FMGE Biochemistry Sample Paper – 2

Duration: 60 Minutes **Maximum Marks: 40**

Instructions

- This paper contains **40 Multiple Choice Questions** modelled on the **Biochemistry** section of FMGE (Part A – Pre-clinical).
- Each correct answer carries **+1 mark**. There is **no negative marking** – attempt all questions.
- Questions follow the clinically integrated pattern of FMGE examination.
- Click a question number in the answer key to navigate to its solution.

FMGE Biochemistry – Q1 to Q40

Q1. A 28-year-old woman presents with angular stomatitis (cracks at the corner of the mouth), glossitis (magenta-coloured tongue), seborrhoeic dermatitis, and corneal neovascularisation. She consumes a diet deficient in dairy products and meat. Which vitamin deficiency best explains this clinical picture?

(A) Riboflavin (vitamin B2)

(B) Thiamine (vitamin B1)

(C) Niacin (vitamin B3)

(D) Pyridoxine (vitamin B6)

Q2. A 60-year-old man is admitted with crushing chest pain and an ECG showing ST elevation in leads II, III, and aVF. Serial enzyme testing is performed. Which LDH isoenzyme profile would be most consistent with a myocardial infarction?

(A) LDH-5 $>$ LDH-1

(B) LDH-3 is the predominant isoenzyme

(C) LDH-1 > LDH-2 ("flipped LDH pattern")

(D) LDH-4 > LDH-1

Q3. A 6-month-old infant presents with progressive psychomotor retardation, exaggerated startle response to sound (hyperacusis), and a cherry-red spot on fundoscopy. Electron microscopy of a skin biopsy shows membranous cytoplasmic bodies in lysosomes. The most likely enzymatic deficiency is:

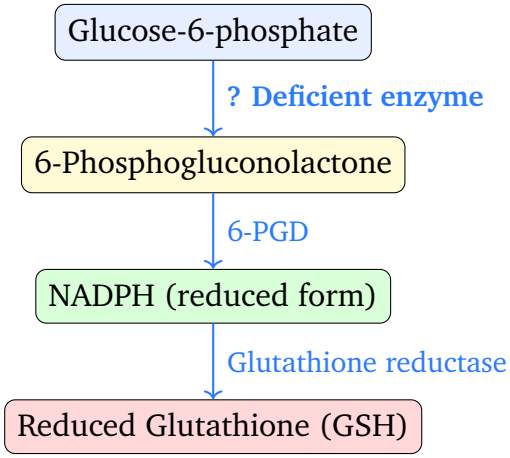
(A) Glucocerebrosidase

(B) Hexosaminidase A

(C) Sphingomyelinase

(D) α-Galactosidase A

Q4. A 25-year-old man of African descent develops acute haemolytic anaemia with haemoglobinuria after taking primaquine for malaria prophylaxis. A peripheral blood smear shows Heinz bodies (denatured haemoglobin precipitates). The biochemical defect responsible for this condition involves the following pathway:



(A) Pyruvate kinase deficiency

(B) Hexokinase deficiency

(C) Glucose-6-phosphate dehydrogenase (G6PD) deficiency

(D) Glutathione synthetase deficiency

Q5. A medical student is studying the water-soluble vitamins. He reads that one vitamin is essential for the synthesis of coenzyme A (CoA) and acyl carrier protein (ACP) and that its deficiency causes the "burning feet syndrome" with paraesthesias and dermatitis. This vitamin is:

(A) Thiamine (vitamin B1)

(B) Niacin (vitamin B3)

(C) Pantothenic acid (vitamin B5)

(D) Biotin (vitamin B7)

Q6. A 2-year-old child presents with massive hepatosplenomegaly, failure to thrive, and progressive neurological deterioration. Bone marrow biopsy reveals "foam cells" (lipid-laden macrophages). Cherry-red spot is noted on fundoscopy. Biochemical analysis shows accumulation of sphingomyelin in tissues. The enzyme deficiency responsible is:

(A) Sphingomyelinase (Niemann-Pick type A)

(B) Glucocerebrosidase

(C) Hexosaminidase A

(D) Arylsulphatase A

Q7. A researcher is studying transcription in eukaryotic cells. She treats cells with α-amanitin (the toxin from Amanita phalloides mushrooms). At low concentrations, α-amanitin specifically inhibits one RNA polymerase, resulting in cessation of mRNA synthesis. Which RNA polymerase is inhibited?

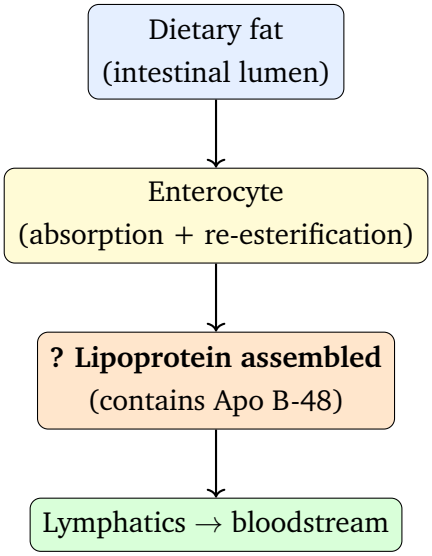
(A) RNA polymerase II

(B) RNA polymerase I

(C) RNA polymerase III

(D) Primase (DNA-dependent RNA polymerase)

Q8. After a fatty meal, dietary triglycerides are absorbed by intestinal enterocytes. The lipoproteins assembled in enterocytes to transport dietary lipids via the thoracic duct (lymphatics) into the circulation are:



(A) VLDL (very low density lipoprotein)

(B) Chylomicrons

(C) HDL (high density lipoprotein)

(D) IDL (intermediate density lipoprotein)

Q9. A 30-year-old man presents to the emergency department in extremis after deliberate ingestion of a rodenticide. He is found to have profound lactic acidosis, elevated venous oxygen saturation, and a characteristic "bitter almond" odour. The toxin acts by binding to a specific metal centre in the mitochondrial electron transport chain. The mechanism of toxicity is:

(A) Inhibition of Complex I by blocking NADH oxidation

(B) Uncoupling of oxidative phosphorylation by acting as a proton ionophore

(C) Inhibition of ATP synthase (Complex V) directly

(D) Binding to Fe^{3+} of cytochrome c oxidase (Complex IV), blocking electron transfer to O_2

Q10. A patient who consumed large quantities of raw egg white daily develops alopecia, perioral dermatitis, and muscle hypotonia. Laboratory workup reveals low plasma levels of a vitamin, as the egg-white protein avidin has bound and prevented its intestinal absorption. This vitamin serves as

a cofactor for all carboxylase enzymes (pyruvate carboxylase, acetyl-CoA carboxylase, propionyl-CoA carboxylase). The deficient vitamin is:

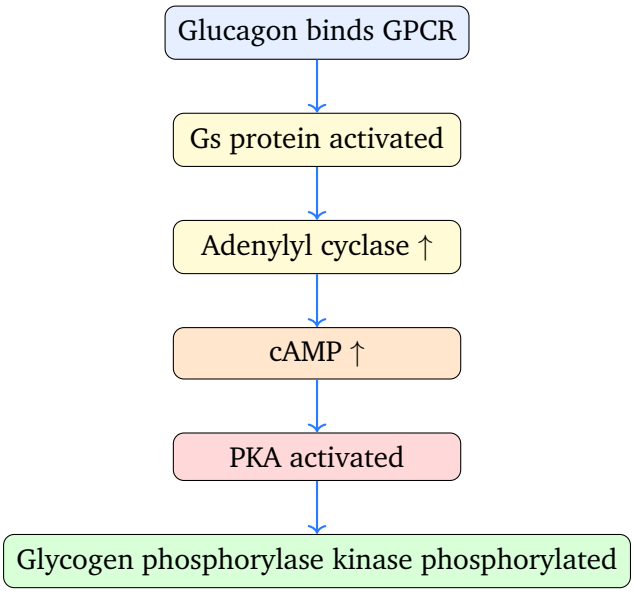
(A) Biotin (vitamin B7)

(B) Riboflavin (vitamin B2)

(C) Folate (vitamin B9)

(D) Thiamine (vitamin B1)

Q11. A patient with type 2 diabetes mellitus has a fasting hyperglycaemia of 220 mg/dL. The endogenous hormone glucagon signals the liver to initiate glycogenolysis via a second-messenger cascade. Which sequence correctly describes the glucagon signalling pathway leading to glycogen breakdown?



(A) Glucagon → GPCR → Gs → adenylyl cyclase → cAMP → PKA → phosphorylase kinase activation

(B) Glucagon → tyrosine kinase receptor → PI3K → Akt → GLUT4 translocation

(C) Glucagon → Gi protein → inhibition of adenylyl cyclase → decreased cAMP

(D) Glucagon → IP_3/DAG pathway → protein kinase C activation

Q12. A 45-year-old obese woman is evaluated for dyslipidaemia. Her physician explains that the rate-limiting enzyme of *de novo* fatty acid synthesis in the cytosol requires biotin and is activated by citrate while being inhibited by palmitoyl-CoA. This enzyme is:

(A) Fatty acid synthase (FAS) complex

(B) Malonyl-CoA decarboxylase

(C) Citrate lyase

(D) Acetyl-CoA carboxylase (ACC)

Q13. A biochemistry professor is discussing enzyme kinetics. She explains that a group of enzymes display sigmoidal (S-shaped) velocity-versus-substrate concentration curves, rather than the hyperbolic curve seen with Michaelis-Menten enzymes. These enzymes respond cooperatively to substrate concentration. Which characteristic best describes allosteric enzymes?

