NEET MDS General Physiology and Biochemistry

Sample Paper – 2

Duration: 11 Minutes **Maximum Marks: 56**

Instructions

- This paper contains **14** Multiple Choice Questions (Single Best Response), modelled on the General Physiology and Biochemistry content of **NEET MDS** conducted by NBEMS.
- The **14-question** length matches the number of Physiology and Biochemistry items you actually meet in the real 240-question paper.
- Each correct answer carries **+4 marks**. There is **negative marking of 1 mark** for every incorrect answer; unattempted questions carry no penalty.
- Every question has **exactly one best answer**. Choose the single most appropriate option.
- Attempt this practice paper in one timed sitting of about **11 minutes**, the pace the real computer-based test demands.

Vitamins and Minerals

Q1. Factors II, VII, IX and X remain biologically inactive until specific glutamate residues on them are converted to gamma-carboxyglutamate. The vitamin that serves as the cofactor for this gamma-carboxylation reaction is:

(A) Vitamin E

(B) Vitamin K

(C) Vitamin B12

(D) Folic acid

Q2. A patient complains that he cannot see in dim light after dusk, though his daytime vision is normal. The deficient vitamin supplies the aldehyde

form that combines with opsin to make rhodopsin in the rods. That vitamin is:

(A) Vitamin D

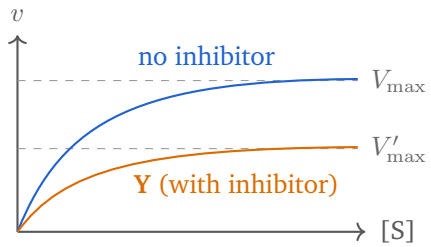
(B) Niacin

(C) Vitamin A

(D) Biotin

Enzymes and Enzyme Kinetics

Q3. The two curves below are from the same enzyme assayed with and without an inhibitor. The inhibited curve marked **Y** plateaus lower than the control, its K_m is unchanged, and piling on extra substrate does not restore the original plateau. The inhibition is:



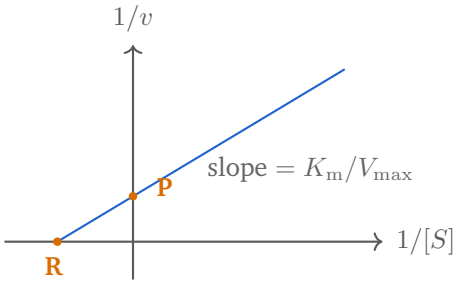
(A) Competitive

(B) Uncompetitive

(C) Allosteric activation

(D) Non-competitive

Q4. The double reciprocal (Lineweaver-Burk) plot below graphs $1/v$ against $1/[S]$ for an enzyme. The intercept marked **P** on the vertical axis and the intercept marked **R** on the horizontal axis respectively equal:



(A) $1/V_{\max}$ and $-1/K_{\mathrm{m}}$

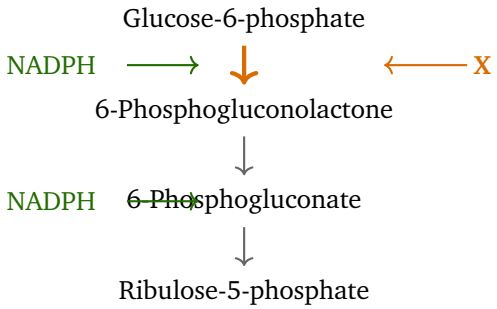
(B) $V_{\max}$ and K_{m}

(C) $1/K_{\mathrm{m}}$ and $-1/V_{\max}$

(D) $K_{\mathrm{m}}/V_{\max}$ and $V_{\max}$

Metabolism of Carbohydrate, Lipid and Protein

Q5. In the outline of the hexose monophosphate shunt below, the step marked **X** is the rate limiting step and its enzyme is deficient in the commonest human enzymopathy, which makes red cells haemolyse after oxidant drugs. The enzyme catalysing **X** is:



(A) Transketolase

(B) Glucose-6-phosphate dehydrogenase

(C) Glucose-6-phosphatase

(D) Pyruvate carboxylase

Q6. After four days of total starvation the blood shows raised acetoacetate and beta-hydroxybutyrate. The organ that synthesises these ketone bodies, and the organ that cannot use them at all, are respectively:

(A) Muscle and brain

(B) Liver and liver

(C) Kidney and heart

(D) Liver and brain

Q7. Palmitate (C16, fully saturated) is completely oxidised to carbon dioxide and water. Counting the 2 ATP equivalents spent on activating the

fatty acid and using the modern P/O ratios of 2.5 for NADH and 1.5 for $FADH_2$, the net ATP yield is about:

(A) 106 ATP

(B) 32 ATP

(C) 12 ATP

(D) 380 ATP

Acid Base Balance and Body Fluids

Q8. A long standing smoker with COPD is drowsy in the dental chair. His blood gas reads pH 7.29, $PaCO_2$ 62 mmHg, bicarbonate 29 mEq/L. The disturbance is:

(A) Metabolic acidosis

(B) Respiratory alkalosis

(C) Respiratory acidosis with renal compensation

(D) Metabolic alkalosis with respiratory compensation

Q9. The serum anion gap is calculated so that a metabolic acidosis can be sorted into two groups. The gap is calculated as:

(A) Sodium plus potassium minus chloride

(B) Chloride plus bicarbonate minus sodium

(C) Sodium plus chloride minus bicarbonate

(D) Sodium minus (chloride plus bicarbonate)

Blood and Haemostasis

Q10. A 12-year-old boy has bled into his knee joints since infancy. His aPTT is markedly prolonged, the prothrombin time and platelet count are normal, and the mixing study corrects. The deficient factor is:

(A) Factor VII

(B) Factor XIII

(C) Factor VIII

(D) Fibrinogen

Q11. The average lifespan of a normal circulating red blood cell is about 120 days, so the marrow must keep replacing them. The hormone that drives that replacement, and the organ that secretes it in the adult, are:

(A) Thrombopoietin from the liver

(B) Erythropoietin from the bone marrow

(C) Erythropoietin from the liver

(D) Erythropoietin from the kidney

Nerve, Muscle and Endocrine Physiology

Q12. The resting membrane potential of a neuron sits at about -70 mV. The pump that maintains the sodium and potassium gradients on which that potential depends moves:

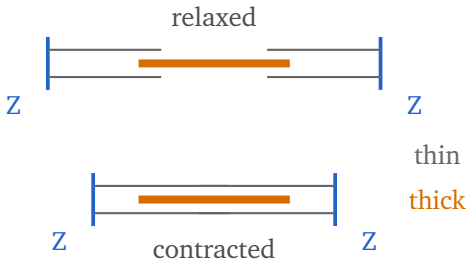
(A) 3 sodium ions in and 2 potassium ions out, without ATP

(B) 3 sodium ions out and 2 potassium ions in, using ATP

(C) 2 sodium ions out and 3 potassium ions in, using ATP

(D) 1 sodium ion out and 1 potassium ion in, using ATP

Q13. The sarcomere below is drawn relaxed and then contracted. According to the sliding filament theory, the change from the upper to the lower drawing happens because:



(A) The thin filaments slide over the thick filaments towards the centre, so the sarcomere shortens while filament lengths stay the same

(B) The thick filaments shorten by folding, pulling the Z lines together

(C) The thin filaments shorten while the thick filaments lengthen

(D) Both sets of filaments shorten equally

Q14. Insulin is released after a carbohydrate meal and lowers blood glucose within minutes. In skeletal muscle and adipose tissue it does this mainly by:

(A) Recruiting GLUT4 transporters from intracellular vesicles to the cell membrane

(B) Increasing hepatic glucose output through glycogenolysis

(C) Opening GLUT2 channels on the pancreatic beta cell

(D) Blocking glucose entry into the brain

Detailed Solutions

Q1.

Solution

Concept – clotting factors are made inactive and then finished off: The liver makes several factors as precursors that only work after one specific chemical modification.

Step 1 – name the modification: Glutamate residues near the amino end of the factor are carboxylated to **gamma-carboxyglutamate**.

Step 2 – name the cofactor: The enzyme gamma-glutamyl carboxylase needs **reduced vitamin K** as its cofactor for that reaction.

Step 3 – explain why it matters: The extra carboxyl groups let the factor chelate calcium, and calcium anchors the factor to the phospholipid surface of the platelet. Without that anchoring the factor floats uselessly in plasma.

Step 4 – list the factors involved: Factors **II, VII, IX and X**, plus protein C and protein S. These are the vitamin K dependent factors.

