coenzyme or prosthetic group for gamma-carboxylase. Calcium-dependent proteins that rely on Vitamin K: Clotting factors II, VII, IX, and X. Anticoagulant factors like protein C and S. Osteocalcin (promotes bone mineralization by depositing calcium). Forms of Vitamin K: Phylloquinone (K1): Found in green vegetables, this form has an isoprenoid side chain with 20 carbons. Menaquinones (K2): Synthesized by intestinal bacteria, these have varying side chain lengths and an isoprenoid side chain containing 30 carbons, which is lipid soluble. (Option B) Menadione (K3): A synthetic form which is water-soluble. Menadione and menadione diacetate can be metabolized into phylloquinone. Vitamin K exists in two forms: Epoxide and Hydroquinone form. Functions : Blood Clotting. Bone Health - Carboxylating glutamate to glutamic acid in proteins like osteocalcin is involved in bone mineralization. Vitamin B1 (Thiamine) (Option A) is important for converting food into energy and maintaining proper nerve function.. Vitamin B9 (Folate (Option B) is crucial for cell division and DNA synthesis. It is particularly important during pregnancy and for overall cell growth. Deficiency in folate can lead to anemia Vitamin D (Option C) is important for maintaining bone health, immune function, and overall well-being. It helps the body absorb calcium and phosphorus from the intestines. Vitamin D deficiency can lead to weakened bones and rickets.

References:

Harper’s Illustrated Biochemistry, 32nd edition, Page 540

↑ *Back to this question in Part A*

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Q65: Due to a prolonged period of consuming maize as a main food source, a 34-year-old man developed diarrhoea and exhibited lesions on his neck, as depicted in

the accompanying image. This condition is caused by a deficiency in: (Q793361)

A. Niacin

B. Zinc

C. Thiamine

D. Iron



Correct Answer: A. Niacin

Explanation: Correct Option A - Niacin The above picture presented above suggests Vitamin B3/niacin deficiency according to clinical features. Pellagra disease is caused by the excessive consumption of a diet made up of maize. Maize/corn has low levels of tryptophan and niacin. Niacin deficiency causes dermatitis, diarrhoea, and dementia. The neck lesion shows characteristic Casal's necklace pattern, as seen in Pellagra. The Casal necklace or Casal collar is a clinical sign of pellagra that shows erythematous pigmented rash of skin near the collarbone region (dermatomes C3 and C4). Incorrect Options: Option B - Zinc deficiency is found in people with severe diarrhea, liver cirrhosis, alcoholism causes Option C - Prolonged deficiency of Thiamine or Vitamin B1 in patients may support sensation loss at extreme level, heart failure symptoms along with hands or feet swelling, demand ischemia related chest pain, double vision, memory loss, Option D - Iron deficiency in the body causes anemia. Hence, red blood cells are unable to produce enough hemoglobin in the body that allows them to carry oxygen. Shortness of breath and tiredness are caused as a result of iron deficiency anemia.

References:

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↑ *Back to this question in Part A*

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Q66: A 42-year-old female came to the emergency department with a bleeding condition and dental caries. Which vitamin supplementation from the list below would be best for treating this condition? (Q820421)

A. Vitamin B1

B. Vitamin B9

C. Vitamin D

D. Vitamin K

Correct Answer: D. Vitamin K

Explanation: Correct Option D - Vitamin K : Vitamin K is essential for blood clotting and proper wound healing. It plays a critical role in the production of clotting factors that prevent excessive bleeding. A deficiency in vitamin K can lead to bleeding disorders, where even minor injuries can result in prolonged bleeding. Additionally, vitamin K is involved in maintaining dental health by supporting the proper mineralization of teeth. Therefore, in this case, the best choice for treating the bleeding condition and dental caries would be vitamin K supplementation. Incorrect Options: Option A - Vitamin B1 (Thiamine) : Vitamin B1 is important for converting food into energy and maintaining proper nerve function. It is not directly associated with bleeding conditions or dental caries. Option B - Vitamin B9 (Folate) : Vitamin B9, also known as folate or folic acid, is crucial for cell division and DNA synthesis. It is particularly important during pregnancy and for overall cell growth. While a deficiency in folate can lead to anemia and other health issues, it is not directly related to bleeding conditions or dental caries. Option C - Vitamin D : Vitamin D is important for maintaining bone health, immune function, and overall well-being. It helps the body absorb calcium and phosphorus from the intestines. While vitamin D deficiency can lead to weakened bones, it is not directly linked to bleeding conditions or dental caries.

↑ *Back to this question in Part A*

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• [12_Heme Synthesis] 032 Heme Synthesis And Porphyrias

« Back to Folder Directory

[12_Heme Synthesis] 032 Heme Synthesis And Porphyrias — Quick Jump

Q1 (p. 1323)	*Q2 (p. 1324)*	*Q3 (p. 1327)*	*Q4 (p. 1329)*	*Q5 (p. 1330)*	*Q6 (p. 1330)*
Q7 (p. 1332)	*Q8 (p. 1333)*	*Q9 (p. 1334)*	*Q10 (p. 1335)*	*Q11 (p. 1336)*	*Q12 (p. 1337)*
Q13 (p. 1338)	*Q14 (p. 1339)*	*Q15 (p. 1340)*	*Q16 (p. 1341)*	*Q17 (p. 1342)*	*Q18 (p. 1343)*
Q19 (p. 1343)	*Q20 (p. 1344)*	*Q21 (p. 1345)*			

Q1: A 7-year-old boy presents with photosensitivity, blistering skin lesions on sun exposure, and dark red urine. The parents are paint factory workers living in an industrial complex. Labs show elevated porphyrins. Which of the following statements about the diagnosis is incorrect? (Q844899)

A. The condition can occur due to exposure to lead, leading to decreased activity of ALA synthase.

B. The red fluorescence of the teeth under UV light is referred to as erythrodontia, due to emission of 600 nm wavelength by porphyrin.

C. Porphyrin has a specific absorption band called Soret band corresponding to 400 nm.

D. Photosensitivity due to the accumulation of porphyrins in the skin which release superoxide radicals causing photooxidative damage.

Correct Answer: A. The condition can occur due to exposure to lead, leading to decreased activity of ALA synthase.

■ *Listen to Audio 1*

Explanation: The case describes porphyria, characterized by photosensitivity, dark red urine, and elevated porphyrins. Lead poisoning can cause similar findings but inhibits ALA dehydratase , ferrochelatase , and coproporphyrinogen oxidase , not ALA synthase . The 4 pyrrole rings in Porphyrins are connected by methenyl bridges (double bonds) capable of absorbing light at 400 nm and emitting light at 600 nm(Option B). This 600 nm wavelength appears as the red fluorescence of the teeth under UV light, which is referred to as Erythrodontia . This property of porphyrins is being used in cancer phototherapy The light absorbed by porphyrins has a specific absorption band called the Soret band (Option C) , which corresponds to around 400 nm as seen on spectrometry. Photosensitivity in porphyria is due to the accumulation of porphyrins in the skin (Option D). Porphyrins absorb light of shorter wavelengths (higher energy) and emit longer wavelengths (lower energy), which in turn releases superoxide radicals causing photooxidative damage.



References:

Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Pages 275–276

↑ *Back to this question in Part A*

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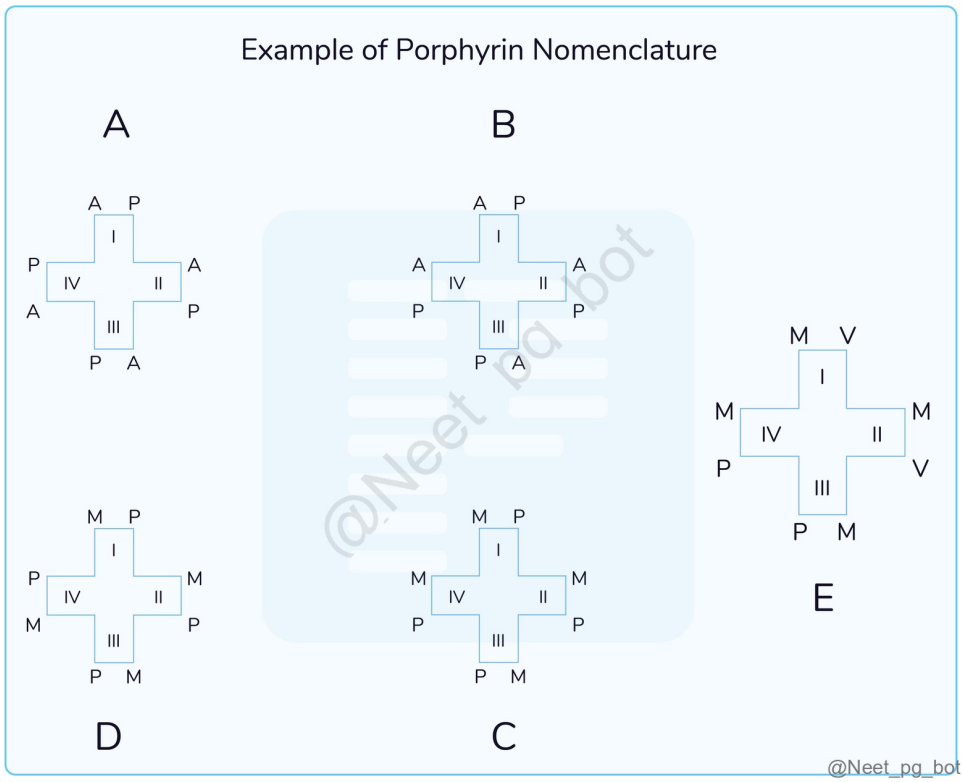
Q2: Which of the following structures correctly represents the type of porphyrin? A= acetyl, P=propionyl, M=methyl, V=vinyl (Q844901)

A. A- Uroporphyrinogen I, B- Uroporphyrinogen III, E- Protoporphyrinogen I

B. D- Coproporphyrinogen III, C- Coproporphyrinogen I, E- Protoporphyrinogen I

C. A- Uroporphyrinogen III, B- Uroporphyrinogen I, E- Protoporphyrinogen III

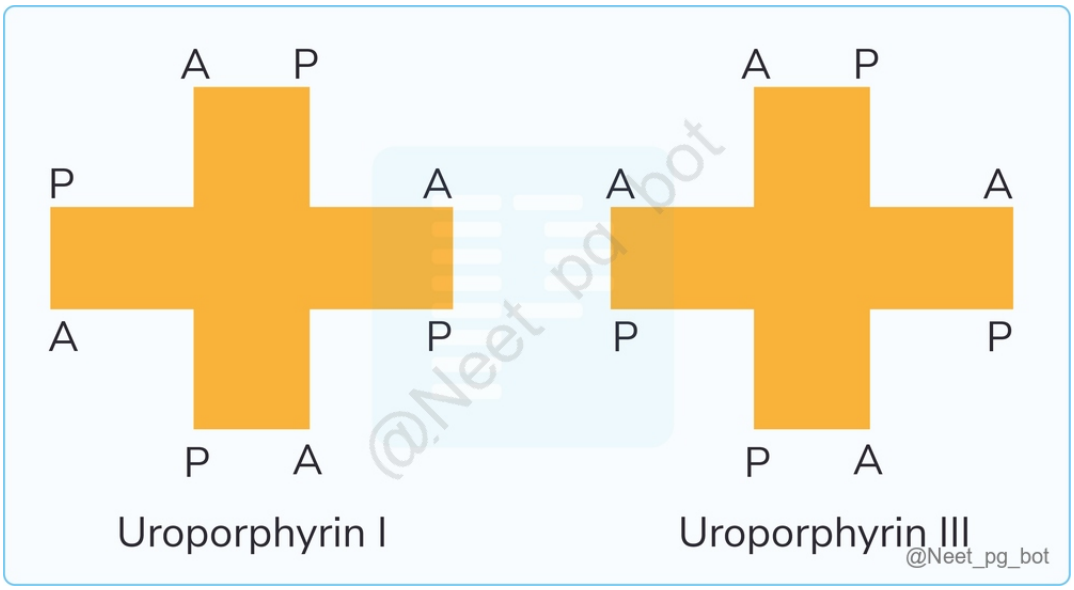
D. D- Coproporphyrinogen I, C- Coproporphyrinogen III, E- Protoporphyrinogen III

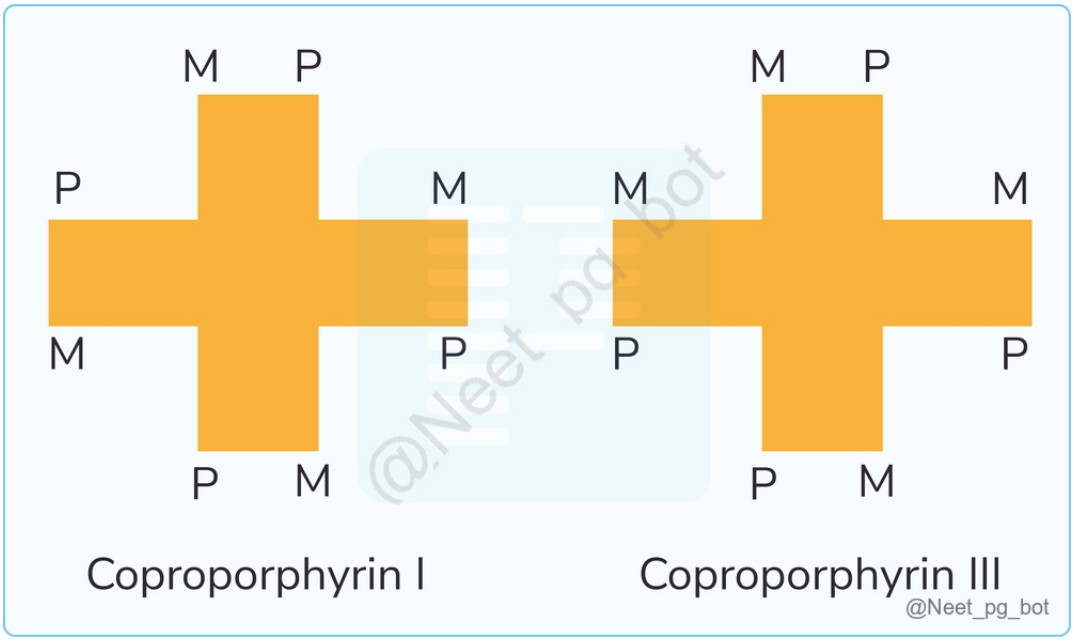


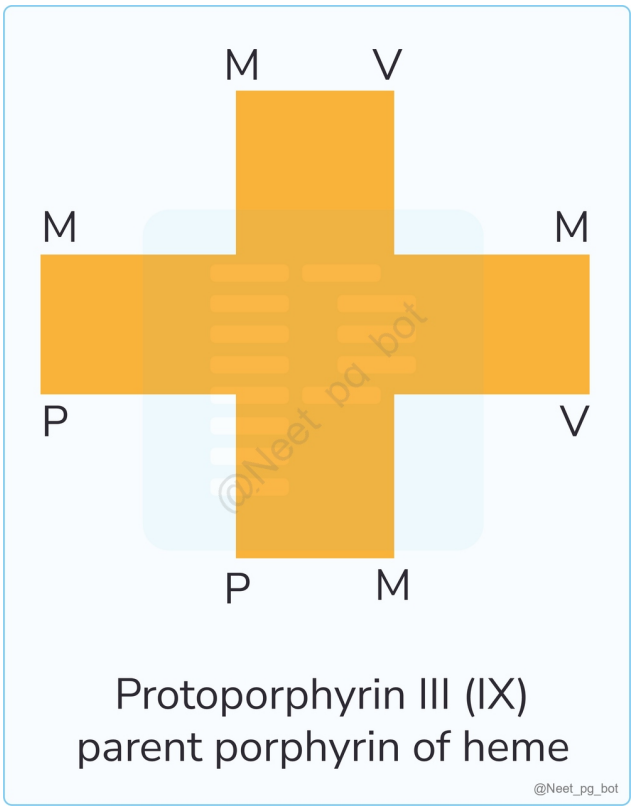
Correct Answer: D. D- Coproporphyrinogen I, C- Coproporphyrinogen III, E- Protoporphyrinogen III

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Explanation: Porphyrins can exist in multiple forms depending on the group in the possible 8 positions of 4 pyrrole rings. Uroporphyrinogen Alternating acetyl (A) and propionyl (P) groups at all 8 positions. Uroporphyrinogen I is when there is symmetry in all the rings. Uroporphyrinogen III is when there is an asymmetry in the last ring. Coproporphyrinogen Instead of acetyl, methyl (M) groups are present. The conversion from the acetyl to the methyl group is done by decarboxylation. Uroporphyrinogen decarboxylase converts uroporphyrinogen to coproporphyrinogen. Protoporphyrinogen In this case, a methyl (M)-vinyl (V) group is present only in rings 1 and 2. Coproporphyrinogen oxidase converts coproporphyrinogen to protoporphyrinogen. Protoporphyrinogen can be converted into protoporphyrin by oxidation (removal of hydrogen) by protoporphyrinogen oxidase.


A P
P A
A P
P A
Uroporphyrin I
A P
A A
P P
P A
Uroporphyrin III
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M P
P M
M P
P M
Coproporphyrin I
M P
M M
P P
P M
Coproporphyrin III
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References:

Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Page 272

↑ *Back to this question in Part A*

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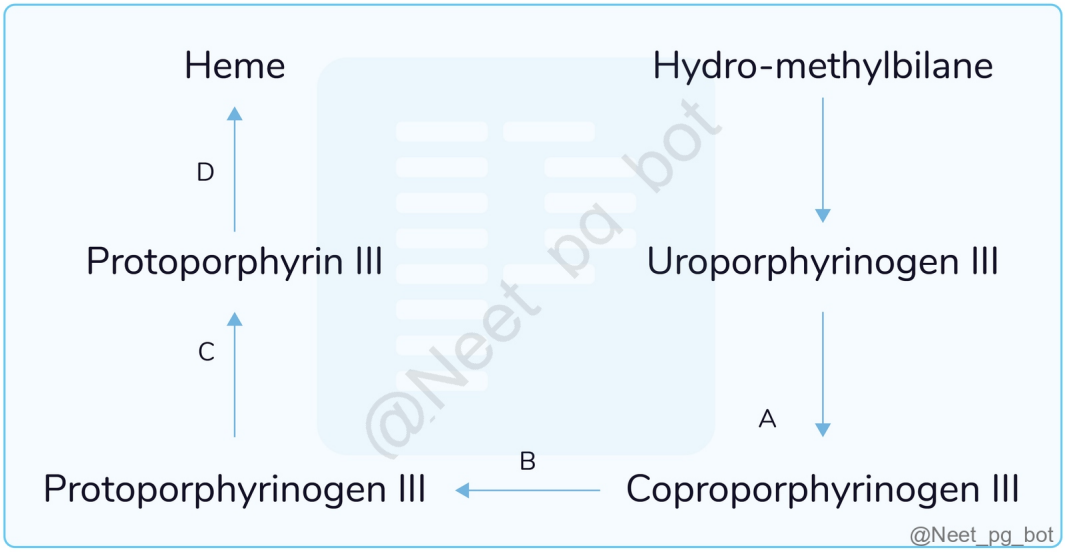
Q3: According to the given image of heme synthesis, which of the following statements is incorrect? (Q844903)

A. Steps C and D occur in Mitochondria, while steps A and B occur in the cytosol.

B. The enzyme involved in step A is Uroporphyrinogen decarboxylase

C. 4 molecules of CO_2 are liberated at step A

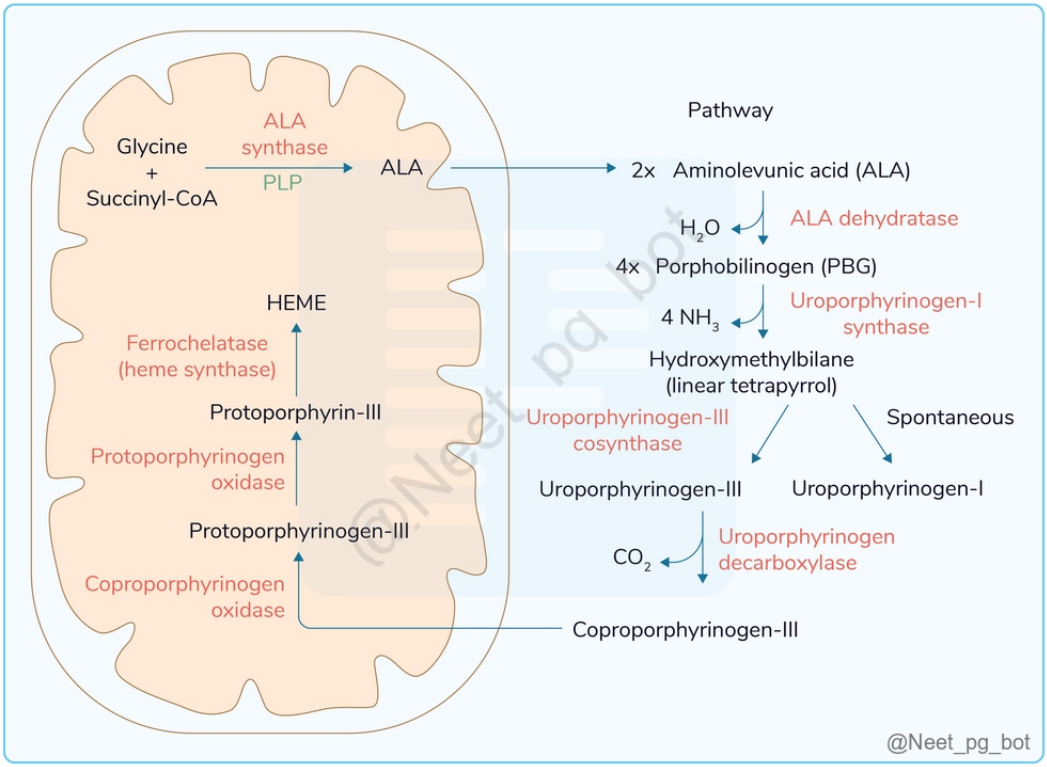
D. Ferrochelatase is the enzyme involved in step D



Correct Answer: A. Steps C and D occur in Mitochondria, while steps A and B occur in the cytosol.

■ *Listen to Audio 1*

Explanation: Step B represents the synthesis of Protoporphyrinogen III from Coproporphyrinogen III, which occurs in mitochondria . Heme Synthesis: Cytosolic steps: 2 – 5 Mitochondrial steps: 1, 6 – 8 Step Reaction Enzyme Key Points 1 Succinyl CoA + Glycine → Delta-aminolevulinic acid (ALA) ALA Synthase Rate-limiting step ; requires pyridoxal phosphate as a cofactor 2 2 ALA → Porphobilinogen (PBG) ALA Dehydratase It involves removal of 2 molecules of water; the enzyme contains zinc and is inhibited by lead 3 4 PBG → Hydroxymethylbilane (HMB) Uroporphyrinogen I Synthase - 4a HMB → Uroporphyrinogen I Spontaneous cyclization - 4b HMB → Uroporphyrinogen III Uroporphyrinogen III Synthase - 5 Uroporphyrinogen III → Coproporphyrinogen III Uroporphyrinogen Decarboxylase (Option B) Decarboxylation, removal of 4 molecules of CO2 (Option C) 6 Coproporphyrinogen III → Protoporphyrinogen III Coproporphyrinogen Oxidase 7 Protoporphyrinogen III → Protoporphyrin III Protoporphyrinogen Oxidase 8 Protoporphyrin III → Heme Ferrochelatase (Option D) Insertion of Fe2+



References:

Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Pages 271–273

↑ *Back to this question in Part A*

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Q4: Which of the following statements about heme synthesis is incorrect? (Q844904)

A. ALA synthase requires pyridoxal phosphate as a cofactor.

B. ALA dehydratase contains zinc as a cofactor and is inhibited by lead.

C. Pyridoxal phosphate deficiency can lead to anemia due to impaired ALA synthase activity.

D. ALA dehydratase is the rate-limiting step of the heme synthesis pathway.

Correct Answer: D. ALA dehydratase is the rate-limiting step of the heme synthesis pathway.

■ *Listen to Audio 1*

Explanation: This statement is incorrect because the rate-limiting enzyme in heme synthesis is ALA synthase , not ALA dehydratase . Statement Key Facts ALA dehydratase is the rate-limiting step of the heme synthesis pathway (Option D): The rate-limiting enzyme is ALA synthase , not ALA dehydratase Converts ALA → Porphobilinogen

(PBG) ALA synthase requires pyridoxal phosphate as a cofactor (Option A) First step of heme synthesis: Succinyl-CoA + Glycine → ALA Requires pyridoxal phosphate (Vitamin B■) as cofactor ALA dehydratase contains zinc and is inhibited by lead (Option B) Catalyzes ALA → Porphobilinogen Lead replaces zinc , inhibiting enzyme activity and causing ALA accumulation Pyridoxal phosphate deficiency can lead to anemia due to impaired ALA synthase activity (Option C) Reduced activity of ALA synthase in Vitamin B■ deficiency Leads to impaired heme synthesis → sideroblastic anemia

References:

Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Pages 271

↑ *Back to this question in Part A*

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Q5: A 35-year-old male presents with microcytic anemia and elevated delta-aminolevulinic acid (ALA). Which statement correctly describes the regulation of ALA synthase I (ALAS I) and ALA synthase II (ALAS II)? (Q844905)

A. ALAS I is primarily expressed in erythroid tissues and regulated by iron.

B. ALAS II is ubiquitously expressed and inhibited by heme.

C. ALAS I is inhibited by heme and primarily expressed in the liver.

D. ALAS II is regulated by heme and is key in hepatic heme synthesis.

Correct Answer: C. ALAS I is inhibited by heme and primarily expressed in the liver.