(A) Follow simple Michaelis-Menten kinetics; K_m fully describes their behaviour

(B) Display sigmoidal kinetics with Hill coefficient $n > 1$; subject to homotropic and heterotropic regulation

(C) Always have only one subunit and one active site

(D) Are irreversibly inhibited by their end-products

Q14. A 55-year-old man with Cushing's syndrome (endogenous hypercortisolism) presents with persistent hyperglycaemia. Cortisol promotes gluconeogenesis in the liver primarily by:

(A) Directly activating glucokinase in hepatocytes

(B) Inhibiting insulin secretion from pancreatic β-cells

(C) Inducing the gene expression of PEPCK (phosphoenolpyruvate carboxykinase) in the liver

(D) Activating glycogen synthase to convert glucose to glycogen

Q15. A 22-year-old man presents with episodic burning pain in the hands and feet (acroparesthesias) since childhood, angiokeratomas (dark-red skin lesions) over the bathing-trunk area, and progressive renal insufficiency. His maternal uncle also has renal disease. Urine shows globotriaosylceramide (Gb3) accumulation. The inheritance is X-linked. The deficient enzyme is:

(A) Sphingomyelinase

(B) Hexosaminidase A

(C) α-Galactosidase A

(D) Glucocerebrosidase

Q16. A 40-year-old man with non-alcoholic fatty liver disease undergoes analysis of hepatic lipid metabolism. It is noted that the β-oxidation of an unsaturated fatty acid (e.g., oleic acid, 18:1Δ^9) requires additional enzymatic steps compared to saturated fatty acids. The extra enzymes required are:

(A) Only thiokinase (fatty acyl-CoA synthetase)

(B) Only enoyl-CoA hydratase

(C) Only 3-hydroxyacyl-CoA dehydrogenase

(D) Enoyl-CoA isomerase (for odd-positioned cis-double bonds) and 2,4-dienoyl-CoA reductase (requiring NADPH)

Q17. A 20-year-old Nigerian man develops acute intravascular haemolysis, jaundice, and dark urine 2 days after starting dapsone for leprosy treatment. His blood film shows Heinz bodies and bite cells. Glutathione levels in erythrocytes are markedly reduced. The underlying genetic defect follows which inheritance pattern and affects which pathway?

(A) Autosomal recessive; affects the glycolytic pathway

(B) X-linked recessive; G6PD deficiency impairs the pentose phosphate pathway, reducing NADPH and GSH

(C) Autosomal dominant; affects haemoglobin structure

(D) X-linked dominant; affects pyruvate kinase

Q18. A researcher studying glycolytic regulation discovers that a key signalling molecule simultaneously activates phosphofructokinase-1 (PFK-1) allosterically and inhibits fructose-1,6-bisphosphatase (FBPase-1). This molecule is synthesised by the bifunctional enzyme PFK-2/FBPase-2 and its levels rise under insulin signalling. This key regulator is:

(A) Fructose-1,6-bisphosphate

(B) Fructose-6-phosphate

(C) Fructose-2,6-bisphosphate

(D) Glucose-1-phosphate

Q19. A 45-year-old non-smoking, non-drinking man with emphysema is found to have severe panacinar emphysema predominantly affecting the lung bases. Liver biopsy shows PAS-positive, diastase-resistant intrahepatic inclusions. Serum electrophoresis shows an absent α_1 band. The most likely diagnosis and molecular defect is:

(A) α_1-Antitrypsin deficiency (ZZ phenotype); misfolded AAT protein accumulates in hepatocytes causing liver disease and uninhibited neutrophil elastase destroys lung parenchyma

(B) Cystic fibrosis; CFTR channel mutation

(C) Hereditary haemochromatosis; HFE gene mutation

(D) Wilson disease; ATP7B copper transporter mutation

Q20. A 3-year-old child with intellectual disability, seizures, and an abnormal urine odour undergoes plasma amino acid analysis. The result shows markedly elevated plasma leucine with no detectable elevation of isoleucine, valine, or phenylalanine. A biochemist notes that this amino acid is unique among the 20 standard amino acids in being purely ketogenic – it is metabolised only to acetyl-CoA and acetoacetate, and never to glucose. This amino acid is:

(A) Isoleucine

(B) Phenylalanine

(C) Leucine

(D) Lysine

Q21. A 32-year-old laboratory technician is found collapsed in the chemical storage room. He is cyanotic, tachycardic, and confused. Despite receiving 100% oxygen via face mask, his venous blood remains bright red (paradoxically high venous pO_2). Blood lactate is 18 mmol/L. The toxin acts on the mitochondrial electron transport chain. The specific site of inhibition and the correct first-line antidote are:

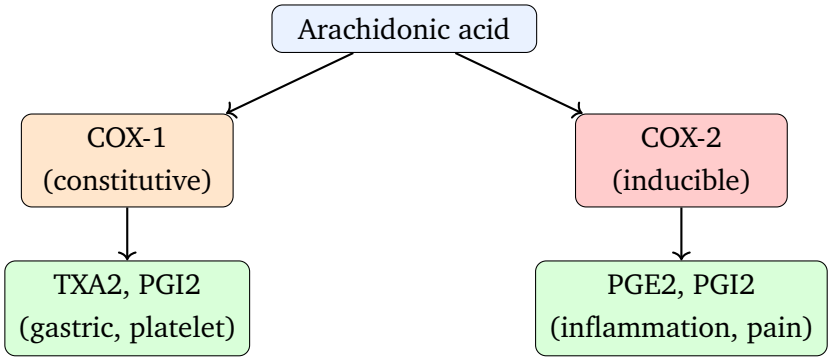
(A) Complex I; treat with rotenone

(B) Complex IV (cytochrome c oxidase, Fe^{3+}); treat with hydroxocobalamin and/or sodium nitrite + thiosulfate

(C) ATP synthase; treat with oligomycin

(D) Complex III; treat with antimycin A

Q22. A pharmaceutical company is developing selective anti-inflammatory agents. They explain the arachidonic acid cascade to investors. Regarding cyclooxygenase (COX) isoenzymes and non-steroidal anti-inflammatory drugs (NSAIDs):



(A) COX-1 is inducible and mediates inflammation; COX-2 is constitutive and protects the gastric mucosa

(B) Celecoxib is a non-selective inhibitor of both COX-1 and COX-2

(C) Aspirin irreversibly inhibits only COX-2 selectively

(D) COX-1 is constitutive (gastric protection, platelet TXA2); COX-2 is inducible (inflammation); selective COX-2 inhibitors (celecoxib) spare gastric mucosa

Q23. A 28-year-old man of Eastern European Jewish ancestry presents with cirrhosis, psychiatric disturbance (personality change, psychosis), and a golden-brown ring at the periphery of the cornea noted on slit-lamp examination (Kayser-Fleischer ring). Serum ceruloplasmin is very low. Urinary copper is elevated. The molecular defect is:

(A) HFE gene mutation; iron overload

(B) HMBS gene mutation; porphyrin accumulation

(C) SERPINA1 gene mutation; α_1-antitrypsin misfolding

(D) ATP7B gene mutation; defective hepatic copper-transporting ATPase leading to copper accumulation in liver, brain, and cornea

Q24. In the urea cycle, ornithine and carbamoyl phosphate condense in the mitochondrial matrix to form citrulline. Citrulline must then cross the inner mitochondrial membrane into the cytoplasm where subsequent steps occur. The intermediate that is exported from the mitochondria in this step is:

(A) Ornithine

(B) Citrulline

(C) Arginine

(D) Argininosuccinate

Q25. A 60-year-old man with rheumatoid arthritis is started on a new biologic therapy. His rheumatologist mentions that his serum CRP (C-reactive protein) is a sensitive marker of inflammation. Regarding the biochemistry of CRP:

(A) CRP is produced by macrophages and is a slow-phase reactant, rising days after inflammation

(B) CRP is a lipid-transport protein synthesised by adipocytes

(C) CRP is synthesised by hepatocytes within 6 hours of inflammatory stimulus; it is an opsonin and activates the classical complement pathway

(D) CRP directly inhibits TNF-α and IL-6 production

Q26. A 10-year-old boy with intellectual disability, self-mutilating behaviour (biting of fingers and lips), choreoathetosis, and gout-like arthritis is found to have markedly elevated serum uric acid. His mother is a carrier but is clinically normal. Biochemical analysis reveals virtually absent activity of a purine salvage enzyme. The disorder is:

(A) Lesch-Nyhan syndrome; HGPRT (hypoxanthine-guanine phosphoribosyltransferase) deficiency; X-linked recessive

(B) Gout; PRPP synthetase overactivity; autosomal dominant

(C) Adenosine deaminase deficiency; SCID

(D) Von Gierke disease; glucose-6-phosphatase deficiency

Q27. During co-transcriptional processing of mRNA in eukaryotic cells, the very first nucleotide at the 5' end of the nascent mRNA is capped with a modified guanosine residue. This modification is important for mRNA stability and translation initiation. The 5' cap structure is:

(A) A poly-A tail added by poly-A polymerase at the 5' end

(B) A 7-methylguanosine (m^7G) cap added in 5'$\rightarrow$5' triphosphate linkage; protects from 5'$\rightarrow$3' exonuclease degradation and is recognised by eIF4E for cap-dependent translation

(C) An IRES (internal ribosome entry site) that replaces the 5' cap in all mRNAs

(D) A 3'-OH hydroxyl group added by terminal transferase

Q28. A biochemist studying one-carbon metabolism notes that a molecule derived from methionine and ATP is the principal **universal methyl group**

donor in the body. This molecule donates methyl groups in the synthesis of creatine, phosphatidylcholine, epinephrine, and in DNA/histone methylation reactions. This molecule is:

(A) Tetrahydrofolate (THF)

(B) 5-Methyltetrahydrofolate (5-methyl-THF)

(C) Homocysteine

(D) S-Adenosylmethionine (SAM)

Q29. A 35-year-old obese woman with metabolic syndrome is counselled about fatty acid synthesis. Her physician explains that in the fed state, excess glucose is converted to fatty acids in the liver. The rate-limiting step of fatty acid synthesis is catalysed by an enzyme located in the cytosol, which is phosphorylated and inactivated by AMP-activated protein kinase (AMPK) during energy deficit. This enzyme is:

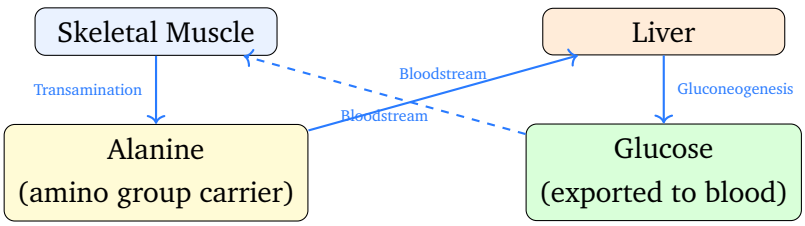
(A) HMG-CoA reductase

(B) Acetyl-CoA carboxylase (ACC)

(C) Citrate lyase

(D) Fatty acid synthase (FAS)

Q30. During prolonged fasting or endurance exercise, the alanine-glucose cycle operates between skeletal muscle and the liver. Which statement best describes the metabolic logic of this cycle?