Step 5 – link it to clinical practice: Warfarin blocks vitamin K epoxide reductase, so vitamin K cannot be recycled to its reduced form, and all four factors fall. That is why a warfarinised patient bleeds after an extraction and why the PT rises first, factor VII having the shortest half life.

Why the other options are wrong:

- A, vitamin E: is a lipid soluble antioxidant that protects membrane fatty acids. It has no carboxylase role.
- C, vitamin B12: is a coenzyme for methylmalonyl-CoA mutase and methionine synthase. Its deficiency gives megaloblastic anaemia and subacute combined degeneration, not a carboxylation defect.
- D, folic acid: carries one-carbon units for purine and thymidylate synthesis. Its deficiency gives megaloblastic anaemia without neurological signs.

Final Answer: Vitamin K $\Rightarrow$ B

Answer: (B) Go Back to Q1

Q2.

Solution

Concept – night vision depends on one pigment: Rods handle vision in dim light, and rods work only if they hold enough rhodopsin.

Step 1 – read the clinical clue: Vision fails in dim light but daytime vision is normal, so the **rods** are failing while the cones are fine. This is **night blindness (nyctalopia)**.

Step 2 – recall what rhodopsin is made of: Rhodopsin is **11-cis-retinal** bound to the protein **opsin**.

Step 3 – trace where retinal comes from: Dietary **vitamin A** (retinol) is oxidised to retinal, the aldehyde form. The question specifically names the aldehyde form, which points straight to vitamin A.

Step 4 – explain the failure: With too little vitamin A there is too little 11-cis-retinal, so rhodopsin cannot be regenerated after each bleaching cycle. The rods then cannot respond to low light and the earliest symptom is night blindness.

Step 5 – note what follows if it continues: Untreated deficiency progresses to conjunctival xerosis, Bitot spots, corneal xerosis and finally keratomalacia, and it also causes keratinising metaplasia of epithelia elsewhere.

Why the other options are wrong:

- A, vitamin D: deficiency gives rickets in children and osteomalacia in adults through defective bone mineralisation. It plays no part in the visual cycle.
- B, niacin: deficiency causes pellagra, that is dermatitis, diarrhoea and dementia, along with a red raw glossitis. Vision is not the presenting problem.
- D, biotin: is the carboxylase coenzyme. Its deficiency is rare and gives dermatitis and alopecia, not night blindness.

Final Answer: Vitamin A $\Rightarrow$ C

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – name the inhibitor from what the two constants do: Each class of inhibitor moves K_m and V_{max} in its own pattern, so the pattern is the diagnosis.

Step 1 – read V_{max} off the graph: Curve **Y** plateaus at V'_{max}, which is clearly below the control plateau. So V_{max} is **decreased**.

Step 2 – read K_m off the graph: Both curves reach half of their own plateau at the same substrate concentration, so the apparent K_m is **unchanged**.

Step 3 – use the substrate clue: Adding more substrate does not restore the plateau, so the inhibitor is not competing for the active site.

Step 4 – match the pattern: V_{max} down with K_m unchanged, not reversed by substrate, is the signature of **non-competitive inhibition**.

Step 5 – confirm with the mechanism: A non-competitive inhibitor binds an **allosteric** site, away from the active site. It binds free enzyme and the enzyme-substrate complex equally well, so substrate cannot displace it. It effectively removes a fraction of enzyme molecules from service, which lowers the ceiling velocity, while the molecules still working bind substrate with their normal affinity, which is why K_m does not move.

Why the other options are wrong:

- A, competitive: raises K_m and leaves V_{max} unchanged, and it *is* overcome by more substrate. The graph shows the opposite on both counts.
- B, uncompetitive: binds only the enzyme-substrate complex, so it lowers **both** K_m and V_{max}. Here K_m has not moved.
- C, allosteric activation: would push the curve **up**, not down. Curve Y sits below the control.

Final Answer: Non-competitive $\Rightarrow$ D

Answer: (D) Go Back to Q3

Q4.

Solution

Concept – why the reciprocal plot exists: The Michaelis-Menten curve is a hyperbola, and you cannot read V_{max} off a hyperbola accurately because it only approaches its plateau. Taking reciprocals turns it into a straight line, and a straight line has intercepts you can read exactly.