■ *Listen to Audio 1*

Explanation: Regulation of ALA Synthase I and II: Feature ALAS I (Non-Erythroid) ALAS II (Erythroid) Expression Location Primarily in the liver Erythroid tissues Regulation by Heme Inhibited by heme Not regulated by heme Regulation by Iron Not regulated by iron Regulated by iron availability Role in Heme Synthesis The rate-limiting enzyme in hepatic heme synthesis Key enzyme in erythroid heme synthesis

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pages 317–318 Textbook of Biochemistry by D.M. Vasudevan, 7th Edition, Page 272

↑ *Back to this question in Part A*

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Q6: A 25-year-old man with microcytic anemia, high iron and ferritin, and ringed sideroblasts has an ALAS2 gene mutation. What is the most likely diagnosis? (Q844906)

A. Iron deficiency anemia

B. Thalassemia

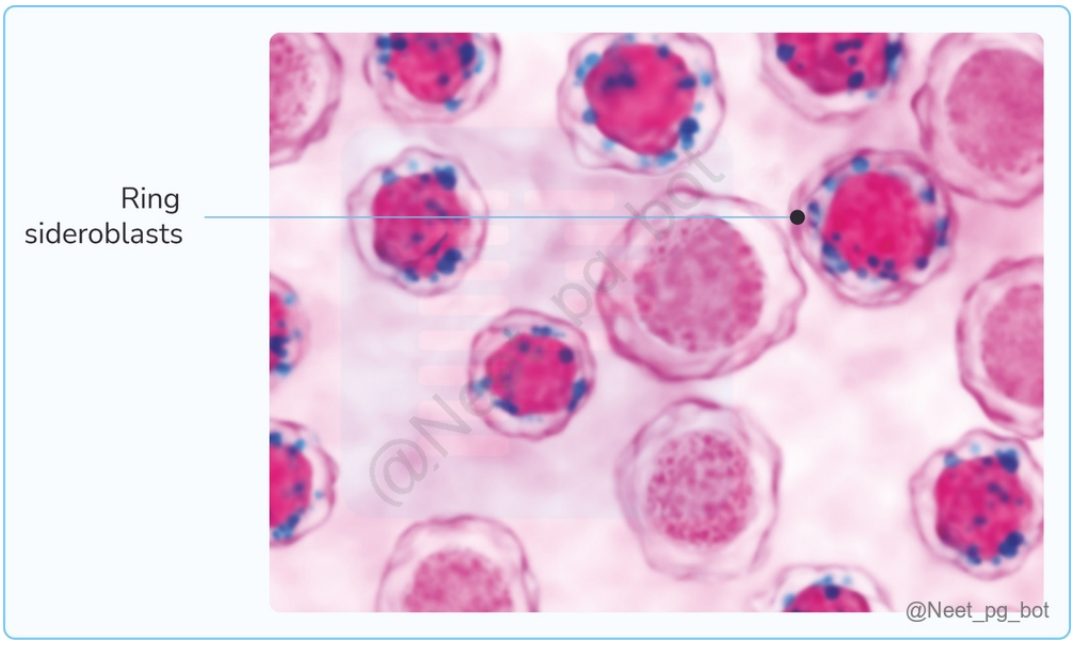
C. X-linked sideroblastic anemia

D. Autosomal recessive sideroblastic anemia

Correct Answer: C. X-linked sideroblastic anemia

■ *Listen to Audio 1*

Explanation: X-linked sideroblastic anemia (XLSA) is caused by mutations in the ALAS2 gene, which is responsible for the enzyme 5-aminolevulinate synthase 2, essential for the first step in heme synthesis. This condition typically presents with microcytic, hypochromic anemia, elevated serum iron and ferritin levels, and ringed sideroblasts in the bone marrow. The confirmed mutation in the ALAS2 gene in the patient points to XLSA, distinguishing it from other forms of anemia. Overactivation of ALAS2 gene causes x linked protoporphyria Lab Finding Type of anemia Usually microcytic hypochromic Serum iron Increased Ferritin Increased Transferrin saturation Increased Bone marrow Ring sideroblasts Peripheral smear Dimorphic RBC population, hypochromia, anisopoikilocytosis Prussian blue stain Iron-loaded mitochondria around the nucleus X-linked sideroblastic anemia (XLSA) is caused by mutations in the ALAS2 gene, affecting heme synthesis, while autosomal recessive sideroblastic anemia (ARSA) results from mutations in genes like SLC25A38 or GLRX5, which impair mitochondrial iron metabolism (Option D) XLSA primarily affects males, whereas ARSA affects both sexes. Both present with microcytic anemia, ringed sideroblasts, and high serum iron, but XLSA typically responds to pyridoxine (vitamin B■), while ARSA does not and is managed with iron chelation or transfusions. Iron Deficiency Anemia (Option A) is characterized by low serum iron and ferritin levels, which do not match the elevated levels seen in the case. Thalassemia (Option B) involves defective globin chain production and normal iron levels, unlike the elevated iron levels and ringed sideroblasts present here.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 320

↑ *Back to this question in Part A*

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Q7: Match the following disorders with the enzyme defects in context to relation synthesis. Enzyme defect Disorder A. Uroporphyrinogen III synthase i) Porphyria cutanea tarda B. Uroporphyrinogen decarboxylase defect ii) Congenital erythropoietic porphyria C. ALA synthase II iii) Acute intermittent porphyria D. Porphobilinogen deaminase iv) X-linked sideroblastic anemia (Q844907)

A. A-ii, B-i, C-iv, D-iii

B. A-iii, B-iv, C-ii, D-i

C. A-iv, B-iii, C-i, D-ii

D. A-i, B-ii, C-iv, D-iii

Correct Answer: A. A-ii, B-i, C-iv, D-iii

■ *Listen to Audio 1*

Explanation: Every defect in the enzyme involved in heme synthesis is associated with a disorder, as shown below: Enzyme defect Disorder ALA synthase II X-linked sideroblastic anemia ALA dehydratase ALA dehydratase

deficienct porphyria Porphobilinogen deaminase (Hydroxymethylbilane synthase) Acute intermittent porphyria Uroporphyrinogen III synthase Congenital erythropoietic porphyria Uroporphyrinogen decarboxylase Porphyria cutanea tarda Coproporphyrinogen oxidase Hereditary coproporphyria Protoporphyrinogen oxidase Porphyria variegata Ferro chelatase Erythropoietic protoporphyria

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 320

↑ *Back to this question in Part A*

@Neet_pg_bot

Q8: Match the following disorder with its clinical classification: Disorder Clinical Manifestations A.Acute intermittent porphyria i) Neuropsychiatric manifestations B.Porphyria Cutanea Tarda ii) Photosensitivity C.Hereditary coproporphyria iii) Both Neuropsychiatric manifestation and photosensitivity D.Congenital erythropoietic porphyria (Q844908)

A. A-i, B-ii, C-iii, D-ii

B. A-i, B-ii, C-ii, D-iii

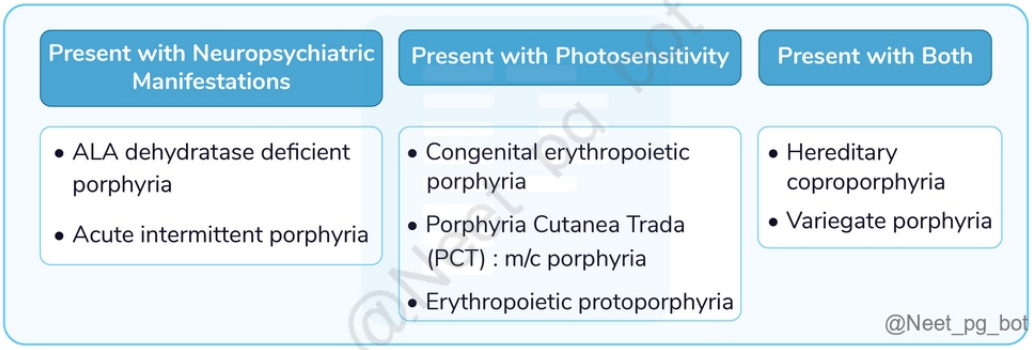
C. A-ii, B-i, C-iii, D-iii

D. A-ii, B-i, C-ii, D-iii

Correct Answer: A. A-i, B-ii, C-iii, D-ii

■ *Listen to Audio 1*

Explanation: Porphyria can be classified into 3 categories based on its important clinical manifestations: Cutaneous photosensitivity is due to the accumulation of porphyrins in the skin, which enhances the production of free radicals upon exposure to light. Neurological complications often arise from severe episodes of acute attacks. These attacks can also trigger neuropsychiatric symptoms like confusion, hallucinations, anxiety, and psychosis . However, these symptoms are typically self-limiting. CEP and PCT present with blistering photosensitivity whereas EP and X linked protoprphyria present with non blistering protoporphyria. Werewolf appearance of blistering cutaneous manifestations





References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 320

https://www.jneurosci.org/content/36/19/5264#

↑ *Back to this question in Part A*

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Q9: Lead poisoning can cause acquired porphyria by inhibiting several enzymes in the heme synthesis pathway. Which of the following enzymes is not affected by lead poisoning? (Q844909)

A. ALA dehydratase

B. Coproporphyrinogen oxidase

C. Ferrochelatase

D. ALA synthase

Correct Answer: D. ALA synthase

■ *Listen to Audio 1*

Explanation: Lead poisoning can lead to acquired porphyria by inhibiting 3 enzymes of the heme synthesis pathway: Enzyme inhibited Features ALA dehydratase (Option A) Mnemonic: LeAD ALA elevation Coproporphyrinogen oxidase (Option B) Coproporphyrin elevation Ferrochelatase (Option C) Elevated Zinc protoporphyrin levels (Most sensitive marker)

References:

Vasudevan Textbook of Biochemistry, 7th Edition, Page 276

↑ *Back to this question in Part A*

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Q10: Which of the following statements about the protoporphyrin ring and its associated metals is incorrect? (Q844898)

A. Heme is a derivative of porphyrin, a cyclic compound formed by the fusion of 4 pyrrole rings with a metallic group in the center

B. Heme-containing proteins such as myoglobin have Fe3+ as the metallic group.

C. Heme-containing enzymes are catalase, peroxidase, and nitric oxide synthase.

D. The metallic group in Chlorophyll is magnesium, while in cytochrome a/a3 is copper.

Correct Answer: B. Heme-containing proteins such as myoglobin have Fe3+ as the metallic group.

■ *Listen to Audio 1*

Explanation: Heme is composed of a protoporphyrin ring with a metallic group (Fe2+) in the center. (Option A) Porphyrin derived proteins Metallic group in the center Heme Heme-containing proteins - Hemoglobin, Myoglobin, some cytochromes Heme-containing enzymes- catalase, peroxidase, nitric oxide synthase (Option C) Iron (Fe2+) Hematin (Oxidized form of Heme) Iron (Fe3+) Cytochrome a/a3 (Option D) Copper Chlorophyll Magnesium

References:

Vasudevan Textbook of Biochemistry Edition 7th Pg. 270, 271, 273

↑ *Back to this question in Part A*

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Q11: An infant presents with severe photosensitivity and recurrent blistering of sun-exposed skin. Parents also report pink-colored urine since birth. On examination, the teeth appear as shown in the image. The most likely diagnosis is: (Q891737)

A. Congenital erythropoietic porphyria (Gunther disease)

B. Erythropoietic protoporphyria

C. Porphyria cutanea tarda

D. Acute intermittent porphyria



Correct Answer: A. Congenital erythropoietic porphyria (Gunther disease)

Explanation: The image shows erythrodontia (reddish-brown discolouration of teeth) , which, together with severe photosensitivity and pink-stained urine, is characteristic of congenital erythropoietic porphyria (Gunther disease) . Congenital erythropoietic porphyria (CEP, Gunther disease) is caused by uroporphyrinogen III synthase deficiency and is inherited in an autosomal recessive manner. The disease leads to the accumulation of uroporphyrin I and

coproporphyrin I , which deposit in teeth, skin, and red blood cells and are excreted in urine. A hallmark feature is erythrodontia , in which teeth show reddish-brown discolouration due to porphyrin deposition and exhibit red fluorescence under UV (Wood's lamp) . Infants often have pink-stained diapers from porphyrinuria. Patients develop severe blistering photosensitivity , leading to scarring and mutilation of sun-exposed areas such as the nose and ears. Porphyrin accumulation in red blood cells causes haemolytic anaemia and splenomegaly . Hypertrichosis may be seen in severe cases. Erythropoietic protoporphyria (Option B): Due to ferrochelatase deficiency , causes immediate painful, non-blistering photosensitivity ; it does not cause erythrodontia or pink-stained diapers. Porphyria cutanea tarda (Option C): Due to uroporphyrinogen decarboxylase deficiency , a hepatic porphyria presenting in adulthood with blistering photosensitivity and skin fragility ; does not present with neonatal erythrodontia. Acute intermittent porphyria (Option D): Due to porphobilinogen deaminase deficiency , presents with acute neurovisceral attacks (abdominal pain, neuropathy, psychiatric symptoms) and no photosensitivity .



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 320

↑ *Back to this question in Part A*

@Neet_pg_bot

Q12: Which porphyria is not classified as a hepatic porphyria? (Q891734)

A. Acute intermittent porphyria

B. Porphyria cutanea tarda

C. Hereditary coproporphyria

D. Erythropoietic protoporphyria

Correct Answer: D. Erythropoietic protoporphyria

Explanation: Erythropoietic protoporphyria (EPP) is classified as an erythropoietic porphyria because the primary site of excess porphyrin production is the bone marrow erythroid cells , not the liver. In contrast, the hepatic porphyrias include acute intermittent porphyria (AIP), porphyria cutanea tarda (PCT), hereditary coproporphyria, variegate porphyria, and ALA dehydratase deficiency porphyria . EPP is caused by ferrochelatase deficiency , the final enzyme in the heme biosynthetic pathway responsible for inserting iron into protoporphyrin IX. Because of this block, protoporphyrin IX accumulates , mainly in erythrocytes and plasma. Clinically, patients develop immediate, painful photosensitivity with burning, itching, and swelling on sun exposure, typically without blistering , unlike porphyria cutanea tarda. Acute neurovisceral attacks (such as abdominal pain and psychiatric symptoms) that characterise the acute hepatic porphyrias are not a feature of EPP. Although EPP is not a hepatic porphyria, the liver is secondarily involved in clearing excess protoporphyrin, and a minority of patients may develop cholestatic liver disease or gallstones . T The diagnostic hallmark is elevated free erythrocyte protoporphyrin , usually with normal urinary porphyrins . Acute intermittent porphyria (Option A): Autosomal dominant; due to porphobilinogen deaminase (hydroxymethylbilane synthase) deficiency ; a hepatic porphyria presenting with acute neurovisceral attacks without photosensitivity. Porphyria cutanea tarda (Option B): Due to uroporphyrinogen decarboxylase deficiency , a hepatic porphyria presenting with blistering photosensitivity and skin fragility , often associated with liver disease or iron overload. Hereditary coproporphyria (Option C): Autosomal dominant; due to coproporphyrinogen oxidase deficiency ; a hepatic (mixed) porphyria causing acute neurovisceral attacks ± photosensitivity .

References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 320, 321

↑ *Back to this question in Part A*

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Q13: An adult female presents with recurrent episodes of abdominal pain, nausea, constipation, tachycardia, hypertension, and peripheral neuropathy. Urine darkens on standing. Elevated urinary ALA and PBG are found. The most likely diagnosis is: (Q891733)

A. Hereditary coproporphyria

B. Variegate porphyria

C. Porphyria cutanea tarda

D. Acute intermittent porphyria (AIP)

Correct Answer: D. Acute intermittent porphyria (AIP)

Explanation: Acute intermittent porphyria (AIP) is caused by hydroxymethylbilane synthase (porphobilinogen deaminase) deficiency . It presents with neurovisceral symptoms - classically severe abdominal pain, autonomic dysfunction (tachycardia, hypertension), peripheral motor neuropathy, and psychiatric symptoms and does not cause photosensitivity . The biochemical hallmark is a marked increase in urinary porphobilinogen (PBG) and delta-aminolevulinic acid (ALA) due to the enzyme block in heme synthesis. A classic clinical clue is urine that darkens on standing , caused by oxidation of PBG to porphobilin, giving a port-wine colour . The abdominal pain is typically severe, diffuse, and without peritoneal signs. The neuropathy is motor-predominant and may progress to respiratory muscle weakness in severe attacks. Precipitating factors include CYP450-inducing drugs (e.g., barbiturates, sulfonamides), fasting , alcohol, and hormonal changes . Hereditary coproporphyria (Option A): Autosomal dominant; due to coproporphyrinogen oxidase deficiency ; presents with acute neurovisceral attacks ± photosensitivity . Variegate porphyria (Option B): Autosomal dominant; due to protoporphyrinogen oxidase deficiency ; causes both neurovisceral and cutaneous symptoms Porphyria cutanea tarda (Option C): Due to uroporphyrinogen decarboxylase deficiency , presents with blistering photosensitivity and skin fragility ; not an acute neurovisceral porphyria .

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 320

↑ *Back to this question in Part A*

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Q14: Which porphyria is not inherited in an autosomal dominant pattern? (Q891731)

A. Acute intermittent porphyria

B. Variegate porphyria

C. Hereditary coproporphyria

D. Erythropoietic protoporphyria

Correct Answer: D. Erythropoietic protoporphyria

Explanation: Most acute hepatic porphyrias - Acute intermittent porphyria, Variegate porphyria, and Hereditary coproporphyria are inherited in an autosomal dominant pattern. Erythropoietic protoporphyria (EPP) is caused by ferrochelatase (FECH) deficiency , the final enzyme in the heme biosynthetic pathway that inserts iron into protoporphyrin IX. Most cases of EPP show a pseudodominant inheritance pattern , typically due to a severe mutation in one FECH allele combined with a low-expression FECH allele on the other, rather than true autosomal dominance. Rarely, EPP may behave as an autosomal recessive disorder . In addition, a distinct X-linked protoporphyria exists due to gain-of-function mutations in ALAS2 . Because of the enzyme block, protoporphyrin IX accumulates in erythrocytes, plasma, and the liver. Clinically, EPP causes immediate, painful, non-blistering

photosensitivity , unlike porphyria cutanea tarda, which produces blistering lesions. About 5-10% of patients may develop cholestatic liver disease due to hepatic deposition of protoporphyrin. The diagnostic hallmark is elevated free (non-zinc-complexed) erythrocyte protoporphyrin . Acute intermittent porphyria (Option A): Autosomal dominant; due to porphobilinogen deaminase (hydroxymethylbilane synthase) deficiency. Variegate porphyria (Option B): Autosomal dominant; due to protoporphyrinogen oxidase deficiency; causes both neurovisceral and cutaneous symptoms. Hereditary coproporphyria (Option C): Autosomal dominant; due to coproporphyrinogen oxidase deficiency; presents with acute neurovisceral attacks ± photosensitivity.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 320

↑ *Back to this question in Part A*

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Q15: Cyclisation of hydroxymethylbilane to the first porphyrinogen (uroporphyrinogen III) is catalysed by which enzyme? (Q891729)

A. Uroporphyrinogen III synthase

B. Uroporphyrinogen decarboxylase

C. Coproporphyrinogen oxidase

D. ALA dehydratase

Correct Answer: A. Uroporphyrinogen III synthase

Explanation: Uroporphyrinogen III synthase catalyses the cyclisation of hydroxymethylbilane (HMB) to form uroporphyrinogen III , the first cyclic tetrapyrrole in heme synthesis. In this reaction, the enzyme causes a specific inversion (“flip”) of the fourth pyrrole ring (Ring D) , producing the asymmetric Type III isomer , which is the only form that can be used for the synthesis of heme, chlorophyll, and vitamin B■■. In the absence of this enzyme, HMB undergoes spontaneous cyclisation to form uroporphyrinogen I , a symmetric, non-physiological isomer that represents a metabolic dead end. Clinically, deficiency of uroporphyrinogen III synthase causes Congenital Erythropoietic Porphyria (Gunther disease) , characterised by accumulation of Type I porphyrins , leading to severe photosensitivity, hemolysis, and reddish-brown discolouration of teeth (erythrodontia) . This enzyme does not require any external cofactors . Uroporphyrinogen decarboxylase (Option B): Acts later in the pathway, converting uroporphyrinogen III to coproporphyrinogen III . Its deficiency causes Porphyria Cutanea Tarda . Coproporphyrinogen oxidase (Option C): A mitochondrial enzyme that converts coproporphyrinogen III to protoporphyrinogen IX ; it does not catalyse the initial cyclisation step. ALA dehydratase (Option D): Also called porphobilinogen synthase , it catalyses the early step converting ALA to porphobilinogen (PBG) , not the cyclisation of HMB.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 316

↑ *Back to this question in Part A*

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Q16: The by-product released during the condensation of porphobilinogen to hydroxymethylbilane by hydroxymethylbilane synthase (PBG deaminase) is: (Q891728)

A. CO■

B. NH■

C. H■O

D. Glutamate

Correct Answer: B. NH■

Explanation: Hydroxymethylbilane synthase (also called porphobilinogen deaminase) catalyses the condensation of four molecules of porphobilinogen (PBG) to form the linear tetrapyrrole hydroxymethylbilane . During this

process, each PBG molecule undergoes deamination , resulting in the release of ammonia (NH■) . Therefore, for every one molecule of hydroxymethylbilane formed, four molecules of NH■ are released . The reaction proceeds via a dipyrromethane cofactor bound to the enzyme, which acts as a primer for chain elongation. Clinically, deficiency of this enzyme causes Acute Intermittent Porphyria (AIP) , characterised by accumulation of PBG and ALA . The unstable hydroxymethylbilane is normally converted by uroporphyrinogen III synthase into uroporphyrinogen III . CO■ (Option A): Carbon dioxide is not released in this step. CO■ is involved earlier in heme synthesis (e.g., in the ALA synthase reaction with succinyl-CoA and glycine), not in the PBG deaminase step. H■O (Option C): Water is not the characteristic by-product of this condensation. The key chemical change here is deamination , which releases NH■ , not H■O. Glutamate (Option D): Glutamate is not a product of this reaction. The substrates are porphobilinogen molecules , and the by-product released is ammonia , not an amino acid.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 316, 317

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: What is the mechanism by which barbiturates worsen porphyria? (Q891719)

A. Inhibition of ferrochelatase

B. Activation of ALA dehydratase

C. Induction (de-repression) of ALA synthase

D. Inhibition of uroporphyrinogen decarboxylase

Correct Answer: C. Induction (de-repression) of ALA synthase

Explanation: Barbiturates are potent inducers of hepatic cytochrome P450 (CYP450) enzymes, which are heme-containing proteins . Increased CYP450 synthesis raises hepatic heme demand , leading to a decrease in the free heme pool . Heme normally exerts negative feedback on ALA synthase (ALAS1) , the rate-limiting enzyme of heme synthesis . When heme levels fall, this inhibition is removed, resulting in de-repression (induction) of ALAS1 . In patients with porphyria, downstream enzymes in the heme pathway (e.g., porphobilinogen deaminase in acute intermittent porphyria) are deficient. Thus, increased ALAS1 activity creates a metabolic bottleneck , causing accumulation of neurotoxic precursors δ-aminolevulinic acid (ALA) and porphobilinogen (PBG). This precipitates acute porphyric attacks with severe abdominal pain, autonomic dysfunction, neuropathy, and psychiatric symptoms. Inhibition of ferrochelatase (Option A): This is the mechanism of lead poisoning , which inhibits ferrochelatase and ALA dehydratase , not the mechanism of barbiturates. Activation of ALA dehydratase (Option B): ALA dehydratase is not activated in porphyria; it is classically inhibited by lead . Barbiturates worsen porphyria by acting upstream at ALAS1 . Inhibition of uroporphyrinogen decarboxylase (Option D): This is the primary defect in porphyria cutanea tarda (PCT) . While some factors can exacerbate PCT, barbiturates specifically worsen porphyria by inducing ALAS1 , not by inhibiting this enzyme.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 321

↑ *Back to this question in Part A*

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Q18: The conversion of succinyl-CoA and glycine to α-amino-β-ketoadipate requires which cofactor? (Q891718)

A. Thiamine pyrophosphate

B. Pyridoxal phosphate (Vitamin B■)

C. Biotin

D. FAD

Correct Answer: B. Pyridoxal phosphate (Vitamin B■)

Explanation: The first step of heme synthesis is catalysed by δ-aminolevulinate synthase (ALAS) in the mitochondrial matrix . This enzyme condenses succinyl-CoA and glycine to form α-amino-β-ketoadipate , an unstable intermediate that rapidly decarboxylates to δ-aminolevulinic acid (ALA) . Pyridoxal phosphate (PLP, Vitamin B■) is an essential cofactor for this reaction. PLP forms a Schiff base with glycine , stabilising reaction intermediates and enabling the condensation. This step is the rate-limiting step of heme synthesis and is feedback-inhibited by heme . Clinically, Vitamin B■ deficiency or inhibition of PLP (e.g., by isoniazid) impairs ALAS activity and can lead to sideroblastic anaemia due to defective heme synthesis. Thiamine pyrophosphate (Option A): TPP (Vitamin B■) is a cofactor for oxidative decarboxylation reactions (e.g., pyruvate dehydrogenase, α-ketoglutarate dehydrogenase), not for ALA synthase. Biotin (Option C): Biotin (Vitamin B■) is required for carboxylation reactions (e.g., pyruvate carboxylase, acetyl-CoA carboxylase), not for condensation of glycine and succinyl-CoA. FAD (Option D): FAD (Vitamin B■ derivative) functions as a redox cofactor in oxidation-reduction reactions and does not participate in the ALAS reaction.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 315, 316

↑ *Back to this question in Part A*

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Q19: Succinyl-CoA, an essential substrate for heme biosynthesis, is derived from which pathway? (Q891717)

A. Glycolysis

B. Fatty acid oxidation

C. TCA cycle (Krebs cycle)

D. Urea cycle

Correct Answer: C. TCA cycle (Krebs cycle)

Explanation: Succinyl-CoA is a key intermediate of the TCA cycle , formed from α-ketoglutarate by the α-ketoglutarate dehydrogenase complex . In heme biosynthesis, succinyl-CoA condenses with glycine in the mitochondrial matrix to form δ-aminolevulinic acid (ALA) , a reaction catalysed by ALA synthase (ALAS) , which is the first and rate-limiting step of heme synthesis and is feedback-inhibited by heme . Although succinyl-CoA can also be generated from odd-chain fatty acids and certain amino acids (valine, isoleucine, methionine) via the propionyl-CoA → methylmalonyl-CoA pathway , the primary metabolic source of succinyl-CoA for cellular metabolism is the TCA cycle . Within the TCA cycle, succinyl-CoA can also be converted to succinate by succinyl-CoA synthetase , producing GTP (or ATP) by substrate-level phosphorylation. Defects in this pathway, particularly in ALA synthase , are associated with sideroblastic anaemias , due to impaired heme synthesis. Glycolysis (Option A): Glycolysis breaks down glucose to pyruvate and does not produce succinyl-CoA . Fatty acid oxidation (Option B): β-oxidation primarily produces acetyl-CoA . Only odd-chain fatty acids can indirectly yield succinyl-CoA, but this is not the main source . Urea cycle (Option D): The urea cycle disposes of nitrogen and is linked to the TCA cycle via fumarate , but it does not generate succinyl-CoA .