(A) Muscle converts pyruvate + amino groups to alanine (via transamination); alanine travels to liver where it is reconverted to pyruvate and then to glucose by gluconeogenesis – non-toxic nitrogen transport

(B) Alanine is oxidised directly in the liver to generate ATP without gluconeogenesis

(C) The cycle transfers fatty acids from muscle to liver for β-oxidation

(D) Alanine synthesised in the liver is transported to muscle to serve as a direct energy substrate

Q31. A molecular biology laboratory uses restriction endonucleases to clone a gene of interest. The scientist selects a Type II restriction enzyme that recognises a 6-base-pair palindromic sequence and cuts within that sequence. The cut produces 4-nucleotide 5'-overhangs ("sticky ends"). Which statement best describes Type II restriction endonucleases?

(A) They require ATP hydrolysis for DNA cleavage and cut at a distant site from recognition

(B) They are single-stranded RNA endonucleases used in CRISPR-Cas9 systems

(C) They methylate DNA at recognition sites to protect host DNA from cleavage

(D) They cleave double-stranded DNA at specific palindromic recognition sequences; produce blunt or sticky (cohesive) ends; used as molecular scissors in recombinant DNA technology

Q32. In the polymerase chain reaction (PCR), a thermostable DNA polymerase is essential so that the enzyme is not denatured during the high-temperature denaturation step. A temperature cycling programme is used: denaturation at 94°C, primer annealing at ~55°C, and extension at 72°C. The thermostable polymerase routinely used was originally isolated from:

(A) E. coli (mesophilic bacterium)

(B) Pyrococcus furiosus (hyperthermophilic archaeon – gives Pfu polymerase)

(C) Thermus aquaticus (thermophilic bacterium from hot springs – gives Taq polymerase)

(D) Bacillus subtilis (spore-forming bacterium)

Q33. A patient with tuberculosis is started on isoniazid (INH) therapy. Six weeks later he develops peripheral neuropathy with burning and tingling in the feet and hands. A key mechanism of INH toxicity involves its interaction with a vitamin:

(A) INH inhibits vitamin B12 absorption leading to subacute combined degeneration

(B) INH chelates zinc and causes acrodermatitis enteropathica

(C) INH is a hydrazide that reacts with pyridoxal phosphate (PLP/vitamin B6), causing relative B6 deficiency; supplementation with pyridoxine prevents peripheral neuropathy

(D) INH inhibits thiamine uptake, causing Wernicke's encephalopathy

Q34. A 55-year-old man from Keshan county in China presents with dilated cardiomyopathy and muscle weakness. Epidemiological studies have linked his disease to a deficiency of a trace mineral that is an essential component of glutathione peroxidase (GPx), the enzyme that reduces hydrogen peroxide and lipid hydroperoxides using glutathione. The deficient mineral is:

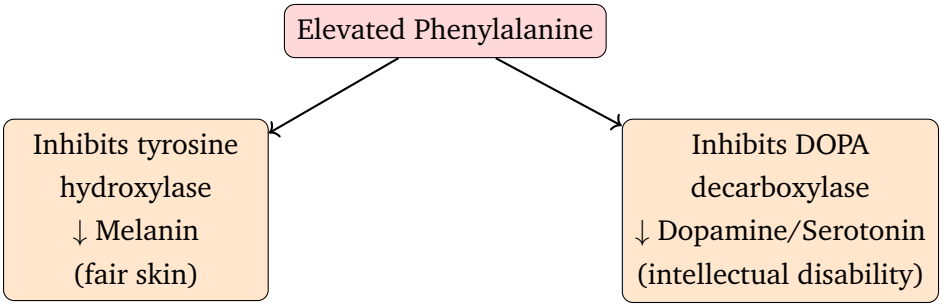
(A) Selenium

(B) Zinc

(C) Copper

(D) Manganese

Q35. A newborn screened at birth is found to have markedly elevated phenylalanine on the Guthrie test. The baby has fair skin and blond hair despite being born to dark-skinned parents. The physician explains the multisystem biochemical consequences of elevated phenylalanine. Which of the following is the most complete explanation of the biochemical pathology in PKU?



(A) Only melanin synthesis is impaired; the neurological features are due to a separate mutation

(B) Elevated phenylalanine activates the urea cycle, causing hyperammonaemia

(C) Phenylalanine is converted to an excess of tyrosine that is neurotoxic

(D) Elevated phenylalanine inhibits tyrosine hydroxylase (reducing melanin, causing hypopigmentation) and inhibits aromatic amino acid decarboxylase (reducing dopamine and serotonin, causing intellectual disability and seizures)

Q36. A biochemistry textbook describes phospholipid metabolism. The most abundant phospholipid in mammalian cell membranes is synthesised via the CDP-choline pathway (Kennedy pathway) or by sequential methylation of phosphatidylethanolamine (PE) using SAM. This phospholipid is also the major component of pulmonary surfactant. It is:

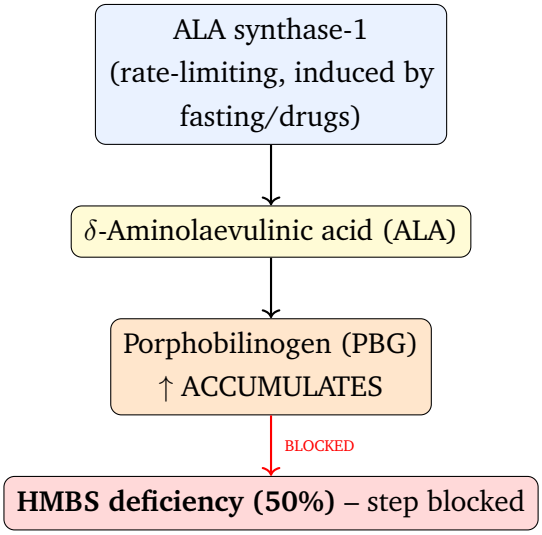
(A) Phosphatidylethanolamine (PE)

(B) Phosphatidylcholine (PC, lecithin)

(C) Phosphatidylserine (PS)

(D) Cardiolipin (diphosphatidylglycerol)

Q37. A 35-year-old woman with intermittent severe abdominal pain, psychiatric disturbance, and motor neuropathy has urine that turns dark on standing. Urine porphobilinogen (PBG) is markedly elevated during acute attacks. The attacks are precipitated by fasting, oral contraceptives, and barbiturates. Acute intermittent porphyria (AIP) is due to:



(A) Uroporphyrinogen III synthase deficiency; causes congenital erythropoietic porphyria

(B) Ferrochelatase deficiency; causes erythropoietic protoporphyria

(C) Porphobilinogen deaminase overactivity; leads to haem overproduction

(D) Hydroxymethylbilane synthase (HMBS/PBG deaminase) deficiency; PBG accumulation; ALA synthase-1 is derepressed by fasting/drugs

Q38. A patient with haemochromatosis has excess iron deposition in the liver. Pathologists performing a Prussian blue stain identify large deposits of an insoluble iron-storage polymer derived from degraded ferritin. What is this storage form of iron, and how does it differ from ferritin?

(A) Transferrin; an iron transport protein in plasma

(B) Myoglobin; an oxygen-storage protein in muscle

(C) Ferritin; a soluble, dynamic iron-storage complex found inside cells

(D) Haemosiderin; an insoluble, partially degraded form of ferritin visible as golden-brown granules on Prussian blue staining, seen in iron overload states

Q39. A medical student studying enzyme mechanisms reads about a class of proteases that employ a Ser-His-Asp catalytic triad at their active site. The serine residue acts as a nucleophile that attacks the scissile pep-

tide bond, forming an acyl-enzyme intermediate. Examples include chymotrypsin, trypsin, thrombin, and elastase. These enzymes belong to which class?

(A) Serine proteases; active-site catalytic triad Ser-His-Asp; charge relay system; nucleophilic serine attacks peptide carbonyl

(B) Cysteine proteases; active-site thiol group; e.g., caspases and papain

(C) Aspartyl (acid) proteases; two aspartate residues; e.g., pepsin and HIV protease

(D) Metalloproteinases; zinc ion at active site; e.g., carboxypeptidase A and collagenase

Q40. A patient with iron-deficiency anaemia has a markedly elevated total iron-binding capacity (TIBC). The physician explains that TIBC reflects the plasma concentration of the iron-transport protein. In the normal state, this protein is approximately 33% saturated with iron. It is synthesised by the liver and is an acute negative-phase reactant (decreases in chronic disease). This protein is:

(A) Ferritin

(B) Transferrin

(C) Lactoferrin

(D) Haemopexin

Detailed Solutions

Q1.

Solution

Concept – Riboflavin (Vitamin B2) deficiency: Riboflavin (B2) is the precursor of the coenzymes **FMN** (flavin mononucleotide) and **FAD** (flavin adenine dinucleotide), essential for oxidative metabolism (Complex I, Complex II, β-oxidation, and many other redox enzymes). The classical deficiency syndrome is the "oro-oculo-genital" syndrome: **angular stomatitis** (cheilosis), **glossitis** (magenta/beefy-red tongue), **seborrhoeic dermatitis** (nasiolabial folds, scrotum/vulva), and importantly **corneal neovascularisation** (vascularisation of the corneal stroma). Riboflavin is found in dairy, meat, eggs, and green vegetables – strict vegetarians or those with malabsorption are at risk.