Step 1 – start from the Michaelis-Menten equation: $v = \dfrac{V_{max}[S]}{K_m + [S]}$

Step 2 – invert both sides: $\dfrac{1}{v} = \dfrac{K_m + [S]}{V_{max}[S]}$

Step 3 – split the numerator: $\dfrac{1}{v} = \dfrac{K_m}{V_{max}[S]} + \dfrac{[S]}{V_{max}[S]}$

Step 4 – simplify the second term: $\dfrac{1}{v} = \dfrac{K_m}{V_{max}} \cdot \dfrac{1}{[S]} + \dfrac{1}{V_{max}}$

Step 5 – compare with $y = mx + c$: Here $y = 1/v$, $x = 1/[S]$, slope $m = K_m/V_{max}$ and intercept $c = 1/V_{max}$.

Step 6 – find intercept P, on the vertical axis: Put $x = 0$. Then $y = c = \mathbf{1/V_{max}}$. That is P.

Step 7 – find intercept R, on the horizontal axis: Put $y = 0$: $0 = \frac{K_m}{V_{max}}x + \frac{1}{V_{max}}$

$\frac{K_m}{V_{max}}x = -\frac{1}{V_{max}}$ $x = -\frac{1}{V_{max}} \cdot \frac{V_{max}}{K_m} = \mathbf{-1/K_m}$

Step 8 – read the answer: P $= 1/V_{max}$ and R $= -1/K_m$, which is why R sits to the *left* of the origin in the figure.

Why the other options are wrong:

- B, V_{max} and K_m: are what you read off the **direct** hyperbolic plot, not off a reciprocal plot. The whole point of taking reciprocals is that the axes now carry $1/v$ and $1/[S]$.
- C, $1/K_m$ and $-1/V_{max}$: simply swaps the two intercepts. Step 6 shows the vertical intercept carries V_{max}, not K_m.
- D, K_m/V_{max} and V_{max}: K_m/V_{max} is the **slope** of the line, not an intercept, and it is already labelled as such on the figure.

Final Answer: $1/V_{max}$ and $-1/K_m \Rightarrow$ A

Answer: (A) Go Back to Q4

Q5.

Solution

Concept – the HMP shunt is not for energy: The pathway exists to make NADPH and ribose-5-phosphate, and it produces no ATP at all.

Step 1 – read the marked step from the diagram: X converts glucose-6-phosphate to 6-phosphogluconolactone, and NADPH comes off it.

Step 2 – name the enzyme: That reaction is catalysed by **glucose-6-phosphate dehydrogenase (G6PD)**.

Step 3 – confirm it is rate limiting: G6PD is the committed and rate limiting step of the shunt. It is controlled by the $NADP^+$ to NADPH ratio, so the pathway runs faster exactly when NADPH has been used up.

Step 4 – explain why the red cell cares: The red cell has no mitochondria, so the HMP shunt is its **only** source of NADPH. NADPH keeps glutathione reduced, and reduced glutathione mops up hydrogen peroxide and other oxidants.

Step 5 – join it to the clinical picture: In G6PD deficiency, an oxidant stress such as primaquine, sulphonamides, nitrofurantoin, fava beans or infection overwhelms the little glutathione available. Haemoglobin is oxidised, precipitates as Heinz bodies, and the cell is destroyed. That is the acute haemolysis described.

Step 6 – confirm the epidemiology clue: G6PD deficiency is X-linked and is the commonest enzyme defect in humans, affecting several hundred million people, which matches "commonest human enzymopathy" exactly.

Why the other options are wrong:

- A, transketolase: works in the **non-oxidative** arm of the shunt, further downstream, and needs TPP. It is not the rate limiting step and it produces no NADPH.
- C, glucose-6-phosphatase: releases free glucose from glucose-6-phosphate in liver and kidney. Its deficiency is von Gierke disease. It is not part of the shunt.
- D, pyruvate carboxylase: is a **gluconeogenic** mitochondrial enzyme converting pyruvate to oxaloacetate. It has nothing to do with the shunt.

Final Answer: Glucose-6-phosphate dehydrogenase $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – ketone bodies are the liver's export fuel: The liver makes them for everyone else and then cannot burn them itself.

Step 1 – explain why they appear in starvation: Glycogen runs out in about a day. Adipose tissue then releases fatty acids, which the liver beta-oxidises to acetyl-CoA far faster than its own TCA cycle can accept.