References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 315, 316

↑ *Back to this question in Part A*

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Q20: Which of the following is not a site for heme biosynthesis? (Q891715)

A. Erythroid precursor cells in bone marrow

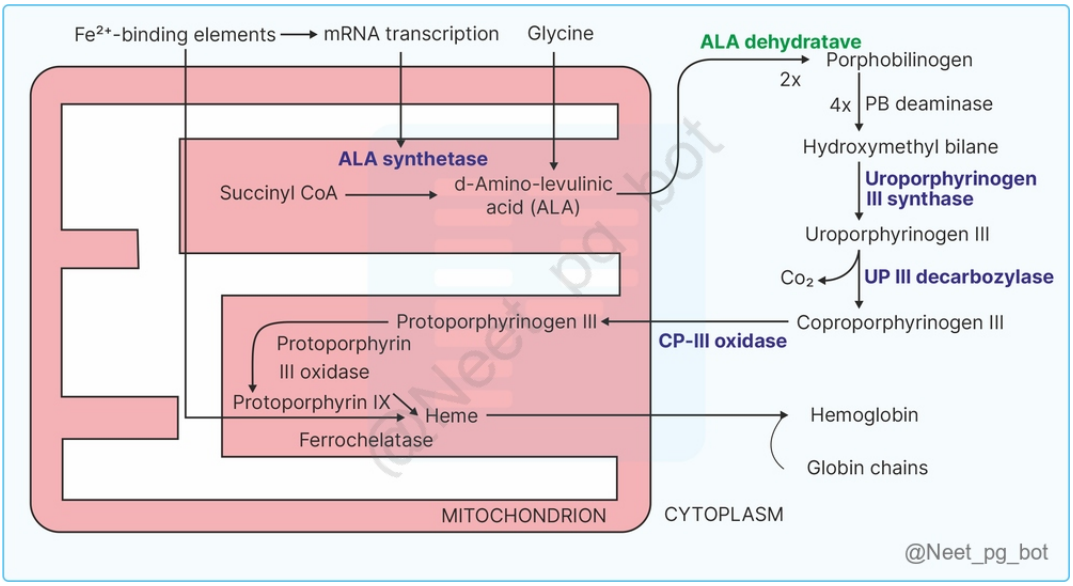
B. Hepatocytes

C. Mature erythrocytes

D. Mitochondria

Correct Answer: C. Mature erythrocytes

Explanation: Mature erythrocytes cannot synthesise heme because they lack mitochondria, a nucleus, and ribosomes , and therefore do not possess the enzymatic machinery required for heme biosynthesis. Heme synthesis is a compartmentalised process , occurring partly in the mitochondria and partly in the cytosol . The first step (condensation of glycine and succinyl-CoA by ALA synthase) and the final steps (including insertion of Fe^{2}■ by ferrochelatase) take place in the mitochondria , while the intermediate steps occur in the cytosol . Approximately 80-85% of total body heme synthesis occurs in erythroid precursor cells in the bone marrow , mainly for haemoglobin production, and the remaining 15-20% occurs in hepatocytes , primarily for synthesis of cytochrome P450 and other hemoproteins . Disruption of this pathway leads to porphyrias . Erythroid precursor cells in bone marrow (Option A): These are the major site of heme synthesis , as they still contain mitochondria and ribosomes and actively produce haemoglobin. Hepatocytes (Option B): The liver is a major site of heme synthesis for cytochromes and other hemoproteins involved in drug metabolism and detoxification. Mitochondria (Option D): Heme biosynthesis begins and ends in the mitochondria , with key enzymes such as ALA synthase and ferrochelatase located there.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Pages 315, 316

↑ *Back to this question in Part A*

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Q21: A patient with anaemia has elevated levels of 2,3-Bisphosphoglycerate (2,3-BPG) in their red blood cells. How does this increased 2,3-BPG level affect the patient's haemoglobin in terms of oxygen delivery? (Q875968)

A. Forms salt bridges with positively charged residues forming a relaxed structure

B. Disrupts salt bridges formed in the taut structure, decreasing oxygen affinity

C. Forms salt bridges with positively charged residues stabilising taut structure

D. Binds to the heme directly, decreasing the oxygen affinity

Correct Answer: C. Forms salt bridges with positively charged residues stabilising taut structure

Explanation: Increased levels of 2,3-BPG stabilise the taut (T) structure of haemoglobin by forming salt bridges with positively charged residues. This stabilisation lowers haemoglobin's affinity for oxygen, promoting oxygen release to tissues, which can be beneficial in conditions like anaemia. Effect of 2,3 BPG on Hb: Reduces the affinity of Hb for oxygen and helps unload oxygen to tissues. A by-product of glycolysis. (Rapaport Leubering Cycle) Mechanism:



References:

Harper’s Illustrated Biochemistry 32nd Edition Pg - 55

↑ *Back to this question in Part A*

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• [12_Heme Synthesis] 033 Previous Year Questions

« Back to Folder Directory

[12_Heme Synthesis] 033 Previous Year Questions — Quick Jump

Q1 (p. 1347) *Q2 (p. 1348)* *Q3 (p. 1349)* *Q4 (p. 1350)* *Q5 (p. 1351)* *Q6 (p. 1351)*
Q7 (p. 1352) *Q8 (p. 1353)* *Q9 (p. 1354)* *Q10 (p. 1355)* *Q11 (p. 1356)* *Q12 (p. 1357)*
Q13 (p. 1358)

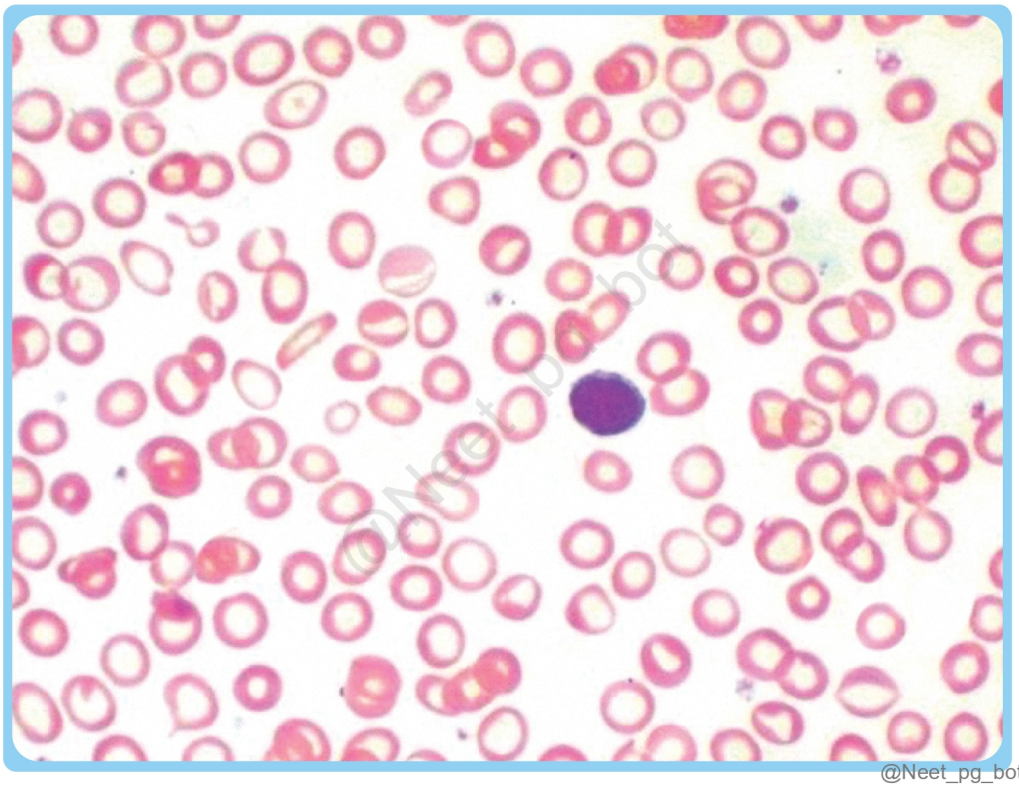
Q1: A 7-year-old child is brought to the clinic with complaints of fatigue and poor concentration. The mother reports that the child has been eating non-food items such as chalk and soil for the past few months. A peripheral blood smear image shows microcytic, hypochromic red blood cells. Which of the following is the most likely diagnosis? (Q866661)

A. Vitamin B12 deficiency

B. Iron deficiency anemia

C. Thalassemia major

D. Lead poisoning



Correct Answer: B. Iron deficiency anemia

Explanation: Pica, characterized by eating non-food items such as chalk and soil, is commonly linked to Iron Deficiency Anemia , which is supported by a peripheral smear showing microcytic, hypochromic red blood cells. Cause: Inadequate iron leading to decreased hemoglobin synthesis. Common in: Children, pregnant women, and individuals with chronic blood loss. Symptoms: Fatigue, pallor, poor concentration, and pica (craving/eating non-food substances like soil or chalk). Peripheral smear: Microcytic, hypochromic red blood cells. Diagnosis: Low serum ferritin, low serum iron, increased total iron-binding capacity (TIBC). Treatment: Oral iron supplementation and addressing the underlying cause. Vitamin B12 deficiency (Option A) causes macrocytic anemia, not microcytic. Thalassemia major (Option C) causes microcytic anemia but usually presents earlier and more severely, with different clinical features. Lead poisoning (Option D) can cause anemia but typically has basophilic stippling on smear and neurological symptoms.

References:

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↑ *Back to this question in Part A*

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Q2: A patient's relatives sent a message on social media to the consulting doctor. He is unaware of the other investigations. However, he mentioned that urinary

Coproporphyrin is positive. What is the probable cause? (Q844265)

A. Lead poisoning

B. Asbestosis

C. Silicosis

D. Mercury poisoning

Correct Answer: A. Lead poisoning

Explanation: Lead poisoning is associated with elevated levels of urinary coproporphyrin, which is a type of porphyrin. Lead inhibits key enzymes in the heme synthesis pathway, particularly ALA dehydratase and ferrochelatase. This inhibition causes a buildup of precursors, including coproporphyrin, in the urine. Elevated urinary coproporphyrin is a characteristic finding in cases of lead poisoning and is used as a diagnostic marker. Lead Poisoning Lead can cause acquired porphyria. Lead inhibits 3 enzymes of heme synthesis Delta ALA dehydratase Copro-pophyrinogen oxidase Ferro chelatase Biochemical features of lead poisoning ■■■■■■■ ■■■■■■■Delta ALA dehydratase: ALA elevation Copro-pophyrinogen oxidase: Elevated coproporphyrin Ferro chelatase: Elevated zinc protoporphyrin levels- most sensitive marker Asbestosis (Option B) and silicosis (Option C) do not increase urinary porphyrins. ■■■■■■■Mercury poisoning (Option D) causes ↑ coproporphyrin I , not specifically coproporphyrin III.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q3: Which of the following is not a product of heme breakdown? (Q839941)

A. Biliverdin

B. Carbon monoxide

C. Ferric Ion

D. Aminolevulinic acid

Correct Answer: D. Aminolevulinic acid

Explanation: Aminolevulinic acid acid is not a product of heme breakdown. It is an intermediate in the biosynthesis of heme, not a breakdown product. The breakdown-products of heme breakdown include: Biliverdin (Option A) : Heme is initially broken down by heme oxygenase to form biliverdin. Bilirubin: Biliverdin is

subsequently reduced to bilirubin by the enzyme biliverdin reductase. Carbon Monoxide (CO) (Option B) : During the breakdown of heme to biliverdin by heme oxygenase, carbon monoxide is produced. Ferric Ion (Fe^{3}) (Option C) : Iron is released in its ferric form (Fe^{3}) during the cleavage of the heme molecule by heme oxygenase.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q4: Which of the following is the rate-limiting enzyme of heme synthesis pathway? (Q820401)

A. HMB synthase

B. δ Amino-Levulinic Acid dehydratase

C. δ Amino-Levulinic Acid synthase

D. Ferrochelatase

Correct Answer: C. δ Amino-Levulinic Acid synthase

Explanation: This enzyme catalyzes the formation of ALA from glycine and succinyl-CoA. It is the rate-limiting enzyme of the heme synthesis pathway. Its activity is tightly regulated by feedback inhibition by heme itself. The rate-limiting enzyme in the heme synthesis pathway is delta-aminolevulinic acid synthase (ALAS) . This enzyme catalyzes the condensation of glycine and succinyl-CoA to form delta-aminolevulinic acid (ALA), which is the first and committed step in the heme biosynthesis pathway. There are two isoforms of ALAS: ALAS1: This isoform is primarily found in the liver and is responsible for heme synthesis for hemoglobin in red blood cells. ALAS2: This isoform is primarily found in erythroid precursor cells and is involved in heme synthesis for hemoglobin in developing red blood cells. HMB synthase (5-Aminolevulinic Acid Synthase) (Option A): This enzyme catalyzes the formation of δ-Aminolevulinic acid (ALA) from succinyl-CoA and glycine. It is not the rate-limiting enzyme but is the first committed step in heme synthesis. δ Amino-Levulinic Acid dehydratase (Porphobilinogen synthase) (Option B): This enzyme catalyzes the condensation of two molecules of ALA to form porphobilinogen. It is not the rate-limiting enzyme either. Ferrochelatase (Option D): This enzyme catalyzes the final step of heme synthesis, inserting an iron ion into the protoporphyrin ring to form heme. It is not the rate-limiting enzyme but is responsible for the incorporation of iron into the heme molecule.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q5: What plasma protein is found in the highest concentration? (Q793458)

A. Bence jones protein

B. Globulin

C. Fibrinogen

D. Albumin

Correct Answer: D. Albumin

Explanation: Albumin is the most abundant protein in plasma. It is manufactured by the liver. Albumin molecules serve as binding proteins for the transport of steroid hormones and fatty acids. Lipids are hydrophobic in nature, so albumin helps their transport in the polar environment of plasma. Bence-Jones proteins (Option A) appear in the urine in many people that have multiple myeloma. Hence, it is not considered as the most abundant protein. Globulins (Option B) are protein groups that are present in our blood and made in our liver by our immune system. It plays an important role in immune function, blood clotting, liver function etc. Various types of globulins are present in our body such as alpha, gamma globulins etc. It is considered as some of the abundant storage protein. Fibrinogen (Option C) is considered the least abundant plasma protein in the blood. Plasma Protein Function Albumin Maintains oncotic pressure, transports hormones and fatty acids Globulins Transport lipids and metal ions, act as antibodies Fibrinogen Essential for blood clotting Prothrombin Precursor to thrombin, which is necessary for blood clotting Complement proteins Part of the immune system, help destroy pathogens Immunoglobulins Antibodies produced by plasma cells, bind to antigens Transferrin Binds to and transports iron Ceruloplasmin Binds to and transports copper Haptoglobin Binds to free hemoglobin, prevents its breakdown Alpha-1-antitrypsin Inhibits enzymes that break down connective tissue Alpha-fetoprotein Produced during fetal development, used as a marker for certain cancers

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: What is the most appropriate dietary recommendation for a middle-aged man diagnosed with a rare variant of pyruvate dehydrogenase deficiency, resulting in high anion gap metabolic acidosis? (Q793470)

A. High protein and low carbohydrate intake

B. High carbohydrate and low fat intake

C. High fat and low carbohydrate intake

D. Only carbohydrate intake henceforth

Correct Answer: C. High fat and low carbohydrate intake

Explanation: This option is the most appropriate because it can help reduce the production of pyruvate, as fats can be converted into ketone bodies, which are an alternative fuel source for the body. A high-fat, low-carbohydrate diet can help mitigate the effects of pyruvate dehydrogenase deficiency and reduce the risk of metabolic acidosis. Pyruvate dehydrogenase deficiency is a neurodegenerative disorder that is associated with abnormal mitochondrial dysfunction. The pyruvate dehydrogenase complex is a multienzyme complex that is located in the matrix of mitochondria. It acts as a catalyst in the oxidative decarboxylation process of pyruvate to acetyl CoA, which is an irreversible reaction. Pyruvate dehydrogenase deficiency is a rare genetic, life-threatening mitochondrial disorder that is associated with lactic acidosis as well as neurological and neuromuscular degeneration. Developmental delay, lethargy, and hypotonia is usually present in the patients with Pyruvate dehydrogenase complex deficiency. As fat cannot be converted to glucose and low carbohydrate intake will also lead to less production of pyruvate . So, this is the best option. Pyruvate dehydrogenase deficiency is a rare genetic disorder that affects the ability of the body to convert pyruvate into acetyl-CoA, which is an important molecule for energy production in the body. Feature Description Inheritance X-linked recessive Incidence Estimated to be 1 in 100,000 to 300,000 live births Symptoms Neurological symptoms such as developmental delay, intellectual disability, seizures, and ataxia; muscle weakness; lactic acidosis; poor feeding; and failure to thrive Diagnosis Blood and urine tests, genetic testing Treatment Supportive care, including a high-fat, low-carbohydrate diet; thiamine supplementation; and medications to control seizures and other symptoms Prognosis The severity of symptoms varies widely, and the prognosis depends on the specific mutation and the age of onset. Some patients have a milder form of the disease and can live relatively normal lives with appropriate treatment, while others have severe intellectual disability and may have a reduced lifespan. Lactic acid is built up in the body when pyruvate dehydrogenase deficiency is present and it causes neurological problems. Hence, high protein and low carbohydrate intake are not allowed in the case of pyruvate dehydrogenase deficiency. As protein is converted to glucose via gluconeogenesis process. (Option A) High carbohydrate and low-fat intake is not recommended as it has carbohydrates that are unable to metabolize completely. (Option B) This option includes only carbohydrate intake, henceforth this cannot be recommended because glucose is unable to metabolize completely. (Option D)

↑ *Back to this question in Part A*

@Neet_pg_bot

Q7: Heme is derived from which of the following amino acids? (Q860419)

A. Glutamine

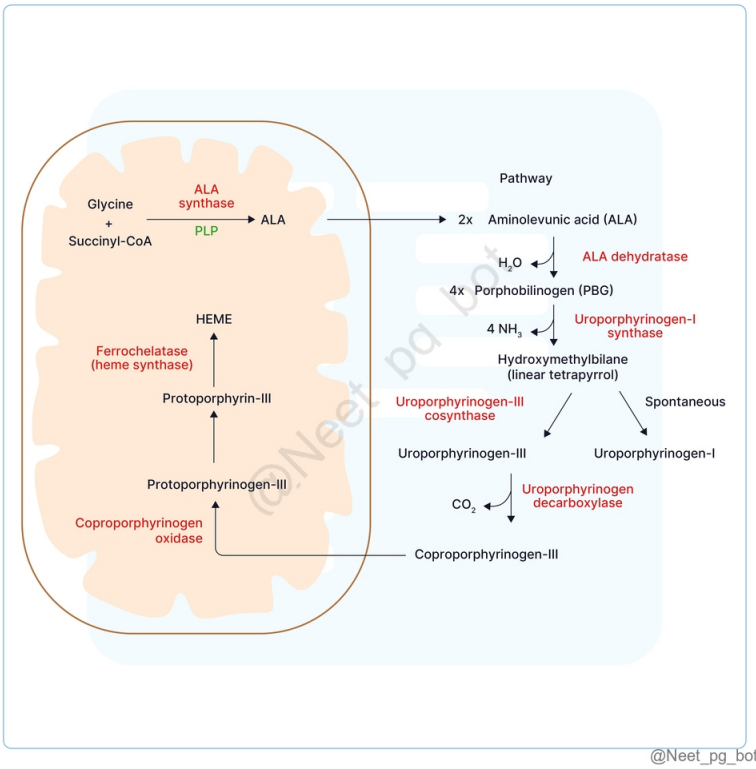
B. Glycine

C. Glutamate

D. Valine

Correct Answer: B. Glycine

Explanation: Glycine combines with succinyl-CoA in the first step of heme synthesis , catalyzed by the enzyme δ-aminolevulinic acid (ALA) synthase . This reaction is the rate-limiting step in heme biosynthesis. Glutamine (Option A): Glutamine is involved in nucleotide biosynthesis and ammonia transport but does not play a direct role in heme synthesis. Glutamate (Option C): Glutamate is primarily involved in the synthesis of glutathione and neurotransmitters but does not contribute to heme synthesis. Valine (Option D): Valine is a branched-chain amino acid involved in protein synthesis and energy production but not in heme biosynthesis.



References:

Vasudevan Textbook of Biochemistry Edition 7th Pg. 271, 272, 273

↑ *Back to this question in Part A*

@Neet_pg_bot

Q8: Which of the following amino acids are necessary for purine ring formation? (Q860424)

A. Glycine, aspartate, valine

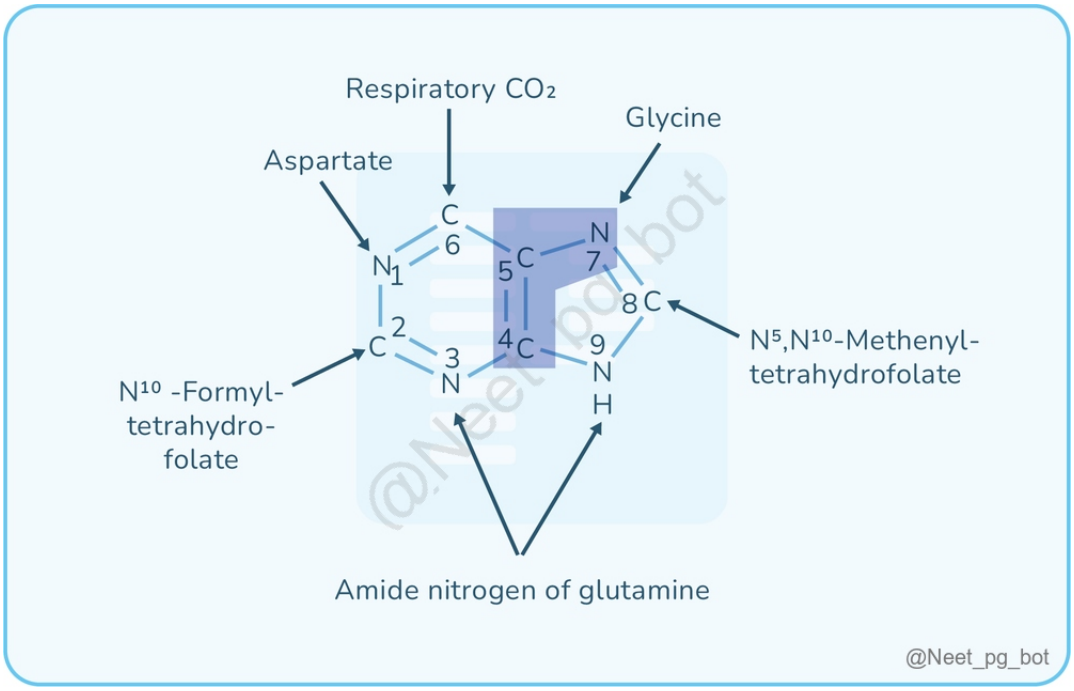
B. Glycine, Aspartate, Glutamine

C. Glycine, Aspartate, Glutamate

D. Glycine, Asparagine, Glutamine

Correct Answer: B. Glycine, Aspartate, Glutamine

Explanation: Purine ring formation requires three amino acids - Glycine, Aspartate, Glutamine and the cofactor N10-formyl tetrahydrofolate (N10- formyl THF) for purine ring formation. (Option B) (Option A, C and D ruled out) The sources of each atom in a purine were found by feeding isotopic precursors of uric acid to pigeons. The C-2 atom is from formyl THF The C-8 atom is from N5N10 methenyl THF The C-6 atom is from respiratory CO2 . N1 is from aspartate N3 and N9 are from Glutamine C4, C5 and N7 are from Glycine



References:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7869020/

Harper’s Illustrated Biochemistry, 32nd edition, pg:338, 339

Vasudevan textbook of biochemistry, 7th edition, pg: 208

↑ *Back to this question in Part A*

@Neet_pg_bot

Q9: Decreased % transferrin saturation is seen in? (Q854613)

A. Hemochromatosis

B. Iron deficiency anemia

C. Megaloblastic anemia

D. Hemosiderosis

Correct Answer: B. Iron deficiency anemia

Explanation: Laboratory findings in iron deficiency anemia: Hemoglobin Indices Low Mean Corpuscular Hemoglobin (MCH), Low Mean Corpuscular Volume (MCV) Peripheral Blood Smear Microcytosis, Hypochromia, Anisocytosis Serum Ferritin Decreased Serum Iron Decreased Transferrin Saturation Decreased Total Iron-Binding Capacity Increased Stool for Occult Blood May be positive Mentzer Index >15 Hemoglobin Electrophoresis Normal Bone Marrow Aspiration/Biopsy Iron stores absent on staining In the rest of the conditions, the % transferrin saturation is normal or increased

References:

https://www.ncbi.nlm.nih.gov/books/NBK448065/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q10: Which part of the haemoglobin molecule does 2,3-Bisphosphoglycerate (2,3-BPG) bind to in order to regulate oxygen release? (Q854619)

A. Alpha chains of haemoglobin

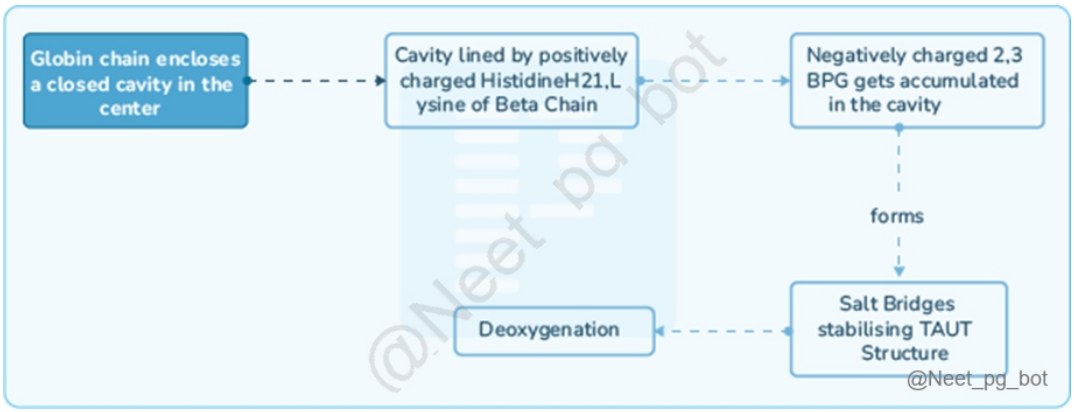
B. Heme iron centre

C. Beta chains of haemoglobin

D. Globin-heme interface

Correct Answer: C. Beta chains of haemoglobin

Explanation: 2,3-BPG binds within the central cavity of the deoxyhaemoglobin tetramer . It interacts with positively charged residues (mainly lysine and histidine) on the β-chains . This binding stabilises the T (tense) state , lowering haemoglobin’s affinity for oxygen and promoting oxygen delivery to tissues. Effect of 2,3 BPG on Hb: Reduces the affinity of Hb for oxygen and helps unload oxygen to tissues. A by-product of glycolysis. (Rapaport Leubering Cycle) Alpha chains of haemoglobin (Option A) : Incorrect because 2,3-BPG binds primarily to residues on the β-chains , not the α-chains. Heme iron centre (Option B) : 2,3-BPG does not bind directly to heme or iron ; it acts allosterically , not at the active oxygen-binding site. Globin-heme interface (Option D) : This is the site of coordination between globin and heme, not where 2,3-BPG binds.