Why the distractors are wrong: (B) Thiamine deficiency causes beriberi (cardiovascular/neurological) or Wernicke's encephalopathy – not angular stomatitis. (C) Niacin deficiency causes pellagra (3Ds: dermatitis on sun-exposed areas, diarrhoea, dementia). (D) Pyridoxine (B6) deficiency causes peripheral neuropathy, sideroblastic anaemia, and convulsions.

Final Answer: Riboflavin (Vitamin B2). $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q1

Q2.

Solution

Concept – LDH isoenzymes in myocardial infarction: Lactate dehydrogenase (LDH) exists as five isoenzymes (tetramers of H and M subunits). **LDH-1** (HHHH) predominates in the **heart**, red blood cells, and kidney. **LDH-5** (MMMM) predominates in the **liver** and skeletal muscle. Normally LDH-2 $>$ LDH-1 in serum. In myocardial infarction, LDH-1 is released in large amounts and surpasses LDH-2 – this **"flipped" LDH pattern** (LDH-1 $>$ LDH-2) is classic for cardiac damage. LDH rises 24–48 h after MI and peaks at 72–96 h. It has been largely replaced by troponin T/I for MI diagnosis but remains high-yield for FMGE.

Why the distractors are wrong: (A) LDH-5 $>$ LDH-1 indicates liver disease or skeletal muscle damage. (B) LDH-3 predominates in lung tissue. (D) LDH-4 predominance is not a recognised pattern for MI.

Final Answer: LDH-1 $>$ LDH-2 ("flipped LDH pattern"). $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – Tay-Sachs disease: Tay-Sachs is an autosomal recessive lysosomal storage disorder caused by deficiency of **hexosaminidase A** (α subunit encoded by the *HEXA* gene). Without this enzyme, **GM2 ganglioside** accumulates in neurons. Classical features: onset at 3–6 months with progressive motor and mental deterioration, exaggerated startle response (hyperacusis), cherry-red spot on fundoscopy (contrast of pale ganglioside-laden macula against the normal perfused fovea), and death by age 2–3 years. It is most prevalent in Ashkenazi Jews (1 in 30 carrier frequency), French-Canadians, and Cajun populations. Membranous cytoplasmic bodies (whorled lamellae) are seen on electron microscopy.

Why the distractors are wrong: (A) Glucocerebrosidase deficiency causes Gaucher disease (hepatosplenomegaly, no neurodegeneration in Type I). (C) Sphingomyelinase deficiency causes Niemann-Pick (cherry-red spot, foam cells – but the primary storage molecule is sphingomyelin, not GM2). (D) α-Galactosidase A deficiency causes Fabry disease (acroparesthesias, angiokeratomas, renal failure).

Final Answer: Hexosaminidase A. $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q3

Q4.

Solution

Concept – G6PD deficiency and the Pentose Phosphate Pathway: Glucose-6-phosphate dehydrogenase (G6PD) catalyses the first and rate-limiting step of the pentose phosphate pathway (PPP), converting G6P to 6-phosphogluconolactone while reducing $NADP^+$ to **NADPH**. NADPH is essential for maintaining **reduced glutathione (GSH)** via glutathione reductase. GSH neutralises oxidative free radicals. In G6PD deficiency, oxidant drugs (primaquine, dapsone, nitrofurantoin) deplete GSH, causing oxidative haemoglobin denaturation (Heinz bodies) and membrane damage (bite cells, haemolysis). G6PD deficiency is the most common enzymopathy worldwide; it is X-linked recessive and confers some protection against malaria.

Why the distractors are wrong: (A) Pyruvate kinase deficiency causes chronic non-spherocytic haemolytic anaemia without Heinz bodies. (B) Hexokinase deficiency is a rare cause of haemolytic anaemia but not oxidant-triggered. (D) Glutathione synthetase deficiency is very rare; the trigger here is G6PD.

Final Answer: Glucose-6-phosphate dehydrogenase (G6PD) deficiency. $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q4

Q5.

Solution

Concept – Pantothenic acid (Vitamin B5): Pantothenic acid is a component of **coenzyme A (CoA)** and the **acyl carrier protein (ACP)** of the fatty acid synthase complex. CoA is indispensable for: acetyl-CoA formation, TCA cycle (succinyl-CoA), β-oxidation (acyl-CoA intermediates), and fatty acid synthesis. Pantothenic acid deficiency is rare (it is ubiquitous in food – "pantothen" means "from everywhere"), but when it occurs (e.g., in prisoners of war), it causes the **"burning feet syndrome"**: dysaesthesia and burning paraesthesias in the feet, along with headache, fatigue, and gastrointestinal symptoms.

Why the distractors are wrong: (A) Thiamine (B1) is a cofactor for TPP-dependent enzymes (PDC, α-KGDH, transketolase); deficiency causes beriberi/Wernicke's. (B) Niacin (B3) is the precursor of $NAD^+/NADP^+$; deficiency causes pellagra. (D) Biotin is the cofactor for carboxylases; deficiency (avidin binding) causes alopecia, dermatitis, and muscle hypotonia – not specifically "burning feet."

Final Answer: Pantothenic acid (Vitamin B5). $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q5

Q6.

Solution

Concept – Niemann-Pick disease Type A: Niemann-Pick disease Type A (acute neuronopathic form) is caused by deficiency of **sphingomyelinase (acid sphingomyelinase)** encoded by the *SMPD1* gene. This enzyme cleaves sphingomyelin into ceramide + phosphocholine. Without it, sphingomyelin accumulates in lysosomes of macrophages (creating foam cells) in the liver, spleen, lungs, and brain. Key clinical features: **hepatosplenomegaly, cherry-red spot, progressive neurodegeneration, and foamy macrophages** in bone marrow and lymph nodes. It predominantly affects Ashkenazi Jews and is fatal in infancy.

Why the distractors are wrong: (B) Glucocerebrosidase deficiency causes Gaucher disease (hepatosplenomegaly, "wrinkled tissue paper" cells, no cherry-red spot in Type I). (C) Hexosaminidase A deficiency causes Tay-Sachs (GM2 accumulation, no foam cells). (D) Arylsulphatase A deficiency causes metachromatic leukodystrophy.

Final Answer: Sphingomyelinase (Niemann-Pick type A). $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q6

Q7.

Solution

Concept – RNA Polymerase II and α-amanitin sensitivity: Eukaryotes have three nuclear RNA polymerases: **RNA Pol I** (synthesises 28S, 18S, 5.8S rRNA – nucleolus), **RNA Pol II** (synthesises pre-mRNA/hnRNA and most snRNAs), and **RNA Pol III** (synthesises tRNA, 5S rRNA, small RNAs). **RNA Pol II** is the target of α-amanitin at low/nanomolar concentrations; RNA Pol III is inhibited at higher concentrations; RNA Pol I is resistant. Since mRNA is the template for protein synthesis, RNA Pol II inhibition kills cells rapidly. α-Amanitin poisoning causes acute hepatic and renal failure after a latent period.

Why the distractors are wrong: (B) RNA Pol I synthesises rRNA in the nucleolus and is resistant to α-amanitin. (C) RNA Pol III (tRNA, 5S rRNA) is inhibited only at high α-amanitin concentrations. (D) Primase is a prokaryotic/eukaryotic RNA polymerase used during DNA replication to synthesise short RNA primers; not relevant here.

Final Answer: RNA polymerase II. $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q7

Q8.

Solution

Concept – Chylomicrons: Dietary triglycerides, digested in the gut by pancreatic lipase, are re-esterified in intestinal enterocytes and packaged into **chylomicrons**. Chylomicrons are assembled in the smooth ER of enterocytes. They contain **Apo B-48** (a truncated form of ApoB synthesised only in the intestine due to mRNA editing by apobec-1), ApoA-I, ApoA-IV, and acquire ApoC-II and ApoE from HDL in lymph. Chylomicrons enter the lymphatic lacteals and travel via the thoracic duct into the systemic circulation, bypassing the portal circulation. LPL (lipoprotein lipase) in peripheral tissues hydrolyses chylomicron triglycerides.

Why the distractors are wrong: (A) VLDL is assembled in the *liver* (contains ApoB-100) and carries endogenous triglycerides. (C) HDL is synthesised by the liver and intestine and participates in reverse cholesterol transport. (D) IDL is a remnant particle formed from VLDL after LPL action.

Final Answer: Chylomicrons. $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q8

Q9.

Solution

Concept – Cyanide poisoning (2,4-Dinitrophenol vs cyanide – this question is about cyanide): The "bitter almond" odour and bright red venous blood indicate **cyanide (CN^-) poisoning**. Cyanide binds tightly to **Fe^{3+} of cytochrome c oxidase (Complex IV)**, the terminal enzyme of the electron transport chain. This blocks electron transfer to O_2, halting the ETC and oxidative phosphorylation. Cells switch to anaerobic glycolysis, producing massive **lactic acidosis**. Paradoxically, venous blood remains oxygenated (bright red) because tissues cannot utilise O_2.

Treatment: Hydroxocobalamin (binds CN^- to form cyanocobalamin) is first-line. Alternatively, sodium nitrite (induces metHb, which scavenges CN^-) + sodium thiosulfate (provides sulphur for rhodanese to convert CN^- to thiocyanate).

Why the distractors are wrong: (A) Rotenone/amobarbital inhibit Complex I; not the mechanism of cyanide. (B) This describes 2,4-dinitrophenol (DNP), which is a proton ionophore uncoupler – it does not block the ETC but dissipates the proton gradient. (C) Oligomycin inhibits ATP synthase (Complex V) directly.

Final Answer: Binding to Fe^{3+} of cytochrome c oxidase (Complex IV), blocking electron transfer to O_2. $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q9

Q10.

