FMGE Biochemistry Sample Paper – 6

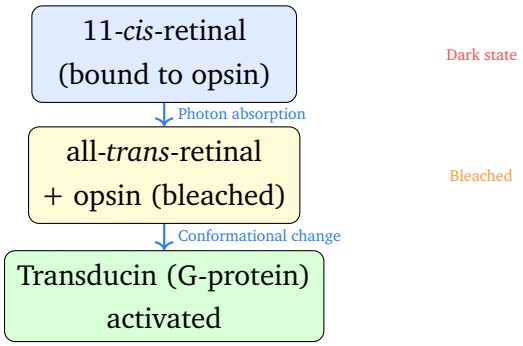
Duration: 60 Minutes **Maximum Marks: 40**

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 55-year-old man undergoes dark-adaptation testing and is found to have elevated threshold for vision in dim light. His serum retinol is low. In a normally functioning rod cell, when a photon strikes rhodopsin, which immediate photochemical event occurs?



(A) Isomerisation of 11-*cis*-retinal to all-*trans*-retinal, activating transducin

(B) Conversion of all-*trans*-retinal to 11-*cis*-retinal

(C) Hydrolysis of rhodopsin by a lysosomal enzyme

(D) Phosphorylation of retinal by rhodopsin kinase in the dark

Q2. An enzyme obeys Michaelis-Menten kinetics with a K_m of 2 mmol/L and V_{max} of 100 nmol/min. When the substrate concentration equals the K_m, which statement correctly describes the reaction velocity?

(A) $v = V_{max}/2 = 50$ nmol/min (first-order behaviour transitions to zero-order)

(B) $v = V_{max} = 100$ nmol/min (enzyme is fully saturated)

(C) $v = 0$ (no substrate binding occurs at K_m)

(D) $v = 2\,V_{max}$ (excess substrate doubles the rate)

Q3. A 7-year-old boy presents with coarse facial features (gargoylism), hepatosplenomegaly, joint stiffness, and cardiac valvular disease. There is no corneal clouding. He has recurrent upper respiratory tract infections. Urine shows elevated dermatan sulfate and heparan sulfate. His elder sister is similarly affected. The enzyme deficiency is:

(A) α-L-Iduronidase (Hurler syndrome, MPS I)

(B) Iduronate-2-sulphatase (Hunter syndrome, MPS II)

(C) Arylsulphatase A (metachromatic leukodystrophy)

(D) Galactosamine-6-sulphatase (Morquio syndrome, MPS IVA)

Q4. A patient with type 2 diabetes is found to have impaired glycogen synthesis after meals. The activated glucose donor used by glycogen synthase to elongate a glycogen chain is formed by which reaction?

(A) Glucose-6-phosphate + UTP $\rightarrow$ UDP-glucose + PP_i (direct)

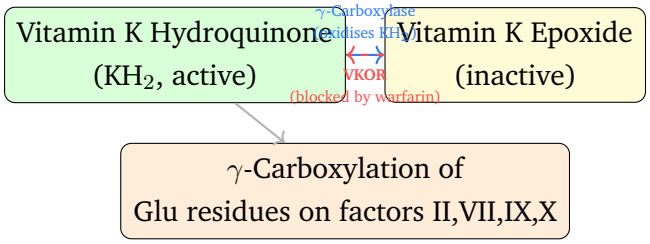
(B) Glucose-1-phosphate + UTP $\rightarrow$ UDP-glucose + PP_i (UDP-glucose pyrophosphorylase)

(C) Glucose + ATP $\rightarrow$ glucose-1-phosphate + ADP

(D) Glucose-6-phosphate $\rightarrow$ glucose-1-phosphate + free phosphate (phosphatase)

Q5. A 68-year-old man on warfarin therapy for atrial fibrillation develops supratherapeutic anticoagulation (INR 7.2) after starting a new antibi-

otic. Warfarin acts by blocking vitamin K recycling. In the normal vitamin K cycle, which enzyme regenerates active vitamin K hydroquinone from vitamin K epoxide?



(A) Vitamin K epoxide reductase (VKOR)

(B) γ-Glutamyl carboxylase

(C) Protein disulfide isomerase

(D) Thioredoxin reductase

Q6. A 4-year-old boy has recurrent episodes of vomiting, irritability, and hyperammonemia. His ammonia rises to 340 μmol/L (normal $<$50). Urine organic acid analysis shows a large peak of argininosuccinic acid. Plasma shows elevated citrulline and argininosuccinate. The enzyme deficiency and the step it catalyses in the urea cycle are:

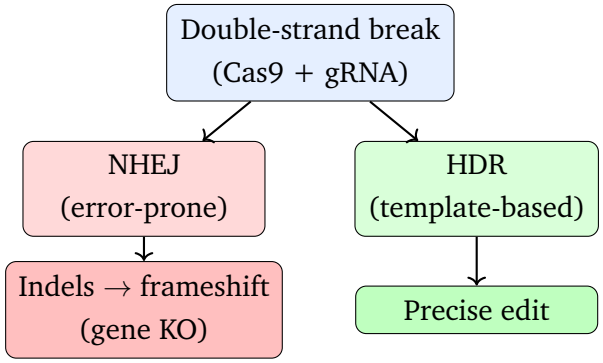
(A) Ornithine transcarbamylase – formation of citrulline from ornithine

(B) Argininosuccinate synthetase – condensation of citrulline with aspartate

(C) Arginase – hydrolysis of arginine to urea + ornithine

(D) Argininosuccinate lyase – cleavage of argininosuccinate to arginine + fumarate

Q7. A molecular biology laboratory uses CRISPR-Cas9 to introduce a targeted deletion in the PCSK9 gene. The guide RNA directs Cas9 to the target sequence. After Cas9 creates a double-strand break (DSB), which repair mechanism introduces small insertions or deletions (indels) that disrupt the reading frame?



(A) Homology-directed repair (HDR) using a donor template

(B) Base excision repair (BER)

(C) Nucleotide excision repair (NER)

(D) Non-homologous end joining (NHEJ)

Q8. A 14-year-old girl presents with recurrent severe abdominal pain (pancreatitis), eruptive xanthomas, and lipaemia retinalis. Fasting triglyceride level is 4500 mg/dL. Plasma appears milky-white (lactescent). After incubation at 4°C overnight, a cream layer forms on top. Lipoprotein electrophoresis shows a chylomicron band only. The primary enzyme/cofactor deficiency in familial type I hyperlipoproteinaemia is:

(A) VLDL receptor deficiency

(B) LDL receptor deficiency (familial hypercholesterolaemia)

(C) Lipoprotein lipase (LPL) deficiency, or ApoC-II deficiency (its activator)

(D) LCAT (lecithin-cholesterol acyltransferase) deficiency

Q9. A hepatocyte generates large quantities of acetyl-CoA from fatty acid oxidation during fasting. Which statement correctly describes why the liver **produces** ketone bodies but **cannot utilise** them?

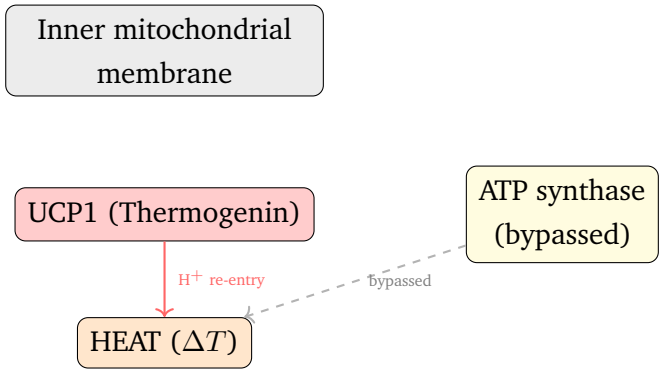
(A) Liver lacks HMG-CoA synthase, so it cannot form HMG-CoA

(B) Liver lacks succinyl-CoA transferase (thiophorase), so it cannot activate acetoacetate to acetoacetyl-CoA

(C) Liver lacks β-hydroxybutyrate dehydrogenase, so it cannot form β-hydroxybutyrate

(D) Liver lacks HMG-CoA lyase, so it cannot form acetoacetate

Q10. A neonate with unusually high heat production in cold weather is examined by a paediatric intensivist. Brown adipose tissue (BAT) contributes significantly to non-shivering thermogenesis in newborns. The key protein in BAT that generates heat instead of ATP is:



(A) Adenine nucleotide translocase (ANT)

(B) Uncoupling protein 1 (UCP1, thermogenin)

(C) Cytochrome c oxidase (Complex IV)

(D) Succinate dehydrogenase (Complex II)

Q11. A 35-year-old woman recovering from surgery in the ICU has a serum albumin of 2.1 g/dL (normal 3.5–5 g/dL). Her nutritional status is being reassessed. Which plasma protein has a half-life of approximately 2 days, making it a more sensitive and rapidly responsive marker of nutritional status than albumin?

(A) Transthyretin (prealbumin)

(B) Transferrin (half-life ∼8–10 days)

(C) Retinol-binding protein (RBP, but requires transthyretin as carrier)

(D) Fibronectin

Q12. Phosphatidylethanolamine (PE) can be converted to phosphatidylcholine (PC) in the liver by sequential methylation. PE is formed from phosphatidylserine (PS) by decarboxylation. Which enzyme catalyses the conversion of phosphatidylserine to phosphatidylethanolamine?