References:

Harper's Illustrated Biochemistry, 32nd Edition, Page 55

↑ *Back to this question in Part A*

@Neet_pg_bot

Q11: What does iron deficiency anemia (IDA) stand for? (Q854624)

A. Low serum ferritin

B. Low transferrin saturation

C. Increased TIBC

D. All of the above

Correct Answer: D. All of the above

Explanation: Iron deficiency anemia (IDA) is characterized by laboratory evidence of depleted iron stores and compensatory changes in iron transport proteins: Low serum ferritin reflects depleted body iron stores. Low transferrin saturation indicates reduced circulating iron available for erythropoiesis. Increased total iron-binding capacity (TIBC) occurs as a compensatory rise in transferrin production by the liver to scavenge available iron. Laboratory findings in iron deficiency anemia: Hemoglobin Indices Low Mean Corpuscular Hemoglobin (MCH), Low Mean Corpuscular Volume (MCV) Peripheral Blood Smear Microcytosis, Hypochromia, Anisocytosis Serum Ferritin Decreased Serum Iron Decreased Transferrin Saturation Decreased Total Iron-Binding Capacity Increased Stool for Occult Blood May be positive Mentzer Index >15 Hemoglobin Electrophoresis Normal Bone Marrow Aspiration/Biopsy Iron stores absent on staining

References:

https://www.ncbi.nlm.nih.gov/books/NBK448065/

↑ *Back to this question in Part A*

Q12: Which of the following is the rate-limiting enzyme of heme synthesis pathway? (Q857891)

A. HMB synthase

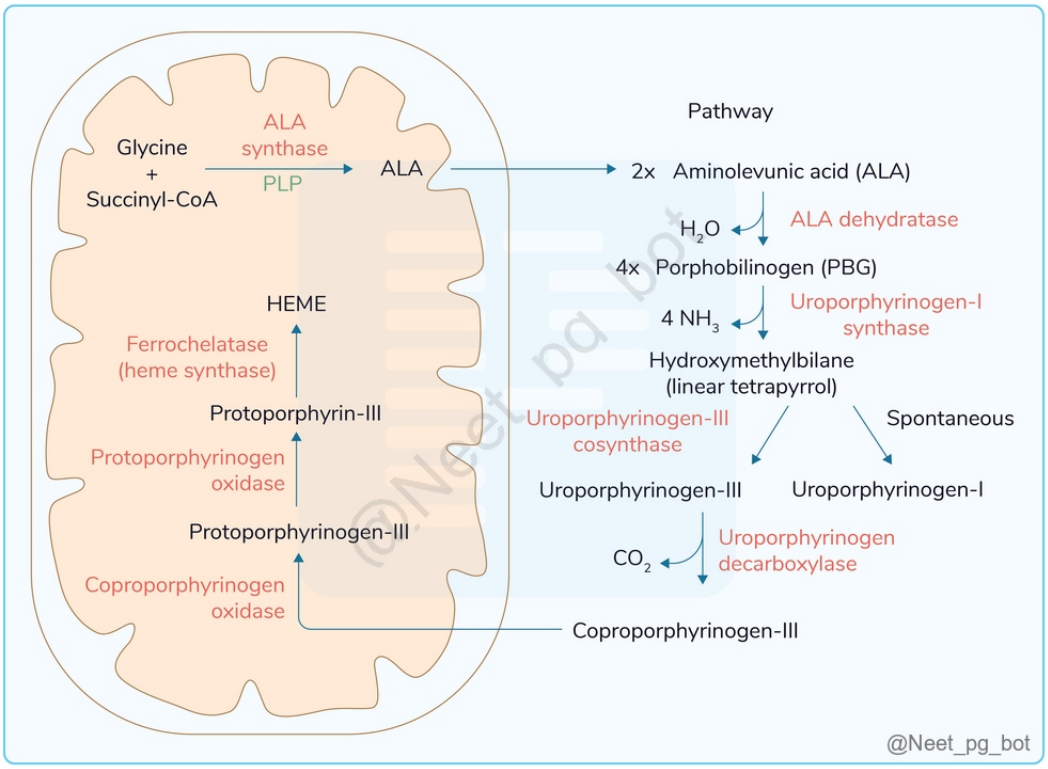
B. δ Amino-Levulinic Acid dehydratase

C. δ Amino-Levulinic Acid synthase

D. Ferrochelatase

Correct Answer: C. δ Amino-Levulinic Acid synthase

Explanation: This enzyme catalyzes the formation of ALA from glycine and succinyl-CoA. It is the rate-limiting enzyme of the heme synthesis pathway. Its activity is tightly regulated by feedback inhibition by heme itself. Heme Synthesis: Step Reaction Enzyme Location Key Points 1 Succinyl CoA + Glycine → Delta-aminolevulinic acid (ALA) ALA Synthase (Option C) Mitochondria Rate-limiting step ; requires pyridoxal phosphate as a cofactor 2 2 ALA → Porphobilinogen (PBG) ALA Dehydratase (Option B) Cytoplasm It involves removal of 2 molecules of water; the enzyme contains zinc and is inhibited by lead 3 4 PBG → Hydroxymethylbilane (HMB) Uroporphyrinogen I Synthase/ HMB synthase (Option A) Cytoplasm - 4a HMB → Uroporphyrinogen I Spontaneous cyclization Cytoplasm - 4b HMB → Uroporphyrinogen III Uroporphyrinogen III Synthase Cytoplasm - 5 Uroporphyrinogen III → Coproporphyrinogen III Uroporphyrinogen Decarboxylase Cytoplasm Decarboxylation, removal of 4 molecules of CO2 6 Coproporphyrinogen III → Protoporphyrinogen III Coproporphyrinogen Oxidase Mitochondria 7 Protoporphyrinogen III → Protoporphyrin III Protoporphyrinogen Oxidase Mitochondria 8 Protoporphyrin III → Heme Ferrochelatase (Option D) Mitochondria Insertion of Fe2+



References:

Vasudevan Textbook of Biochemistry Edition 7th Pg. 271, 272, 273

↑ *Back to this question in Part A*

@Neet_pg_bot

Q13: A female is diagnosed with lead poisoning . Which of the following enzyme defects is primarily responsible for the hematologic abnormalities observed in this condition? (Q857898)

A. ALA synthase

B. Uroporphyrinogen decarboxylase

C. Heme synthase

D. Coproporphyrinogen oxidase

Correct Answer: C. Heme synthase

Explanation: Lead (Pb^{2}■) inhibits ferrochelatase , the final enzyme in the heme biosynthesis pathway , which catalyzes: Protoporphyrin IX + Fe^{2}■ → Heme Inhibition prevents iron incorporation into protoporphyrin IX,

causing accumulation of free protoporphyrin and resulting in microcytic, hypochromic anemia . Although lead also inhibits ALA dehydratase , the hematologic effects (anemia) are primarily due to ferrochelatase inhibition ALA synthase (A): Rate-limiting step of heme synthesis, not affected by lead Uroporphyrinogen decarboxylase (B): Defective in porphyria cutanea tarda , unrelated to lead poisoning. Coproporphyrinogen oxidase (D): Defective in hereditary coproporphyria , also unrelated to lead toxicity.

References:

Vasudevan Textbook of Biochemistry Edition 7th Pg. 276

↑ *Back to this question in Part A*

@Neet_pg_bot

• [13_Amino Acid and Protein Metabolism] 034 Previous Year Questions

« Back to Folder Directory

[13_Amino Acid and Protein Metabolism] 034 Previous Year Questions — Quick Jump

Q1 (p. 1360) *Q2 (p. 1360)* *Q3 (p. 1362)* *Q4 (p. 1362)* *Q5 (p. 1363)* *Q6 (p. 1364)*

Q1: Which of the following is deficient in a patient with phenylketonuria with normal phenyl alanine hydroxylase levels? (Q844228)

A. Tyrosine transaminase

B. Dopa decarboxylase

C. Tyrosine hydroxylase

D. Dihydrobiopterine reductase

Correct Answer: D. Dihydrobiopterine reductase

Explanation: In malignant phenylketonuria , the enzyme phenylalanine hydroxylase is normal, but there is a deficiency of tetrahydrobiopterin (BH■) due to lack of dihydrobiopterin reductase . Without BH■, phenylalanine cannot be converted to tyrosine, leading to high phenylalanine levels despite normal PAH. Classic phenylketonuria (PKU) results from a deficiency of phenylalanine hydroxylase (PAH) , which converts phenylalanine → tyrosine using tetrahydrobiopterin (BH■) as a cofactor. However, in some cases, PAH enzyme activity is normal , but there is a deficiency of dihydrobiopterin reductase (DHPR) , which regenerates BH■ from BH■ . This leads to a functional deficiency of BH■ , resulting in accumulation of phenylalanine and reduced synthesis of dopamine, norepinephrine, and serotonin , since BH■ is also a cofactor for tyrosine hydroxylase and tryptophan hydroxylase . Clinical features: Intellectual disability (if untreated) Movement disorders Seizures and hypomyelination (due to neurotransmitter deficiency) Tyrosine transaminase (Option A): Deficiency causes tyrosinemia type II , not PKU. Dopa decarboxylase (Option B): Converts L-DOPA to dopamine; not linked to PKU pathology. Tyrosine hydroxylase (Option C): Catalyzes dopamine synthesis, but its deficiency leads to catecholamine deficiency, not phenylalanine accumulation

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: A patient presents with dermatitis, dementia, and cognitive decline. Which micronutrient deficiency is most likely responsible? (Q844235)

A. Niacin

B. Thiamine

C. Tryptophan

D. Riboflavin

Correct Answer: A. Niacin

Explanation: The Clinical scenario of Dermatitis, Dementia and Cognitive decline suggests Niacin deficiency (Pellagra) Pellagra is characterized by the classic triad of symptoms: Dermatitis Bright red erythema- in sun-exposed areas (face, ankles, feet) Casal's necklace- ↑pigmentation around the neck Diarrhoea Dementia {{caption_text}} Incorrect Options: Thiamine (Option B) (Vitamin B1) deficiency typically presents with symptoms related to the nervous system, such as peripheral neuropathy, cardiovascular abnormalities, and Wernicke-Korsakoff syndrome. Tryptophan (Option C) plays a crucial role in various metabolic processes. It serves as a precursor for Niacin (Vitamin B3) and Serotonin. Thus symptoms like mood alteration, decreased/increased appetite is also present. Riboflavin (Option D) deficiency causes cheilosis, glossitis, seborrheic dermatitis and proliferation of bulbar conjunctival capillaries.



References:

Harper’s Illustrated Biochemistry, 32nd Edition, Pg.542,543 Textbook of Biochemistry- Vasudevan, 7th Edition, Pg. 239-242,477,479,481,482

↑ *Back to this question in Part A*

@Neet_pg_bot

Q3: What is true about the Hayflick limit? (Q856475)

A. Human cells can divide a finite number of times only

B. Free radical oxidation level is unrestricted

C. Telomerase activity is unrestricted

D. RNA polymerase can function for a finite number of time only

Correct Answer: A. Human cells can divide a finite number of times only

Explanation: The Hayflick limit , discovered by Dr. Leonard Hayflick, states that normal human cells have a finite capacity for replication. Telomeres, protective caps at chromosome ends, shorten with each cell division due to incomplete DNA replication. Shortened telomeres eventually lead to cellular senescence, preventing uncontrolled cell division and potential cancer. Some cells, like stem cells and certain cancers, express telomerase, extending telomeres and bypassing the Hayflick limit. In normal human cells, telomerase activity is limited, leading to replicative senescence. The Hayflick limit , the maximum times cells can divide, is influenced by several factors: Telomere Length: Shortening telomeres indicate reduced division capacity. Telomerase Activity: Higher activity extends telomeres, bypassing the limit. DNA Damage: Accumulated damage triggers senescence, limiting division. Cellular Stress: Various stressors accelerate senescence, reducing the limit. Genetic Factors: Variations affect telomere maintenance and DNA repair. Environmental Factors: Lifestyle, pollutants, and stress impact cellular health, affecting the limit. Free radical oxidation level is unrestricted (Option B): The Hayflick limit is not directly related to free radical oxidation levels. Telomerase activity is unrestricted (Option C): Telomerase activity is typically restricted in normal human cells, unlike stem cells and certain cancers. RNA polymerase can function for a finite number of times only (Option D): The Hayflick limit primarily involves telomere shortening, not RNA polymerase function.

References:

Hayflick limit

↑ *Back to this question in Part A*

@Neet_pg_bot

Q4: A female patient presented with pain and redness in the great toe. The serum uric acid level is 9.4mg/dl. The physician prescribed a drug for the treatment. Which of the following enzyme is likely to be inhibited by this drug? (Q859691)

A. Phosphoribosyl transferase

B. Thymidylate synthase

C. Xanthine oxidase

D. Dihydrofolate reductase

Correct Answer: C. Xanthine oxidase

Explanation: The clinical scenario describes a female patient with pain and redness in the great toe, which suggests gout, a condition caused by elevated uric acid levels. The patient's serum uric acid level of 9.4 mg/dl is above the normal range, indicating hyperuricemia, which can lead to the formation of uric acid crystals in the joints, causing pain and inflammation. The physician has prescribed a drug that helps manage gout by reducing uric acid levels. The drug prescribed is allopurinol, which is known to inhibit xanthine oxidase, the enzyme responsible for converting hypoxanthine to xanthine and xanthine to uric acid. By inhibiting xanthine oxidase, allopurinol decreases the production of uric acid, effectively managing gout by lowering uric acid levels in the body. Phosphoribosyl transferase (Option A): Involved in purine metabolism, but not the target of allopurinol. Thymidylate synthase (Option B): Involved in DNA synthesis, not related to gout treatment. Dihydrofolate reductase (Option D): Involved in folate metabolism, and inhibited by drugs like methotrexate, not relevant to gout treatment.

References:

Harper’s Illustrated Biochemistry (23rd Edition), Page 81,82.

Vasudevan Textbook of Biochemistry (7th Edition), Page 62.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q5: Which of the following post-translational modifications is common to collagen, hemoglobin, and immunoglobulins? (Q860100)

A. Hydroxylation

B. Subunit aggregation

C. Poly A tail addition

D. DNA mismatch repair

Correct Answer: B. Subunit aggregation

Explanation: Collagen forms a functional triple helix through aggregation of three polypeptide chains, stabilized by hydrogen bonds and hydroxyproline residues. Hemoglobin is a functional tetramer, composed of two α-globin and two β-globin chains, which aggregate to carry oxygen efficiently. Immunoglobulins (antibodies) consist of two heavy and two light chains, which aggregate to form the functional Y-shaped molecule. In all three proteins, subunit aggregation is a crucial post-translational event for proper folding, stability, and biological function. Incorrect Options: Hydroxylation (Option A): This is a post-translational modification involving the addition of hydroxyl groups to amino acids. While hydroxylation is important in collagen synthesis, it is not a common modification for hemoglobin and immunoglobulins. Poly A tail addition (Option C): This is a post-transcriptional modification that involves adding a polyadenine tail to the 3' end of mRNA. It helps in mRNA stability and translation efficiency but does not pertain to the protein structures in question. DNA mismatch repair (Option D): DNA mismatch repair is a genomic maintenance process that corrects errors in DNA replication. It is unrelated to protein post-translational modifications.

References:

Harper's Illustrated Biochemistry, 32nd Edition, Pg. 35-40

Textbook of Biochemistry- Vasudevan, 7th Edition, Pg. 604

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: A 2-month-old infant presents with poor feeding, vomiting, and lethargy. Laboratory studies show hyperammonemia and elevated levels of orotic acid in the urine. Which of the following is the most likely enzyme deficiency? (Q866671)

A. Carbamoyl phosphate synthetase I deficiency

B. Ornithine transcarbamylase (OTC) deficiency

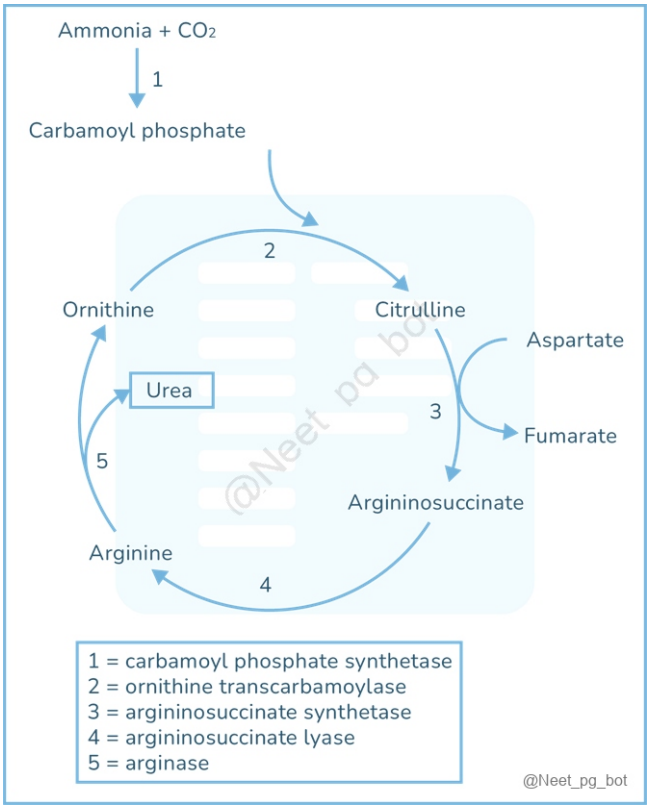
C. Orotidine 5'-phosphate decarboxylase deficiency

D. Argininosuccinate lyase deficiency

Correct Answer: B. Ornithine transcarbamylase (OTC) deficiency

Explanation: The infant's symptoms and laboratory findings are highly suggestive of Type 2 Hyperammonemia , or Ornithine Transcarbamylase (OTC) Deficiency. Hyperammonemia type 2 is an X-linked deficiency caused by a defect in ornithine transcarbamoylase (this reaction occurs in mitochondria), which forms citrulline in the Krebs cycle. Mothers with this condition may have hyperammonemia and avoid high-protein foods. Elevated blood, cerebrospinal fluid, and urine glutamine levels are due to increased glutamine synthesis in response to high ammonia levels. Orotic aciduria - Due to the channelling of carbamoyl phosphate into pyrimidine synthesis. Feature Type 1 Hyperammonemia Type 2 Hyperammonemia Defective Enzyme Carbamoyl Phosphate Synthetase

I (CPS1) (Option A) Ornithine Transcarbamylase (OTC) Inheritance Autosomal recessive X-linked recessive Mechanism The inability to synthesize carbamoyl phosphate blocks the urea cycle. Accumulation of carbamoyl phosphate in the mitochondria, which then spills over into the cytoplasm. Metabolic Consequences Elevated ammonia and glutamate Normal orotic acid and uracil Low citrulline. Elevated ammonia, glutamate, and glutamine. High uracil and/or orotic acid (excess carbamoyl phosphate enters pyrimidine synthesis). Normal citrulline. Clinical Presentation Symptoms coincide with increased protein intake. CNS : Lethargy, drowsiness, coma, Convulsions, vomiting, respiratory stimulation, hyperventilation. Similar to Type 1. Hepatomegaly. Orotidine 5'-phosphate decarboxylase deficiency (Option C): this is orotic aciduria (megaloblastic anaemia, growth retardation), but ammonia is normal. Argininosuccinate lyase deficiency (Option D): causes hyperammonemia but with elevated citrulline/argininosuccinate, not orotic acid.



↑ *Back to this question in Part A*

@Neet_pg_bot

• [14_Clinical Biochemistry] 035 Previous Year Questions

« Back to Folder Directory

[14_Clinical Biochemistry] 035 Previous Year Questions — Quick Jump

Q1 (p. 1366)	*Q2 (p. 1367)*	*Q3 (p. 1367)*	*Q4 (p. 1368)*	*Q5 (p. 1369)*	*Q6 (p. 1369)*
Q7 (p. 1371)	*Q8 (p. 1372)*	*Q9 (p. 1372)*	*Q10 (p. 1373)*	*Q11 (p. 1374)*	*Q12 (p. 1375)*
Q13 (p. 1376)	*Q14 (p. 1379)*	*Q15 (p. 1380)*	*Q16 (p. 1381)*	*Q17 (p. 1382)*	*Q18 (p. 1383)*
Q19 (p. 1384)	*Q20 (p. 1385)*	*Q21 (p. 1386)*	*Q22 (p. 1386)*	*Q23 (p. 1387)*	*Q24 (p. 1388)*
Q25 (p. 1388)	*Q26 (p. 1389)*				

Q1: Which of the following uses CRISPR-Cas9 technology? (Q860081)

A. DNA Editing

B. Post-transcriptional Modification

C. DNA Induction

D. DNA Editing Solution

Correct Answer: A. DNA Editing

Explanation: CRISPR cas9 is used in editing parts of the genome by removing, adding or altering sections of the DNA sequence. It is currently the simplest, most versatile and precise method of genetic manipulation. C-clustered R-regulatory I-interspaced S-short P-palindromic R-repeats NHEJ and HEJ are the two mechanisms employed by the CRISPR-Cas9 System. It is a powerful tool for inducing precise double-stranded breaks in DNA. CRISPR-Cas9 System Components: Guide RNA (gRNA): Directs the Cas9 enzyme to a specific DNA sequence. Cas9 Enzyme: Induces double-stranded breaks (DSBs) at the target site. Repair Mechanisms: Non-homologous end Joining (NHEJ): This technique directly ligates broken DNA ends without a template, often leading to small insertions or deletions. It can also cause gene knockout by disrupting the coding sequence. Homologous End Joining (HEJ): Uses a homologous template for precise repair of Double strand breaks and enables accurate gene knock-in or correction of mutations. Post-transcriptional Modification (Option B): This process involves changes to RNA after it has been transcribed from DNA, such as splicing, capping, and polyadenylation. It does not involve CRISPR-Cas9 technology, which primarily targets DNA rather than RNA. DNA Induction (Option C): This term is not commonly used in the context of CRISPR-Cas9 technology. CRISPR-Cas9 is focused on directly editing DNA sequences rather than inducing changes. DNA Editing Solution (Option D): While CRISPR-Cas9 is a powerful tool for DNA editing, the term "DNA Editing Solution" is vague and not specific to the CRISPR-Cas9 system itself.

References:

Brock Biology of Microorganisms 15th Edition, Page 298

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8215069/

Harper’s Illustrated Biochemistry 32nd Edition, Page 454

↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: A child presents with abdominal distension and an enlarged liver by 8 cm below the costal margin. The liver is smooth on palpation. Which of the following is the most likely diagnosis? (Q866675)

A. Glycogen Storage Disease (GSD)

B. Lysosomal Storage Disease (LSD)

C. Hepatocellular Carcinoma (HCC)

D. Autoimmune Hepatitis

Correct Answer: A. Glycogen Storage Disease (GSD)

Explanation: A child with abdominal distension and massive hepatomegaly (8 cm below the costal margin) points to a metabolic or infiltrative cause. Smooth liver suggests uniform enlargement without focal mass or nodularity. In GSD (e.g., Von Gierke disease, Pompe, Cori, Anderson), hepatomegaly results from excessive glycogen accumulation in hepatocytes. Often presents in infancy/early childhood with hypoglycemia, lactic acidosis, hyperlipidemia (depending on subtype), but in some types, the main clue is a large smooth liver . Incorrect Options: Lysosomal Storage Disease (LSD) (Option B): Also causes hepatomegaly, but often with coexisting splenomegaly, coarse facial features, and developmental regression (e.g., Gaucher, Niemann–Pick). Hepatocellular carcinoma (HCC) (Option C): Rare in a young child without pre-existing liver disease; typically causes a firm, irregular nodular liver. Autoimmune hepatitis (Option D): Usually causes mild to moderate hepatomegaly with jaundice, fatigue, elevated transaminases; not massive smooth enlargement.

↑ *Back to this question in Part A*

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Q3: A patient presents with fatigue, anemia, and hepatomegaly. Hemoglobin electrophoresis reveals the presence of HbS. What will be the appropriate treatment? (Q866656)

A. Hydroxyurea

B. Blood transfusion

C. Iron supplementation

D. Vitamin B12 supplementation

Correct Answer: A. Hydroxyurea

Explanation: The presence of HbS confirms Sickle Cell Anemia , and hydroxyurea is the primary treatment as it boosts fetal hemoglobin (HbF) levels, reducing red blood cell sickling and related complications. Cause: Mutation in β-globin gene → production of abnormal hemoglobin S (HbS). Pathophysiology: HbS polymerizes under low oxygen → sickling of RBCs → vaso-occlusion and hemolysis. Symptoms: Anemia, fatigue, pain crises, splenomegaly/hepatomegaly, increased infection risk. Diagnosis: Hemoglobin electrophoresis showing HbS. Treatment: Hydroxyurea: Increases fetal hemoglobin (HbF), reducing sickling episodes. Blood transfusions for severe anemia/crisis. Supportive care: pain management, infection prevention. Incorrect Options: Blood transfusion (Option B) may be used in severe cases but are not the first-line chronic therapy. Iron supplementation (Option C) and Vitamin B12 supplementation (Option D) are not specific treatments for sickle cell anemia.