Solution

Concept – Biotin deficiency (avidin in raw egg whites): Biotin (Vitamin B7) is the cofactor for all four mammalian carboxylase enzymes: (i) **pyruvate carboxylase** (pyruvate $\rightarrow$ OAA; gluconeogenesis), (ii) **acetyl-CoA carboxylase (ACC)** (acetyl-CoA $\rightarrow$ malonyl-CoA; fatty acid synthesis), (iii) **propionyl-CoA carboxylase** (propionyl-CoA $\rightarrow$ methylmalonyl-CoA; odd-chain FA and BCAA catabolism), and (iv) **3-methylcrotonyl-CoA carboxylase** (leucine catabolism). Biotin is covalently attached to the enzyme by biotinidase. **Avidin** in raw egg whites binds biotin with extraordinarily high affinity ($K_d \approx 10^{-15}$ M), preventing intestinal absorption. Deficiency signs: alopecia, seborrhoeic dermatitis, perioral/nasal rash, muscle hypotonia, and lactic acidosis (pyruvate carboxylase failure).

Why the distractors are wrong: (B) Riboflavin (B2) is not bound by avidin and serves as FAD/FMN – deficiency causes angular stomatitis. (C) Folate is not bound by avidin – deficiency causes megaloblastic anaemia and neural tube defects. (D) Thiamine is not bound by avidin – deficiency causes beriberi/Wernicke's.

Final Answer: Biotin (Vitamin B7). $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – Glucagon signalling cascade and glycogen breakdown: Glucagon binds its **G-protein coupled receptor (GPCR)** on hepatocytes, activating the stimulatory G-protein ($\mathbf{G_s}$). G_s activates **adenylyl cyclase**, converting ATP to **cyclic AMP (cAMP)**. cAMP binds the regulatory subunits of **protein kinase A (PKA)**, releasing active catalytic subunits. PKA phosphorylates and activates **phosphorylase kinase**, which in turn phosphorylates and activates **glycogen phosphorylase b** $\rightarrow$ **a**. The net result is hepatic glycogenolysis and glucose release.

Why the distractors are wrong: (B) The PI3K-Akt-GLUT4 pathway is the *insulin* signalling pathway in muscle and adipose – not glucagon in liver. (C) Glucagon activates (not inhibits) adenylyl cyclase via G_s – G_i inhibits adenylyl cyclase (e.g., as activated by somatostatin). (D) The IP_3/DAG pathway (PKC activation) is downstream of G_q-linked receptors (e.g., vasopressin V1, angiotensin II); glucagon acts via G_s and cAMP.

Final Answer: Glucagon $\rightarrow$ GPCR $\rightarrow$ Gs $\rightarrow$ adenylyl cyclase $\rightarrow$ cAMP $\rightarrow$ PKA $\rightarrow$ phosphorylase kinase activation. $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q11

Q12.

Solution

Concept – Acetyl-CoA carboxylase (ACC) – rate-limiting enzyme of fatty acid synthesis: Acetyl-CoA carboxylase (ACC) catalyses the carboxylation of acetyl-CoA to **malonyl-CoA**, the committed, rate-limiting step of *de novo* fatty acid synthesis in the cytosol. It is a **biotin-dependent** enzyme. Regulation: *allosteric activation* by **citrate** (signals high energy and acetyl-CoA availability); *allosteric inhibition* by **palmitoyl-CoA** (product feedback); *hormonal*: **insulin** promotes dephosphorylation and activation; **glucagon/epinephrine** activate AMPK, which phosphorylates and **inhibits** ACC. ACC also polymerises into active filaments when activated by citrate.

Why the distractors are wrong: (A) Fatty acid synthase (FAS) is a multi-enzyme complex that uses malonyl-CoA (provided by ACC) to extend the acyl chain – it is not the rate-limiting enzyme. (B) Malonyl-CoA decarboxylase reverses ACC's

action. (C) Citrate lyase provides acetyl-CoA in the cytosol from mitochondrial citrate – it is upstream of ACC.

Final Answer: Acetyl-CoA carboxylase (ACC). $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q12

Q13.

Solution

Concept – Allosteric (Cooperative) Enzymes: Allosteric enzymes are regulatory enzymes that differ fundamentally from simple Michaelis-Menten enzymes. They display **sigmoidal** (S-shaped) v vs [S] curves instead of hyperbolic curves. The Hill coefficient (n) is > 1 for positive cooperativity (binding of one substrate molecule facilitates binding of the next). They are regulated by: (i) **homotropic effectors** – the substrate itself (cooperative binding), and (ii) **heterotropic effectors** – molecules different from the substrate that bind allosteric sites to activate or inhibit the enzyme. Examples: haemoglobin (cooperative O_2 binding), ATCase (aspartate transcarbamoylase), phosphofructokinase-1. They are typically multisubunit proteins.

Why the distractors are wrong: (A) Michaelis-Menten kinetics apply to simple enzymes – allosteric enzymes follow Hill kinetics. (C) Allosteric enzymes typically have multiple subunits (oligomers). (D) Allosteric inhibition by end-products (feedback inhibition) is common but not always irreversible – it is usually reversible.

Final Answer: Sigmoidal kinetics with Hill coefficient $n > 1$; homotropic and heterotropic regulation. $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q13

Q14.

Solution

Concept – Cortisol and gluconeogenesis via PEPCK induction: Cortisol (glucocorticoid) promotes gluconeogenesis by acting as a transcriptional activator. It binds cytosolic glucocorticoid receptors (GR), and the cortisol-GR complex translocates to the nucleus and binds GREs (glucocorticoid response elements). **PEPCK (phosphoenolpyruvate carboxykinase)** is the key gluconeogenic enzyme that converts OAA to PEP (bypassing the irreversible pyruvate kinase step). Cortisol also induces glucose-6-phosphatase and amino acid transporters, promoting substrate delivery for gluconeogenesis. Chronically elevated cortisol (Cushing's

syndrome) causes steroid diabetes.

Why the distractors are wrong: (A) Cortisol does not activate glucokinase – it actually promotes gluconeogenesis and glycogenolysis. (B) Cortisol does have some anti-insulin effects, but the primary mechanism for gluconeogenesis is PEPCK gene induction. (D) Cortisol promotes glycogen breakdown (glycogenolysis) rather than synthesis.

Final Answer: Inducing the gene expression of PEPCK in the liver. $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q14

Q15.

Solution

Concept – Fabry disease (α-galactosidase A deficiency): Fabry disease is an X-linked lysosomal storage disorder caused by deficiency of **α-galactosidase A** (encoded by the *GLA* gene on Xq22.1). The enzyme cleaves terminal α-galactose residues from **globotriaosylceramide (Gb3/ceramide trihexoside)**. Without this enzyme, Gb3 accumulates in vascular endothelium, myocardium, renal glomeruli and tubuli, and the dorsal root ganglia. Clinical features: **episodic neuropathic pain** (burning acroparesthesias – worse with exercise, fever), **angiokeratomas** (bathing-trunk distribution), **progressive renal failure, cardiomyopathy, and stroke**. Males are severely affected; heterozygous females have variable expression. Enzyme replacement therapy (agalsidase alfa/beta) is available.

Why the distractors are wrong: (A) Sphingomyelinase deficiency = Niemann-Pick (foam cells, cherry-red spot, not acroparesthesias). (B) Hexosaminidase A deficiency = Tay-Sachs (GM2 ganglioside, cherry-red spot, neurodegeneration). (D) Glucocerebrosidase deficiency = Gaucher disease (hepatosplenomegaly, bone crises).

Final Answer: α-Galactosidase A. $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q15

Q16.

Solution

Concept – β-oxidation of unsaturated fatty acids: The standard β-oxidation spiral is designed for *fully saturated* fatty acyl-CoAs. When a *cis* double bond is encountered: (i) For **monounsaturated** FAs (e.g., oleic acid 18:1Δ^9-cis): after 3 turns of β-oxidation, a *cis*-Δ^3 intermediate is produced. **Enoyl-CoA isomerase** (3-cis to 2-trans enoyl-CoA isomerase) converts this to the *trans*-Δ^2 form needed

by enoyl-CoA hydratase, bypassing the first β-oxidation step (acyl-CoA dehydrogenase) and thus one $FADH_2$ is not produced in that round. (ii) For **polyunsaturated** FAs (e.g., linoleic acid $18{:}2\Delta^{9,12}$): in addition to enoyl-CoA isomerase, **2,4-dienoyl-CoA reductase** (using NADPH) is needed to handle the second double bond.

Why the distractors are wrong: (A) Thiokinase activates all fatty acids to acyl-CoA – it is not unique to unsaturated FA oxidation. (B) Enoyl-CoA hydratase is part of the standard β-oxidation cycle. (C) 3-Hydroxyacyl-CoA DH is part of standard β-oxidation.

Final Answer: Enoyl-CoA isomerase and 2,4-dienoyl-CoA reductase (NADPH). $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q16

Q17.

Solution

Concept – G6PD deficiency: inheritance and mechanism: G6PD deficiency is the most common human enzyme deficiency, affecting ~400 million people worldwide. It follows **X-linked recessive** inheritance (gene on Xq28); males are most severely affected, while females are usually carriers (with Lyon effect causing variable expression). The enzyme G6PD catalyses the first step of the **pentose phosphate pathway (PPP)**, generating **NADPH**. NADPH is required by glutathione reductase to regenerate **reduced glutathione (GSH)**. GSH is the primary antioxidant protecting erythrocytes (which lack mitochondria). In G6PD deficiency, oxidant stress (dapsone, primaquine, fava beans) overwhelms the reduced GSH capacity, causing oxidative haemoglobin denaturation (**Heinz bodies**), erythrocyte membrane damage (**bite cells**), and intravascular haemolysis.

Final Answer: X-linked recessive; G6PD deficiency impairs the pentose phosphate pathway, reducing NADPH and GSH. $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q17

Q18.