(A) Phosphatidylethanolamine N-methyltransferase (PEMT)

(B) Phosphatidylserine decarboxylase (PSD)

(C) CDP-choline:diacylglycerol phosphocholine transferase

(D) Sphingomyelin synthase

Q13. A 9-month-old infant presents with vomiting and severe hypoglycaemia 30–40 minutes after ingestion of fruit juice. He has hepatomegaly and elevated liver enzymes. His older sibling died of liver failure. Urine shows elevated fructose-1-phosphate. The deficient enzyme in hereditary fructose intolerance (HFI) and how it differs from benign essential fructosuria are:

(A) Fructokinase deficiency in HFI; aldolase B deficiency in essential fructosuria

(B) Fructose-1,6-bisphosphatase in HFI; fructokinase in essential fructosuria

(C) Aldolase B in HFI (fructose-1-phosphate accumulates, liver damage); fructokinase in essential fructosuria (fructose in urine, benign)

(D) Aldolase A in HFI; aldolase B in essential fructosuria

Q14. A biochemistry student is asked to identify the structure of the most abundant saturated fatty acid in the human body, which is also the end-product of de novo fatty acid synthesis by fatty acid synthase (FAS). Which of the following correctly describes palmitic acid?

(A) 18-carbon saturated fatty acid (stearic acid, C18:0)

(B) 16-carbon saturated fatty acid (hexadecanoic acid, C16:0)

(C) 16-carbon monounsaturated fatty acid (palmitoleic acid, C16:1)

(D) 14-carbon saturated fatty acid (myristic acid, C14:0)

Q15. Niemann-Pick disease type A results from sphingomyelinase deficiency, causing sphingomyelin accumulation. In normal cells, sphingomyelin

is synthesised by transfer of a phosphocholine headgroup from phosphatidylcholine to ceramide. What is the other product of this reaction catalysed by sphingomyelin synthase?

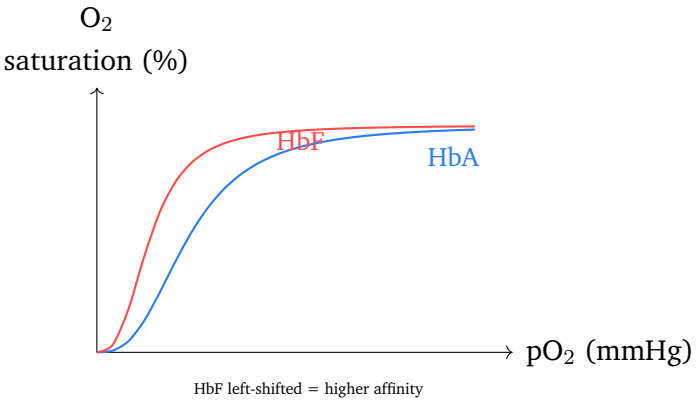
(A) Phosphatidylethanolamine

(B) Diacylglycerol (DAG)

(C) Ceramide-1-phosphate

(D) Lysophosphatidylcholine

Q16. A neonatologist explains to a medical student why fetal haemoglobin (HbF) has a higher oxygen affinity than adult haemoglobin (HbA) in the placental circulation. Which molecular mechanism accounts for the higher O_2 affinity of HbF?



(A) HbF has α-chains replaced by ζ-chains, which bind O_2 more tightly

(B) HbF has a higher iron content per haem group

(C) HbF has β-chains replaced by ϵ-chains in embryonic life only

(D) HbF has γ-chains (instead of β-chains) that bind 2,3-BPG less effectively, reducing its allosteric inhibition

Q17. An infant presents with persistent hyperinsulinism and hyperammonaemia (HI/HA syndrome). Genetic testing reveals a gain-of-function mutation in the enzyme that links amino acid catabolism to the TCA cycle via reversible oxidative deamination of glutamate. This enzyme is:

(A) Glutamate dehydrogenase (GDH) – glutamate $\rightleftharpoons$ α-ketoglutarate + NH_3

(B) Glutamine synthetase – glutamate + NH_3 $\rightarrow$ glutamine

(C) Alanine aminotransferase (ALT) – alanine + α-KG $\rightarrow$ pyruvate + glutamate

(D) Glutaminase – glutamine $\rightarrow$ glutamate + NH_3

Q18. During prolonged fasting, the liver switches from glycolysis to gluconeogenesis. Citrate, exported from mitochondria to the cytoplasm, serves as a key allosteric signal. Which TWO regulatory effects does citrate exert in the cytoplasm?

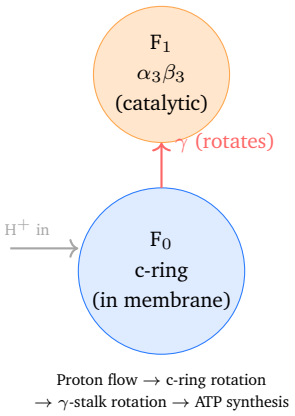
(A) Activates PFK-1 (stimulates glycolysis) and inhibits acetyl-CoA carboxylase (inhibits FA synthesis)

(B) Inhibits glycogen synthase and activates pyruvate kinase

(C) Inhibits PFK-1 (inhibits glycolysis) and activates acetyl-CoA carboxylase (stimulates FA synthesis)

(D) Activates HMG-CoA reductase and inhibits pyruvate dehydrogenase

Q19. ATP synthase (Complex V) uses the proton-motive force (PMF) generated by the ETC to synthesise ATP. The binding-change mechanism proposed by Paul Boyer states that the three β-subunits cycle through conformational states. Which subunit rotation drives this conformational change?



(A) The α-subunits rotate relative to the β-subunits

(B) The β-subunits rotate around a fixed γ-subunit

(C) The γ-subunit (central stalk) rotates inside the $\alpha_3\beta_3$ ring, driven by c-ring rotation

(D) The F_0 c-ring rotates without moving the γ-subunit

Q20. A patient with abetalipoproteinaemia cannot absorb fat-soluble vitamins and has steatorrhoea, acanthocytosis, and ataxia. His condition results from a mutation in MTP (microsomal triglyceride transfer protein), which is essential for VLDL assembly. Which apolipoproteins are structural components of VLDL secreted from the liver?

(A) ApoA-I and ApoA-II (predominantly HDL apolipoproteins)

(B) ApoC-I and ApoE only (no ApoB)

(C) ApoB-48, ApoC-II, and ApoE (intestinal lipoprotein pattern)

(D) ApoB-100, ApoC-II, and ApoE

Q21. A 45-year-old obese patient with type 2 diabetes has elevated serum free fatty acids. Insulin suppresses lipolysis in adipose tissue. The hormone-sensitive lipase (HSL) is regulated by a cAMP-PKA cascade. Which statement correctly describes how insulin inhibits lipolysis?

(A) Insulin activates adenylyl cyclase, raising cAMP, which activates protein phosphatase to dephosphorylate HSL

(B) Insulin directly binds HSL and inhibits its catalytic domain

(C) Insulin activates phosphodiesterase (via Akt), lowering cAMP, reducing PKA activity, and decreasing HSL phosphorylation (active form)

(D) Insulin promotes fatty acid oxidation, consuming fatty acids before they leave the adipocyte

Q22. Glucuronic acid is synthesised from glucose and serves important conjugation and structural functions in the body. Which of the following best describes the activated form of glucuronic acid used in conjugation reactions?

(A) UDP-glucuronic acid (formed from UDP-glucose by UDP-glucose dehydrogenase)

(B) Glucuronic acid-1-phosphate (formed directly from glucose-1-phosphate)

(C) ADP-glucuronate (formed by ADP-glucose pyrophosphorylase)

(D) CMP-glucuronate (analogous to CMP-sialic acid)

Q23. A 3-year-old child has hepatomegaly, hypoglycaemia with fasting, and accumulation of limit dextrins in the liver. Muscle function is normal. Enzyme assay confirms deficiency of the debranching enzyme (amylo-1,6-glucosidase + glucan-1,4-1,4-glucan transferase). Which glycogen storage disease does this represent, and which is considered the mildest hepatic GSD?

(A) Type I (Von Gierke) is this child; Type VI is the mildest

(B) Type II (Pompe) is this child; Type V is the mildest

(C) Type V (McArdle) is this child; Type I is the mildest

(D) Type III (Cori) is this child; Type VI (Hers/phosphorylase kinase deficiency) is the mildest hepatic GSD

Q24. A 25-year-old woman presents with deep venous thrombosis and is found to have elevated plasma homocysteine (65 μmol/L, normal $<$15). She is deficient in vitamin B6. Which of the following metabolic pathways of homocysteine requires vitamin B6 (as pyridoxal phosphate)?

(A) Remethylation of homocysteine to methionine via methionine synthase (requires B12 and 5-methyl-THF)

(B) Remethylation via betaine-homocysteine methyltransferase (BHMT) in the liver

(C) Transsulfuration: homocysteine + serine $\rightarrow$ cystathionine via cystathionine β-synthase (CBS, requires B6)

(D) Oxidation of homocysteine to homocysteic acid (non-enzymatic, O_2-dependent)

Q25. A strict vegan who has avoided all animal products for 12 years presents with subacute combined degeneration of the spinal cord. His serum B12 is undetectable. Which statement about body vitamin B12 stores is most accurate?

(A) Body stores are approximately 50 μg, sufficient for only 1–2 months

(B) Stores are in red blood cells and depleted within weeks of dietary abstinence

(C) B12 is not stored; it is utilised immediately upon absorption

(D) Liver stores 2–5 mg, sufficient for 3–5 years; hence deficiency takes years to develop in adult vegans

Q26. A pharmacologist studies calcium signalling downstream of the IP_3 pathway. Calmodulin is a ubiquitous calcium-sensing protein that, upon binding Ca^{2+}, activates multiple target enzymes. How many calcium ions does calmodulin bind, and what is the structural motif used for calcium binding?

(A) Four Ca^{2+} ions via four EF-hand (helix-loop-helix) motifs; the Ca^{2+}-calmodulin complex activates CaM kinases, eNOS, and phosphodiesterase

(B) Two Ca^{2+} ions via two zinc-finger domains

(C) Eight Ca^{2+} ions via annexin repeats

(D) One Ca^{2+} ion via a C2 domain (like protein kinase C)

Q27. In the genetic code, 61 codons encode amino acids but only $\sim$45 tRNA species exist in humans. Francis Crick proposed the wobble hypothesis to explain how one tRNA can read multiple codons. At the wobble position, the modified nucleoside inosine (I) can base-pair with which three bases?