↑ *Back to this question in Part A*

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Q4: A neonate presents with seizures and is found to have a cherry red spot on fundus examination. Enzyme assay reveals deficiency of hexosaminidase A. Which of the following substances is most likely to be accumulated in this patient? (Q866323)

A. GM1 ganglioside

B. GM2 ganglioside

C. Sphingomyelin

D. Glucocerebroside

Correct Answer: B. GM2 ganglioside

Explanation: A neonate presenting with seizures and a cherry red spot likely has Tay-Sachs Disease, a lysosomal storage disorder caused by hexosaminidase A deficiency, leading to the accumulation of GM2 ganglioside in neurons. Tay-Sachs Disease Inheritance Autosomal recessive Cause Deficiency of hexosaminidase A enzyme Accumulation GM2 ganglioside in ganglion cells and neurons Clinical Manifestations Initial symptoms include loss of head control, inability to sit, exaggerated startle response to sound, and a disproportionately large head. Progressive features include spasticity, deafness, and blindness. Not associated with organomegaly. Fundus Finding Cherry-red spot is caused by GM2 ganglioside accumulation in the ganglion cells of the retina, creating a contrast with the normal red fovea. Incorrect Options: GM1 ganglioside (Option A): Accumulates in GM1 gangliosidosis, caused by β-galactosidase deficiency. Sphingomyelin (Option C): Accumulates in Niemann-Pick disease, caused by sphingomyelinase deficiency. Glucocerebroside (Option D): Accumulates in Gaucher disease,

caused by glucocerebrosidase deficiency.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q5: In uncontrolled diabetes, in which of the following cells there is decreased glucose uptake due to insulin deficiency: (Q865446)

A. RBC

B. Skeletal muscle

C. Liver

D. Brain

Correct Answer: B. Skeletal muscle

Explanation: In uncontrolled diabetes, insulin deficiency leads to decreased glucose uptake in cells that rely on insulin for glucose transport. Skeletal muscle cells are insulin-dependent, meaning they require insulin to facilitate the uptake of glucose via glucose transporters (e.g., GLUT4). In the absence of sufficient insulin, glucose uptake in skeletal muscle decreases, leading to elevated blood glucose levels. RBC (Option A) RBCs do not depend on insulin for glucose uptake. They use GLUT1 transporters, which allow glucose to enter the cells independently of insulin levels. Therefore, their glucose uptake is not significantly affected by insulin deficiency. Liver (Option C) The liver can take up glucose independently of insulin to some extent, especially under the influence of other hormones like glucagon. While insulin does regulate hepatic glucose uptake and production, the primary effect of insulin deficiency in the liver is increased glucose output (e.g., gluconeogenesis), not a significant decrease in uptake. Brain (Option D) Brain cells primarily use GLUT3 and GLUT1 transporters, which are insulin-independent. The brain can continue to take up glucose even in the absence of insulin.

References:

Harper's Illustrated Biochemistry, 31st Edition, Page 576.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: For mycobacterium species differentiation, technique used is/are? Line probe assay MIRU (Mycobacterium Interspersed Rapatitive Units) Spoligotyping RFLP (Q865405)

A. 1, 2, 3

B. 2, 3, 4

C. 1, 2, 4

D. 1, 2, 3, 4

Correct Answer: D. 1, 2, 3, 4

Explanation: All of the listed techniques can be used for Mycobacterium species differentiation , particularly for Mycobacterium tuberculosis complex (MTBC) and non-tuberculous mycobacteria (NTM). TB lab investigations: Investigations Purpose Advantages Limitations Sputum smear Initial screening for pulmonary TB. Samples are stained using the Ziehl-Neelsen method or Auramine-O stain to visualize acid-fast bacilli (AFB) under a microscope. Quick and inexpensive. Can provide results within a few hours. Low sensitivity, especially in cases of extrapulmonary TB or in patients with HIV. Cannot differentiate between Mycobacterium tuberculosis and non-tuberculous mycobacteria (NTM). Culture Methods Confirm the diagnosis of TB and assess drug susceptibility. Solid Media: Sputum is inoculated onto solid culture media like Löwenstein-Jensen or Middlebrook agar. Liquid Media: Automated systems like BACTEC or MGIT are used to detect growth faster. The gold standard for TB diagnosis. Allows for drug susceptibility testing. Time-consuming; results can take weeks. Requires specialized laboratory facilities. Xpert MTB/RIF Detects Mycobacterium tuberculosis and rifampicin resistance. Method : A cartridge-based system that performs nucleic acid amplification (NAAT). Sputum or other specimens are processed directly in the cartridge. Rapid results (within 2 hours). High sensitivity and specificity. Simultaneously detects rifampicin resistance. More expensive than smear microscopy. Does not provide full drug susceptibility testing. LAMP (Loop-Mediated Isothermal Amplification) Detects DNA of Mycobacterium tuberculosis. Method : Amplifies DNA at a constant temperature without the need for thermal cycling. Sputum or other specimens are used. Rapid results (within 1-2 hours). High specificity and sensitivity. Can be performed at the point of care. Used for patients with HIV with CD4+ counts of less than 100 cells/μL irrespective of signs and symptoms of TB. Less widely available than PCR-based tests. Cannot detect drug resistance directly. TrueNat Point-of-care test for TB detection. Method : Uses a micro-PCR technology to amplify DNA. Results are available within 60 minutes. Portable and easy to use in peripheral health facilities. Provides results quickly, facilitating immediate treatment decisions. Line Probe Assay (LPA) (Option 1) Detects TB and drug resistance (rifampicin and isoniazid). Method : Sputum samples are processed, and DNA is extracted. Amplified DNA is hybridized to specific probes on a membrane strip. Rapid results (within 1-2 days). Can detect multiple mutations associated with drug resistance. Requires a laboratory setup and trained personnel. Not suitable for all strains of TB. RFLP (Option 4) IS6110 restriction fragment length polymorphism (RFLP) analysis is widely used for molecular epidemiological studies of tuberculosis. Restriction enzymes Nru I, EcoN I, Pst I, and Pvu II were used to analyze IS6110 fingerprints in Mycobacterium tuberculosis strains H37Rv (virulent) and H37Ra (avirulent). MIRU (Mycobacterial Interspersed Repetitive Units) (Option 2) is a genotyping method based on variable-number tandem repeats (VNTRs) found at specific loci in the Mycobacterium genome. By analyzing the number of repeat units at these loci, MIRU provides a unique fingerprint for different strains. Spoligotyping (Option 3) is a molecular technique that targets the direct repeat (DR) region of the Mycobacterium tuberculosis complex (MTBC) genome. This region consists of short, conserved repeats interspersed with unique spacer sequences. By detecting the presence or absence of these spacers, spoligotyping can differentiate among various strains of MTBC.

References:

Genotyping Mycobacterium tuberculosis isolates with few copies of IS6110: Value of additional genetic markers - ScienceDirect

IS6110-based restriction fragment length polymorphism (RFLP) analysis of Mycobacterium tuberculosis H37Rv and H37Ra - PubMed

TB lab investigations

↑ *Back to this question in Part A*

@Neet_pg_bot

Q7: Which of the following is caused by the deficiency of the enzyme tyrosine transaminase? (Q860430)

A. Type I Tyrosinemia

B. Type II Tyrosinemia

C. Alkaptonuria

D. Type III Tyrosinemia

Correct Answer: B. Type II Tyrosinemia

Explanation: Type II tyrosinemia is caused by a deficiency of tyrosine transaminase (also known as tyrosine aminotransferase), which leads to the accumulation of tyrosine and its metabolites. This condition primarily affects the eyes, skin, and nervous system, with symptoms including corneal ulcers and intellectual disability. Disorders of Tyrosine Metabolism Disorder Deficiency Enzyme Key Clinical Features Tyrosinemia type I (Option A ruled out) Fumarylacetoacetate hydrolase Jaundice, hepatomegaly, hypoglycemia, HCC, liver cirrhosis Often presents with Fanconi syndrome and renal failure Elevated tyrosine and succinylacetone (pathognomic) Tyrosinemia type II (Option B) Tyrosine aminotransferase Painful corneal erosions, palmoplantar hyperkeratosis Also known as oculocutaneous tyrosinemia Elevated tyrosine Tyrosinemia type III (Option D ruled out) p-Hydroxyphenylpyruvate dioxygenase Recurrent seizures, intermittent ataxia Elevated tyrosine and p-hydroxyphenylpyruvate Alkaptonuria (Black urine disease) (Option C ruled out) Homogentisate 1,2-dioxygenase Dark urine, ochronosis, arthritis Black or brown pigmentation of sclera (Osler’s sign) and ear cartilage Elevated homogentisic acid

References:

Harper’s Illustrated Biochemistry, 32nd Edition Pg. 296

Pubmed

↑ *Back to this question in Part A*

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Q8: An infant presents to the Pediatrics OPD with a distended abdomen and jaundice. The mother reports that the baby has shown an increased appetite in recent months. During the clinical examination, the physician notes hepatomegaly. Which of the following enzyme deficiencies is most likely responsible for this condition? (Q860357)

A. Glycogen Synthase

B. Glycogen phosphorylase

C. Glucose-6-Phosphatase

D. Galactose-1-Phosphate Uridyl Transferase

Correct Answer: C. Glucose-6-Phosphatase

Explanation: Glucose-6-Phosphatase deficiency causes Von Gierke disease (Glycogen Storage Disease type I). It leads to the inability to convert glucose-6-phosphate to glucose, causing accumulation of glycogen in the liver and kidney. This results in hepatomegaly, marked abdominal distension, jaundice, chronic hunger (increased appetite), and hypoglycemia. Glycogen synthase (Option A): A deficiency in glycogen synthase leads to Glycogen Storage Disease Type 0 , characterized by inability to store glycogen , leading to hypoglycemia without hepatomegaly or jaundice . Glycogen Phosphorylase (Option B): Causes Hers disease (GSD Type VI), which presents with milder hypoglycemia and hepatomegaly, but no jaundice or severe symptoms . Galactose-1-Phosphate Uridyl Transferase (Option D) : It's deficiency causes Classic Galactosemia which presents with vomiting, cataracts, failure to thrive , and liver failure , not isolated hepatomegaly with increased appetite.

References:

Nelson Textbook of Pediatrics, 21st Edition, Pg. 788 and 789

↑ *Back to this question in Part A*

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Q9: A 1-month-old infant presents with recurrent vomiting, diarrhea, jaundice, and failure to thrive shortly after breastfeeding began. Examination shows hepatomegaly, and urine tests are positive for reducing substances but negative for glucose. What is the most likely diagnosis? (Q860334)

A. Lactose Intolerance

B. Duodenal Atresia

C. Galactosemia

D. Pyloric Stenosis

Correct Answer: C. Galactosemia

Explanation: This presentation is classic for galactosemia, caused by deficiency of galactose-1-phosphate uridyl transferase, leading to accumulation of galactose-1-phosphate in liver, kidneys, and brain. Symptoms begin soon after exposure to breast milk (contains lactose → glucose + galactose). Typical features include vomiting, diarrhea, jaundice, hepatomegaly, failure to thrive, and urine positive for reducing substances but glucose negative, confirming non-glucose sugar excretion. A) Lactose intolerance : Congenital form is rare, does not cause jaundice or hepatomegaly, and usually presents later. B) Duodenal atresia: Presents in first days of life with bilious vomiting, no diarrhea or hepatomegaly. D) Pyloric stenosis : Causes projectile non-bilious vomiting at 3–6 weeks, no jaundice or reducing substances, and no liver involvement.

References:

Nelson Textbook of Pediatrics, 21st Edition, Pg. 788 and 789

https://www.ncbi.nlm.nih.gov/books/NBK532285/

↑ *Back to this question in Part A*

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Q10: An infant presents with distended abdomen, jaundice, increased appetite, and hepatomegaly. Which enzyme deficiency is most likely responsible for this condition? (Q860329)

A. Glucose-6-phosphatase

B. Glycogen phosphorylase

C. Galactose-1-phosphate uridyl transferase

D. Glycogen synthase

Correct Answer: C. Galactose-1-phosphate uridyl transferase

Explanation: This presentation is classic for Galactosemia , caused by deficiency of galactose-1-phosphate uridyl transferase (GALT) . The enzyme is essential for converting galactose-1-phosphate → UDP-galactose , allowing galactose to enter glycolysis. Its deficiency leads to accumulation of galactose-1-phosphate and galactitol in the liver, kidney, and brain. Clinical features include: Jaundice and hepatomegaly (due to hepatic injury) Failure to thrive and hypoglycemia Vomiting after milk intake (lactose → glucose + galactose) Cataracts (from galactitol accumulation) Increased appetite and abdominal distension from hepatomegaly Glucose-6-phosphatase (A): Causes Von Gierke's disease (GSD I) with severe fasting hypoglycemia, lactic acidosis, and hepatomegaly, but no jaundice or cataracts. Glycogen phosphorylase (B): Deficiency leads to Hers disease (GSD VI) — mild hypoglycemia and hepatomegaly, without jaundice. Glycogen synthase (D): Causes GSD 0, presenting with

fasting hypoglycemia and no hepatomegaly.

References:

Harper’s Illustrated Biochemistry (23rd Edition), Page 174.

Vasudevan Textbook of Biochemistry (7th Edition), Page 129.

↑ *Back to this question in Part A*

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Q11: A 7-year-old child of short stature presents with skin patches and learning difficulties. Diagnostic workup indicates a defect in DNA repair specifically related to double-strand breaks. What is the most likely diagnosis? (Q860325)

A. Bloom Syndrome

B. Xeroderma Pigmentosum

C. Cockayne Syndrome

D. Lynch Syndrome

Correct Answer: A. Bloom Syndrome

Explanation: Bloom Syndrome Bloom syndrome is caused by a defect in the BLM gene, which encodes a RecQ helicase essential for DNA repair, particularly double-strand break repair. Clinical Features : Physical: Short stature, narrow face, small jaw, large ears, and high-pitched voice. Skin: Red rash on sun-exposed areas (face, arms, hands) with dilated blood vessels; patches of lighter/darker skin. Development: Fertility and learning difficulties; growth and developmental delays. Infections: Frequent ear and lung infections. Comorbidities: Increased risk of diabetes and chronic obstructive pulmonary disease (COPD). Cancer Risk: Early-onset cancers, especially squamous cell skin cancer, leukemia, lymphoma, and gastrointestinal cancers. Xeroderma pigmentosum (Option B): It is an autosomal recessive disorder characterized by sun sensitivity, with approximately 50% of patients having acute burning on minimal sun exposure. UV radiation causes the cross-linking of pyrimidine residues , preventing normal DNA replication. Nucleotide excision repair (NER) prevents this DNA damage. Inherited loss-of-function mutations in any of these genes give rise to xeroderma pigmentosum. The onset of freckling (lentigines) on sun-exposed skin before age two years in most patients. At an early age, there is a significantly increased risk of sunlight-induced cutaneous neoplasms (basal cell carcinoma, squamous cell carcinoma, melanoma) . Cockayne Syndrome (Option C): Cockayne syndrome is associated with defective transcription-coupled nucleotide excision repair, resulting in photosensitivity, growth failure, and neurodevelopmental abnormalities. Double-strand break repair is not affected. Lynch Syndrome (Option D): Lynch syndrome, also known as hereditary non-polyposis colorectal cancer (HNPCC), involves a defect in mismatch repair genes (MLH1, MSH2, MSH6, PMS2). It is not associated with childhood growth failure or learning difficulties.

References:

https://www.cancer.gov/publications/dictionaries/cancer-terms/def/bloom-syndrome

Robbins and Cotran, Pathologic Basis of Disease, 10th edition, Page no. 314

Fitzpatrick's Dermatology, 9th edition, Page no. 2343, 2347

↑ *Back to this question in Part A*

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Q12: A 16-year-old patient presents with pellagra-like skin eruptions during the spring season for the past 5 years. Laboratory tests reveal normal levels of 5-Hydroxyindoleacetic Acid (HIAA), high aminoaciduria, and normal urinary indole levels. What is the most likely diagnosis? (Q860099)

A. Carcinoid tumor

B. Nutritional pellagra

C. Hartnup disorder

D. Essential fructosuria

Correct Answer: C. Hartnup disorder

Explanation: The patient's presentation is consistent with Hartnup disease (Pellagra-like dermatosis) , a rare inborn error of metabolism. This condition is associated with impaired transport of monoamine monocarboxylic amino acids across the brush border epithelium of the intestine and renal tubules. Hartnup disorder Inheritance Autosomal recessive Pathogenesis Mutation in SLC6A19 gene; Impaired transport of neutral amino acids across intestinal and kidney brush border epithelium Clinical features Photosensitivity Pellagra-like rash (Decreased Niacin due to tryptophan deficiency) Neurologic symptoms (ataxia, emotional instability) - Decreased serotonin due to tryptophan deficiency Biochemical defect Deficiency of nicotinamide synthesis Diagnosis Aminoaciduria (neutral amino acids) in urine and plasma Demonstrate SLC6A19 defect Treatment Administration of nicotinamide/nicotinic acid Protection from sunlight Carcinoid tumor (Option A): Carcinoid tumors cause increased serotonin production, which is metabolized to 5-HIAA and excreted in urine. The normal 5-HIAA levels in this patient rule out carcinoid syndrome. Nutritional pellagra (Option B): Nutritional pellagra is caused by niacin (vitamin B3) deficiency and is characterized by the "4 Ds": diarrhea, dermatitis, dementia, and death. However, this patient has high aminoaciduria, which is not typical of nutritional pellagra. Essential fructosuria (Option D): It is caused by a deficiency of fructokinase, leading to the excretion of unmetabolized fructose in the urine. It does not cause pellagra-like symptoms or high aminoaciduria.

References:

Nelson Textbook of Paediatrics Edition 21st Pg. 707, 708, 3501

↑ *Back to this question in Part A*

Q13: Which of the following substances is used in a tube for collecting blood samples specifically for measuring alcohol concentration? (Q860092)

A. Heparin + EDTA

B. Sodium fluoride + Potassium dichromate

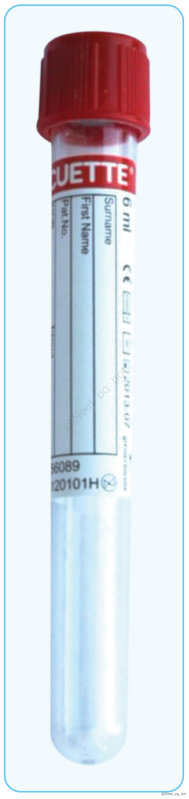
C. Heparin

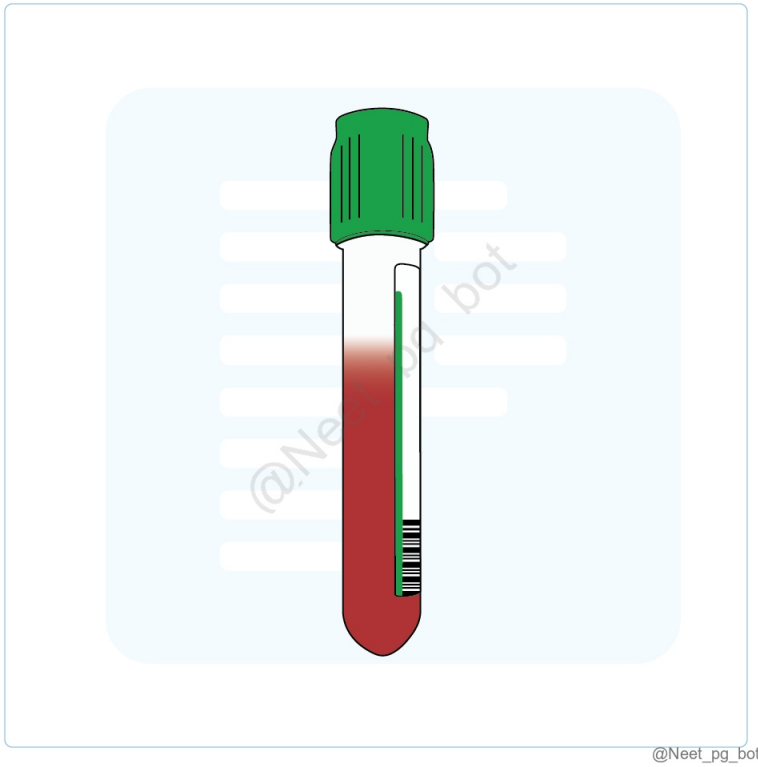
D. Sodium citrate

Correct Answer: B. Sodium fluoride + Potassium dichromate

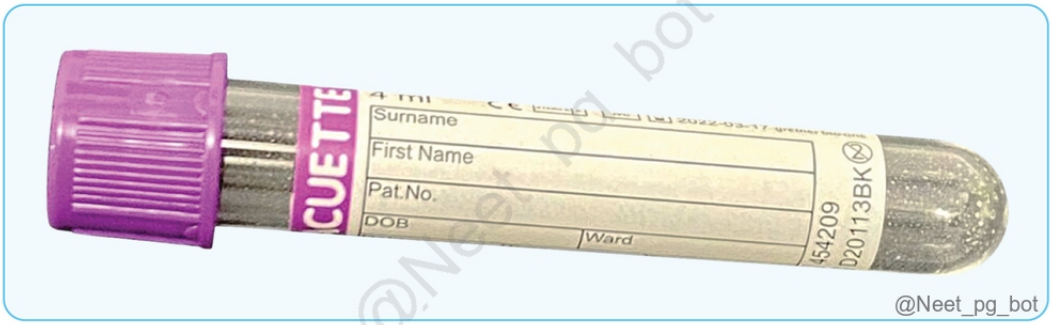
Explanation: Sodium fluoride is an antiglycolytic agent that inhibits the breakdown of glucose, and Potassium dichromate is a reagent that stabilizes the sample for accurate alcohol concentration measurement. This combination is specifically used for alcohol testing because it prevents the metabolism of alcohol in the sample. Blood Collection Tubes Tube Type Additives & Mechanism Key Applications Coagulation Tube {{caption_text}} Sodium citrate binds calcium (Factor IV) to prevent clotting; anticoagulation reversed by adding calcium. Used for coagulation studies. Must be filled completely. Serum Tube {{caption_text}} Glass/silica activates clotting (Factor XII) . Some include gel separators to prevent cellular contamination. Used for clinical chemistry, immunology, and serology. Heparin Tube Sodium/lithium heparin inhibits thrombin and Factor Xa to prevent clotting. Common in biochemical tests. EDTA Tube {{caption_text}} EDTA chelates calcium irreversibly, preventing clotting. Preferred for hematology. Stable for enzymatic studies. Glycolytic Inhibitor Tube {{caption_text}} Sodium fluoride inhibits glycolysis; may also include citrate, oxalate, or EDTA to prevent clotting. Used to measure glucose and lactate levels. Heparin + EDTA (Option A): Both heparin and EDTA are anticoagulants , they are not specifically used for preserving blood samples for alcohol concentration measurement. They lack the ability to inhibit glycolysis, which can alter alcohol levels in the sample. Heparin (Option C) : Heparin is an anticoagulant used to prevent blood clotting, but it is not suitable for alcohol concentration tests as it does not preserve the sample for this specific purpose. Sodium citrate (Option D) : Sodium citrate is an anticoagulant used mainly for coagulation studies, but it is not used for measuring blood alcohol concentration.


BD Vacutainer®
Buff. Na Citrate
0.109M, 3.2%
REF 363083
LOT 21241322 2013-02
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Surname
First Name
6 ml
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References:

https://www.ncbi.nlm.nih.gov/books/NBK555991/

↑ *Back to this question in Part A*

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Q14: A 45-year-old man presents with deep labored breathing and altered sensorium. His arterial blood gas shows a metabolic acidosis. The following serum electrolyte values are obtained: Na■: 145 mmol/L K■: 4 mmol/L Cl■: 90 mmol/L HCO■■: 15 mmol/L Calculate the anion gap (AG) and interpret the finding. (Q854606)

A. 16 mmol/L – Normal anion gap metabolic acidosis

B. 40mmol/L – High anion gap metabolic acidosis

C. 14 mmol/L – Normal anion gap metabolic acidosis

D. 32 mmol/L – Mixed metabolic acidosis

Correct Answer: B. 40mmol/L – High anion gap metabolic acidosis

Explanation: Formula: Anion Gap (AG)=(Na++K+)–(Cl–+HCO3–) AG=(145+4)–(90+15)=149–105=44 mmol/L (Note: K■ is often omitted in clinical practice — if omitted, AG = 145 - (90 + 15) = 40 mmol/L, still markedly elevated.) A normal AG is approximately 8–12 mmol/L (without K■). Here, AG ≈ 40–44 mmol/L, indicating a high anion gap metabolic acidosis (HAGMA). Causes of High Anion Gap Metabolic Acidosis (Mnemonic: MUDPILES): M – Methanol U – Uremia (renal failure) D – Diabetic ketoacidosis P – Paraldehyde / Propylene glycol I – Isoniazid / Iron L – Lactic acidosis E – Ethylene glycol S – Salicylate toxicity

References:

Vasudevan’s Textbook of Biochemistry for Medical Students, 7th edition, page 399

↑ *Back to this question in Part A*

@Neet_pg_bot

Q15: Which of the following disorders is characterized by the presence of a cherry red spot on the retina? (Q860068)

A. Tay-Sachs disease

B. Fabry's disease

C. Gaucher's disease

D. Von Gierke's disease

Correct Answer: A. Tay-Sachs disease

Explanation: Tay-Sachs disease is a lysosomal storage disorder characterized by progressive neurodegeneration, developmental delay, and a cherry-red spot on the retina. Tay-Sachs Disease Inheritance Autosomal recessive Cause Deficiency of hexosaminidase A enzyme Accumulation GM2 ganglioside in ganglion cells and neurons Clinical Manifestations Initial symptoms include loss of head control, inability to sit, exaggerated startle response to sound, and a disproportionately large head. Progressive features include spasticity, deafness, and blindness. Not associated with organomegaly. Fundus Finding Cherry-red spot is caused by GM2 ganglioside accumulation in the ganglion cells of the retina, creating a contrast with the normal red fovea. Fabry's disease (Option B): Fabry's disease is caused by a deficiency of alpha-galactosidase A, leading to the accumulation of globotriaosylceramide. It presents with angiokeratomas, neuropathic pain, and renal involvement but does not typically feature a cherry-red spot on the retina. Gaucher's disease (Option C): Gaucher's disease is due to glucocerebrosidase deficiency, causing glucocerebroside accumulation in macrophages. It presents with hepatosplenomegaly, bone pain, and cytopenias but is not associated with a cherry-red spot. Von Gierke's disease (Option D): Von Gierke's disease (glycogen storage disease type I) results from a deficiency of glucose-6-phosphatase. It causes severe fasting hypoglycemia, hepatomegaly, and lactic acidosis but does not involve retinal findings.