Solution

Concept – Fructose-2,6-bisphosphate as the key regulator of glycolysis/gluconeogenesis: Fructose-2,6-bisphosphate (F-2,6-BP) is a *regulatory* metabolite (not an intermediate of glycolysis or gluconeogenesis). It is synthesised and degraded by the **bifunctional enzyme PFK-2/FBPase-2**. F-2,6-BP is

the most potent allosteric **activator of PFK-1** (glycolysis ON) and simultaneously the most potent allosteric **inhibitor of FBPase-1** (gluconeogenesis OFF). Under insulin signalling, PFK-2 domain is active (dephosphorylated) $\rightarrow$ F-2,6-BP rises $\rightarrow$ glycolysis stimulated. Under glucagon signalling, PKA phosphorylates the bi-functional enzyme $\rightarrow$ FBPase-2 domain active $\rightarrow$ F-2,6-BP falls $\rightarrow$ gluconeogenesis favoured.

Why the distractors are wrong: (A) F-1,6-BP is an intermediate and also allosterically activates PFK-1 and pyruvate kinase, but it is not the key regulator described. (B) F-6-P is the substrate of PFK-1. (D) G-1-P is involved in glycogenolysis/glycogen synthesis, not the PFK-1/FBPase-1 switch.

Final Answer: Fructose-2,6-bisphosphate. $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q18

Q19.

Solution

Concept – α_1-Antitrypsin (AAT) deficiency: **α_1-Antitrypsin (AAT)** is a serine protease inhibitor (serpin) synthesised predominantly by hepatocytes. Its primary target is **neutrophil elastase** in the lung. In the ZZ (homozygous) phenotype, the **Z-mutation** (Glu342Lys) causes the AAT protein to misfold and polymerise, accumulating as **PAS-positive, diastase-resistant inclusions** in hepatocyte ER $\rightarrow$ **liver disease** (neonatal hepatitis, cirrhosis, hepatocellular carcinoma). Simultaneously, insufficient AAT reaches the lung $\rightarrow$ unopposed elastase destroys alveolar walls $\rightarrow$ **panacinar emphysema** (predominantly lower lobe, in non-smokers). Serum electrophoresis shows absent or very low α_1 fraction (AAT is the main α_1-globulin).

Why the distractors are wrong: (B) Cystic fibrosis involves the CFTR chloride channel – prominent bronchiectasis, mucus plugging, pancreatic insufficiency, no PAS inclusions. (C) Hereditary haemochromatosis involves iron overload; the HFE mutation causes transferrin receptor dysregulation – no inclusions in hepatocyte ER. (D) Wilson disease involves copper accumulation (ATP7B mutation) – Kayser-Fleischer rings, low ceruloplasmin.

Final Answer: α_1-Antitrypsin deficiency (ZZ phenotype). $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q19

Q20.

Solution

Concept – Leucine as the purely ketogenic amino acid: Amino acids are classified by the metabolic fate of their carbon skeletons. **Leucine** is the only *purely ketogenic* amino acid among all 20 standard amino acids – its catabolism yields **acetyl-CoA and acetoacetate** exclusively, with no net glucogenic intermediates. (Lysine is sometimes listed alongside leucine as purely ketogenic, but in most Indian exam curricula, leucine is the classic single answer for this question.) In contrast, isoleucine and phenylalanine are *both* glucogenic and ketogenic. Leucine is also the most powerful stimulator of mTOR (muscle protein synthesis) and the BCAA responsible for neurotoxicity in MSUD.

Why the distractors are wrong: (A) Isoleucine is *both* glucogenic and ketogenic (yields succinyl-CoA + acetyl-CoA). (B) Phenylalanine is *both* glucogenic (via tyrosine $\rightarrow$ fumarate) and ketogenic (via acetoacetate). (D) Lysine is purely ketogenic (yields only acetyl-CoA/acetoacetate), but the question's clinical scenario points specifically to isolated leucine elevation, making leucine the intended answer.

Final Answer: Leucine. $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q20

Q21.

Solution

Concept – Cyanide poisoning: site of action and antidote: Cyanide (CN^-) binds with high affinity to the **ferric (Fe^{3+}) form of cytochrome c oxidase** (Complex IV), the terminal electron acceptor in the mitochondrial ETC. This irreversibly blocks electron transfer to molecular O_2, halting oxidative phosphorylation in all cells. Clinical hallmarks: **lactic acidosis** (anaerobic glycolysis only), **paradoxically bright-red venous blood** (tissues cannot utilise O_2), and the smell of bitter almonds (HCN gas). High-risk groups: smoke inhalation victims, industrial chemical workers.

Antidote strategy: (1) **Hydroxocobalamin** (cobalt in vitamin B12 analogue avidly scavenges CN^- to form cyanocobalamin – preferred). (2) **Sodium nitrite** (induces metHaemoglobin, Fe^{3+} competes with cytochrome oxidase for CN^-) followed by **sodium thiosulfate** (rhodanese converts CN^- + thiosulfate $\rightarrow$ thiocyanate, renally excreted).

Final Answer: Complex IV (cytochrome c oxidase, Fe^{3+}); treat with hydroxocobalamin and/or sodium nitrite + thiosulfate. $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q21

Q22.

Solution

Concept – COX isoenzymes and NSAIDs: Arachidonic acid (released from membrane phospholipids by phospholipase A2) is converted by cyclooxygenase (COX) enzymes to prostaglandins. **COX-1** is constitutively expressed in most tissues; it produces **thromboxane A2** (platelet aggregation) and **prostacyclin PGI2** (gastric mucosal protection, vasodilation). **COX-2** is induced by inflammatory stimuli (TNF-α, IL-1β); it produces **PGE2 and PGI2** at inflammatory sites (pain, fever, vasodilation). **Traditional NSAIDs** (aspirin, ibuprofen, naproxen) inhibit *both* COX-1 and COX-2, explaining gastric side effects. **Celecoxib** is a *selective COX-2 inhibitor*, sparing COX-1-mediated gastric protection (but increases thrombotic risk by not inhibiting TXA2).

Why the distractors are wrong: (A) The roles are reversed – COX-1 is constitutive; COX-2 is inducible. (B) Celecoxib is a selective COX-2 inhibitor, not non-selective. (C) Aspirin irreversibly inhibits *both* COX-1 and COX-2 (acetylates the active site serine); it is not selective for COX-2.

Final Answer: COX-1 constitutive (gastric/platelet); COX-2 inducible (inflammation); selective COX-2 inhibitors spare gastric mucosa. $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q22

Q23.

Solution

Concept – Wilson disease (ATP7B mutation): Wilson disease is an autosomal recessive disorder of copper metabolism caused by mutations in **ATP7B**, which encodes a **P-type copper-transporting ATPase** in hepatocytes. ATP7B has two roles: (i) incorporates copper into ceruloplasmin for secretion, and (ii) excretes excess copper into bile. Without functional ATP7B, **copper accumulates** in the liver (hepatitis, cirrhosis), brain (basal ganglia – dysarthria, dystonia, psychiatric disturbance), cornea (**Kayser-Fleischer rings** – golden-brown deposits of copper in Descemet's membrane), and kidneys (Fanconi syndrome). **Serum ceruloplasmin is low** (paradoxically, because un-incorporated apo-ceruloplasmin is rapidly degraded). **24-hour urinary copper is elevated.** Treatment: penicillamine or trientine (copper chelators), zinc (blocks intestinal copper absorption).

Why the distractors are wrong: (A) HFE mutation causes hereditary haemochromatosis (iron overload, no KF rings). (B) HMBS mutation causes acute intermittent porphyria (abdominal pain, neuropsychiatric, no KF rings). (C) SERPINA1 mutation causes α_1-antitrypsin deficiency (emphysema, liver disease, no KF rings).

Final Answer: ATP7B gene mutation; defective copper-transporting ATPase. $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q23

Q24.

Solution

Concept – Citrulline transport in the urea cycle: In the urea cycle: (1) Carbamoyl phosphate + Ornithine $\xrightarrow{\text{OTC (mitochondria)}}$ **Citrulline**. (2) **Citrulline is then exported from the mitochondrial matrix to the cytoplasm** via a specific antiporter (citrulline/ornithine carrier). (3) In the cytoplasm: Citrulline + Aspartate $\xrightarrow{\text{AS synthetase, ATP}}$ Argininosuccinate. Steps 3 onwards occur in the cytoplasm. Ornithine is returned from the cytoplasm back into the mitochondria in exchange for citrulline. The compartmental division of the urea cycle is clinically important: OTC deficiency (most common urea cycle disorder) is X-linked and causes citrulline to be trapped in the mitochondria with ornithine accumulating.

Why the distractors are wrong: (A) Ornithine enters the mitochondria (the exchange transporter moves ornithine IN and citrulline OUT). (C) Arginine is a later cytoplasmic intermediate. (D) Argininosuccinate is formed in the cytoplasm from citrulline + aspartate.

Final Answer: Citrulline. $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q24

Q25.

Solution

Concept – C-reactive protein (CRP): C-reactive protein (CRP) is the prototypical acute-phase reactant. Key facts: (i) Synthesised exclusively by **hepatocytes** in response to IL-6 (and IL-1β, TNF-α). (ii) Plasma levels rise within **6 hours** of inflammation or tissue injury (much faster than ESR, which takes 24–48 hours). (iii) Half-life is $\sim$19 hours; falls rapidly when inflammation resolves. (iv) Functions as an **opsonin** (binds phosphocholine on bacteria/damaged cells) and **activates the classical complement pathway**. (v) In steady state, normal serum CRP < 5 mg/L; in severe sepsis, levels can reach > 500 mg/L. CRP is used to monitor response to anti-inflammatory therapy (e.g., biologics in rheumatoid arthritis).

Final Answer: CRP is synthesised by hepatocytes within 6 hours; it is an opsonin and activates complement. $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q25

Q26.

Solution

Concept – Lesch-Nyhan syndrome (HGPRT deficiency): Lesch-Nyhan syndrome is caused by virtually complete deficiency of **HGPRT (hypoxanthine-guanine phosphoribosyltransferase)**, encoded on the X chromosome (Xq26.2-Xq27.1). HGPRT is the key salvage enzyme that recycles hypoxanthine and guanine back to IMP and GMP using PRPP. Without it: (i) hypoxanthine and guanine are oxidised to **uric acid** (gout, nephrolithiasis); (ii) PRPP accumulates and drives *de novo* purine synthesis, compounding uric acid overproduction. The **neurological** features (choreoathetosis, intellectual disability, **compulsive self-mutilation**) are unique and distinguish it from ordinary gout. Inheritance: X-linked recessive; females are carriers, males are affected.