Step 2 – explain why the TCA cycle cannot cope: Oxaloacetate is being pulled off into gluconeogenesis, so acetyl-CoA has nothing to condense with and it piles up.

Step 3 – name the site of synthesis: Surplus acetyl-CoA is diverted to ketogenesis in the **hepatic mitochondria**, with HMG-CoA synthase as the rate limiting enzyme. The products are acetoacetate, beta-hydroxybutyrate and a little acetone.

Step 4 – name the organ that cannot use them: The **liver** lacks **thiophorase (succinyl-CoA acetoacetate CoA transferase)**, the enzyme needed to reactivate acetoacetate. So the liver cannot oxidise the very fuel it makes, and every ketone

body it produces is exported.

Step 5 – check the option list against that: The question asks for the maker and the organ that cannot use them at all. Maker = liver, non-user = liver. Option B says liver and liver.

Step 6 – head off the usual trap: The brain *can* use ketone bodies. After a few days of starvation it derives a large share of its energy from them, which is exactly what spares muscle protein. So brain is not the answer to the second half.

Why the other options are wrong:

- A, muscle and brain: muscle does not synthesise ketone bodies, it **consumes** them. Both halves are wrong.
- C, kidney and heart: the kidney makes only a trivial amount, and cardiac muscle is one of the best ketone body **users** in the body.
- D, liver and brain: the first half is right, but the brain adapts to burn ketone bodies in prolonged starvation, so it is not the organ that cannot use them.

Final Answer: Liver and liver $\Rightarrow$ B

Answer: (B) Go Back to Q6

Q7.

Solution

Concept – count the cycles first, then the products: Beta-oxidation chops two carbons off at a time, so the arithmetic is fixed once you know the chain length.

Step 1 – count the acetyl-CoA produced: Palmitate has 16 carbons. $16 \div 2 = 8$ acetyl-CoA.

Step 2 – count the cycles needed: It takes $n - 1$ cycles to make n acetyl-CoA. $8 - 1 = 7$ cycles.

Step 3 – count the reducing equivalents from the cycles: Each cycle yields 1 $FADH_2$ and 1 NADH. $7 \times 1 = 7$ $FADH_2$ $7 \times 1 = 7$ NADH

Step 4 – convert those to ATP: 7 $FADH_2 \times 1.5 = 10.5$ ATP 7 NADH $\times 2.5 = 17.5$ ATP $10.5 + 17.5 = 28$ ATP

Step 5 – convert the acetyl-CoA to ATP: One acetyl-CoA through the TCA cycle gives 3 NADH, 1 $FADH_2$ and 1 GTP. $3 \times 2.5 = 7.5$ $1 \times 1.5 = 1.5$ $1 \times 1 = 1$ $7.5+1.5+1 = 10$ ATP per acetyl-CoA. $8 \times 10 = 80$ ATP

Step 6 – add the two halves: $28 + 80 = 108$ ATP

Step 7 – subtract the activation cost: Activating palmitate to palmitoyl-CoA

splits ATP to AMP plus pyrophosphate, which costs **2 ATP equivalents**, not one. $108 - 2 = \mathbf{106}$ ATP

Step 8 – sanity check the result: Palmitate has 16 carbons against glucose's 6 and is far more reduced, so a yield several times that of glucose is exactly what you expect. This is why fat is the body's dense fuel store.

Why the other options are wrong:

- B, 32 ATP: is the yield from one molecule of **glucose**, not palmitate. It is the classic distractor here.
- C, 12 ATP: is roughly one turn of the TCA cycle on the older ratios, that is a small fraction of what palmitate delivers.
- D, 380 ATP: is far too high. Even before subtracting activation the total only reaches 108.

Final Answer: 106 ATP $\Rightarrow$ A

Answer: (A) Go Back to Q7

Q8.

Solution

Concept – read an ABG in a fixed order: pH first, then find the value moving in the same direction as the pH, then check the other for compensation.

Step 1 – read the pH: pH 7.29 is below 7.35, so this is an **acidosis**.

Step 2 – read the $PaCO_2$: 62 mmHg is well above the normal 35 to 45. Retained CO_2 makes carbonic acid, which drives the pH down. So CO_2 is moving *with* the pH and is the **primary** problem. The disturbance is **respiratory**.