References:

Ghai Essential Pediatrics, 10th Edition, Pg.693

Tay-sach disease

https://www.ncbi.nlm.nih.gov/books/NBK564432/#:~:text=Tay%20Sachs%20disease%20manifests%20in,as%20one%20week%20of%20life

↑ *Back to this question in Part A*

@Neet_pg_bot

Q16: A 40-year-old female working as a senior executive leading a sedentary lifestyle presents to the OPD with palmoplantar pigmentation. X-ray spine shows calcification of intervertebral discs. On adding Benedict's reagent to the urine sample, it gives a greenish-brown precipitate and comes positive. What is the most likely enzyme deficiency in this patient? (Q860064)

A. Phenylalanine hydroxylase

B. Tyrosine hydroxylase

C. Homogentisic acid oxidase

D. PHPP dioxygenase

Correct Answer: C. Homogentisic acid oxidase

Explanation: The patient is most likely suffering from alkaptonuria, a condition caused by homogentisic acid oxidase deficiency. This leads to the accumulation of homogentisic acid, which is excreted in the urine. When exposed to air, the urine darkens, and a positive Benedict's test (which detects reducing sugars) can produce a greenish-brown precipitate. The palmoplantar pigmentation and intervertebral disc calcification (also known as ochronosis) are characteristic of this condition. Alkaptonuria Definition Rare autosomal recessive genetic disorder caused by a deficiency of homogentisate 1,2 dioxygenase (HGD) , an enzyme in the tyrosine metabolism pathway. Mechanism and Pathophysiology Defective tyrosine metabolism → homogentisic acid (HGA) accumulation → ochronotic pigment deposition → systemic disease (ochronosis). Deficiency: HGD fails to convert HGA to maleylacetoacetate. Consequences: HGA oxidizes into melanin-like polymers → connective tissue deposition (ochronosis). Affects joints, heart valves, kidneys, and vessels. Clinical Features Triad: Homogentisic aciduria, ochronosis, ochronotic osteoarthropathy. Pediatric signs: Dark urine (oxidized HGA). Musculoskeletal : Arthritis (spine, hips, knees), kyphoscoliosis, spinal stenosis. Cardiac : A ortic stenosis, mitral valve disease, coronary artery disease. Renal/Metabolic: Stones (renal, gallbladder, prostate). Neurological : Peripheral neuropathy, stroke. Evaluation Clinical scoring: AKU Severity Score Index (AKUSSI). Diagnostic tests: Gold standard: Urinary HGA quantification via gas chromatography-mass spectrometry. Genetic testing for HGD mutations. Imaging (CT/MRI): Joint involvement, valvular calcification. Echocardiography: Valvular abnormalities. Management Symptomatic : Pain control, physiotherapy, joint surgery. Diet : Low-protein diet (limited efficacy). Medications: Vitamin C : Reduces HGA oxidation but not urinary excretion. Nitisinone : Inhibits tyrosine metabolism, reduces HGA >95%. Side effects: tyrosinemia, leukopenia, thrombocytopenia. Prognosis and Complications Prognosis : Normal life expectancy; reduced quality of life (pain, sleep issues, disability). Complications : Stones (renal,

gallbladder, salivary). Tendon/ligament ruptures. Osteopenia, fractures. Aortic calcification/stenosis. Phenylalanine hydroxylase (Option A): Phenylalanine hydroxylase deficiency causes phenylketonuria (PKU), which is associated with intellectual disability, seizures, and a musty odor. The symptoms described here are not typical of PKU. Tyrosine hydroxylase (Option B): Tyrosine hydroxylase deficiency is associated with a condition called hereditary tyrosinemia type 2, which can cause eye and skin problems but does not present with the urine color change or intervertebral disc calcification seen in this case. PHPP dioxygenase (Option D): Deficiency in 4-hydroxyphenylpyruvate dioxygenase causes type III tyrosinemia, which is associated with elevated levels of tyrosine in the blood and urine, but does not typically present with palmoplantar pigmentation or calcification of intervertebral discs.

References:

Vasudevan Textbook of Biochemistry (7th Edition), Page 237, 238

https://www.ncbi.nlm.nih.gov/books/NBK560571/

Harper's Illustrated Biochemistry, 32nd Edition, Page 296, 304

https://pubmed.ncbi.nlm.nih.gov/10942115/#:~:text=Tyrosinemia%20type%20III%20(OMIM%20276710,of%20tyrosine%20and%20phenolic%20metabolites.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q17: A 3-month-old baby presents with severe hepatomegaly, cataracts in both eyes, lethargy, and hypotonia. Based on these symptoms, which enzyme deficiency is most likely involved? (Q860054)

A. Galactokinase

B. Hepatic Kinase

C. Hepatic Glucose-6-Phosphatase

D. Galactose-1-Phosphate Uridyl Transferase

Correct Answer: D. Galactose-1-Phosphate Uridyl Transferase

Explanation: Galactose-1-Phosphate Uridyl Transferase deficiency causes classic galactosemia, a severe metabolic disorder characterized by hepatomegaly, cataracts, lethargy, and hypotonia due to the accumulation of toxic metabolites like galactose-1-phosphate and galactitol. Galactosemia Enzyme Deficiency Galactose-1-phosphate uridylyltransferase (GALT) - Classical type Galactokinase- benign disease with only oil drop cataract. (Option A ruled out) Epimerase- SNHL + symptoms of classical type (Low galactose-based diet can be given which is contraindicated in the classical type) Pathophysiology Accumulation of galactose-1-phosphate (toxic causing jaundice, and liver damage) Inability to metabolize galactose from milk Damage to liver, kidney, and brain Clinical Features Hepatic: Jaundice, hepatomegaly GI: Vomiting, diarrhea, failure to thrive CNS: Seizures,

intellectual disability Eyes: Oil drop cataracts Increased risk of E. coli sepsis Diagnosis Reducing substance in urine (positive)(Positive Benedict test but negative glucose oxidase test) Direct enzyme (GALT) assay Genetic testing (Perinatal diagnosis is possible) Management Lactose-free diet Breast Milk contraindicated Soy-based or elemental formulas Calcium supplementation Hepatic Glucose-6-Phosphatase (Option C) : Deficiency of it causes glycogen storage disease type I (Von Gierke disease), which presents with hepatomegaly, hypoglycemia, and lactic acidosis. Cataracts are not a feature of this condition.

References:

Nelson Textbook of Pediatrics, 21st Edition, Pg. 788 and 789

↑ *Back to this question in Part A*

@Neet_pg_bot

Q18: A couple brings their 4-year-old child to the outpatient department (OPD) due to self-mutilating behavior and poor growth. Which of the following associated enzyme deficiencies could explain these symptoms? (Q860052)

A. HGPRT deficiency

B. Homogentisic Aciduria

C. Phenylalanine Hydroxylase deficiency

D. Adenosine deaminase deficiency

Correct Answer: A. HGPRT deficiency

Explanation: Hypoxanthine-guanine phosphoribosyltransferase (HGPRT) deficiency causes Lesch-Nyhan syndrome, a rare X-linked recessive disorder. It is characterized by self-mutilating behavior, hyperuricemia, gout, neurological abnormalities, and poor growth. HGPRT is critical for p urine salvage , and its deficiency leads to an overproduction of uric acid. Lesch-Nyhan Syndrome Clinical Features Metabolic: Severe gout, sodium urate crystal deposition causing j oint swelling and kidney damage . Neurological: Poor muscle control. Involuntary movements (resembling Huntington's disease). Facial grimacing, repetitive limb movements. Behavioral: Self-mutilation (lip and finger biting, starting ~2nd year). Other: Moderate developmental delays , potential megaloblastic anemia (from poor vitamin B12 utilization). Management Metabolic: Allopurinol for gout; lithotripsy for kidney stones. Neurological: Symptomatic treatment with drugs like carbidopa/levodopa, diazepam, phenobarbital, or haloperidol. Homogentisic Aciduria (Option B): Homogentisic aciduria, or alkaptonuria, results from homogentisate 1,2-dioxygenase deficiency. It leads to darkened urine, ochronosis (dark pigmentation of connective tissues), and arthritis but does not cause self-mutilation or poor growth. Phenylalanine Hydroxylase deficiency (Option C): Phenylalanine hydroxylase deficiency causes phenylketonuria (PKU), which presents with intellectual disability, seizures, and hypopigmentation. Self-mutilation and hyperuricemia are not associated with this condition. Adenosine deaminase deficiency (Option D): A rare genetic disorder caused by ADA gene mutations, leading to a lack of B and T lymphocytes and severe immune deficiency. Symptoms, often appearing before 6 months, include recurrent infections, chronic diarrhea, rashes, and delayed growth. Without treatment,

life-threatening infections and certain cancers may occur.

References:

https://www.ninds.nih.gov/health-information/disorders/lesch-nyhan-syndrome

Vasudevan Textbook of Biochemistry (7th Edition), Page 237, 238

Harper’s Illustrated Biochemistry, 32nd Edition, Pg 334-336

https://www.cancer.gov/publications/dictionaries/cancer-terms/def/adenosine-deaminase-deficient-severe-combined-immunodeficiency

↑ *Back to this question in Part A*

@Neet_pg_bot

Q19: Zellweger syndrome is associated with which cellular organelle? (Q860043)

A. Peroxisomes

B. Nucleosomes

C. Lysosomes

D. Ribosomes

Correct Answer: A. Peroxisomes

Explanation: Zellweger syndrome is a peroxisomal biogenesis disorder caused by mutations in PEX genes. It results in defective peroxisome formation, leading to an inability to metabolize very long-chain fatty acids (VLCFAs) and plasmalogens. Clinical features include hypotonia, seizures, hepatomegaly, and craniofacial abnormalities. Zellweger Spectrum Disorder Rare autosomal recessive disorder with defective peroxisome biogenesis affecting VLCFA metabolism. Includes Zellweger syndrome, neonatal adrenoleukodystrophy, infantile Refsum disease, and rhizomelic chondrodysplasia punctata. Etiology Caused by mutations in PEX genes encoding peroxins, primarily PEX1/PEX6 Functional peroxisomal disturbances: Zellweger spectrum: Generalized peroxisomal dysfunction. Adrenoleukodystrophy: Mutations in ABCD1, impacting VLCFA transport. Rhizomelic chondrodysplasia punctata: Multiple enzymatic defects. Pathophysiology Peroxisomes: Membrane-bound organelles with >50 enzymes for fatty acid metabolism, abundant in liver/kidney. Mutated PEX genes disrupt peroxisome formation, leading to VLCFA accumulation, reduced steroid biosynthesis, and neuronal damage. Major abnormalities: Brain: Demyelination, neuronal injury. Liver: Fibrosis. Kidney: Cortical cysts. Clinical Features Multisystem involvement with severity depending on age of onset: Neonatal-Infantile: Hypotonia, feeding issues, seizures, facial dysmorphism, ocular and hearing abnormalities, hepatomegaly. Childhood: Developmental delay, FTT, hepatic dysfunction, adrenal insufficiency, neuroregression. Adolescent-Adult: Neuroregression, ataxia, neuropathy, adrenal insufficiency, leukodystrophy. Diagnosis Initial: Clinical features + elevated VLCFA on newborn screening. Biochemical tests: Elevated VLCFA, bile acid intermediates, and phytanic/pristanic acid; reduced plasmalogens. Genetic testing: Confirms PEX mutations. Prenatal diagnosis and

genetic counseling recommended. Management No cure; supportive care focuses on improving quality of life. Therapies : Docosahexaenoic acid Lorenzo oil: Reduces VLCFA but doesn't halt disease progression. Cholic acid : Approved for hepatic dysfunction. Supportive Measures: Hearing aids for hearing loss. Ophthalmologic care for vision issues. Antiepileptics for seizures. Vitamin supplementation: Fat-soluble vitamins (A, D, E, K). Cortisone for adrenal insufficiency. Gastrostomy for feeding difficulties. Nucleosomes (Option B): Disorders involving nucleosomes are linked to chromatin remodeling or epigenetic regulation, not Zellweger syndrome. Lysosomes (Option C): Lysosomal storage diseases (e.g., Tay-Sachs disease, Gaucher disease) result from enzyme deficiencies affecting lysosomal function. Zellweger syndrome does not involve lysosomes. Ribosomes (Option D): Diseases involving ribosomes include ribosomopathies like Diamond-Blackfan anemia, which are unrelated to Zellweger syndrome.

References:

https://www.ncbi.nlm.nih.gov/sites/books/NBK560676/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q20: On centrifugation of a cell, LDH will get deposited in: (Q859706)

A. Mitochondria

B. Lysosome

C. Cytoplasm

D. Golgi complex

Correct Answer: C. Cytoplasm

Explanation: Lactate dehydrogenase (LDH) is primarily located in the cytoplasm. The enzyme exists in various isoforms, with LDH-5 being the most abundant in the cytoplasm. About 99% of LDH activity is found in the cytoplasm, where it plays a critical role in the conversion of lactate to pyruvate and vice versa, part of the cell's metabolic processes. While a small fraction of LDH can be found in the nucleus (involved in DNA binding and transcription), the vast majority of LDH activity occurs in the cytoplasm. Mitochondria (Option A): LDH is not primarily located in the mitochondria. Mitochondria contain enzymes like pyruvate dehydrogenase and enzymes involved in the citric acid cycle, but LDH, which is involved in anaerobic metabolism, is predominantly found in the cytoplasm. Lysosome (Option B): LDH is not found in significant amounts in the lysosome. Lysosomes are involved in cellular digestion and degradation processes, and while some enzymes may be active there, LDH is not typically one of them. It is primarily cytoplasmic Golgi complex (Option D) is responsible for modifying, sorting, and packaging proteins for secretion or use within the cell, but it is not a location where LDH is predominantly found. LDH is mainly active in the cytoplasm and plays its role in energy metabolism rather than processing or transporting cellular components like the Golgi.

References:

Phosphotyrosine-containing Lactate dehydrogenase - PubMed

Transport-exclusion pharmacology to localize lactate dehydrogenase activity within cells

↑ *Back to this question in Part A*

@Neet_pg_bot

Q21: Which of the following enzyme defects leads to Pompe's disease? (Q857351)

A. Lysosomal alpha glucosidase

B. β-hexosaminidase -A

C. β-galactocerebrosidase

D. β-glucocerebrosidase

Correct Answer: A. Lysosomal alpha glucosidase

Explanation: Pompe's Disease (Glycogen storage disease Type II) Enzyme Deficiency Lysosomal α1→4 and α1→6 glucosidase (acid maltase) Affected Gene GAA Clinical Features Muscle hypotonia, death due to heart failure within 2 years of age (seen in juvenile) Muscle dystrophy (seen in adults) Mode of Inheritance Autosomal recessive Management Supportive care Enzyme replacement therapy Chest x-ray at regular intervals Obtain an initial echocardiogram to evaluate the extent of cardiomyopathy Medical management may be useful based on the stage of cardiomyopathy. Avoid drastic changes in fluid status, either through dehydration or fluid overload.

References:

Harper's Biochemistry 32e pg: 174

Vasudevan Textbook of Biochemistry (7th Edition), Page 129.

Pompe disease diagnosis and management guideline - PMC

↑ *Back to this question in Part A*

@Neet_pg_bot

Q22: Xanthurenic aciduria may be treated with high doses of which vitamin? (Q857350)

A. Vitamin B6

B. Vitamin B12

C. Vitamin B5

D. Vitamin B7

Correct Answer: A. Vitamin B6

Explanation: The metabolic disorders which respond to vitamin B6 therapy are: Xanthurenic aciduria Homocystinuria Vitamin B6 (Pyridoxal Phosphate - PLP): Amino Acid Metabolism PLP acts as a coenzyme for many reactions in amino acid metabolism. Transamination Catalyzed by amino transferases (e.g., Alanine transaminase), converting amino acids to keto acids. Decarboxylation Required for decarboxylation of amino acids, forming important neurotransmitters and metabolites. Neurotransmitter Synthesis GABA from glutamate (important for inhibitory neurotransmission). Histamine from histidine (mediator of allergy). Serotonin from 5-hydroxy tryptophan (mood regulation). Taurine from cysteine. Sulfur Containing Amino Acids Involved in methionine and cysteine metabolism. Homocysteine Metabolism Converts homocysteine to cystathionine; deficiency leads to increased homocysteine, associated with myocardial infarction risk. Heme Synthesis Required for ALA synthase, the rate-limiting enzyme in heme biosynthesis. Niacin Production Required for the conversion of tryptophan to niacin (via kynurenine). Glycogenolysis PLP is a coenzyme for phosphorylase, which converts glycogen to glucose-1-phosphate.

References:

Vasudevan’s Textbook of Biochemistry for Medical Students, 7th edition, page 483

↑ *Back to this question in Part A*

@Neet_pg_bot

Q23: Deficiency of which of the following causes Barth’s syndrome? (Q857325)

A. Cardiolipin

B. Sphingomyelin

C. Glycolipids

D. Cerebroside

Correct Answer: A. Cardiolipin

Explanation: Barth’s syndrome: Cause Mutations affecting cardiolipin metabolism (decreased or altered cardiolipin levels), causing defects in mitochondrial function. Clinical Features Cardioskeletal myopathy (affects both heart and muscle). Mitochondrial dysfunction in aging and pathologic conditions.

References:

Harper’s Biochemistry 32nd edition, page 211

↑ *Back to this question in Part A*

@Neet_pg_bot

Q24: Liver produces ketones but cannot use it due to the deficiency of which of the following? (Q857321)

A. Thiophorase

B. Alkaline phosphatase

C. Alanine transaminase

D. Thoiolase

Correct Answer: A. Thiophorase

Explanation: Liver cannot use ketone bodies due to the deficiency of Thiphorase KETOSIS Ketone Bodies Produced in the liver Utilized by extrahepatic tissues due to the absence of Thiophorase enzyme in the liver Tissues that use Ketone Bodies Almost all tissues except: Liver Red blood cells (RBCs) Tissues that prefer ketone bodies: Heart muscle Renal cortex Placenta Tissues that use ketone bodies only in the absence of glucose: Skeletal muscle Brain Additional Tissues Using Ketone Bodies Intestinal mucosal cells Adipocytes Enzyme Involved Thiophorase : Converts acetoacetate to acetoacetyl CoA Fatty Acids as Primary Energy Source During Starvation Skeletal muscles Heart Liver Energy Production Pathway Acetoacetyl CoA enters the beta-oxidation pathway to produce energy

References:

Vasudevan Biochemistry 7th edition. Pg.164

Harper Biochemistry 32nd edition. Pg. 221

↑ *Back to this question in Part A*

@Neet_pg_bot

Q25: Identify the test: (Q854626)

A. Rothera’s test

B. Benedict’s test

C. Fehling’s test

D. Barfoed’s test



Correct Answer: A. Rothera’s test

Explanation: Rothera's test: The presence of ketosis can be established by the detection of ketone bodies in urine by Rothera's test. Method-Saturate 5 mL of urine with solid ammonium sulfate. Add a few drops of freshly prepared sodium nitroprusside followed by 2 mL of liquor ammonia along the sides of the test tube. Development of a purple ring indicates the presence of ketone bodies in urine.

References:

Vasudevan’s Textbook of Biochemistry for Medical Students, 7th Edition, Page 166

↑ *Back to this question in Part A*

@Neet_pg_bot

Q26: What happens to chloride levels in sweat and pancreatic secretions in cystic fibrosis? (Q854625)

A. Chloride is raised in sweat

B. Chloride is decreased in sweat

C. Chloride is decreased in pancreatic secretions

D. None of the above

Correct Answer: A. Chloride is raised in sweat

Explanation: In cystic fibrosis, CFTR channels are defective. In sweat glands, CFTR normally reabsorbs chloride so its dysfunction leads to high chloride in sweat . In pancreatic ducts, CFTR normally secretes chloride and bicarbonate so its dysfunction causes low chloride and thick secretions , contributing to duct obstruction and pancreatic insufficiency. Cystic fibrosis: Inheritance Autosomal recessive disorder. Genetic Cause Mutations in the CFTR gene (cystic fibrosis transmembrane regulator). CFTR Function Cyclic AMP-regulated chloride (Cl^-) transporter. Symptoms Chronic bacterial infections in airways and sinuses. Fat maldigestion due to pancreatic exocrine insufficiency. Infertility in males due to abnormal development of the vas deferens. Diagnostic Feature Elevated chloride levels in sweat (>60 mmol/L). Note: Chloride decreased in pancreatic secretions (Option C) TRUE, but not the best single answer.

References:

Vasudevan’s Textbook of Biochemistry for Medical Students, 7th edition, page 486

↑ *Back to this question in Part A*

@Neet_pg_bot

• [15_Biochemistry] 036 Previous Year Questions

« Back to Folder Directory

[15_Biochemistry] 036 Previous Year Questions — Quick Jump

Q1 (p. 1391) *Q2 (p. 1391)*

Q1: In a case presented in the outpatient department (OPD), a patient reported experiencing abdominal pain and diarrhea. Upon further examination, the patient exhibited symptoms of cutaneous flushing, extensive wheezing, and tricuspid insufficiency. Subsequent contrast enhanced MRI scans indicated the presence of endocardial fibrosis. As a result of this condition, which substance is likely to be excreted excessively by the patient? (Q793465)

A. Vanillylmandelic acid

B. Imidazoleacetic acid

C. 5-hydroxy indole acetic acid

D. Choline

Correct Answer: C. 5-hydroxy indole acetic acid

Explanation: Nowadays, carcinoid tumor incidence is rising. The major clinical symptoms include flushing, diarrhea, bronchospasm as well as carcinoid heart disease. Expression of its amine or hormones is variable, so it makes diagnosis of disease late or erroneous. 5-Hydroxy indoleacetic acid (5-HIAA) is considered as important biomarkers for the disease, while Chromogranin A(CgA), pancreatic polypeptide are also used as non-specific biomarkers for the disease. Primary metabolic substrate of serotonin is 5-Hydroxy indoleacetic acid (5-HIAA). Serotonin gets broken into 5-hydroxy indoleacetic acid (5-HIAA) within the liver. Incorrect Choices: a. Neuroblastoma are diagnosed by the presence of urinary vanillylmandelic acid and homo vanillic acid (HVA) as biomarkers. b. Breast tumors are caused by imidazole-acetic acid and its derivatives. d. Dietary intake of choline in excess can cause risk of colon and rectum cancer.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: The best method for the detection of mutations with low allele frequency is: (Q855560)

A. Droplet digital PCR

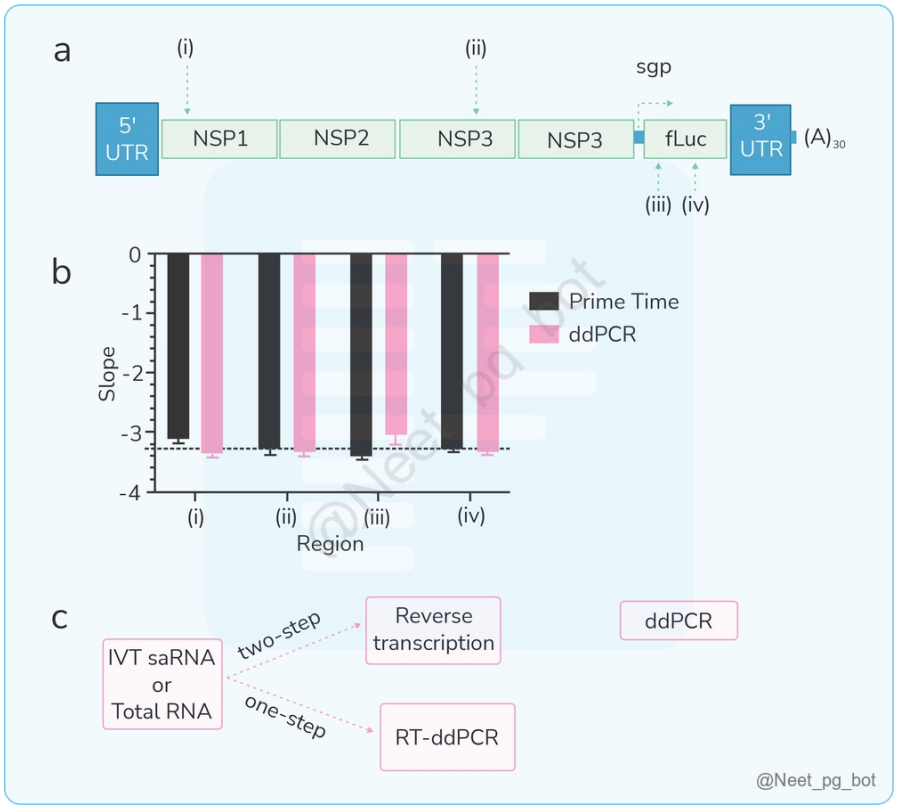
B. FISH

C. Nested PCR

D. Sanger sequencing

Correct Answer: A. Droplet digital PCR

Explanation: Droplet Digital PCR (ddPCR) is a highly sensitive quantitative technique for detecting mutations with very low allele frequencies. It partitions a sample into thousands of droplets, each containing a single DNA molecule. PCR is performed on each droplet, and fluorescent probes detect the presence or absence of the mutation. Specialized equipment analyzes the droplets to count fluorescent signals, determining mutation frequency. ddPCR is highly accurate and valuable for detecting low-frequency mutations in clinical samples. {{caption_text}} Learning Objective: Assay Type Advantages Main Limitations Real-time PCR Rapid results Cost effectiveness Limited number of DNA sequences Digital droplet PCR High sensitivity (detection of low allele frequency,0.005-0.1) Limited number of DNA sequences Next-generation sequencing Larger number of DNA sequences. Detection of coexisting mutations. Higher allele frequency required compared to ddPCR (0.1%). Higher costs More complex data analysis and interpretation.



References:

pmc.ncbi.nlm.nih.gov/articles/PMC8013307/

↑ *Back to this question in Part A*

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• [16_Concepts] 037 Previous Year Questions

« Back to Folder Directory

[16_Concepts] 037 Previous Year Questions — Quick Jump

Q1 (p. 1394) *Q2 (p. 1395)* *Q3 (p. 1395)* *Q4 (p. 1396)* *Q5 (p. 1397)* *Q6 (p. 1397)* *Q7 (p. 1398)*

Q1: Cholera is known to cause diarrhea through its interaction with specific receptors in the human body. Which of the following is known to make it easier for cholera toxin to enter the body through the intestinal mucosa? (Q820337)

A. GLUT 1

B. GM2 ganglioside

C. Cephalin

D. GM1 ganglioside

Correct Answer: D. GM1 ganglioside

Explanation: Correct Option D - GM1 ganglioside: Cholera toxin is produced by the bacterium Vibrio cholerae and is responsible for causing the characteristic symptoms of cholera, including severe diarrhea. Cholera toxin enters the body through the intestinal mucosa and interacts with specific receptors on the surface of intestinal cells. The receptor that facilitates the entry of cholera toxin into the body is the GM1 ganglioside receptor. GM1 ganglioside is a glycosphingolipid, a type of molecule found in the lipid bilayer of cell membranes. Cholera toxin binds to GM1 ganglioside on the surface of intestinal cells, which triggers the toxin to be internalized into the cell through endocytosis. Once inside the cell, the toxin can exert its effects, leading to the disruption of ion transport and fluid balance, ultimately causing severe diarrhea. Option A - GLUT1 (Glucose Transporter 1): GLUT1 is a protein responsible for transporting glucose across cell membranes. It is not directly involved in the interaction between cholera toxin and intestinal cells. Option B - GM2 ganglioside: While GM2 ganglioside is a type of glycosphingolipid, it is not the receptor specifically associated with cholera toxin binding and entry into intestinal cells. Option C - Cephalin: Cephalin is a type of phospholipid and is not known to be directly involved in the interaction between cholera toxin and intestinal cells.