(A) I pairs only with C (strict Watson-Crick pairing)

(B) I pairs with U and G (two bases)

(C) I pairs with U, C, and A (wobble position is position 34 of anticodon)

(D) I pairs with A, G, and U (all pyrimidines and purines)

Q28. A patient with HIV is being initiated on antiretroviral therapy. The HIV reverse transcriptase has unique features exploited by antiviral drugs.

Which statement correctly describes the mechanism of reverse transcriptase and its vulnerability?

(A) Reverse transcriptase is an RNA-directed RNA polymerase; NRTIs mimic ribonucleotides

(B) Reverse transcriptase has 3'→5' proofreading exonuclease activity, keeping mutation rate low

(C) Reverse transcriptase integrates the proviral DNA into the host genome (integrase function)

(D) Reverse transcriptase is an RNA-dependent DNA polymerase with RNase H activity and **no** proofreading → high mutation rate; NRTIs and NNRTIs are key drug targets

Q29. A researcher studying oxidative stress measures lipid peroxidation products in plasma from ICU patients. Lipid peroxidation is initiated by free radicals attacking polyunsaturated fatty acids (PUFAs). Which marker is considered the most specific and reliable *in vivo* indicator of lipid peroxidation?

(A) Malondialdehyde (MDA) – measured by TBARS assay (thiobarbituric acid reactive substances)

(B) 4-Hydroxynonenal (4-HNE) – reactive aldehyde from ω-6 PUFA peroxidation

(C) Isoprostanes (8-iso-$PGF_{2\alpha}$) – formed from arachidonic acid by non-enzymatic free radical peroxidation *in vivo*

(D) Diene conjugates – early products of PUFA peroxidation (less specific)

Q30. The fidelity of translation is maintained in part by aminoacyl-tRNA synthetases, which must select the correct amino acid to attach to its cognate tRNA. The two-step proofreading mechanism of aminoacyl-tRNA synthetases includes which activities?

(A) Pre-transfer editing (hydrolysis of incorrect aminoacyl-adenylate before tRNA transfer) and post-transfer editing (hydrolysis of mischarged

tRNA product); error rate $\sim 10^{-5}$

(B) Proofreading by a 3'→5' exonuclease analogous to DNA polymerase

(C) Recognition of incorrect tRNA by a GTP-dependent quality-control factor (EF-Tu)

(D) Ribosomal A-site rejection of near-cognate aminoacyl-tRNAs (kinetic proofreading only)

Q31. During co-translational protein folding in the endoplasmic reticulum (ER), disulfide bonds must form between specific cysteine residues. Which ER-resident enzyme catalyses both the formation and the rearrangement (isomerisation) of disulfide bonds to allow proteins to reach their native, correctly folded conformation?

(A) BiP (GRP78) – the Hsp70 chaperone; binds exposed hydrophobic regions

(B) Protein disulfide isomerase (PDI) – catalyses disulfide bond formation and isomerisation; re-oxidised by Ero1

(C) Calnexin/calreticulin – lectin chaperones that bind monoglucosylated glycans

(D) ERp57 – a PDI-family member that works with calnexin/calreticulin (not the primary enzyme)

Q32. A 6-year-old child with a diet severely deficient in fresh fruits and vegetables presents with muscle weakness and easy fatiguability. Carnitine levels in plasma are low. L-Carnitine is synthesised endogenously from two amino acids, and the final hydroxylation step requires two micronutrients. Which of the following correctly identifies the precursor amino acids and the cofactors required?

(A) Methionine + cysteine; cofactors are vitamin B6 and SAM

(B) Glycine + serine; cofactors are THF and vitamin B12

(C) Lysine + methionine; cofactors are vitamin C (ascorbate) and Fe^{2+} (for the hydroxylase steps)

(D) Arginine + proline; cofactors are α-ketoglutarate and oxygen

Q33. A 25-year-old woman of Northern European descent presents with haemolytic anaemia, jaundice, and mild splenomegaly. Her blood film shows spherocytes and increased osmotic fragility. Coombs' test is negative. Family history reveals autosomal dominant inheritance. The defect in hereditary spherocytosis (HS) involves which protein component of the erythrocyte membrane skeleton?

(A) Haemoglobin chains (as in sickle cell disease)

(B) Glucose-6-phosphate dehydrogenase (G6PD) – X-linked, causes episodic haemolysis

(C) Spectrin, ankyrin, band 3, or protein 4.2 – impaired vertical linkage between lipid bilayer and skeleton $\rightarrow$ membrane vesiculation $\rightarrow$ spherocytes

(D) Pyruvate kinase – AR disorder, causes haemolytic anaemia without spherocytes

Q34. A diabetic patient in ketoacidotic crisis is found to have blood glucose 520 mg/dL and serum ketones "strongly positive" on dipstick. However, a point-of-care β-hydroxybutyrate test shows 7.2 mmol/L. The nurse is confused because the traditional nitroprusside (Rothera) dipstick test result seems inconsistently lower than the clinical picture. The explanation is:

(A) Nitroprusside reacts with β-hydroxybutyrate but not acetoacetate

(B) Nitroprusside reacts with all three ketone bodies equally

(C) Nitroprusside detects β-hydroxybutyrate and acetoacetate but not acetone

(D) Nitroprusside (Rothera's test) reacts with acetoacetate and acetone but **not** β-hydroxybutyrate; in severe DKA, β-OHB predominates, so urine ketone sticks underestimate true ketonaemia

Q35. A 52-year-old man with familial hypercholesterolaemia is started on atorvastatin. After 3 months, his LDL-C has fallen from 240 to 130 mg/dL. Which mechanism best explains the LDL-lowering effect of statins?

(A) Statins directly bind and inactivate LDL particles in the plasma

(B) Statins inhibit cholesterol absorption from the gut (like ezetimibe)

(C) Statins activate lipoprotein lipase (LPL), increasing VLDL clearance

(D) Statins inhibit HMG-CoA reductase $\rightarrow$ reduced hepatic cholesterol synthesis $\rightarrow$ upregulation of LDL receptors $\rightarrow$ increased LDL clearance from plasma

Q36. A 70-year-old man is admitted with uraemia, serum urea of 180 mg/dL (BUN 84 mg/dL), and creatinine of 9.5 mg/dL. His BUN-to-creatinine ratio is 8.8. In contrast, a patient with upper GI haemorrhage and dehydration has a BUN of 80 mg/dL and creatinine of 1.2 mg/dL (ratio $\sim$67). What is the significance of an elevated BUN:creatinine ratio ($>$20:1)?

(A) Suggests pre-renal azotaemia (dehydration, haemorrhage, increased protein catabolism) – increased urea reabsorption by the kidney disproportionate to creatinine

(B) Suggests intrinsic renal disease (glomerulonephritis, ATN) with proportional rise in both BUN and creatinine

(C) Indicates hepatic failure (reduced urea synthesis lowers BUN relative to creatinine)

(D) Suggests post-renal obstruction, where creatinine rises faster than BUN

Q37. A cell biologist studying nucleocytoplasmic transport shows that a large protein bearing a nuclear localisation sequence (NLS) is imported into the nucleus via importins in a Ran-GTP-dependent manner. What is the approximate diameter of the nuclear pore complex (NPC) and how many distinct nucleoporin (Nup) proteins form it?

(A) 20 nm diameter; composed of 8 different nucleoporins (minimal pore)

(B) 50 nm diameter; composed of ∼100 different nucleoporins

(C) 200 nm diameter; composed of ∼50 nucleoporins, all integral membrane proteins

(D) ∼120 nm diameter; composed of ∼30 different nucleoporins (∼500 copies total per pore); FG-Nups form the selective permeability barrier

Q38. A surgeon orders a prothrombin time (PT) and activated partial thromboplastin time (aPTT) pre-operatively. The PT is prolonged (22 seconds; normal 12–14 s) but the aPTT is normal. This pattern points to a defect in which coagulation pathway, and which specific factor deficiency could cause this?

(A) Intrinsic pathway defect (Factor VIII or IX deficiency – haemophilia A or B)

(B) Extrinsic/common pathway defect; isolated Factor VII deficiency would prolong PT only (Factor VII is in the extrinsic pathway and has the shortest half-life of all clotting factors)

(C) Common pathway defect due to fibrinogen deficiency (would prolong both PT and aPTT)

(D) Platelet defect (thrombocytopenia would not affect PT or aPTT)

Q39. A 28-year-old woman found unconscious in a house fire is brought to the emergency department. Pulse oximetry reads 98% (SpO_2) but she is cyanotic and confused. Carboxyhaemoglobin (COHb) level is 42%. Which biochemical explanation best accounts for tissue hypoxia in CO poisoning despite apparently normal pulse oximetry?

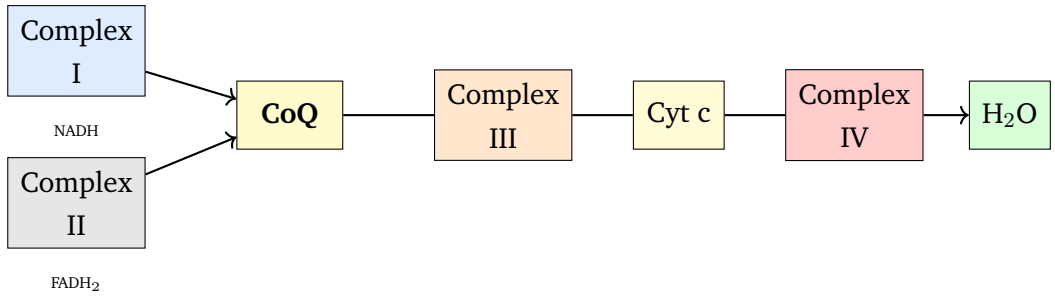
(A) CO destroys haem and causes haemolytic anaemia, reducing oxygen-carrying capacity

(B) CO binds haemoglobin at the haem iron with ∼240 times the affinity of O_2, forming COHb; CO also causes a left shift in the O_2-Hb dissociation curve in remaining oxyhaemoglobin, impairing O_2 delivery; pulse oximetry cannot distinguish COHb from OxyHb

(C) CO reacts with 2,3-BPG, displacing it from haemoglobin and causing a right shift

(D) CO only inhibits cytochrome oxidase and does not affect haemoglobin

Q40. In the mitochondrial electron transport chain (ETC), coenzyme Q (ubiquinone, CoQ_{10}) plays a central role as a mobile electron carrier. From which two ETC complexes does CoQ accept electrons, and to which complex does it donate them?