Why the distractors are wrong: (B) PRPP synthetase overactivity causes hyperuricaemia and gout but NOT self-mutilation; it is autosomal. (C) Adenosine deaminase deficiency causes SCID (immunodeficiency), not gout. (D) Von Gierke disease causes glycogen storage and hypoglycaemia, not the neurological-gout syndrome.

Final Answer: Lesch-Nyhan syndrome; HGPRT deficiency; X-linked recessive. $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q26

Q27.

Solution

Concept – 5' m^7G cap of mRNA: The **5' cap** is the first co-transcriptional modification of pre-mRNA in eukaryotes. It consists of a **7-methylguanosine (m^7G)** residue connected to the 5'-end of the transcript via an unusual **5'→5' triphosphate linkage** (not the standard 3'→5' phosphodiester bond). The cap is added by capping enzyme shortly after RNA polymerase II initiates transcription (when the nascent RNA is ∼20–30 nt long). Functions of the m^7G cap: (i) **protects mRNA from 5'→3' exonuclease degradation**; (ii) is recognised by **eIF4E** (cap-binding protein) for **cap-dependent translation initiation**; (iii) promotes nuclear export of mRNA; (iv) facilitates splicing of the first intron.

Why the distractors are wrong: (A) Poly-A tail is added at the *3'* end by poly-A polymerase, not the 5' end. (C) IRES elements are used by certain viral mRNAs (picornaviruses, HCV) as an alternative translation initiation mechanism – not

universal. (D) 3'-OH is the normal end of RNA transcripts – it has nothing to do with the 5' cap.

Final Answer: 7-methylguanosine (m^7G) cap in 5'→5' triphosphate linkage; recognised by eIF4E. ⇒ $\boxed{B}$

Answer: (B) Go Back to Q27

Q28.

Solution

Concept – S-Adenosylmethionine (SAM) as universal methyl donor: SAM is formed by the reaction of **methionine + ATP** (catalysed by methionine adenosyltransferase), with release of pyrophosphate and orthophosphate. SAM is the principal one-carbon donor for virtually all **transmethylation reactions** in the body: DNA methylation (CpG islands), histone methylation (epigenetics), RNA methylation, synthesis of creatine (from guanidinoacetate), synthesis of **epinephrine** (from norepinephrine, by PNMT), synthesis of **phosphatidylcholine** (by PEMT – methylation of PE), carnitine synthesis, and neurotransmitter inactivation. After donating its methyl group, SAM becomes **S-adenosylhomocysteine (SAH)**, which is then hydrolysed to **homocysteine** + adenosine.

Why the distractors are wrong: (A) THF accepts and transfers one-carbon units (formyl, methylene, methyl) but is not itself the universal methyl donor – 5-methyl-THF donates its methyl to homocysteine via methionine synthase to regenerate methionine. (B) 5-methyl-THF is the methyl donor for one specific reaction (remethylation of homocysteine) – not the universal donor. (C) Homocysteine is the product after SAM donates its methyl group.

Final Answer: S-Adenosylmethionine (SAM). ⇒ $\boxed{D}$

Answer: (D) Go Back to Q28

Q29.

Solution

Concept – ACC as rate-limiting enzyme of fatty acid synthesis and AMPK regulation: Acetyl-CoA carboxylase (ACC) is the committed step of fatty acid synthesis, converting acetyl-CoA to malonyl-CoA (requires biotin, CO_2, ATP). ACC resides in the **cytosol**. Energy-sensing regulation: **AMPK (AMP-activated protein kinase)** is activated when the AMP:ATP ratio rises (during energy deficit – starvation, exercise). AMPK **phosphorylates and inactivates ACC**, thereby shutting down fatty acid synthesis (an ATP-consuming process) and – since malonyl-CoA

inhibits carnitine palmitoyl transferase I (CPT-I) – removing the inhibition on β-oxidation. This switches the cell from anabolic (fat storage) to catabolic (fat oxidation) mode. Metformin (antidiabetic drug) activates AMPK, partly explaining its insulin-sensitising effects.

Why the distractors are wrong: (A) HMG-CoA reductase is the rate-limiting step of *cholesterol* synthesis (target of statins), not fatty acid synthesis. (C) Citrate lyase provides cytosolic acetyl-CoA from mitochondrial citrate – upstream of ACC. (D) Fatty acid synthase (FAS) carries out the elongation steps using malonyl-CoA – it is not the rate-limiting (committed) step.

Final Answer: Acetyl-CoA carboxylase (ACC). $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q29

Q30.

Solution

Concept – Alanine-glucose (Cahill) cycle: The **alanine-glucose cycle** (Cahill cycle) operates between **skeletal muscle** and **liver** during fasting and exercise. In muscle: (i) Protein catabolism releases amino acids. (ii) Branched-chain amino acid (BCAA) transamination generates glutamate $\rightarrow$ glutamate donates its amino group to pyruvate (from glycolysis) via **alanine aminotransferase (ALT)** $\rightarrow$ **alanine** is formed. (iii) Alanine enters the bloodstream (a non-toxic nitrogen carrier; unlike NH_3). In liver: (iv) Alanine undergoes transamination (ALT) $\rightarrow$ pyruvate + glutamate. Pyruvate enters **gluconeogenesis** $\rightarrow$ glucose exported back to muscle. (v) The amino group enters the urea cycle. This cycle serves dual purpose: glucose supply to muscle and non-toxic nitrogen transport from muscle to liver.

Why the distractors are wrong: (B) Alanine is not oxidised directly for ATP in the liver – it is transaminated to pyruvate which enters gluconeogenesis. (C) The Randle cycle (glucose-fatty acid cycle) governs fat/glucose balance; fatty acids are not transferred in the alanine cycle. (D) The direction is muscle $\rightarrow$ liver (alanine), liver $\rightarrow$ muscle (glucose) – not alanine from liver to muscle.

Final Answer: Muscle converts pyruvate + amino groups to alanine; alanine travels to liver for gluconeogenesis and urea cycle – non-toxic nitrogen transport. $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q30

Q31.

Solution

Concept – Type II restriction endonucleases: Type II restriction endonucleases are the workhorses of recombinant DNA technology. They: (i) recognise **specific palindromic DNA sequences** (typically 4–8 bp; e.g., EcoRI recognises GAATTC); (ii) **cleave within or adjacent to** the recognition sequence; (iii) generate either **blunt ends** (e.g., EcoRV) or **sticky/cohesive ends** (5'-overhangs: e.g., EcoRI; 3'-overhangs: e.g., KpnI); (iv) require only Mg^{2+} as cofactor – **no ATP hydrolysis** (unlike Type I and III which require ATP). Sticky ends are used for directional cloning because complementary overhangs from different DNA sources can be ligated.

Why the distractors are wrong: (A) Type I and Type III restriction enzymes require ATP and cut at distant sites – not Type II. (B) CRISPR-Cas9 uses single-guide RNA; it is an RNA-guided DNA nuclease – not a restriction endonuclease. (C) DNA methyltransferases (part of the restriction-modification system) methylate the host DNA to protect it – they are separate enzymes, not the endonucleases themselves.

Final Answer: Type II restriction endonucleases cleave dsDNA at specific palindromic sequences; produce blunt or sticky ends; used as molecular scissors. $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q31

Q32.

Solution

Concept – Taq polymerase and PCR: Polymerase chain reaction (PCR) was developed by Kary Mullis (Nobel Prize 1993). The key breakthrough was using a **thermostable DNA polymerase** isolated from ***Thermus aquaticus***, a bacterium living in hot springs (Yellowstone National Park, ~70–75°C). This **Taq polymerase** remains active at the 94°C denaturation step, eliminating the need to add fresh enzyme each cycle. Taq polymerase lacks 3'→5' proofreading exonuclease activity (higher error rate); for high-fidelity applications, **Pfu polymerase** (from *Pyrococcus furiosus*) with proofreading activity is used.

Why the distractors are wrong: (A) E. coli DNA polymerase I/III are mesophilic (37°C optimum) and are denatured at 94°C – not suitable for PCR. (B) Pfu polymerase (from Pyrococcus furiosus) is indeed used in PCR but is not the original/standard Taq polymerase. (D) Bacillus subtilis is a mesophilic spore-former – not the source of any thermostable polymerase.

Final Answer: Thermus aquaticus (gives Taq polymerase). $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q32

Q33.

Solution

Concept – Isoniazid (INH) and vitamin B6 (pyridoxine) deficiency: Isoniazid (INH), the cornerstone of TB treatment, is a **hydrazide**. INH reacts chemically with **pyridoxal phosphate (PLP/B6)**, the active form of pyridoxine, forming hydrazones that are excreted in urine. This creates a **functional B6 deficiency**. PLP is the cofactor for: (i) aminotransferases (ALT, AST), (ii) decarboxylases (DOPA decarboxylase $\rightarrow$ dopamine; glutamate decarboxylase $\rightarrow$ GABA), (iii) δ-aminolaevulinic acid (ALA) synthase. PLP deficiency from INH causes: **peripheral neuropathy** (most common), sideroblastic anaemia, and rarely convulsions. **Prevention:** co-administer **pyridoxine (25–50 mg/day)** with INH, especially in those at high risk (diabetics, alcoholics, malnourished, pregnant).

Why the distractors are wrong: (A) INH does not inhibit B12 absorption; B12 deficiency causes subacute combined degeneration of the cord – a different picture. (B) Zinc chelation and acrodermatitis enteropathica is not associated with INH. (D) INH does not inhibit thiamine – Wernicke's encephalopathy is caused by thiamine deficiency in alcoholics.

Final Answer: INH reacts with PLP (vitamin B6) causing relative B6 deficiency; supplement pyridoxine. $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q33

Q34.