Step 3 – read the bicarbonate: 29 mEq/L is above the normal 22 to 26. A raised bicarbonate pushes pH *up*, so it is opposing the acidosis rather than causing it.

Step 4 – name that opposition: The kidney retains bicarbonate and excretes hydrogen ions to buffer the retained CO_2. That is **renal (metabolic) compensation**.

Step 5 – confirm it is only partial: The pH is still 7.29 and has not returned to 7.40, so compensation is incomplete. This is chronic respiratory acidosis with partial renal compensation.

Step 6 – fit the clinical story: COPD causes alveolar hypoventilation, so CO_2 accumulates over years. The drowsiness is CO_2 narcosis. Take care with sedation and with high flow oxygen in such a patient.

Why the other options are wrong:

- A, metabolic acidosis: would show a **low** bicarbonate and a low CO_2. Here both are high.
- B, respiratory alkalosis: needs a pH above 7.45 with a low CO_2. Both values here are the reverse.
- D, metabolic alkalosis: would give an alkaline pH. The pH here is 7.29, which is acidic, so the primary disturbance cannot be an alkalosis.

Final Answer: Respiratory acidosis with renal compensation $\Rightarrow$ C

Answer: (C) Go Back to Q8

Q9.

Solution

Concept – plasma must be electrically neutral: Total cations equal total anions. The laboratory only measures some of each, and the anion gap is the accounting difference that is left over.

Step 1 – list the measured cation: Sodium is the dominant measured cation.

Step 2 – list the measured anions: Chloride and bicarbonate are the two measured anions.

Step 3 – write the formula: Anion gap $= Na^+ - (Cl^- + HCO_3^-)$.

Step 4 – state the normal value: About 8 to 12 mEq/L. That residue is made up of unmeasured anions, mainly albumin, phosphate, sulphate and organic acids.

Step 5 – explain how it sorts an acidosis: If acid is added to the blood, the added acid's anion is usually unmeasured, so bicarbonate falls while chloride stays put and the gap **widens**. Causes include diabetic ketoacidosis, lactic acidosis, renal failure and methanol, ethylene glycol or salicylate poisoning.

Step 6 – give the other group: If bicarbonate is lost directly rather than titrated away, chloride is retained to keep neutrality, so the gap stays **normal**. Causes include diarrhoea and renal tubular acidosis. This is hyperchloraemic acidosis.

Step 7 – note the albumin correction: Albumin is the main unmeasured anion, so a low albumin lowers the measured gap and can hide a real high gap acidosis. Add roughly 2.5 mEq/L to the gap for every 1 g/dL the albumin is below normal.

Why the other options are wrong:

- A, Na plus K minus Cl: adds potassium but drops bicarbonate. Bicarbonate is the value that actually moves in an acidosis, so omitting it defeats the whole purpose of the calculation.
- B, Cl plus HCO_3 minus Na: is the correct formula with the sign reversed, so

it returns a negative number and is not what is used.
- C, Na plus Cl minus HCO_3: adds chloride to sodium instead of subtracting it, mixing an anion in with the cation. It is not a gap at all.

Final Answer: Sodium minus (chloride plus bicarbonate) $\Rightarrow$ D

Answer: (D) Go Back to Q9

Q10.

Solution

Concept – let each test point to one limb of the cascade: PT screens the extrinsic limb, aPTT screens the intrinsic limb, and the platelet count screens primary haemostasis. Read them together.

Step 1 – use the normal platelet count: Primary haemostasis is intact, so this is not a platelet problem.

Step 2 – use the normal PT: The extrinsic limb (factor VII) and the common pathway (X, V, II, fibrinogen) are working. If the common pathway were faulty, the PT would also be prolonged.

Step 3 – use the prolonged aPTT: Only the **intrinsic** limb is faulty, that is factor VIII, IX, XI or XII.

Step 4 – use the mixing study: The aPTT corrects when normal plasma is added, so the patient is **missing a factor** rather than carrying an inhibitor. An inhibitor such as a lupus anticoagulant would not correct.

Step 5 – use the clinical picture: Bleeding into joints (haemarthrosis) since infancy in a boy is the classic picture of an X-linked recessive severe factor deficiency.