(A) Accepts from Complex I (NADH) and Complex II ($FADH_2$); donates to Complex III; also accepts from ETF-dehydrogenase (β-oxidation) and other flavoproteins

(B) Accepts from Complex III only; donates to Complex I

(C) Accepts from Complex II only; donates to Cytochrome c directly (by-passing Complex III)

(D) Accepts from Cytochrome c; donates to Complex I (reverse electron transfer)

Detailed Solutions

Q1.

Solution

Concept – Rhodopsin bleaching cycle: In rod photoreceptors, 11-*cis*-retinal is covalently bound via a Schiff base to the protein opsin, forming rhodopsin. When a photon is absorbed, 11-*cis*-retinal undergoes **photoisomerisation to all-*trans*-retinal**. This conformational change activates the G-protein **transducin**, which in turn activates phosphodiesterase to hydrolyse cGMP, closing Na^+ channels and hyperpolarising the rod cell. All-*trans*-retinal is then released from opsin (bleaching), reduced to all-*trans*-retinol, transported to the RPE, re-isomerised to 11-*cis*-retinal, and returned to regenerate rhodopsin. Vitamin A (retinol) deficiency impairs regeneration of 11-*cis*-retinal, causing night blindness.

Why distractors are wrong: (B) Isomerisation goes *cis*→*trans* on light absorption, not the reverse. (C) Lysosomes are not involved in the visual cycle. (D) Rhodopsin kinase phosphorylates *activated* (bleached) rhodopsin to quench the signal – not in the dark.

Final Answer: Isomerisation of 11-*cis*-retinal to all-*trans*-retinal, activating transducin. $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q1

Q2.

Solution

Concept – Michaelis-Menten equation and K_m: The Michaelis-Menten equation is $v = V_{max}[S]/(K_m + [S])$. When $[S] = K_m$: $v = V_{max} \cdot K_m/(K_m + K_m) = V_{max}/2$. In this question, $v = 100/2 = 50$ nmol/min. At $[S] = K_m$, the enzyme is half-saturated. When $[S] \gg K_m$, the reaction approaches zero-order (rate = V_{max}, independent of $[S]$). When $[S] \ll K_m$, the reaction is first-order (rate $\approx (V_{max}/K_m)[S]$, proportional to $[S]$). K_m reflects substrate affinity: low K_m = high affinity.

Why distractors are wrong: (B) $v = V_{max}$ only when $[S] \gg K_m$ (saturating concentrations). (C) $v = 0$ only when $[S] = 0$. (D) v can never exceed V_{max} – a ceiling defined by total enzyme concentration and k_{cat}.

Final Answer: $v = V_{max}/2 = 50$ nmol/min. $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q2

Q3.

Solution

Concept – Hunter syndrome (MPS II): Hunter syndrome (mucopolysaccharidosis type II) is caused by deficiency of **iduronate-2-sulphatase**, leading to accumulation of **heparan sulfate and dermatan sulfate** in lysosomes. It is the only MPS disorder with **X-linked recessive** inheritance, affecting males (brothers and maternal uncles). Key distinguishing feature: **NO corneal clouding** (unlike Hurler MPS I, where α-L-iduronidase deficiency also causes corneal clouding). Both Hurler and Hunter present with gargoylism, hepatosplenomegaly, intellectual disability, joint stiffness, and cardiac disease.

Why distractors are wrong: (A) α-L-iduronidase deficiency causes Hurler (MPS I), which *does* cause corneal clouding and is autosomal recessive. (C) Arylsulphatase A deficiency causes metachromatic leukodystrophy (MLD), a white matter disease. (D) Galactosamine-6-sulphatase deficiency causes Morquio syndrome (MPS IVA), characterised by skeletal dysplasia without intellectual disability.

Final Answer: Iduronate-2-sulphatase (Hunter syndrome, MPS II). $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q3

Q4.

Solution

Concept – UDP-glucose as activated glucose donor in glycogen synthesis: Glycogen synthase elongates glycogen chains by transferring glucose from **UDP-glucose** to the non-reducing end of a glycogen chain (forming α-1,4-glycosidic bonds). UDP-glucose is synthesised by **UDP-glucose pyrophosphorylase (UTP:glucose-1-phosphate uridylyltransferase)**: glucose-1-phosphate + UTP $\rightarrow$ UDP-glucose + PP_i. Inorganic pyrophosphate (PP_i) is immediately hydrolysed by pyrophosphatase, driving the reaction forward. Glucose-6-phosphate must first be converted to glucose-1-phosphate by phosphoglucomutase before UDP-glucose can be formed.

Why distractors are wrong: (A) The substrate is glucose-1-phosphate, not glucose-6-phosphate. (C) Hexokinase/glucokinase forms glucose-6-phosphate, not glucose-1-phosphate. (D) There is no phosphatase converting glucose-6-P $\rightarrow$ glucose-1-P; phosphoglucomutase performs this isomerisation.

Final Answer: Glucose-1-phosphate + UTP $\rightarrow$ UDP-glucose + PP_i. $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q4

Q5.

Solution

Concept – Vitamin K cycle and VKOR: Vitamin K hydroquinone (KH_2, the active reduced form) is oxidised to vitamin K epoxide during γ-carboxylation of Glu residues on coagulation factors II, VII, IX, X and anticoagulant proteins C and S. Vitamin K epoxide reductase complex (**VKORC1**) regenerates vitamin K quinone and then KH_2, completing the cycle. **Warfarin** inhibits VKOR, trapping vitamin K in the epoxide form and preventing regeneration of active KH_2, thereby blocking γ-carboxylation. Without carboxylation, these clotting factors cannot bind calcium or phospholipid surfaces and are inactive.

Why distractors are wrong: (B) γ-Glutamyl carboxylase uses KH_2 as cofactor but does not regenerate it. (C) Protein disulfide isomerase catalyses disulfide bond formation in the ER (not the vitamin K cycle). (D) Thioredoxin reductase reduces thioredoxin (an antioxidant system, not the vitamin K cycle).

Final Answer: Vitamin K epoxide reductase (VKOR). $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q5

Q6.

Solution

Concept – Argininosuccinate lyase (ASL) deficiency: In the urea cycle, argininosuccinate lyase cleaves argininosuccinate into **arginine + fumarate**. ASL deficiency causes argininosuccinate to accumulate in plasma and urine (argininosuccinic aciduria). It presents with hyperammonaemia, intellectual disability, trichorrhexis nodosa (brittle hair), and hepatic dysfunction. Citrulline is elevated (as it is the precursor of argininosuccinate). The condition is distinguishable from other urea cycle disorders by the characteristic argininosuccinate peak on amino acid chromatography.

Why distractors are wrong: (A) OTC deficiency causes citrullinuria and elevated orotic acid in urine, not argininosuccinate. (B) Argininosuccinate synthetase deficiency causes citrullinaemia type I (citrulline accumulates). (C) Arginase deficiency causes argininaemia with elevated arginine, spasticity, and relatively mild hyperammonaemia.

Final Answer: Argininosuccinate lyase – cleavage of argininosuccinate to arginine + fumarate. $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q6

Q7.

Solution

Concept – CRISPR-Cas9 and NHEJ repair: After Cas9 creates a precise double-strand break (DSB) at the target site, the cell's DNA repair machinery must seal the break. **Non-homologous end joining (NHEJ)** is the predominant repair pathway in most mammalian cells: the broken ends are ligated without a homologous template, often introducing small insertions or deletions (**indels**) at the break site. If an indel disrupts the reading frame (frameshift), a premature stop codon is generated, effectively knocking out the gene. **HDR** (homology-directed repair) uses a provided donor template for precise editing and is less frequent.

Why distractors are wrong: (A) HDR produces precise edits (e.g., correction mutations) and requires a template; it does not introduce indels. (B) BER corrects damaged bases (e.g., 8-oxoguanine), not DSBs. (C) NER removes bulky adducts (UV-induced thymine dimers, crosslinks), not DSBs.

Final Answer: Non-homologous end joining (NHEJ). $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q7

Q8.

Solution

Concept – Familial type I hyperlipoproteinaemia (hyperchylomicronaemia): Familial type I is characterised by massive hypertriglyceridaemia (>1000 mg/dL), creamy lactescent plasma, and eruptive xanthomas. It results from **lipoprotein lipase (LPL) deficiency** or, less commonly, **ApoC-II deficiency** (ApoC-II is the obligatory co-activator of LPL on endothelial surfaces). Without LPL activity, chylomicrons cannot be cleared from the circulation. Plasma incubated at 4°C overnight shows a cream layer on top (chylomicrons) with clear plasma below. There is **no increased risk of atherosclerosis** (because LDL is not elevated). Treatment is dietary fat restriction (<20 g/day).

Why distractors are wrong: (A) VLDL receptor deficiency is extremely rare and causes a different lipoprotein pattern. (B) LDL receptor deficiency causes familial hypercholesterolaemia (type IIa) with elevated LDL, not chylomicronaemia. (D) LCAT deficiency causes hypoHDL, corneal opacification, and haemolytic anaemia (Norum disease), not chylomicronaemia.