Solution

Concept – Selenium and glutathione peroxidase (Keshan disease): Selenium is an essential trace mineral incorporated as **selenocysteine** (the 21st amino acid) into the active site of **glutathione peroxidase (GPx)**. GPx catalyses: $2GSH + H_2O_2 \rightarrow GSSG + 2H_2O$, neutralising **reactive oxygen species (ROS)**. Selenium is also a component of thioredoxin reductase and iodothyronine deiodinase (T4 $\rightarrow$ T3 conversion). **Selenium deficiency** causes: (i) **Keshan disease** – dilated cardiomyopathy, endemic to selenium-poor soils of Keshan county, China; (ii) Kashin-Beck disease (osteochondropathy); (iii) immune impairment.

Why the distractors are wrong: (B) Zinc is a cofactor for carbonic anhydrase, carboxypeptidase, alcohol dehydrogenase, and > 300 other enzymes; deficiency

causes acrodermatitis enteropathica and growth retardation. (C) Copper is a component of ceruloplasmin, cytochrome c oxidase, and superoxide dismutase; deficiency causes anaemia and Menkes disease. (D) Manganese is a cofactor for arginase, pyruvate carboxylase, and mitochondrial superoxide dismutase (Mn-SOD); deficiency is rare.

Final Answer: Selenium. $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q34

Q35.

Solution

Concept – Multi-system biochemical pathology of PKU: In **phenylketonuria (PKU)**, phenylalanine hydroxylase (PAH) deficiency causes accumulation of **phenylalanine (Phe)**. Elevated Phe causes multi-system pathology through **competitive inhibition of enzymes** that normally use tyrosine or tryptophan: (i) **Inhibition of tyrosine hydroxylase** (tyrosine $\rightarrow$ DOPA $\rightarrow$ dopamine): reduces **melanin synthesis** in melanocytes $\rightarrow$ **fair skin, blond hair, blue eyes** (hypopigmentation). (ii) **Inhibition of DOPA decarboxylase (AADC)** and **tryptophan hydroxylase**: reduces **dopamine and serotonin** biosynthesis $\rightarrow$ **intellectual disability, seizures, microcephaly**. (iii) Accumulated Phe is shunted to phenylpyruvate, phenylacetate (phenylketones) $\rightarrow$ musty/mousy urine odour $\rightarrow$ positive Guthrie test and ferric chloride test.

Treatment: Low-Phe diet (sufficient for protein synthesis but no excess) with tyrosine supplementation (now essential). Sapropterin (BH4 cofactor supplement) for BH4-responsive PKU variants.

Final Answer: Elevated Phe inhibits tyrosine hydroxylase (hypopigmentation) and aromatic amino acid decarboxylase (reduced dopamine/serotonin $\rightarrow$ intellectual disability/seizures). $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q35

Q36.

Solution

Concept – Phosphatidylcholine (lecithin): **Phosphatidylcholine (PC)**, also known as lecithin, is the **most abundant phospholipid** in mammalian cell membranes, comprising ~40–50% of total phospholipids. Biosynthetic pathways: (i) **CDP-choline (Kennedy) pathway**: choline $\xrightarrow{\text{CK}}$ phosphocholine $\xrightarrow{\text{CCT}}$ CDP-choline $\xrightarrow{\text{CPT}}$ PC + CMP. CCT (CTP:phosphocholine cytidylyltransferase) is the

rate-limiting step. (ii) **PEMT pathway**: phosphatidylethanolamine is sequentially methylated 3 times using **SAM** by phosphatidylethanolamine N-methyltransferase (PEMT) – mainly in liver. PC is the major component of pulmonary surfactant (with sphingomyelin) and of bile lipids (enables cholesterol solubilisation). **Lecithin:sphingomyelin (L:S) ratio** in amniotic fluid ≥ 2 indicates fetal lung maturity.

Why the distractors are wrong: (A) Phosphatidylethanolamine (PE) is the second most abundant phospholipid. (C) Phosphatidylserine (PS) is found mainly in the inner leaflet of cell membranes; its externalisation signals apoptosis. (D) Cardiolipin (diphosphatidylglycerol) is unique to the inner mitochondrial membrane (and bacterial membranes).

Final Answer: Phosphatidylcholine (PC, lecithin). $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q36

Q37.

Solution

Concept – Acute Intermittent Porphyria (AIP) – HMBS deficiency: AIP is the most common acute hepatic porphyria. It is caused by ~50% deficiency of **hydroxymethylbilane synthase (HMBS)**, also called porphobilinogen deaminase. This causes **porphobilinogen (PBG)** and ALA to accumulate during attacks. Crucially, **ALA synthase-1 (ALAS-1)**, the rate-limiting enzyme of haem biosynthesis, is normally repressed by free haem. In AIP, because haem production is impaired, ALAS-1 is **derepressed** $\rightarrow$ more ALA and PBG are produced. Precipitants (fasting, drugs, hormones) further induce ALAS-1 via increased mRNA transcription. Clinical: **abdominal pain** (colicky, severe), **neuropsychiatric** (confusion, psychosis), **peripheral motor neuropathy**, **dark/port-wine urine** (PBG oxidises to porphyrins). No photosensitivity (because porphyrins don't accumulate in skin).

Why the distractors are wrong: (A) Uroporphyrinogen III synthase deficiency causes congenital erythropoietic porphyria (CEP) – severe cutaneous photosensitivity, haemolysis – not acute neurological attacks. (B) Ferrochelatase deficiency causes erythropoietic protoporphyria (EPP) – painful photosensitivity – not acute attacks. (C) PBG deaminase (HMBS) *deficiency* (not overactivity) is the cause of AIP.

Final Answer: HMBS (PBG deaminase) deficiency; PBG accumulation; ALAS-1 derepressed by fasting/drugs. $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q37

Q38.

Solution

Concept – Haemosiderin vs ferritin in iron storage: Iron is stored intracellularly in two forms: (i) **Ferritin**: a soluble, dynamic iron-storage complex; 24 subunits of H and L chains form a hollow shell (apoferritin) that can store up to 4,500 iron atoms as ferric oxyhydroxide. Ferritin is rapidly mobilisable and reflects total body iron stores (serum ferritin correlates with iron status). (ii) **Haemosiderin**: an insoluble, partially degraded and aggregated form of ferritin; iron is less readily mobilisable. It appears as **golden-brown, granular deposits** in macrophages and hepatocytes and stains bright blue with **Prussian blue (Perls' stain)** – used histologically to diagnose iron overload (haemochromatosis, haemosiderosis).

Why the distractors are wrong: (A) Transferrin is the plasma iron-transport glycoprotein (not a storage form). (B) Myoglobin is the muscle oxygen-binding protein (contains iron in haem – not a storage form). (C) While ferritin is indeed a storage form, the question describes the insoluble Prussian-blue-positive form seen in iron overload, which is haemosiderin.

Final Answer: Haemosiderin; insoluble, degraded ferritin; Prussian blue staining; seen in iron overload. $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q38

Q39.

Solution

Concept – Serine proteases: catalytic triad and mechanism: Serine proteases are a large family of enzymes characterised by an active site **catalytic triad: Ser-His-Asp**. The charge relay system works as follows: Asp (negatively charged) stabilises His (imidazole), which acts as a base to deprotonate Ser's hydroxyl group, making it a powerful nucleophile. Nucleophilic Ser attacks the **electrophilic carbonyl** of the scissile peptide bond $\rightarrow$ tetrahedral intermediate $\rightarrow$ acyl-enzyme intermediate (amino acid product released) $\rightarrow$ water attacks the acyl-enzyme (deacylation) $\rightarrow$ carboxylic acid product released, Ser regenerated. Examples: **chymotrypsin** (cleaves after aromatic/large hydrophobic residues), **trypsin** (cleaves after Arg/Lys), **elastase** (cleaves after small aliphatic residues), **thrombin** (cleaves fibrinogen), plasmin, kallikrein.

Why the distractors are wrong: (B) Cysteine proteases use a thiol group (Cys) as the nucleophile – examples: caspases, papain, cathepsin B. (C) Aspartyl proteases use two Asp residues – examples: pepsin, renin, HIV protease. (D) Metalloproteinases (Zn^{2+}) – examples: carboxypeptidase A and B, collagenase, neprilysin.

Final Answer: Serine proteases; catalytic triad Ser-His-Asp; nucleophilic serine mechanism. $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q39

Q40.

Solution

Concept – Transferrin (iron transport protein): Transferrin is a β_1-globulin glycoprotein ($M_r \approx 79$ kDa) synthesised by hepatocytes. It has **two iron-binding sites**, each binding one Fe^{3+} ion. In normal health, transferrin is approximately **33% saturated** (i.e., only 1/3 of the binding capacity carries iron). **TIBC** (total iron-binding capacity) reflects total transferrin concentration in plasma. Changes in iron status: in **iron deficiency**, the liver produces more transferrin (elevated TIBC) and saturation falls ($< 20\%$). In **chronic disease** (anaemia of chronic disease), ferritin is high (iron sequestered) and transferrin is low (negative acute-phase reactant). In **haemochromatosis**, transferrin saturation is $> 50 - -60\%$.

Why the distractors are wrong: (A) Ferritin is the *intracellular* iron-storage protein – elevated in iron overload. (C) Lactoferrin is an iron-binding protein in secretions (milk, tears, saliva) and neutrophil granules; it is part of innate immunity, not plasma transport. (D) Haemopexin binds free haem (haem scavenger) in plasma – not iron transport per se.

Final Answer: Transferrin. $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q40

Answer Key – FMGE Biochemistry Sample Paper 2

1	A	2	C	3	B	4	C	5	C
6	A	7	A	8	B	9	D	10	A
11	A	12	D	13	B	14	C	15	C
16	D	17	B	18	C	19	A	20	C
21	B	22	D	23	D	24	B	25	C
26	A	27	B	28	D	29	B	30	A
31	D	32	C	33	C	34	A	35	D
36	B	37	D	38	D	39	A	40	B