Step 6 – pick between VIII and IX: Both haemophilia A (factor VIII) and haemophilia B (factor IX) give this pattern, but haemophilia A is about **five times commoner** and is the expected single best answer. Factor VIII is the choice.

Step 7 – note the dental relevance: Such a patient needs factor VIII cover before extraction, plus tranexamic acid and local measures. Never rely on pressure alone.

Why the other options are wrong:

- A, factor VII: sits in the extrinsic limb, so its deficiency prolongs the **PT**. The PT here is normal.
- B, factor XIII: cross-links fibrin after the clot has formed, so **both** PT and aPTT are normal. Its deficiency shows as delayed rebleeding and needs a urea clot solubility test.
- D, fibrinogen: sits in the common pathway, so its deficiency prolongs **both**

PT and aPTT. The normal PT rules it out.

Final Answer: Factor VIII ⇒ C

Answer: (C) Go Back to Q10

Q11.

Solution

Concept – red cell numbers are held steady by a feedback loop: Cells are destroyed at a fixed rate, so production must be sensed and matched.

Step 1 – take the lifespan given: A red cell survives about **120 days**, after which the ageing cell is removed by macrophages of the spleen, liver and marrow.

Step 2 – work out the replacement load: About one one-hundred-twentieth of the red cell mass, roughly 1 percent, must be replaced every single day. That is around 2 million cells per second.

Step 3 – name the hormone: Erythropoietin drives that replacement. It acts on the burst forming and colony forming erythroid progenitors in the marrow, pushing them to divide and mature and preventing their apoptosis.

Step 4 – name the source in the adult: Erythropoietin is made by the **peritubular interstitial fibroblasts of the renal cortex**, so the kidney is the answer.

Step 5 – name the trigger: Tissue hypoxia stabilises hypoxia inducible factor in those cells, which switches on the erythropoietin gene. Anaemia, high altitude or lung disease therefore raise erythropoietin.

Step 6 – confirm with the disease: In chronic kidney disease the fibroblasts are lost, erythropoietin falls, and the patient becomes anaemic despite a normal marrow. That anaemia responds to recombinant erythropoietin, which proves the kidney is the source.

Why the other options are wrong:

- A, thrombopoietin from the liver: thrombopoietin is indeed largely hepatic, but it regulates **platelets** through megakaryocytes, not red cells.
- B, erythropoietin from the bone marrow: names the right hormone but the wrong source. The marrow is the **target** of erythropoietin, not its factory.
- C, erythropoietin from the liver: the liver does make a small share, around 10 percent, and is the main source in the **fetus**. In the adult the kidney is the answer, which is what the question asks for.

Final Answer: Erythropoietin from the kidney ⇒ D

Answer: (D) Go Back to Q11

Q12.

Solution

Concept – the resting potential rests on gradients, and gradients cost energy: The membrane potential is not maintained by the pump directly. The pump maintains the gradients, and the gradients plus the membrane's permeability set the potential.

Step 1 – name the pump: The **sodium-potassium ATPase (Na^+/K^+ ATPase)** in the plasma membrane.

Step 2 – state its stoichiometry: For every ATP hydrolysed it moves **3 sodium ions out** of the cell and **2 potassium ions in**.

Step 3 – note that it needs ATP: Both ions are moved **against** their concentration gradients, so this is primary active transport and ATP is compulsory. Ouabain and digoxin inhibit it.

Step 4 – state the gradients it creates: Sodium ends up high outside (about 145 mEq/L) and low inside (about 12 mEq/L). Potassium ends up high inside (about 140 mEq/L) and low outside (about 4 mEq/L).

Step 5 – explain how that gives -70 mV: At rest the membrane is far more permeable to potassium than to sodium, through leak channels. Potassium therefore diffuses out down its gradient, leaving the impermeant intracellular anions behind, and the inside turns negative. The potential settles near the potassium equilibrium potential, pulled slightly positive by the small sodium leak.

Step 6 – add the pump's direct contribution: Since 3 positive charges leave for every 2 that enter, the pump exports net positive charge. It is **electrogenic** and contributes a few millivolts of extra negativity itself, but its main role is keeping the gradients topped up.

Why the other options are wrong:

- A, 3 sodium in and 2 potassium out without ATP: has the directions reversed, which would destroy the gradients rather than build them, and no energy source is offered for uphill transport.
- C, 2 sodium out and 3 potassium in: has the numbers swapped. That would import net positive charge and make the pump electrogenic in the wrong direction.
- D, 1 for 1: would be electroneutral, so the pump would contribute nothing to the potential directly, and it is simply not the stoichiometry of this pump.