Final Answer: Lipoprotein lipase (LPL) deficiency, or ApoC-II deficiency. $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q8

Q9.

Solution

Concept – Hepatic ketogenesis without ketone utilisation: The liver generates ketone bodies (acetoacetate, β-hydroxybutyrate) from excess acetyl-CoA in the mitochondria via HMG-CoA synthase $\rightarrow$ HMG-CoA $\rightarrow$ HMG-CoA lyase $\rightarrow$ acetoacetate $\rightarrow$ β-OHB. However, the liver **cannot utilise** ketone bodies as fuel because it **lacks succinyl-CoA transferase** (also called thiophorase or 3-oxoacid CoA-transferase). This enzyme activates acetoacetate to acetoacetyl-CoA in extrahepatic tissues (brain, heart, muscle, kidney): acetoacetate + succinyl-CoA $\rightarrow$ acetoacetyl-CoA + succinate. Without this enzyme, the liver cannot incorporate acetoacetate into the TCA cycle.

Why distractors are wrong: (A) The liver does have HMG-CoA synthase (mitochondrial isoform for ketogenesis). (C) The liver does have β-hydroxybutyrate dehydrogenase (β-OHB $\rightarrow$ acetoacetate). (D) The liver does have HMG-CoA lyase.

Final Answer: Liver lacks succinyl-CoA transferase (thiophorase), so it cannot activate acetoacetate. $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q9

Q10.

Solution

Concept – UCP1 (Thermogenin) in brown adipose tissue: Brown adipose tissue (BAT) contains abundant mitochondria with **uncoupling protein 1 (UCP1, thermogenin)** in the inner mitochondrial membrane. UCP1 creates a proton conductance pathway that allows H^+ to re-enter the mitochondrial matrix **without passing through ATP synthase**, dissipating the proton-motive force as **heat** rather than ATP. This non-shivering thermogenesis is particularly important in neonates and hibernating animals. UCP1 is activated by free fatty acids (liberated by norepinephrine-stimulated lipolysis) and inhibited by purine nucleotides (GDP, ATP). BAT is highly vascularised (brown from cytochromes and Fe) and appears in the interscapular region, mediastinum, and around major vessels.

Why distractors are wrong: (A) ANT (adenine nucleotide translocase) exchanges ATP^{4-} for ADP^{3-} across the inner membrane – it is a carrier, not an uncoupler. (C) Complex IV reduces O_2 to H_2O and pumps protons; it does not uncouple. (D) Complex II is succinate dehydrogenase, an ETC entry point.

Final Answer: Uncoupling protein 1 (UCP1, thermogenin). $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q10

Q11.

Solution

Concept – Transthyretin (prealbumin) as nutritional marker: Transthyretin (TTR, prealbumin) is a tetrameric plasma protein synthesised in the liver. It transports **thyroxine (T4)** and, when bound to retinol-binding protein (RBP), also facilitates **retinol (vitamin A) transport**. Its plasma half-life is approximately **2 days**, far shorter than albumin (18–20 days) or transferrin (8–10 days). This short half-life makes it a **sensitive early indicator** of nutritional status changes and acute-phase response. Levels fall rapidly in protein malnutrition (kwashiorkor), liver disease, and acute inflammation (negative acute-phase reactant). Widely used in ICU nutrition assessment.

Why distractors are wrong: (B) Transferrin has a half-life of ∼8–10 days; it is a better marker than albumin but slower than transthyretin. (C) RBP (half-life ∼12 hours) is actually the most rapidly responsive, but it circulates bound to transthyretin, so their levels are correlated and both fall together. (D) Fibronectin is a matrix protein, not a standard nutritional marker.

Final Answer: Transthyretin (prealbumin). $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q11

Q12.

Solution

Concept – Phosphatidylserine decarboxylation to phosphatidylethanolamine: Phosphatidylserine decarboxylase (PSD), located in the inner mitochondrial membrane, catalyses: Phosphatidylserine (PS) $\rightarrow$ Phosphatidylethanolamine (PE) + CO_2. This is the mitochondrial route for PE synthesis. PE can then be converted to phosphatidylcholine (PC) by three sequential SAM-dependent methylation steps catalysed by **PEMT (phosphatidylethanolamine N-methyltransferase)**. Alternatively, PC is synthesised *de novo* via the CDP-choline (Kennedy) pathway. The PS$\rightarrow$PE$\rightarrow$PC pathway is an important alternative (salvage) route for PC synthesis in the liver.

Why distractors are wrong: (A) PEMT methylates PE to PC (three steps, each using SAM) – it is downstream. (C) CDP-choline:diacylglycerol phosphocholine transferase is the final step of the Kennedy pathway for PC synthesis from choline. (D) Sphingomyelin synthase transfers a phosphocholine from PC to ceramide to make sphingomyelin.

Final Answer: Phosphatidylserine decarboxylase (PSD). $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q12

Q13.

Solution

Concept – Hereditary fructose intolerance vs essential fructosuria: Hereditary fructose intolerance (HFI) is caused by **aldolase B deficiency**. Fructokinase converts fructose → fructose-1-phosphate normally, but aldolase B (expressed in liver, kidney, intestine) cannot cleave fructose-1-phosphate. Accumulation of fructose-1-phosphate is toxic to hepatocytes (inhibits glycogenolysis and gluconeogenesis, causes hypoglycaemia) and causes liver damage, jaundice, and eventual cirrhosis. **Essential fructosuria** is caused by **fructokinase deficiency**: fructose cannot be phosphorylated in the liver, so it remains in the blood and is excreted in urine. It is **completely benign** (no fructose-1-phosphate accumulates).

Why distractors are wrong: (A) Reverses the enzymes incorrectly. (B) Fructose-1,6-bisphosphatase deficiency causes a different disorder (impaired gluconeogenesis, lactic acidosis). (D) Aldolase A is the muscle/RBC isoform of aldolase; it does not metabolise fructose-1-phosphate efficiently.

Final Answer: Aldolase B in HFI; fructokinase in essential fructosuria. ⇒ $\boxed{C}$

Answer: (C) Go Back to Q13

Q14.

Solution

Concept – Palmitic acid (C16:0): Palmitic acid is the most abundant saturated fatty acid in the human body and the primary end-product of *de novo* fatty acid synthesis by the multi-enzyme fatty acid synthase (FAS) complex. Its systematic name is **hexadecanoic acid** (16 carbons, no double bonds – saturated, C16:0). De novo synthesis begins with acetyl-CoA (starter unit) and adds 7 malonyl-CoA units (2 carbons each) to build a 16-carbon chain. Palmitic acid is the precursor for longer-chain fatty acids (C18, C20) by elongases. It is abundant in palm oil, dairy products, and animal fats.

Why distractors are wrong: (A) Stearic acid is C18:0 (18 carbons, saturated). (C) Palmitoleic acid is C16:1 ω-7 (palmitic with one double bond). (D) Myristic acid is C14:0 (14 carbons, saturated; found in nutmeg).

Final Answer: 16-carbon saturated fatty acid (hexadecanoic acid, C16:0). ⇒ $\boxed{B}$

Answer: (B) Go Back to Q14

Q15.

Solution

Concept – Sphingomyelin synthesis and diacylglycerol (DAG): Sphingomyelin synthase catalyses the transfer of a phosphocholine headgroup from **phosphatidylcholine (PC)** to **ceramide**: Ceramide + PC $\rightarrow$ Sphingomyelin + **Diacylglycerol (DAG)**. DAG is the other product of this reaction. Sphingomyelin is a major component of the myelin sheath and plasma membranes. Ceramide itself (sphingosine + fatty acid, amide bond) is the backbone of all sphingolipids. Sphingomyelin is hydrolysed back to ceramide + phosphocholine by **sphingomyelinase** (deficient in Niemann-Pick disease).

Why distractors are wrong: (A) Phosphatidylethanolamine is not a product of sphingomyelin synthase. (C) Ceramide-1-phosphate is formed by ceramide kinase (a different reaction). (D) Lysophosphatidylcholine results from phospholipase A_2 cleavage of PC, not from sphingomyelin synthase.

Final Answer: Diacylglycerol (DAG). $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q15

Q16.

Solution

Concept – Haemoglobin F (HbF) and 2,3-BPG: HbF ($\alpha_2\gamma_2$) has γ-chains instead of β-chains. At position 143, β-chains have **His**$_{143}$ (a key 2,3-BPG binding residue in the central cavity of deoxyHbA), whereas γ-chains have **Ser**$_{143}$. This single amino acid difference means 2,3-BPG **binds HbF much less effectively**. Since 2,3-BPG stabilises the deoxy (low-affinity, T-state) conformation of haemoglobin, less 2,3-BPG binding to HbF means HbF stays in the oxy (high-affinity, R-state) conformation more readily. This gives HbF a higher O_2 affinity, enabling the fetus to extract O_2 from maternal HbA in the placenta.

Why distractors are wrong: (A) HbF has γ-chains, not ζ-chains. ζ-chains are embryonic and form haemoglobin Gower 1 in very early development. (B) Iron content per haem is the same (Fe^{2+}). (C) ϵ-Chains are embryonic (haemoglobin Gower 2); HbF is the fetal form throughout gestation and early neonatal life.

Final Answer: HbF has γ-chains that bind 2,3-BPG less effectively, reducing allosteric inhibition, raising O_2 affinity. $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q16

Q17.

Solution

Concept – Glutamate dehydrogenase (GDH) and hyperinsulinism-hyperammonaemia (HI/HA) syndrome: Glutamate dehydrogenase (GDH) is a mitochondrial enzyme that catalyses the reversible oxidative deamination of L-glutamate: Glutamate $\rightleftharpoons$ α-ketoglutarate + NH_3 + NADH (or NADPH). GDH is **allosterically activated by ADP and leucine** (signals low energy / amino acid excess) and **inhibited by GTP and ATP**. It links amino acid catabolism to the TCA cycle via α-ketoglutarate. **Gain-of-function mutations** in GDH (relieving GTP inhibition) cause HI/HA syndrome: excess GDH activity increases glutamate deamination $\rightarrow$ more α-ketoglutarate fuels TCA $\rightarrow$ excess ATP $\rightarrow$ stimulates insulin release **and** increases NH_3 production.