References:

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↑ *Back to this question in Part A*

@Neet_pg_bot

Q2: Which of the following minerals is best known for insulin secretion stabilisation? (Q820373)

A. Cadmium

B. Copper

C. Iron

D. Zinc

Correct Answer: D. Zinc

Explanation: Correct Option D - Zinc: Zinc is an essential trace mineral that plays a crucial role in numerous biological processes, including insulin secretion and stabilization. It is involved in the synthesis, storage, and release of insulin from pancreatic beta cells. Zinc also helps in maintaining the structure and stability of insulin molecules. Therefore, of the options provided, zinc is the best-known mineral for insulin secretion stabilization. Incorrect Options: Option A - Cadmium: Cadmium is a toxic heavy metal that can have detrimental effects on health, including disrupting various physiological processes. However, it is not known for its role in insulin secretion stabilization. Option B - Copper: Copper is an essential trace mineral that plays a role in various bodily functions, including the formation of red blood cells and maintaining connective tissues. While it is involved in several enzymatic reactions, it is not primarily known for its role in insulin secretion stabilization. Option C - Iron: Iron is vital for oxygen transport in the blood and is a key component of hemoglobin. It is not primarily associated with insulin secretion stabilization.

↑ *Back to this question in Part A*

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Q3: A 13-year-old boy who accompanied his father to a fish farming region in his hometown accidentally consumes a white odourless chemical. He was under some medication and post-meal he developed toxicity of the drug. Which of the the following toxin is most likely to cause this condition? (Q820415)

A. Alkyl guanides

B. BAL

C. Antimycin A

D. Strychnine

Correct Answer: C. Antimycin A

Explanation: Antimycin A Antimycin A is commonly used in fish farming as a piscicide (a chemical toxic to fish) to selectively remove undesirable fish species. It is a colorless to white crystalline compound and is relatively odorless. It is a potent respiratory chain uncoupler. When consumed, it inhibits cellular respiration. It is a mitochondrial toxin that inhibits Complex III of the electron transport chain. This inhibition leads to decreased ATP production and increased oxidative stress. This can amplify the toxicity of some medications- especially those metabolized via oxidative pathways (e.g. anticonvulsants, antibiotics, or chemotherapeutics). Alkyl guanides (Option A) are a broad class of compounds, some of which can be toxic, but not specifically associated with fish farming or the described scenario. Also, they aren't primarily known for causing drug toxicity by interfering with metabolism in the same direct way as Antimycin A. BAL (British Anti-Lewisite; Dimercaprol) (Option B): This is a chelating agent , not a toxin, and is used as an antidote for heavy metal poisoning. Strychnine (Option D): It's a plant-derived neurotoxin (not used in fish farming). It causes convulsions via spinal inhibition of glycine , not mitochondrial toxicity. It does not primarily cause drug toxicity by interfering with drug metabolism in the way Antimycin A does. Its symptoms would be distinctly neurological, not primarily drug accumulation.

References:

.https://pmc.ncbi.nlm.nih.gov/articles/PMC6441541/ https://pmc.ncbi.nlm.nih.gov/articles/PMC1217497/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q4: In which of the following locations can cardiolipin be found? (Q830710)

A. Outer mitochondrial membrane

B. Inner mitochondrial membrane

C. Cytosol

D. Mitochondrial matrix

Correct Answer: B. Inner mitochondrial membrane

Explanation: Correct Option B - Inner mitochondrial membrane: This is the correct option. Cardiolipin is a distinctive phospholipid abundant in the inner mitochondrial membrane. It plays a role in the structural integrity of the mitochondria and is involved in several mitochondrial functions, including electron transport chain activity. Incorrect Options: Option A - Outer mitochondrial membrane: The outer mitochondrial membrane primarily consists of phospholipids like phosphatidylcholine and phosphatidylethanolamine. Cardiolipin is not a major component of the outer mitochondrial membrane. Option C - Cytosol: Cardiolipin is not typically found in the cytosol. It is mainly confined to the mitochondrial membrane. Option D - Mitochondrial matrix: Cardiolipin is primarily located in the inner mitochondrial membrane and is not a major component of the mitochondrial matrix.

↑ *Back to this question in Part A*

@Neet_pg_bot

Q5: What is the primary mechanism behind tissue damage in long-standing diabetes? (Q852558)

A. Insulin resistance

B. Sorbitol accumulation

C. Hyperglycemia

D. Decreased insulin secretion

Correct Answer: C. Hyperglycemia

Explanation: Hyperglycaemia is the primary mechanism behind tissue damage in long-standing diabetes . Chronic high blood glucose levels lead to various pathophysiological changes , including: Advanced Glycation End Products (AGEs): Hyperglycaemia causes non-enzymatic glycation of proteins and lipids, leading to oxidative stress, inflammation, and vascular damage . Polyol Pathway Activation: Excess glucose is converted into sorbitol , which accumulates in tissues (e.g., retina, nerves, kidneys), leading to osmotic stress and oxidative damage . Protein Kinase C (PKC) Activation: Promotes vascular dysfunction, inflammation, and increased permeability , contributing to complications like diabetic retinopathy and nephropathy . Hexosamine Pathway Dysregulation: Alters gene expression, promoting vascular and renal damage . Insulin resistance (Option A) is incorrect because it plays a role in type 2 diabetes , but the primary cause of tissue damage is chronic hyperglycaemia , not insulin resistance itself. Sorbitol accumulation (Option B) is incorrect because while the polyol pathway contributes to complications like diabetic neuropathy and cataracts , it is not the sole mechanism behind widespread tissue damage. Decreased insulin secretion (Option D) is incorrect because it leads to hyperglycaemia , but it is the high glucose levels that drive long-term tissue damage, not insulin deficiency directly.

References:

https://pmc.ncbi.nlm.nih.gov/articles/PMC6679814/

https://pmc.ncbi.nlm.nih.gov/articles/PMC2996922/

https://pubmed.ncbi.nlm.nih.gov/19489690/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q6: A 13-year-old boy who accompanied his father to a fish farming region in his hometown accidentally consumes a white odourless chemical. He was under some medication and post-meal he developed toxicity of the drug. Which of the following toxin is most likely to cause this condition? (Q857895)

A. Alkyl guanides

B. BAL

C. Antimycin A

D. Strychnine

Correct Answer: C. Antimycin A

Explanation: Antimycin A Antimycin A is commonly used in fish farming as a piscicide (a chemical toxic to fish) to selectively remove undesirable fish species. It is a colorless to white crystalline compound and is relatively odorless. It is a potent respiratory chain uncoupler. When consumed, it inhibits cellular respiration. It is a mitochondrial toxin that inhibits Complex III of the electron transport chain. This inhibition leads to decreased ATP production and increased oxidative stress. This can amplify the toxicity of some medications- especially those metabolized via oxidative pathways (e.g. anticonvulsants, antibiotics, or chemotherapeutics). Alkyl guanides (Option A) are a broad class of compounds, some of which can be toxic, but not specifically associated with fish farming or the described scenario. Also, they aren't primarily known for causing drug toxicity by interfering with metabolism in the same direct way as Antimycin A. BAL (British Anti-Lewisite; Dimercaprol) (Option B): This is a chelating agent , not a toxin, and is used as an antidote for heavy metal poisoning. Strychnine (Option D): It's a plant-derived neurotoxin (not used in fish farming). It causes convulsions via spinal inhibition of glycine , not mitochondrial toxicity. It does not primarily cause drug toxicity by interfering with drug metabolism in the way Antimycin A does. Its symptoms would be distinctly neurological, not primarily drug accumulation.

References:

Vasudevan 7th edition , pg no 266

↑ *Back to this question in Part A*

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Q7: Complex IV is inhibited by --? (Q859423)

A. Rotenone

B. Cyanide

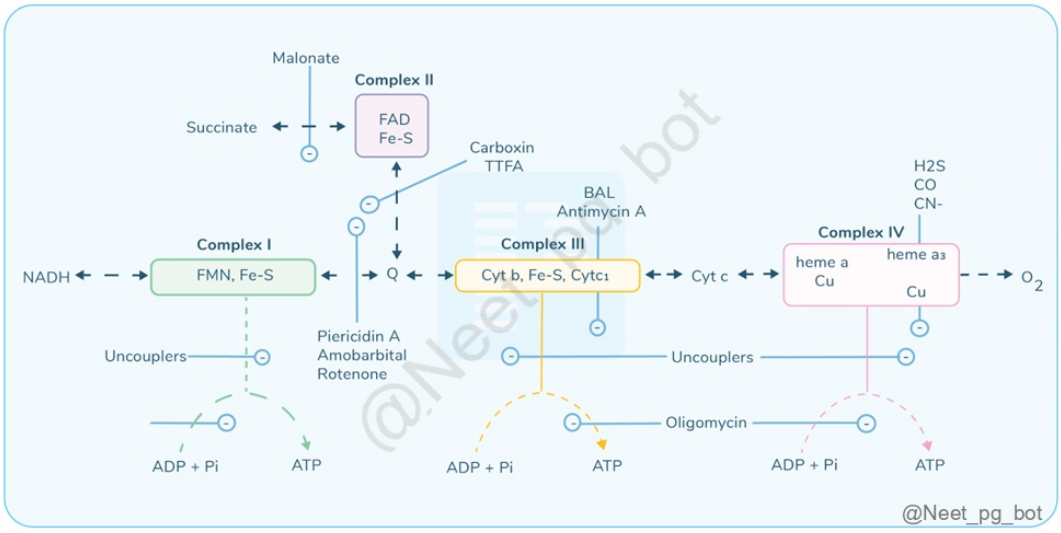
C. Carboxin

D. Antimycin A

Correct Answer: B. Cyanide

Explanation: Complex IV, also known as cytochrome c oxidase, is the final enzyme in the electron transport chain, where it catalyzes the transfer of electrons to oxygen, resulting in water formation. Cyanide inhibits Complex IV by binding to the iron in the heme group of cytochrome c oxidase, thereby preventing the transfer of electrons to

oxygen. Inhibitors of the Electron Transport Chain (ETC): Inhibitor Target Site Mechanism Rotenone (Option A) Amobarbital Piericidin A Complex I (NADH dehydrogenase) Blocks electron transfer from Complex I to ubiquinone■ TTFA (Thenoyltrifluoroacetone) Carboxin (Option C) Complex II (Succinate dehydrogenase) Blocks electron transfer from Complex II to ubiquinone■ Malonate Complex II Malonate is a competitive inhibitor of Complex II. Antimycin A (Option D) BAL Complex III Cytochrome bc1 complex Blocks electron transfer from Complex III to Cytochrome C. Cyanide (CN−) (Option B) Carbon monoxide (CO) Hydrogen Sulfide (H2S) Complex IV (Cytochrome c Oxidase) Prevents electron transfer from Complex IV to oxygen■ Oligomycin ATP Synthase (Complex V) Blocks the proton channel of ATP synthase, preventing ATP synthesis■ Atractyloside Adenine Nucleotide Translocase Blocks ADP/ATP exchange across the mitochondrial membrane■ {{caption_text}}



References:

Harper’s Illustrated Biochemistry, 32nd edition, pg: 127, 128.

Inhibitors of Oxidative phosphorylation

↑ *Back to this question in Part A*

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• [17_Miscellaneous] 038 Previous Year Questions

« Back to Folder Directory

[17_Miscellaneous] 038 Previous Year Questions — Quick Jump

Q1 (p. 1400)	*Q2 (p. 1400)*	*Q3 (p. 1401)*	*Q4 (p. 1402)*	*Q5 (p. 1406)*	*Q6 (p. 1406)*
Q7 (p. 1407)	*Q8 (p. 1408)*	*Q9 (p. 1408)*	*Q10 (p. 1409)*	*Q11 (p. 1410)*	*Q12 (p. 1411)*
Q13 (p. 1412)	*Q14 (p. 1413)*	*Q15 (p. 1414)*	*Q16 (p. 1415)*	*Q17 (p. 1416)*	

Q1: Nitrogenous waste is excreted from the body in the form of? (Q859710)

A. Urea

B. Nitric oxide

C. Ammonia

D. Glutamine

Correct Answer: A. Urea

Explanation: Ammonia is produced during the breakdown of amino acids and by gut bacteria in humans. However, ammonia is highly toxic, especially to the brain, if its levels in the blood rise too high. To prevent this toxicity, the body converts ammonia into urea through the urea cycle . This process occurs in the liver, where ammonia is safely transformed into urea, a less toxic substance. Urea is then excreted from the body through the urine, effectively removing excess nitrogenous waste from the body. Incorrect Options: Nitric oxide (Option B): A signaling molecule involved in vasodilation and other processes, not nitrogenous waste excretion. Ammonia (Option C): While ammonia is produced during amino acid breakdown, it is toxic at high levels and is converted into urea for excretion. Glutamine (Option D): An amino acid involved in nitrogen transport but not directly excreted as a nitrogenous waste product.

References:

The Urea Cycle Step by Step

↑ *Back to this question in Part A*

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Q2: Which of the following substances, at the given concentration, makes water unsuitable for human consumption according to safety standards? (Q866677)

A. Fluoride – 0.8 mg/L

B. Cadmium – 0.3 mg/L

C. Chloride - 200 mg/L

D. Calcium – 7 mg/L

Correct Answer: B. Cadmium – 0.3 mg/L

Explanation: The unsafe substance here is cadmium at 0.3 mg/L , because it exceeds permissible limits for drinking water. WHO & BIS (Bureau of Indian Standards) limits: Fluoride : desirable ~1.0 mg/L (max 1.5 mg/L) → 0.8 mg/L is safe. Cadmium : safe limit is 0.003 mg/L (3 µg/L) → 0.3 mg/L is 100× higher , toxic. Chloride : permissible up to 250 mg/L → 200 mg/L is safe. Calcium : desirable up to 75 mg/L (max 200 mg/L) → 7 mg/L is safe. Toxicity of cadmium in water: Chronic exposure → kidney damage (proximal tubular dysfunction), bone demineralisation (Itai-Itai disease in Japan), carcinogenic effects. Often from industrial waste, batteries, and electroplating runoff.

↑ *Back to this question in Part A*

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Q3: In a village of Assam, a man is observed walking with the help of a stick and has genu valgum. Many people in the same village present with similar symptoms. What is the most probable cause of this condition? (Q866664)

A. Calcium deficiency

B. Vitamin C deficiency

C. Skeletal fluorosis

D. Vitamin D deficiency (Osteomalacia)



Correct Answer: C. Skeletal fluorosis

Explanation: The presence of genu valgum and multiple affected individuals in an Assam village suggests Skeletal Fluorosis , a condition common in areas with high fluoride levels in drinking water, leading to bone pain, stiffness, joint deformities, and walking difficulty. Cause: Chronic ingestion of high levels of fluoride, usually through drinking water. Endemic Areas: Common in parts of India, especially Assam, Andhra Pradesh, and Rajasthan. Symptoms: Bone pain and joint stiffness Genu valgum (knock knees), difficulty walking May progress to crippling deformities X-ray findings: I ncreased bone density (osteosclerosis) Prevention: Use of fluoride-free water, defluoridation techniques Treatment: Primarily supportive; address deformities and improve nutrition. Fluorine content in water Related symptoms <0.5mg/L Dental caries >1.5mg/L Dental fluorosis 3-6mg/L Skeletal fluorosis >10mg/L Crippling fluorosis Calcium deficiency (Option A) and Vitamin D deficiency (Osteomalacia) (Option D) cause bone pain and weakness, but are less likely to occur in clusters. Vitamin C deficiency (scurvy) (Option B) affects collagen synthesis and presents with bleeding gums, petechiae, not typically genu valgum.

References:

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↑ *Back to this question in Part A*

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Q4: A 5-year-old child is brought to the clinic with abdominal pain, irritability, and developmental delay. On examination, the child has pallor and a bluish line on the

gums. Laboratory tests reveal microcytic anemia, and a peripheral blood smear shows red blood cells with basophilic stippling. Which of the following is the most likely diagnosis? (Q866662)

A. Iron deficiency anemia

B. Sideroblastic anemia

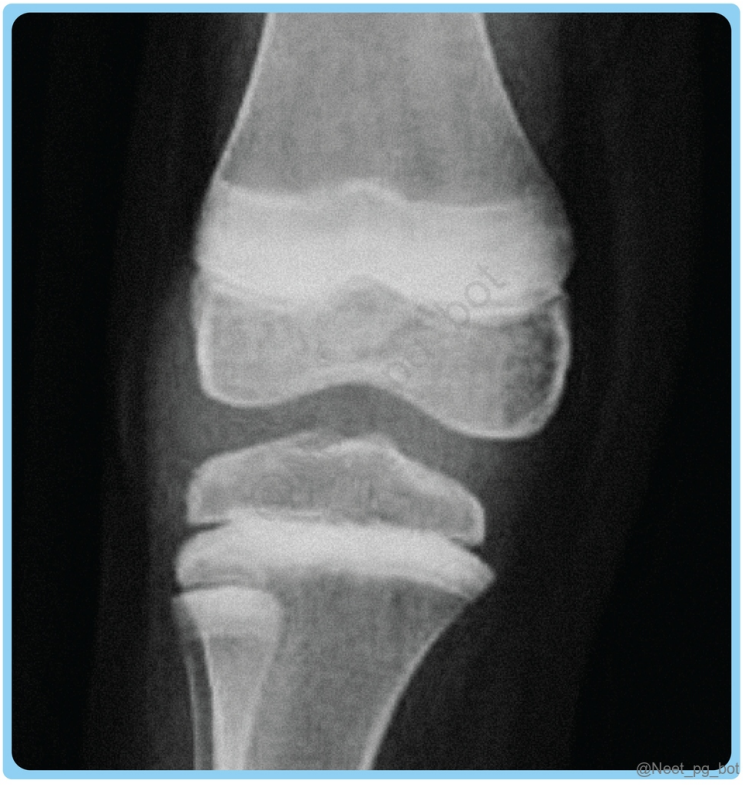
C. Lead poisoning

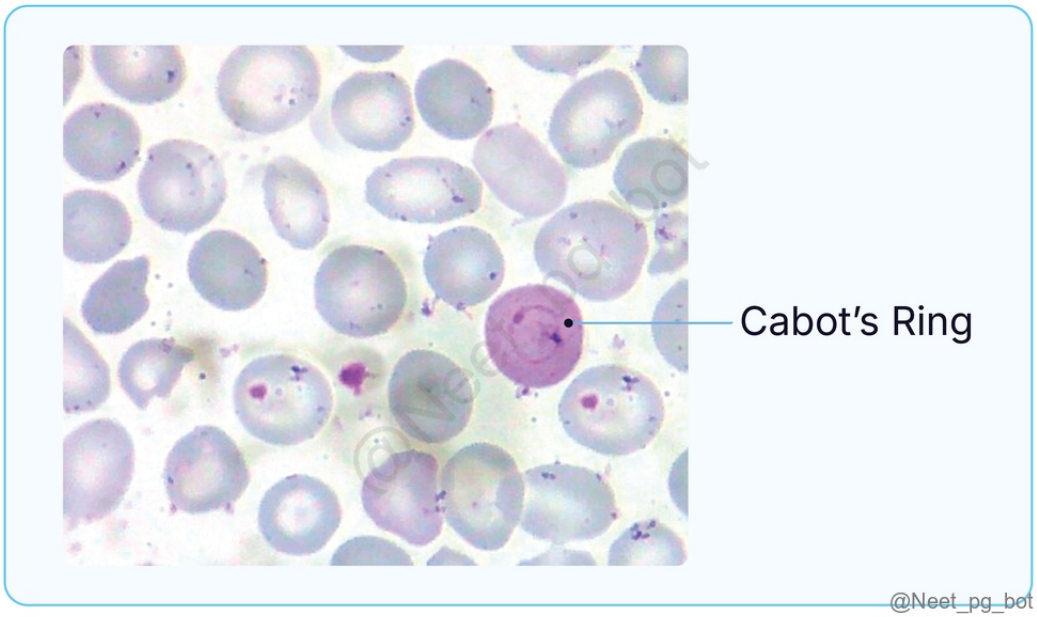
D. Thalassemia minor

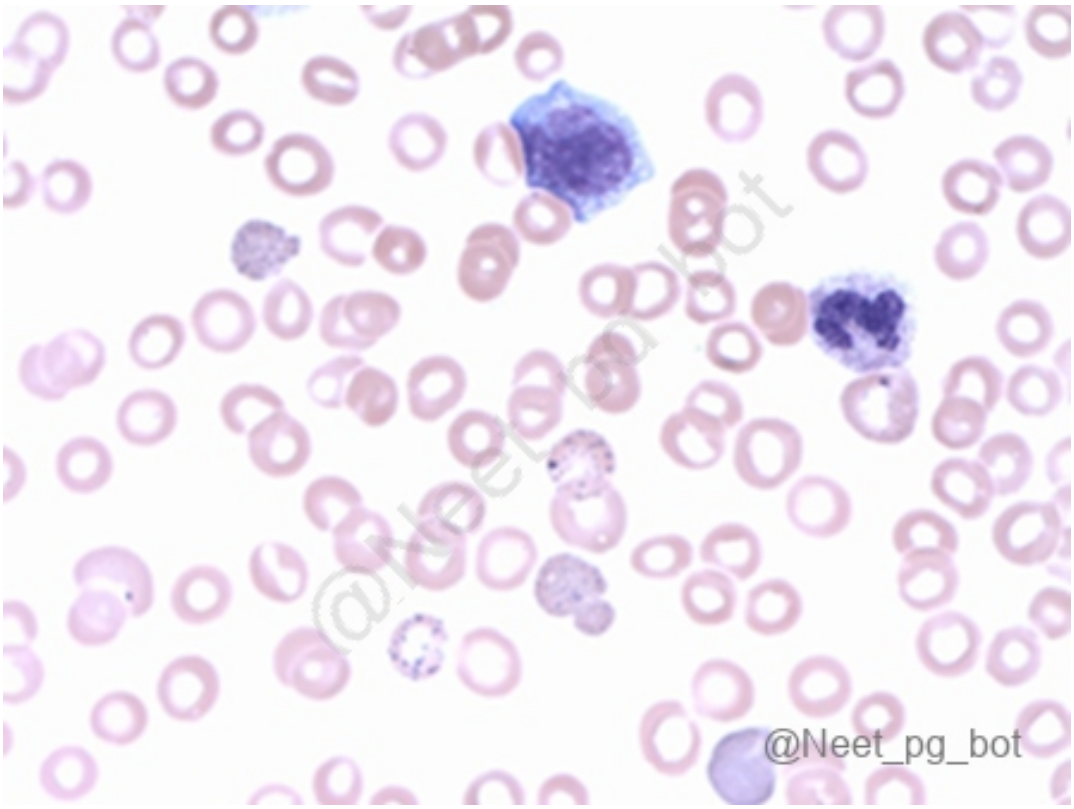
Correct Answer: C. Lead poisoning

Explanation: Classic signs of Lead Poisoning include abdominal pain, irritability, developmental delay, pallor, and a bluish line on the gums (Burtonian's or Burton's line), along with laboratory findings of microcytic anemia and basophilic stippling on the peripheral smear. Source Exposure to lead from paint, contaminated soil, or old pipes. Enzymes inhibited Ferrochelatase ALA-dehydratase Coproporphyrinogen oxidase). Clinical features Facial pallor: Circumoral (vasospasm) → Earliest and most specific feature Lead line or Burtonian lines: Stippled blue line observed on the gums, resulting from a subepithelial deposit of granules. Lead osteopathy/Lead line (Bone-line): Calcium deposition occurs at the growth plates of bones (specifically the lower end of the femur and the upper end of the tibia). Lead palsy: Predominantly motor features → (dry belly ache, wrist drop - radial nerve), foot drop - common peroneal nerve). Blood picture: Microcytic-hypochromic anemia, Cabot rings in RBCs, Sideroblasts (nucleated red cells), Anisocytosis, Poikilocytosis, Polycythemia Basophilic stippling or Punctate basophilia Colic and constipation (Painter's Colic or Devonshire colic) → Spasmodic pain relieved by pressure (at night). Saturnine gout (due to decreased excretion of uric acid) Encephalopathy Reproductive system (amenorrhea, dysmenorrhea, abortion, sterility derangments) CVS and Kidney (Hypertension, arteriolar degeneration, nephritis). Treatment Remove lead exposure and chelation therapy (e.g., EDTA, dimercaprol). Iron deficiency anemia (Option A) does not present with basophilic stippling or Burton's line. Sideroblastic anemia (Option B) shows ring sideroblasts in bone marrow but not Burton's line or neurological symptoms. ■■■■■■■Thalassemia minor (Option D) has microcytic anemia but lacks lead poisoning features.


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References:

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↑ *Back to this question in Part A*

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Q5: If you need to diagnose a syndromic illness such as meningitis, which of the following diagnostic techniques is most appropriate? (Q866328)

A. Multiplex PCR

B. Uniplex PCR

C. Attributed PCR

D. Nested PCR

Correct Answer: A. Multiplex PCR

Explanation: When diagnosing a syndromic illness such as meningitis, which may be caused by various pathogens including bacteria, viruses, and fungi, Multiplex PCR is used because it can simultaneously detect multiple causative agents. Amplifies multiple DNA targets in one reaction using several primer sets. Saves time and reduces costs by testing multiple pathogens simultaneously. Commonly used in clinical diagnostics and genetic testing. Requires careful primer design to avoid interference. Offers high sensitivity and specificity when well optimized. Ideal for syndromic testing where multiple pathogens may be involved. Uniplex PCR (Option B) detects only one target per reaction, making it less efficient for syndromic testing. Attributed PCR (Option C) is not a commonly used term in molecular diagnostics. Nested PCR (Option D) is a two-step PCR designed to increase sensitivity and specificity but usually targets a single pathogen per test.

↑ *Back to this question in Part A*

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Q6: Cyanide and carbon monoxide act on which of the heme proteins: (Q865454)

A. Cytochrome c oxidase

B. Myoglobin

C. Catalase

D. NADH dehydrogenase

Correct Answer: A. Cytochrome c oxidase

Explanation: Cyanide (CN■) and carbon monoxide (CO) are potent inhibitors of cytochrome c oxidase (Complex IV of the mitochondrial electron transport chain). Cyanide: Binds irreversibly to the heme iron (Fe^{3}■) in cytochrome c oxidase, blocking electron transfer to oxygen. Carbon monoxide: Binds competitively to the heme iron (Fe^{2}■) in cytochrome c oxidase, displacing oxygen (O■). Myoglobin (Option B) CO binds to myoglobin (causing cherry-red tissues), but this is not the primary lethal mechanism. Catalase (Option C) Not a target of

cyanide/CO toxicity (catalase breaks down H■O■, not involved in O■ utilization). NADH dehydrogenase (Complex I) (Option D) Inhibited by rotenone, not cyanide/CO.