Final Answer: 3 sodium out and 2 potassium in, using ATP $\Rightarrow$ B

Answer: (B) Go Back to Q12

Q13.

Solution

Concept – muscle shortens without any filament shortening: This is the single idea the sliding filament theory of Huxley contributes, and it is counter-intuitive enough to be examined every year.

Step 1 – read the figure: The Z lines are closer together in the lower drawing, so the sarcomere has shortened.

Step 2 – measure the filaments in the figure: The orange thick filament is drawn exactly the same length in both panels, and each thin filament is the same length in both. Only their **overlap** has increased.

Step 3 – state the mechanism: Myosin heads on the thick filament bind actin, swing (the power stroke), and drag the thin filament towards the centre of the sarcomere. The heads then detach when fresh ATP binds and repeat the cycle.

Step 4 – follow what that does to the bands: The **I band** narrows and the **H zone** narrows, because both are regions without overlap. The **A band**, which is the length of the thick filament, does **not** change. This band behaviour is the direct proof that no filament shortens.

Step 5 – recall the trigger: Calcium released from the sarcoplasmic reticulum binds troponin C, tropomyosin shifts off the actin binding sites, and cross-bridge cycling can begin.

Step 6 – pick the option: Thin filaments slide inwards, sarcomere shortens, filament lengths unchanged. That is option A.

Why the other options are wrong:

- B, thick filaments fold and shorten: this was the old pre-Huxley belief. It is disproved by the A band staying a constant width throughout contraction.
- C, thin shorten while thick lengthen: neither filament changes length, and nothing in the figure lengthens.
- D, both shorten equally: again contradicts the constant A band, and the figure shows both filaments drawn identically in the two panels.

Final Answer: Thin filaments slide over thick, filament lengths unchanged $\Rightarrow$ A

Answer: (A) Go Back to Q13

Q14.

Solution

Concept – insulin clears glucose by opening doors that already exist: The transporters are pre-made and stored inside the cell, which is why insulin can act within minutes rather than hours.

Step 1 – name the two insulin sensitive tissues: Skeletal muscle and **adipose tissue**. Both carry the **GLUT4** transporter.

Step 2 – state where GLUT4 sits when insulin is absent: GLUT4 is parked in **intracellular vesicles**, away from the surface, so glucose uptake by these tissues is low while fasting.

Step 3 – follow the signal: Insulin binds its receptor, a tyrosine kinase. The receptor autophosphorylates, phosphorylates IRS-1, and activates PI3-kinase and then Akt.

Step 4 – state what Akt does: Akt drives the GLUT4 vesicles to fuse with the plasma membrane. This is **translocation** or recruitment.

Step 5 – state the result: Many more transporters now sit in the membrane, so facilitated diffusion of glucose into the cell rises sharply and blood glucose falls. Removing insulin reverses it, the transporters being internalised again.

Step 6 – add insulin's other actions for completeness: It also stimulates glycogen synthesis in muscle and liver, stimulates lipogenesis, inhibits lipolysis, inhibits gluconeogenesis and glycogenolysis, and promotes potassium entry into cells. Every one of these lowers blood glucose or stores fuel.

Step 7 – note the clinical hook: Type 2 diabetes is largely a failure of this GLUT4 translocation step, that is insulin resistance, rather than an absence of insulin.

Why the other options are wrong:

- B, increasing hepatic glucose output: is exactly backwards. That is what **glucagon** does, and it raises blood glucose. Insulin *suppresses* glycogenolysis and gluconeogenesis.
- C, opening GLUT2 on the beta cell: GLUT2 is insulin **independent**. It is the beta cell's glucose sensor and it lets glucose in to trigger insulin release. It is upstream of insulin, not a target of it.
- D, blocking glucose entry into the brain: the brain uses GLUT1 and GLUT3, both insulin independent, precisely so that neurons keep receiving glucose whatever the insulin level. Blocking brain glucose would be lethal.

Final Answer: Recruiting GLUT4 to the cell membrane $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q14

Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	B	2	C	3	D	4	A	5	B
6	B	7	A	8	C	9	D	10	C
11	D	12	B	13	A	14	A		