Why distractors are wrong: (B) Glutamine synthetase consumes NH_3 to make glutamine – its deficiency would increase ammonia, but it is not linked to hyperinsulinism. (C) ALT is a transaminase; its mutation does not cause hyperinsulinism. (D) Glutaminase releases NH_3 from glutamine; gain-of-function would cause hyperammonaemia but not hyperinsulinism.

Final Answer: Glutamate dehydrogenase (GDH). $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q17

Q18.

Solution

Concept – Citrate as dual allosteric regulator: Citrate, exported from the mitochondria to the cytoplasm via the tricarboxylate carrier, signals a replete TCA cycle and energy surplus. In the cytoplasm, citrate exerts two key allosteric effects: (1) **Inhibits PFK-1** (phosphofructokinase-1), the rate-limiting enzyme of glycolysis – signals that the cell has sufficient carbon for the TCA cycle, so glycolysis should slow. (2) **Activates acetyl-CoA carboxylase (ACC)**, the rate-limiting enzyme of fatty acid synthesis – citrate is cleaved by ATP-citrate lyase to yield acetyl-CoA (the substrate for ACC) AND allosterically activates ACC, promoting malonyl-CoA production and fatty acid synthesis. This perfectly coordinates energy sensing with biosynthesis.

Why distractors are wrong: (A) Citrate *inhibits* PFK-1 (not activates) and *activates* ACC (not inhibits). (B) Citrate does not inhibit glycogen synthase or activate pyruvate kinase. (D) HMG-CoA reductase is regulated by cholesterol and insulin/glucagon ratio, not by citrate directly.

Final Answer: Inhibits PFK-1 and activates acetyl-CoA carboxylase (ACC). $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q18

Q19.

Solution

Concept – ATP synthase binding-change mechanism (γ-subunit rotation): ATP synthase (F_0-F_1-ATPase) has two domains: F_0 (membrane-embedded, contains the c-ring proton channel) and F_1 (extrinsic, catalytic, $\alpha_3\beta_3\gamma\delta\epsilon$ composition). Proton flow through the c-ring causes the **c-ring to rotate**. This rotates the attached **γ-subunit (central stalk)**, which acts as a cam inside the $\alpha_3\beta_3$ hexamer. The γ-subunit rotation drives the three β-subunits sequentially through three conformational states: **Open (O)** – releases ATP; **Loose (L)** – binds ADP + P_i loosely; **Tight (T)** – synthesises ATP with high affinity. Each 360° revolution synthesises 3 ATP; approximately 10 protons pass per revolution ($\sim$3.3 H^+/ATP).

Why distractors are wrong: (A) The α-subunits are fixed; only the γ-subunit (central stalk) and c-ring rotate. (B) The β-subunits are fixed relative to each other; it is the γ-subunit that rotates *inside* them. (D) The c-ring rotation is directly linked to γ-subunit rotation – they are mechanically coupled.

Final Answer: The γ-subunit (central stalk) rotates inside the $\alpha_3\beta_3$ ring, driven by c-ring rotation. $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q19

Q20.

Solution

Concept – VLDL structure and apolipoproteins: VLDL (very low-density lipoprotein) is assembled in the hepatocyte ER and Golgi and secreted into the bloodstream to transport triglycerides from the liver to peripheral tissues. Its obligatory structural apolipoprotein is **ApoB-100** (synthesised only in the liver; ApoB-48 is the intestinal isoform, found in chylomicrons). VLDL also carries **ApoC-II** (which activates LPL in capillaries) and **ApoE** (which mediates receptor-mediated uptake). MTP (microsomal triglyceride transfer protein) is essential for loading triglycerides onto ApoB-100 during VLDL assembly; its deficiency (abetalipoproteinaemia) prevents VLDL and chylomicron secretion.

Why distractors are wrong: (A) ApoA-I and ApoA-II are the major apolipoproteins of HDL. (B) ApoC-I and ApoE alone is incomplete – ApoB-100 is the obligatory structural protein. (C) ApoB-48 is exclusively intestinal (chylomicrons), not hepatic (VLDL).

Final Answer: ApoB-100, ApoC-II, and ApoE. $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q20

Q21.

Solution

Concept – Insulin inhibition of lipolysis via phosphodiesterase/PKA pathway: Hormone-sensitive lipase (HSL) is activated by phosphorylation via PKA (when cAMP is high, e.g., during fasting or catecholamine stimulation). Insulin inhibits lipolysis through: (1) **Activation of phosphodiesterase (PDE)** via the PI3K-Akt pathway $\rightarrow$ PDE hydrolyses cAMP to AMP $\rightarrow$ cAMP falls $\rightarrow$ PKA activity decreases $\rightarrow$ less HSL phosphorylation $\rightarrow$ less active HSL. (2) **Activation of protein phosphatase 2A (PP2A)** $\rightarrow$ dephosphorylates HSL (converting it to inactive form). Net effect: suppression of free fatty acid release from adipocytes.

Why distractors are wrong: (A) Insulin does *not* activate adenylyl cyclase; it does the opposite. (B) Insulin does not directly bind HSL's catalytic domain. (D) Insulin promotes glucose uptake and lipogenesis in adipocytes but does not directly promote β-oxidation.

Final Answer: Insulin activates phosphodiesterase (via Akt), lowering cAMP, reducing PKA activity and HSL phosphorylation. $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q21

Q22.

Solution

Concept – UDP-glucuronic acid (activated form for conjugation): Glucuronic acid is synthesised from glucose: Glucose-6-P $\rightarrow$ Glucose-1-P $\rightarrow$ UDP-glucose $\rightarrow$ **UDP-glucuronic acid** (catalysed by UDP-glucose dehydrogenase, which uses 2 NAD^+). The activated form for all conjugation and biosynthetic reactions is **UDP-glucuronic acid**. It is used by UDP-glucuronosyltransferases (UGTs) to conjugate: bilirubin (in the liver), steroid hormones, thyroid hormones, bile acids, and drugs/xenobiotics. UDP-glucuronic acid is also the building block for glycosaminoglycans: hyaluronic acid, heparan sulfate, dermatan sulfate, and chondroitin sulfate.

Why distractors are wrong: (B) Glucuronic acid-1-phosphate is not a biologically relevant activated intermediate in this pathway. (C) ADP-glucuronate does not exist in glucuronate metabolism (ADP-glucose is used in starch synthesis in plants). (D) CMP-sialic acid is the activated donor for sialic acid in glycoprotein

biosynthesis – a different sugar nucleotide.

Final Answer: UDP-glucuronic acid. $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q22

Q23.

Solution

Concept – Cori disease (GSD III) and Hers disease (GSD VI): Cori disease (GSD Type III) is caused by deficiency of the **debranching enzyme** (amylo-1,6-glucosidase + oligo-1,4→1,4-glucan transferase). Without debranching, glycogenolysis stops at branch points, accumulating **limit dextrins**. Clinical features: hepatomegaly, fasting hypoglycaemia (milder than Type I), and sometimes myopathy (if muscle is also affected). Hepatic glycogen has abnormal structure. **Hers disease (GSD Type VI)** is caused by hepatic phosphorylase kinase (or hepatic phosphorylase) deficiency. It presents with hepatomegaly and mild fasting hypoglycaemia, is the **mildest hepatic GSD**, and often improves spontaneously.

Why distractors are wrong: (A) Von Gierke (Type I) is glucose-6-phosphatase deficiency, not debranching enzyme. (B) Pompe (Type II) is acid maltase (lysosomal glucosidase) deficiency causing cardiomyopathy and hypotonia. (C) McArdle (Type V) is muscle phosphorylase deficiency causing exercise intolerance, not liver disease.

Final Answer: Type III (Cori) is this child; Type VI (Hers) is the mildest hepatic GSD. $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q23

Q24.

Solution

Concept – Homocysteine metabolism: transsulfuration (B6-dependent): Homocysteine has two metabolic fates: (1) **Remethylation** back to methionine: via **methionine synthase** (requires B12 as cofactor and 5-methyl-THF as methyl donor) or via **BHMT** (betaine-homocysteine methyltransferase, using betaine as methyl donor in liver only). (2) **Transsulfuration**: homocysteine + serine → cystathionine, catalysed by **cystathionine β-synthase (CBS)**, which requires **pyridoxal phosphate (PLP, vitamin B6)**. Cystathionine is then cleaved by cystathionine γ-lyase (also B6-dependent) to cysteine + α-ketobutyrate. Vitamin B6 deficiency impairs transsulfuration, and B12/folate deficiency impairs remethylation – both cause hyperhomocysteinaemia.

Why distractors are wrong: (A) Methionine synthase requires B12 and 5-methyl-THF (not B6). (B) BHMT uses betaine (trimethylglycine) as methyl donor and does not require B6. (D) Non-enzymatic oxidation is not a regulated metabolic pathway.

Final Answer: Transsulfuration via cystathionine β-synthase (CBS) requires vitamin B6 (PLP). $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q24

Q25.

Solution

Concept – Vitamin B12 body stores: The body stores approximately **2–5 mg** of vitamin B12, primarily in the **liver**. Given daily requirements of $\sim$2–3 μg/day, these stores are sufficient for **3–5 years** of complete dietary abstinence. Vitamin B12 is also efficiently recycled via **enterohepatic circulation** (secreted in bile and reabsorbed in the terminal ileum). Consequently, adult vegans who eliminate all animal products take *years* to develop deficiency, whereas individuals with malabsorption (e.g., pernicious anaemia with intrinsic factor deficiency, or terminal ileal disease such as Crohn's) may deplete stores within 3–5 years. Neonates born to B12-deficient mothers are at risk because fetal stores are low.