References:

Harper's Illustrated Biochemistry, 31st Edition, Page 123.

↑ *Back to this question in Part A*

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Q7: Ascent to high altitude causes ? (Q865438)

A. Metabolic acidosis

B. Metabolic alkalosis

C. Respiratory acidosis

D. Respiratory alkalosis

Correct Answer: D. Respiratory alkalosis

Explanation: At high altitudes, hypoxia triggers hyperventilation, leading to enhanced CO■ removal and results in respiratory alkalosis. Changes during Acclimatization Respiratory At high altitudes, lower oxygen levels stimulate peripheral chemoreceptors, increasing pulmonary ventilation. This leads to enhanced CO2 removal, causing respiratory alkalosis. Kidneys respond by excreting more bicarbonate to stabilize blood pH, causing a sustained increase in ventilation and supporting prolonged acclimatization. Haematological At high altitudes, lower PO2 boosts erythropoietin production, increasing red blood cell count. This also raises 2,3-diphosphoglycerate (2,3-DPG) levels in red blood cells. Initially, alkalosis shifts the oxyhemoglobin dissociation curve to the left but increased 2,3-DPG shifts it to the right, improving oxygen release to tissues. Cardiovascular Hypoxia activates the sympathoadrenal axis, increasing heart rate, cardiac output, and blood pressure. Increased cardiac output leads to an increase in blood flow, and hypoxia causes vasodilation in the systemic circulation, further contributing to increased blood flow and oxygen supply to the tissues. Tissue In acclimatization, skeletal muscles undergo several changes, including an increase in the number of capillaries and mitochondria and an increase in myoglobin content. Further, chemicals released from the hypoxic tissues stimulate angiogenesis. Metabolic acidosis (Option A) involves decreased bicarbonate levels due to conditions like diarrhea or renal failure, not high altitude. Metabolic alkalosis (Option B) can occur later as a compensatory renal response to respiratory alkalosis but is not the initial effect. Respiratory acidosis (Option C) results from hypoventilation and CO■ retention .

References:

Guyton and Hall Textbook of Medical Physiology, 14th Edition, Page 554,555

Ganong's Review of Medical Physiology, 26th Edition, Page 637,638

GK Pal Comprehensive Textbook of Medical Physiology, Page. 948

↑ *Back to this question in Part A*

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Q8: A child presents with hepatomegaly and a cherry-red spot on the macula. Neurological regression is also noted. These findings are suggestive of a lysosomal storage disorder. Which of the following enzyme deficiencies is most likely responsible? (Q865412)

A. Arylsulfatase A

B. Hexosaminidase A

C. Sphingomyelinase

D. Galactocerebrosidase

Correct Answer: C. Sphingomyelinase

Explanation: A child presenting with hepatomegaly, a cherry-red spot on the macula, and neurological regression is indicative of sphingomyelinase deficiency, the enzyme deficiency responsible for Niemann-Pick Disease. Disease Enzyme Deficiency Lipid Accumulating Clinical Symptoms Tay-Sachs disease (Option B ruled out) Hexosaminidase A, S Cer—Glc—Gal(NeuAc) GalNAc GM2 Ganglioside Mental retardation, blindness, muscular weakness Fabry disease α-Galactosidase Cer—Glc—Gal—Gal Globotriaosylceramide Skin rash, kidney failure (symptoms manifest fully only in males; X-linked recessive) Metachromatic leukodystrophy (Option A ruled out) Arylsulfatase A Cer—Gal—OSO3 3-Sulfogalactosylceramide Mental retardation and psychological disturbances in adults; demyelination Krabbe disease (Option D ruled out) Galactocerebrosidase Cer—Gal Galactosylceramide Mental retardation; myelin almost absent Gaucher disease β-Glucosidase Cer—Glc Glucosylceramide Enlarged liver and spleen, erosion of long bones, mental retardation in infants Niemann-Pick disease types A, B (Option C) Sphingomyelinase Cer—P—choline Sphingomyelin Enlarged liver and spleen, mental retardation; fatal in early life Farber disease Ceramidase Acyl—Sphingosine Ceramide Hoarseness, dermatitis, skeletal deformation, mental retardation; fatal in early life Abbreviations: Cer, ceramide; Gal, galactose; Glc, glucose; NeuAc, N-acetylne

References:

Harper’s Illustrated Biochemistry, 32nd Edition, Page 245.

↑ *Back to this question in Part A*

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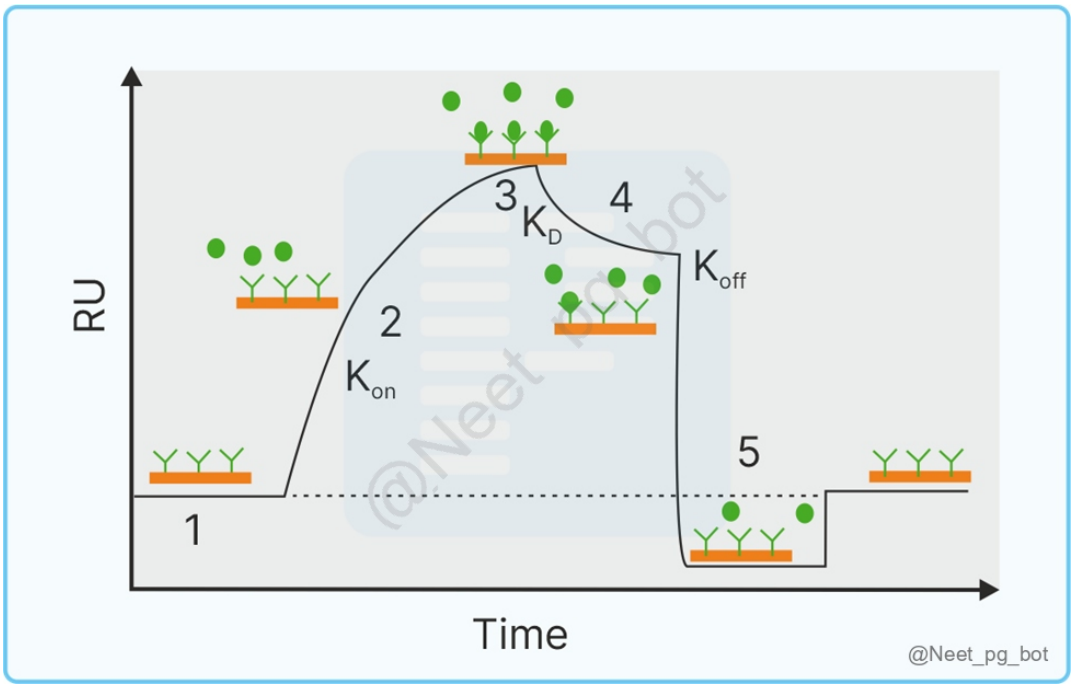
Q9: Name the graph and mention its application. (Q865396)

A. ELISA, Antibody concentration

B. ELISA, Antibody affinity

C. Plasma resonance, Ab concentration

D. Plasma resonance, Ab affinity



Correct Answer: D. Plasma resonance, Ab affinity

Explanation: The given graph is Plasma resonance or Surface Plasmon Resonance (SPR) , which is used to determine the determine the strength (affinity) and speed (kinetics) of antibody-antigen interactions. ELISA (Option A, B) for antibody concentration typically produces a standard curve with a sigmoidal shape, plotting absorbance against antibody concentration. This method uses colorimetric detection to quantify the amount of antibody or antigen present in a sample, making it a reliable and widely used tool in diagnostics and research

References:

Full article: Determining the affinities of high-affinity antibodies using KinExA and surface plasmon resonance

↑ *Back to this question in Part A*

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Q10: Which test should be done for detecting metabolite levels in inborn errors of metabolism in neonates? (Q839926)

A. Tandem mass spectroscopy

B. Complete blood count (CBC)

C. Urinalysis

D. Genetic testing

Correct Answer: A. Tandem mass spectroscopy

Explanation: Correct Option A - Tandem mass spectroscopy: Tandem mass spectroscopy (MS/MS) is widely used for newborn screening for metabolic disorders. It allows for the detection of multiple metabolic diseases from a single blood sample. It is highly sensitive and can identify various metabolic abnormalities by analyzing specific metabolites and their precursors. Incorrect Options: Option B - Complete blood count (CBC): CBC is a common blood test used to evaluate overall health and detect a wide range of disorders, including anemia, infection, and many other diseases. However, it is not specifically designed for metabolic disease screening. Option C - Urinalysis: Useful for evaluating kidney function and urinary tract issues but not comprehensive enough for metabolic disease screening Option D - Genetic testing: Identifies specific genetic mutations, often used when there is a high suspicion of a particular disorder, but not a first-line screening tool due to cost and specificity.

References:

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↑ *Back to this question in Part A*

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Q11: In a 14-year-old male, a medical condition has been identified as a result of a defect in a gene responsible for DNA repair. The individual is experiencing increased pigmentation on the skin and is at a heightened risk of developing skin cancers, according to the physician. Which of the following mechanisms is most likely to be impaired in this particular condition? (Q859573)

A. Nonhomologous end joining repair defect

B. Nucleotide excision repair defect

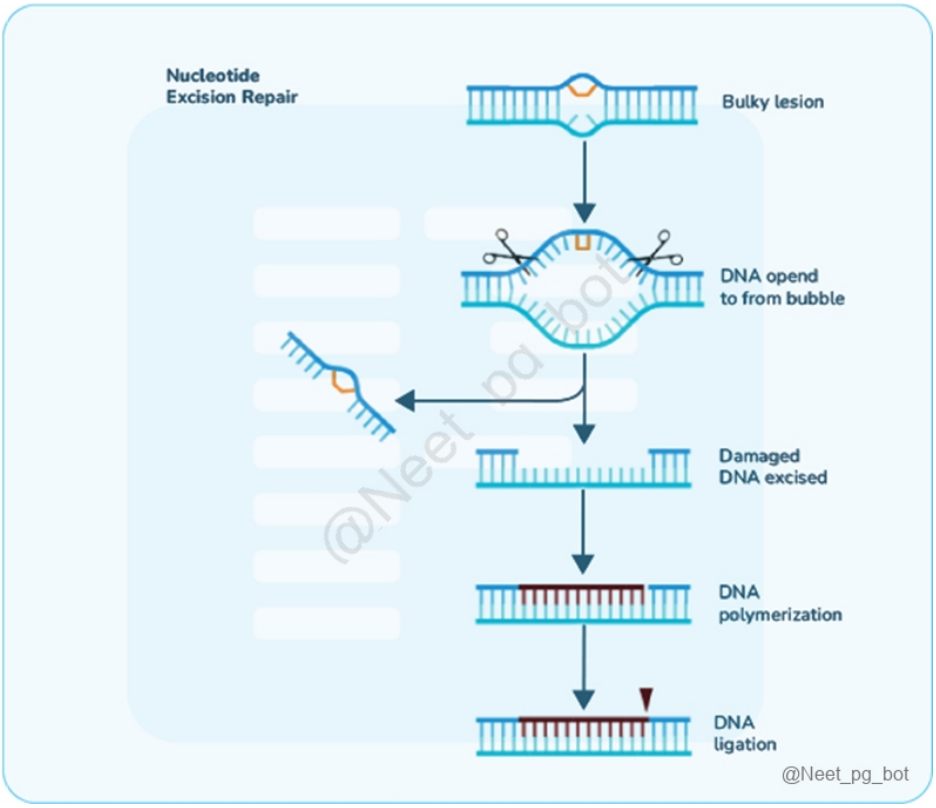
C. DNA base excision repair defect

D. DNA mismatch repair defect

Correct Answer: B. Nucleotide excision repair defect

Explanation: In this 14-year-old male, the condition described involves increased pigmentation on the skin and a heightened risk of skin cancers. These symptoms are consistent with Xeroderma Pigmentosum (XP) , a disorder

caused by an inability to repair UV-induced DNA damage. UV radiation can cause the formation of pyrimidine dimers (thymine dimers) in the skin cells, which, if not repaired, lead to mutations and an increased risk of cancer. The key defective mechanism in XP is Nucleotide Excision Repair (NER) . Nucleotide excision repair: Nucleotide excision repair is required when chemical damage to a segment occurs. UV mediated Pyr Pyr dimerization is observed in a segment. 30 bases are removed and then the correct bases are added Recognition and Excision: In bacteria, UvrABC proteins (uvrABC excinuclease) recognize these dimers and cleave the damaged strand on both the 5′ and 3′ sides of the lesion. A short oligonucleotide containing the dimer is excised, leaving a gap in the DNA strand. The gap is subsequently filled in by DNA polymerase I and sealed by DNA ligase. Nucleotide Excision Repair in Humans: A related nucleotide excision repair (NER) pathway exists in humans, addressing UV-induced damage encountered during DNA synthesis. Transcription-coupled repair, a type of NER, specifically targets lesions that obstruct RNA synthesis. Clinical application: Pyrimidine dimers can form in human skin cells exposed to unfiltered sunlight. In xeroderma pigmentosum (XP), a genetic disorder, the inability to repair UV-induced DNA damage leads to the accumulation of mutations and a heightened risk of developing early and multiple skin cancers. XP results from defects in genes coding for proteins necessary for NER of UV damage {{caption_text}}



References:

Vasudevan 7th edition, pg 583

Lippincott 7th edition, 1219-1222

↑ *Back to this question in Part A*

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Q12: A child with mental retardation, blindness, and muscle weakness was investigated and was found to be deficient for Hexosaminidase-A enzyme. She is most likely suffering from .. (Q859572)

A. Krabbe's disease

B. Fabry's disease

C. Tay-sachs disease

D. Gaucher's disease

Correct Answer: C. Tay-sachs disease

Explanation: The child in the clinical scenario is showing signs of mental retardation, blindness, and muscle weakness , which strongly suggest a lysosomal storage disorder . Upon investigation, the child was found to be deficient in hexosaminidase A enzyme. The disease most likely causing these symptoms is Tay-Sachs disease .It is an inborn error of metabolism due to the failure of degradation of gangliosides, which is caused by a deficiency in the enzyme hexosaminidase A. Lipid Storage Diseases (Sphingolipidoses): They are a group of lysosomal storage diseases. Disease Enzyme deficiency Features Tay-Sachs disease (Option C) Hexosaminidase A Mental retardation Blindness Muscular weakness Cherry red spots in the macula Fabry disease (Option B ruled out) α-Galactosidase Skin rash Kidney failure (full symptoms only in males X-linked recessive) Krabbe disease (Option A ruled out) β-Galactosidase Mental retardation Near absence of myelin Predominant neurological manifestation and cranial nerves causing deafness and blindness. Gaucher disease (Option D ruled out) β-Glucosidase Enlarged liver and spleen Erosion of long bones Mental retardation in infants Anemia Thrombocytopenia Niemann-Pick disease Sphingomyelinase Enlarged liver and spleen Mental retardation Fatal in early life Cherry red spot in macula Farber disease Ceramidase Hoarseness Dermatitis Skeletal deformation Mental retardation Fatal in early life

References:

Harper’s illustrated Biochemistry 32e pg 245

https://www.ncbi.nlm.nih.gov/books/NBK564432/

↑ *Back to this question in Part A*

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Q13: Nitric oxide is synthesized from (Q859428)

A. L-arginine

B. L-citrulline

C. Glycine

D. Lysine

Correct Answer: A. L-arginine

Explanation: Nitric oxide (NO■) is a highly reactive free radical with an unpaired electron, making it highly reactive. It is uncharged and has a short half-life (approximately 0.1 second). Synthesis of Nitric Oxide : NO■ is synthesized from arginine by the enzyme nitric oxide synthase (NOS) . NOS contains heme , FAD , FMN , and tetrahydrobiopterine . The process utilizes NADPH and molecular oxygen . Calmodulin is required to modulate NOS activity. The guanidino nitrogen of arginine is incorporated into NO■, while one oxygen atom from molecular oxygen is incorporated into NO■ and the other into citrulline . This makes NOS a di-oxygenase enzyme. Isoenzymes of NOS : Mechanism of Action of Nitric Oxide : NO■ diffuses to adjacent smooth muscle cells and activates guanylate cyclase . This leads to an increase in cyclic GMP levels, which activates protein kinase in smooth muscle. This causes the dephosphorylation of myosin light chains , resulting in relaxation of smooth muscle (vasodilation) in blood vessels, leading to lower blood pressure . L-citrulline (Option B) : L-citrulline is a byproduct of nitric oxide synthesis, as it is produced from L-arginine during the enzymatic conversion to NO■. It is not the direct precursor for nitric oxide. Glycine (Option C) : Glycine is an amino acid, but it is not involved in the synthesis of nitric oxide. It serves other functions in the body, such as being a neurotransmitter. Lysine (Option D) : L-lysine is another amino acid, but it does not play a direct role in the synthesis of nitric oxide.

References:

Vasudevan 7th edition Pg no. 227, 228

↑ *Back to this question in Part A*

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Q14: What is the order of the band in the sarcomere? (Q854616)

A. I-Z-M-A-H

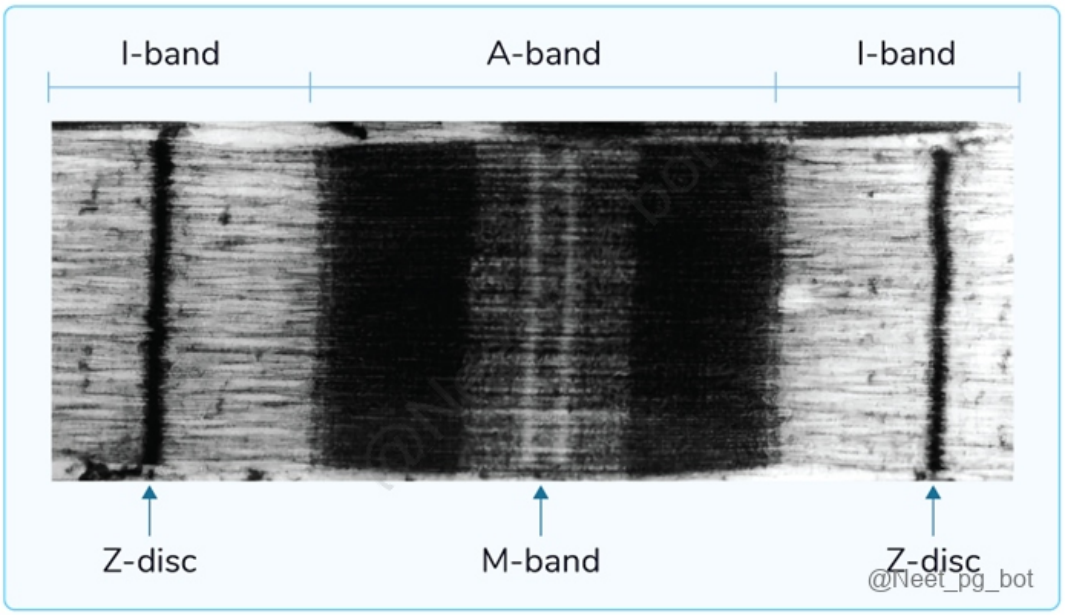
B. Z-H-M-A-I

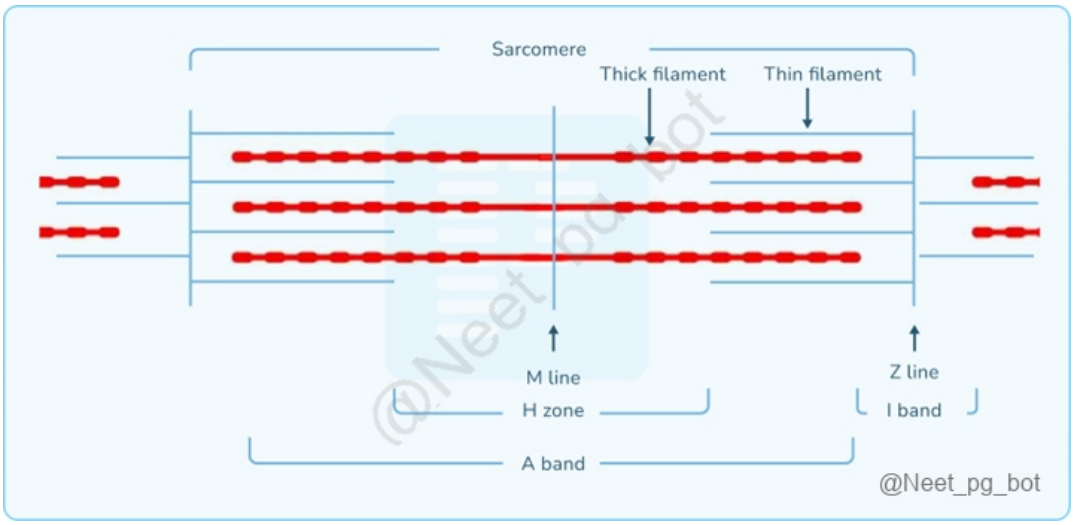
C. Z-I-A-H-M

D. Z-H-I-A-M

Correct Answer: C. Z-I-A-H-M

Explanation: {{caption_text}} Sarcomere The functional unit of a myofibril, composed of alternating dark A bands and light I bands. Z Line A dense, narrow line that bisects the I band; contains 2 actin molecules. I Band The light band alternating with the dark A band; bisected by the Z line. A Band The dark band in the sarcomere; includes the central H zone and is composed of thick filaments. H Zone The lighter region in the center of the A band. M Line The dark line in the middle of the H zone; contains M protein.





References:

Vasudevan's Textbook of Biochemistry for Medical Students, 7th edition, page 724

↑ *Back to this question in Part A*

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Q15: The development of cataracts in patients with Diabetes Mellitus is primarily due to the accumulation of which of the following substances in the lens? (Q852565)

A. Sorbitol

B. Dulcitol

C. Mannitol

D. Galactitol

Correct Answer: A. Sorbitol

Explanation: Sorbitol accumulation in the lens is the primary cause of diabetic cataracts . In diabetes mellitus , chronic hyperglycaemia leads to the activation of the polyol pathway , where: Glucose is converted to sorbitol by aldose reductase . Sorbitol is slowly metabolised to fructose by sorbitol dehydrogenase . In the lens, sorbitol accumulates , causing: Osmotic stress → water influx → lens swelling Oxidative stress → protein damage → cataract formation Diabetic cataracts typically present as snowflake cataracts in younger individuals or as accelerated senile cataracts . Dulcitol (Option B) is incorrect because dulcitol (galactitol) accumulates in galactosemia , not diabetes. It causes galactose-induced cataracts due to galactokinase or GALT deficiency . Mannitol (Option C) is incorrect because mannitol is an exogenous osmotic diuretic and does not accumulate in the lens. Galactitol (Option D) is incorrect because it is another name for dulcitol , and its accumulation is linked to galactosemia , not diabetes.

References:

https://pmc.ncbi.nlm.nih.gov/articles/PMC2903955/

https://pmc.ncbi.nlm.nih.gov/articles/PMC11556822/

↑ *Back to this question in Part A*

@Neet_pg_bot

Q16: Which of the following enzymes is typically elevated in prostate cancer? (Q852562)

A. Acid phosphatase

B. Alkaline phosphatase

C. Lactate dehydrogenase

D. Alpha-fetoprotein

Correct Answer: A. Acid phosphatase

Explanation: Acid phosphatase , specifically prostatic acid phosphatase (PAP) , is an enzyme that is elevated in prostate cancer , particularly in advanced or metastatic cases . PAP was historically used as a tumour marker for prostate cancer , though prostate-specific antigen (PSA) is now the preferred marker. Significance: Elevated in prostate cancer with bone metastases Can be used to monitor disease progression Less commonly used now due to the superior sensitivity of PSA Alkaline Phosphatase (Option B) is incorrect because while alkaline phosphatase

(ALP) is elevated in bone metastases , it is not specific for prostate cancer and can also be seen in liver disease and bone disorders. Lactate Dehydrogenase (Option C) is incorrect because LDH is a non-specific marker of cell turnover and is elevated in various malignancies, not just prostate cancer. Alpha-Fetoprotein (Option D) is incorrect because AFP is primarily a marker for hepatocellular carcinoma and germ cell tumours , not prostate cancer.

References:

Robbins Pathology, 10th Edition, Page 976

↑ *Back to this question in Part A*

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Q17: A patient presents with a history of vomiting. Arterial blood gas analysis reveals the following: pH : 7.5 pCO■ : 48 mm Hg HCO■■ : 30 mm Hg What is the most likely acid-base abnormality? (Q852561)

A. Metabolic alkalosis

B. Metabolic acidosis

C. Respiratory acidosis

D. Respiratory alkalosis

Correct Answer: A. Metabolic alkalosis

Explanation: Parameter Value Normal Range Interpretation pH 7.5 7.35–7.45 ↑ → Alkalosis HCO■■ 30 mEq/L 22–26 mEq/L ↑ → Metabolic cause pCO■ 48 mmHg 35–45 mmHg ↑ → Respiratory compensation (retaining CO■) Since both pH and HCO■■ are elevated, the primary disturbance is metabolic alkalosis. The rise in pCO■ indicates compensatory hypoventilation by the lungs to retain CO■ (acid) and partially correct pH. These findings suggest a primary metabolic alkalosis with respiratory compensation . Common Causes of Metabolic Alkalosis: Vomiting or gastric suction → loss of H■ and Cl■ from stomach acid Diuretic use (loop or thiazide) → increased H■ and K■ loss Hyperaldosteronism → promotes H■ secretion Excess bicarbonate administration Mechanism in This Case: Vomiting leads to loss of H■ (as HCl) from the stomach. The body compensates by increasing bicarbonate (HCO■■) in plasma. Respiratory system responds by hypoventilation , raising pCO■ . Metabolic Acidosis (Option B) is incorrect because metabolic acidosis would present with a low pH (<7.35) and a low HCO■■ , which is not the case here. Respiratory Acidosis (Option C) is incorrect because respiratory acidosis is characterized by low pH and high pCO■ , but in this case, the pH is elevated. Respiratory Alkalosis (Option D) is incorrect because respiratory alkalosis involves low pCO■ due to hyperventilation, whereas this patient has elevated pCO■ as a compensatory response.

References:

Ghai Textbook of Paediatrics Edition 10, Page 71, 72, 73, 74

↑ *Back to this question in Part A*

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