Why distractors are wrong: (A) Total stores are 2–5 mg (not micrograms) – sufficient for years. (B) B12 is not stored in red blood cells; it is stored in hepatocytes. (C) B12 is actively stored in the liver and undergoes enterohepatic circulation.

Final Answer: Liver stores 2–5 mg, sufficient for 3–5 years. $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q25

Q26.

Solution

Concept – Calmodulin structure and function: Calmodulin (CaM) is a ubiquitous calcium-sensing protein of 148 amino acids. It contains **four EF-hand (helix-loop-helix) motifs**, each capable of binding one Ca^{2+} ion – giving **four Ca^{2+} binding sites** in total. Binding of four Ca^{2+} ions causes a conformational change, exposing hydrophobic patches that allow calmodulin to bind and activate target proteins including: **CaM kinases II and IV**, eNOS (nitric oxide synthase), phosphodiesterase (PDE), myosin light-chain kinase (MLCK), and calcineurin (PP2B). Calmodulin itself has no enzymatic activity – it acts as a calcium sensor that modulates its target proteins.

Why distractors are wrong: (B) Two Ca^{2+} via zinc-finger domains describes zinc finger transcription factors, not calmodulin. (C) Annexin repeats bind phospholipids and Ca^{2+} (different family). (D) C2 domains are found in PKC and synaptotagmin – they bind 2 Ca^{2+} per C2 domain and are not calmodulin.

Final Answer: Four Ca^{2+} ions via four EF-hand motifs; activates CaM kinases, eNOS, and phosphodiesterase. $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q26

Q27.

Solution

Concept – Wobble hypothesis (Crick 1966): The **wobble hypothesis** explains how a single tRNA species can recognise multiple synonymous codons. The **5' base of the anticodon** (position 34, the "wobble position") pairs with the **3' base of the codon** (codon position 3). Wobble pairing is less strict than Watson-Crick pairing. Inosine (I, a modified nucleoside formed by deamination of adenosine) at position 34 can pair with **U, C, or A** at codon position 3 (three different bases), allowing one tRNA to decode up to three codons. This reduces the number of tRNA molecules needed to decode all 61 sense codons.

Why distractors are wrong: (A) Strict Watson-Crick: I would pair only with C – but the wobble hypothesis allows I to pair with U, C, and A. (B) Pairing with U and G is the wobble pairing for G (guanosine at position 34 can pair with U or C). (D) I can pair with U, C, A – but NOT G. Pairing with "all purines and pyrimidines" overstates it.

Final Answer: Inosine (I) pairs with U, C, and A (wobble position 34 of anticodon). $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q27

Q28.

Solution

Concept – HIV reverse transcriptase mechanism and drug targets: HIV reverse transcriptase (RT) is an **RNA-dependent DNA polymerase** (reverse transcriptase). It transcribes the viral RNA genome into DNA: (1) RNA template $\rightarrow$ RNA:DNA hybrid (using RT's DNA polymerase activity); (2) RNA degraded (using RT's inherent **RNase H** activity); (3) second DNA strand synthesised $\rightarrow$ dsDNA. HIV RT has **no proofreading 3'$\rightarrow$5' exonuclease activity** $\rightarrow$ error rate $\sim 10^{-4}$ per base per replication cycle $\rightarrow$ enormous genetic diversity and rapid drug resistance.

It is targeted by **NRTIs** (nucleoside/nucleotide RT inhibitors – chain terminators) and **NNRTIs** (non-nucleoside RT inhibitors – bind allosteric pocket). Integrase (not RT) integrates the proviral DNA.

Why distractors are wrong: (A) RT is an RNA-dependent DNA polymerase (not RNA→RNA polymerase); NRTIs mimic deoxyribonucleotides (DNA building blocks). (B) RT has NO proofreading – this is key to its error-prone nature. (C) Integration is performed by HIV *integrase* (target of integrase strand transfer inhibitors, INSTIs).

Final Answer: RNA-dependent DNA polymerase with RNase H; no proofreading; targets of NRTIs and NNRTIs. $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q28

Q29.

Solution

Concept – Lipid peroxidation biomarkers and isoprostanes: Lipid peroxidation is a free radical chain reaction: initiation ($OH^{\bullet}$ or other ROS attacks PUFA C-H bond) $\rightarrow$ propagation (lipid radical $L^{\bullet} \rightarrow LOO^{\bullet} \rightarrow LOOH \rightarrow$ more $L^{\bullet}$) $\rightarrow$ termination (antioxidants: vitamin E, vitamin C, GPx/selenium). Products include malondialdehyde (MDA), 4-hydroxynonenal (4-HNE), and isoprostanes. **Isoprostanes (especially 8-iso-$PGF_{2\alpha}$)** are prostaglandin-like compounds formed from arachidonic acid by non-enzymatic free radical peroxidation *in vivo*. They are considered the **gold standard** biomarker of oxidative stress because they are specific, chemically stable, not susceptible to artefactual formation ex vivo, and measurable in plasma and urine.

Why distractors are wrong: (A) MDA (TBARS assay) is non-specific – also reacts with non-lipid aldehydes and can form artefactually during sample processing. (B) 4-HNE is a specific lipid peroxidation product but less well-validated as an in vivo marker than isoprostanes. (D) Diene conjugates form early in peroxidation but are unstable and less specific.

Final Answer: Isoprostanes (8-iso-$PGF_{2\alpha}$) – the most specific in vivo marker. $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q29

Q30.

Solution

Concept – Aminoacyl-tRNA synthetase proofreading: Aminoacyl-tRNA synthetases (aaRS) ensure accurate amino acid–tRNA pairing with an error rate of $\sim 10^{-5}$ through a **two-step proofreading mechanism**: **Step 1 (activation)**: Amino acid + ATP $\rightarrow$ aminoacyl-adenylate (AA-AMP) + PP_i. If the wrong AA is activated (pre-transfer editing), the aminoacyl-adenylate is hydrolysed before transfer. **Step 2 (transfer)**: AA-AMP + tRNA $\rightarrow$ aminoacyl-tRNA + AMP. If the wrong AA is transferred to the tRNA (post-transfer editing), an editing (hydrolytic) domain on the aaRS cleaves the mischarged tRNA. This two-site mechanism (synthetic site + editing site) achieves the necessary fidelity.

Why distractors are wrong: (B) Aminoacyl-tRNA synthetases do not have a 3'$\rightarrow$5' exonuclease like DNA polymerase – they have dedicated hydrolytic editing domains. (C) EF-Tu (a GTPase) does provide kinetic proofreading at the ribosome A-site, but this is a separate quality-control step *after* tRNA charging. (D) Ribosomal kinetic proofreading is real but is distinct from the two-step editing of aaRS.

Final Answer: Pre-transfer and post-transfer editing by aminoacyl-tRNA synthetases; error rate $\sim 10^{-5}$. $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q30

Q31.

Solution

Concept – Protein disulfide isomerase (PDI): Protein disulfide isomerase (PDI) is an ER-resident enzyme (retained by KDEL sequence) of the thioredoxin superfamily. It has four thioredoxin-like domains (two catalytically active: a and a', two inactive: b and b') and catalyses: (1) **Oxidation**: formation of new disulfide bonds from free Cys-SH groups (thiol oxidation). (2) **Isomerisation**: rearrangement of incorrect (non-native) disulfide bonds to the correct (native) pattern. (3) **Reduction**: reduction of non-native disulfides. PDI is re-oxidised by **Ero1 (ER oxidoreductin 1)**, which transfers electrons to O_2. PDI is essential for the folding of disulfide-containing secretory and membrane proteins.

Why distractors are wrong: (A) BiP/GRP78 (Hsp70 family) binds exposed hydrophobic regions of misfolded proteins (not disulfide bonds). (C) Calnexin and calreticulin are lectin chaperones that recognise monoglucosylated N-linked glycans – they do not directly catalyse disulfide bond chemistry. (D) ERp57 is a PDI-family member that works with calnexin/calreticulin specifically for glycoprotein substrates; PDI is the primary enzyme for disulfide bond isomerisation generally.

Final Answer: Protein disulfide isomerase (PDI). $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q31

Q32.

Solution

Concept – L-Carnitine synthesis: L-Carnitine is synthesised endogenously from the amino acids **lysine and methionine** (methionine provides the methyl groups via SAM). The pathway: (1) Trimethyllysine $\rightarrow$ hydroxytrimethyllysine (requires **trimethyllysine hydroxylase**, which needs **vitamin C and Fe^{2+}**); (2) several further steps; (3) γ-butyrobetaine $\rightarrow$ L-carnitine (requires **γ-butyrobetaine hydroxylase**, also needing **vitamin C and Fe^{2+}**). The liver and kidney are the main synthesis sites. Carnitine is essential for importing long-chain fatty acids into the mitochondrial matrix (as acylcarnitine esters) for β-oxidation. Deficiency causes myopathy, hypoglycaemia, and accumulation of long-chain acylcarnitines.

Why distractors are wrong: (A) Methionine + cysteine: cysteine is not used in carnitine synthesis. (B) Glycine + serine with THF/B12 is the one-carbon metabolism pathway, not carnitine synthesis. (D) Arginine + proline with α-ketoglutarate describes prolyl hydroxylase (collagen synthesis), not carnitine synthesis.

Final Answer: Lysine + methionine; cofactors are vitamin C and Fe^{2+}. $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q32

Q33.

Solution

Concept – Hereditary spherocytosis (HS): membrane skeleton defects: Hereditary spherocytosis results from defects in proteins that maintain the **vertical linkage** between the lipid bilayer and the underlying membrane skeleton. Affected proteins include **spectrin** (α and β, encoded by SPTA1 and SPTB), **ankyrin** (ANK1, links spectrin to band 3), **band 3** (SLC4A1, the anion exchanger that anchors the skeleton to the bilayer), and **protein 4.2** (stabilises ankyrin-band 3 interaction). Without proper vertical linkage, membrane fragments (lipid bilayer vesicles) are shed at the spleen $\rightarrow$ spherocytes $\rightarrow$ trapped and destroyed in the spleen. Laboratory: **positive osmotic fragility test**, negative direct Coombs' (not immune haemolysis), elevated MCHC.

Why distractors are wrong: (A) Haemoglobin chain defects cause haemoglobinopathies (sickle cell, thalassaemia) – red cell shape and osmotic

fragility are different. (B) G6PD deficiency is X-linked and causes episodic haemolysis with Heinz bodies, not spherocytes. (D) Pyruvate kinase deficiency (autosomal recessive) causes haemolytic anaemia with echinocytes/irregular cells, not spherocytes.

Final Answer: Spectrin, ankyrin, band 3, or protein 4.2 – impaired vertical linkage $\rightarrow$ membrane vesiculation $\rightarrow$ spherocytes. $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q33

Q34.

Solution

Concept – Nitroprusside (Rothera's) test and ketone body detection: The nitroprusside reaction (Rothera's test) detects ketones in urine/serum. **Sodium nitroprusside** reacts with **acetoacetate** (producing a purple colour) and also with **acetone**, but **does NOT react with β-hydroxybutyrate (β-OHB)**. This is clinically important because in **severe DKA**, the high NADH:NAD^+ ratio (from accelerated β-oxidation) drives the equilibrium strongly towards β-OHB: β-OHB:acetoacetate ratio can be 3:1 to 10:1 in severe DKA. Therefore, dipstick/Rothera's tests can severely **underestimate** the degree of ketosis. Enzymatic β-OHB assays are the gold standard for DKA monitoring.

Why distractors are wrong: (A) This reverses the reactivity – nitroprusside reacts with acetoacetate, NOT β-OHB. (B) Nitroprusside does *not* react equally with all three ketone bodies. (C) Nitroprusside does *not* detect β-OHB.

Final Answer: Nitroprusside reacts with acetoacetate and acetone but **not** β-OHB; in severe DKA, β-OHB predominates. $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q34

Q35.

Solution

Concept – Statin mechanism of action: Statins (atorvastatin, rosuvastatin, simvastatin, etc.) are competitive inhibitors of **HMG-CoA reductase**, the rate-limiting enzyme of the mevalonate pathway (cholesterol synthesis). Reduced hepatic cholesterol synthesis lowers intracellular cholesterol $\rightarrow$ SREBP-2 (sterol regulatory element-binding protein 2) is activated $\rightarrow$ transcription of **LDL receptor (LDLR) gene** is upregulated $\rightarrow$ more LDL receptors on hepatocyte surface $\rightarrow$ increased endocytosis and clearance of LDL particles from plasma. Statins also reduce VLDL secretion and have pleiotropic anti-inflammatory effects.

Why distractors are wrong: (A) Statins act on the liver, not on plasma LDL particles directly. (B) Cholesterol absorption inhibition is the mechanism of ezetimibe (targets NPC1L1). (C) LPL activation is the mechanism of fibrates (PPAR-α agonists), which reduce VLDL/TG.

Final Answer: Statins inhibit HMG-CoA reductase $\rightarrow$ upregulation of LDL receptors $\rightarrow$ increased LDL clearance. $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q35

Q36.

Solution

Concept – BUN:creatinine ratio in pre-renal vs renal azotaemia: Urea (BUN) is produced in the liver from amino acid catabolism and excreted by the kidney. Creatinine is produced from creatine phosphate in muscle at a constant rate and excreted by glomerular filtration (not reabsorbed significantly). Normal BUN:creatinine ratio = 10:1 to 20:1. **BUN:Cr >20:1** indicates **pre-renal azotaemia**: in dehydration or reduced renal perfusion, the kidney avidly reabsorbs sodium and water (and urea along with water in the proximal tubule) but not creatinine, so BUN rises disproportionately. Also elevated in GI haemorrhage (protein load from blood digestion) and high protein catabolism. **BUN:Cr ≈10:1** suggests intrinsic renal disease (proportional GFR reduction).

Why distractors are wrong: (B) Intrinsic renal disease causes proportional rises in both BUN and creatinine $\rightarrow$ ratio remains 10–15:1. (C) Liver failure reduces urea production $\rightarrow$ low BUN $\rightarrow$ low BUN:Cr ratio. (D) Post-renal obstruction raises both BUN and creatinine; creatinine does not rise faster.

Final Answer: Pre-renal azotaemia – disproportionate BUN rise due to increased tubular urea reabsorption. $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q36

Q37.

Solution

Concept – Nuclear pore complex (NPC) structure: The nuclear pore complex (NPC) is the largest protein complex in eukaryotic cells, with a molecular mass of ~120 MDa in vertebrates. Key facts: **Diameter ~120 nm** (outer diameter); composed of **~30 different nucleoporin (Nup) proteins**, assembled into ~500 copies per NPC (total ~500–1000 protein copies per pore). **FG-Nups** (phenylalanine-glycine repeat nucleoporins) line the central channel and create a selective per-

meability barrier via a hydrogel or selective phase mechanism. Small molecules (<40 kDa) diffuse freely; large proteins require nuclear localisation sequences (NLS) and importin/exportin (karyopherin) proteins driven by the **Ran-GTP/GDP gradient**.

Why distractors are wrong: (A) 20 nm is too small (that is approximately the size of a ribosome). (B) ∼100 different Nup proteins significantly overstates the number of distinct Nup proteins. (C) 200 nm is too large and most nucleoporins are not integral membrane proteins.

Final Answer: ∼120 nm diameter; ∼30 different nucleoporins; FG-Nups create selective permeability barrier. ⇒ $\boxed{D}$

Answer: (D) Go Back to Q37

Q38.

Solution

Concept – Coagulation pathways and PT vs aPTT: Prothrombin time (PT) tests the **extrinsic pathway** (Factor VII) + common pathway (X, V, II, fibrinogen). **aPTT** tests the **intrinsic pathway** (XII, XI, IX, VIII) + common pathway. Isolated **prolonged PT with normal aPTT** indicates a defect **specific to the extrinsic pathway** – i.e., **Factor VII deficiency**. Factor VII has the shortest plasma half-life (∼4–6 hours) of all clotting factors, so it is the first to fall in vitamin K deficiency (e.g., early warfarin therapy) or liver disease. The common pathway defect (Factors X, V, II, or fibrinogen deficiency) would prolong *both* PT and aPTT.

Why distractors are wrong: (A) Factor VIII or IX deficiency (haemophilia A or B) prolongs aPTT with normal PT (intrinsic pathway defect – opposite pattern). (C) Fibrinogen deficiency is a common pathway defect; it prolongs both PT and aPTT plus thrombin time. (D) Platelet dysfunction (thrombocytopenia or platelet function disorder) affects bleeding time and PFA-100, not PT or aPTT.

Final Answer: Extrinsic/common pathway; isolated Factor VII deficiency prolongs PT only. ⇒ $\boxed{B}$

Answer: (B) Go Back to Q38

Q39.

Solution

Concept – CO poisoning: carboxyhaemoglobin and left shift: Carbon monoxide (CO) binds haemoglobin at the Fe^{2+} of haem with approximately **240 times the affinity of O_2**, forming **carboxyhaemoglobin (COHb)**. COHb cannot carry

O_2, reducing O_2-carrying capacity. Additionally, CO causes a **left shift** of the O_2-Hb dissociation curve in the remaining oxyhaemoglobin (unbound subunits have higher O_2 affinity – Haldane effect), impairing O_2 release to tissues. CO also directly inhibits **cytochrome c oxidase (Complex IV)**, impairing cellular respiration. Pulse oximetry falsely reads COHb as OxyHb (both absorb light similarly at 660 nm). Treatment: 100% O_2 (displaces CO competitively), or hyperbaric O_2 in severe cases.

Why distractors are wrong: (A) CO does not destroy haem or cause haemolysis; it binds reversibly to Fe^{2+}. (C) CO does not react with 2,3-BPG; it causes a left shift (not right shift) of the O_2-Hb curve. (D) CO affects *both* haemoglobin and cytochrome oxidase.

Final Answer: CO binds Hb with $\sim$240$\times$ affinity of O_2; left shifts O_2-Hb curve; pulse oximetry cannot distinguish COHb from OxyHb. $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q39

Q40.

Solution

Concept – Coenzyme Q (ubiquinone) as mobile electron carrier: Coenzyme Q (CoQ, ubiquinone) is a **lipid-soluble quinone** embedded in the inner mitochondrial membrane. It acts as a **mobile electron carrier** that collects electrons from multiple sources and delivers them to Complex III (ubiquinol:cytochrome c oxidoreductase). Sources of electrons for CoQ: (1) **Complex I (NADH-ubiquinone oxidoreductase)** – from mitochondrial NADH; (2) **Complex II (succinate dehydrogenase)** – from $FADH_2$ (succinate oxidation); (3) **ETF-ubiquinone oxidoreductase (ETFDH)** – from the electron-transferring flavoprotein (ETF), which accepts electrons from acyl-CoA dehydrogenases during β-oxidation, as well as other mitochondrial dehydrogenases (GPDH, DHODH). CoQ can be reduced (ubiquinol, QH_2) and re-oxidised by Complex III.

Why distractors are wrong: (B) CoQ accepts from Complex I and II, donates to Complex III (not vice versa). (C) CoQ accepts from Complex II and others, but always donates to Complex III. (D) Cytochrome c accepts electrons from Complex III and donates to Complex IV – the opposite direction.

Final Answer: Accepts from Complex I (NADH), Complex II ($FADH_2$), and ETFDH; donates to Complex III. $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q40

Answer Key – FMGE Biochemistry Sample Paper 6

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