NEET MDS General Physiology and Biochemistry

Sample Paper – 1

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. A patient presents with swollen bleeding gums, loose teeth and perifollicular haemorrhages. The deficient vitamin acts as a cofactor for the hydroxylation of proline and lysine during collagen synthesis. That vitamin is:

(A) Vitamin A

(B) Vitamin K

(C) Vitamin C

(D) Vitamin D

Q2. The active coenzyme form of vitamin B1 (thiamine), required by pyruvate dehydrogenase for the oxidative decarboxylation of pyruvate, is:

(A) Thiamine pyrophosphate (TPP)

(B) Flavin adenine dinucleotide (FAD)

(C) Pyridoxal phosphate (PLP)

(D) Nicotinamide adenine dinucleotide (NAD^+)

Enzymes and Enzyme Kinetics

Q3. The Michaelis constant (K_m) of an enzyme is defined as the substrate concentration at which:

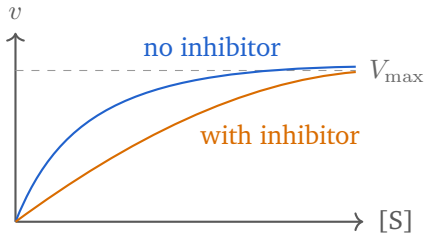
(A) The enzyme is completely saturated with substrate

(B) The reaction velocity is half of V_{max}

(C) The reaction velocity falls to zero

(D) V_{max} is doubled

Q4. The curves below show the same enzyme assayed with and without an inhibitor. The inhibitor raises the apparent K_m while V_{max} is unchanged, and the effect is overcome by adding more substrate. This pattern indicates:



(A) Non-competitive inhibition

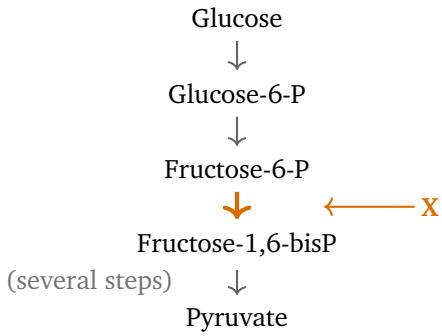
(B) Competitive inhibition

(C) Uncompetitive inhibition

(D) Irreversible inhibition

Metabolism of Carbohydrate, Lipid and Protein

Q5. In the outline of glycolysis below, the step marked **X** is irreversible and is the rate limiting step of the whole pathway. The enzyme catalysing **X** is:



(A) Phosphofructokinase-1

(B) Hexokinase

(C) Pyruvate kinase

(D) Aldolase

Q6. The rate limiting enzyme of the urea cycle, which is located in the mitochondrial matrix and requires N-acetylglutamate as an obligatory allosteric activator, is:

(A) Arginase

(B) Ornithine transcarbamoylase

(C) Argininosuccinate synthetase

(D) Carbamoyl phosphate synthetase I

Q7. Using the currently accepted P/O ratios, the net ATP yield from the complete aerobic oxidation of one molecule of glucose to carbon dioxide and water is approximately:

(A) 2 ATP

(B) 12 ATP

(C) 30 to 32 ATP

(D) 100 ATP

Acid Base Balance and Body Fluids

Q8. An arterial blood gas report reads: pH 7.30, $PaCO_2$ 30 mmHg, bicarbonate 14 mEq/L. The correct interpretation is:

(A) Respiratory acidosis

(B) Respiratory alkalosis

(C) Metabolic acidosis with respiratory compensation

(D) Metabolic alkalosis with respiratory compensation

Q9. In a healthy 70 kg adult male, total body water is about 42 litres. The volume held in the intracellular fluid compartment is approximately:

(A) 14 litres

(B) 28 litres

(C) 3.5 litres

(D) 42 litres

Blood and Haemostasis

Q10. A patient booked for extraction has a prolonged bleeding time but a normal clotting time. This combination points to a defect in:

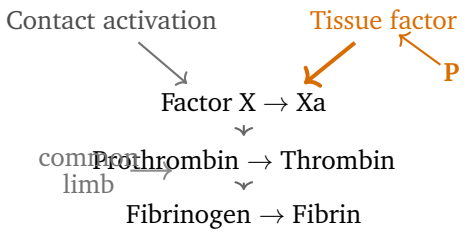
(A) Platelet number or platelet function

(B) Factor VIII

(C) Factor IX

(D) The conversion of fibrinogen to fibrin

Q11. In the outline of the coagulation cascade below, the limb marked **P** is triggered by tissue factor and is the limb assessed by the prothrombin time (PT). Limb **P** is the:



(A) Intrinsic pathway

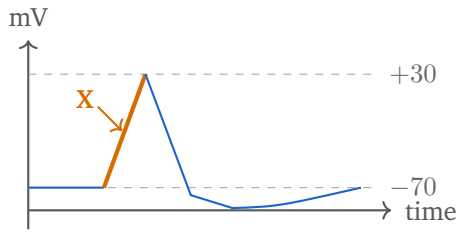
(B) Extrinsic pathway

(C) Common pathway

(D) Fibrinolytic pathway

Nerve, Muscle and Endocrine Physiology

Q12. The trace below shows a nerve action potential. The rapid upstroke marked **X**, which carries the membrane from threshold towards $+30$ mV, is produced by:



(A) Efflux of potassium through voltage-gated potassium channels

(B) Influx of chloride through ligand-gated channels

(C) Efflux of calcium from the sarcoplasmic reticulum

(D) Influx of sodium through voltage-gated sodium channels

Q13. The neurotransmitter released at the somatic neuromuscular junction, which binds nicotinic receptors on the motor end plate and is broken down by acetylcholinesterase in the synaptic cleft, is:

(A) Noradrenaline

(B) Dopamine

(C) Gamma-aminobutyric acid (GABA)

(D) Acetylcholine

Q14. Parathyroid hormone is secreted in response to a fall in plasma calcium. Its combined actions on bone, kidney and gut are:

(A) Increased bone resorption, increased renal calcium reabsorption and increased activation of vitamin D

(B) Decreased bone resorption, increased renal calcium excretion and decreased activation of vitamin D

(C) Increased bone deposition, increased renal phosphate reabsorption and no effect on vitamin D

(D) Decreased bone resorption, decreased renal calcium reabsorption and increased calcitonin release

Detailed Solutions

Q1.

Solution

Concept – vitamin C and collagen: Collagen is only strong once its proline and lysine residues are hydroxylated.

Step 1 – name the enzymes involved: Prolyl hydroxylase and lysyl hydroxylase carry out that hydroxylation.

Step 2 – identify their cofactor: Both enzymes need **ascorbic acid (vitamin C)** to keep their iron in the reduced ferrous state.

Step 3 – state what fails without it: Without hydroxylation the collagen triple helix cannot cross-link properly, so it is weak and unstable.

Step 4 – link to the clinical picture: Weak collagen in blood vessel walls gives bleeding gums, loose teeth, perifollicular haemorrhages and poor wound healing. This is **scurvy**.

Why the other options are wrong:

- A, vitamin A: deficiency causes night blindness, xerophthalmia and keratinising metaplasia, not a collagen defect.
- B, vitamin K: is needed for the gamma-carboxylation of clotting factors II, VII, IX and X. Its deficiency does cause bleeding, but through a coagulation defect and not through collagen.
- D, vitamin D: deficiency causes rickets and osteomalacia by impairing calcium absorption and bone mineralisation.

Final Answer: Vitamin C $\Rightarrow$ C

Answer: (C) Go Back to Q1

Q2.

Solution

Concept – each B vitamin has its own coenzyme form: The vitamin is the dietary molecule; the coenzyme is the working form the enzyme actually uses.

Step 1 – take vitamin B1: Thiamine is phosphorylated to **thiamine pyrophosphate (TPP)**.

Step 2 – find where TPP works: TPP is the coenzyme for oxidative decarboxylations, including pyruvate dehydrogenase, alpha-ketoglutarate dehydrogenase and

transketolase.

Step 3 – confirm against the question: The question names pyruvate dehydrogenase, so the coenzyme required is TPP.

Step 4 – note the deficiency: Without TPP, pyruvate cannot enter the TCA cycle, lactate accumulates, and the patient develops beriberi or Wernicke encephalopathy.

Why the other options are wrong:

- B, FAD: is the coenzyme of vitamin B2 (riboflavin). It is present in the pyruvate dehydrogenase complex but is not the vitamin B1 form the question asks for.
- C, PLP: is the coenzyme of vitamin B6 (pyridoxine), used in transamination and decarboxylation of amino acids.
- D, NAD^+: is the coenzyme of vitamin B3 (niacin), used as a hydride acceptor in oxidation reactions.

Final Answer: Thiamine pyrophosphate $\Rightarrow$ A

Answer: (A) Go Back to Q2

Q3.

Solution

Concept – what K_m actually measures: K_m is a substrate concentration, not a velocity, and it reports how tightly an enzyme binds its substrate.

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

Step 2 – substitute $[S] = K_m$: $v = \dfrac{V_{max}\,K_m}{K_m + K_m} = \dfrac{V_{max}\,K_m}{2K_m}$

Step 3 – simplify: $v = \dfrac{V_{max}}{2}$

Step 4 – read the meaning back: So K_m is exactly the substrate concentration giving half-maximal velocity. A *low* K_m therefore means high affinity, because little substrate is needed to reach half speed.

Why the other options are wrong:

- A, full saturation: happens as $[S]$ approaches infinity, where v approaches V_{max}, not at K_m.
- C, velocity zero: occurs when $[S] = 0$, which tells you nothing about the enzyme's affinity.

- D, V_{max} doubled: V_{max} is a fixed ceiling for a given enzyme concentration. Substrate cannot double it.

Final Answer: The substrate concentration at half of V_{max} $\Rightarrow$ $\boxed{B}$

Answer: (B) Go Back to Q3

Q4.

Solution

Concept – read inhibition from what happens to K_m and V_{max}: The two constants move in a different pattern for each type of inhibitor, so the pattern names the type.

Step 1 – read V_{max} from the graph: Both curves plateau at the same height, so V_{max} is **unchanged**.

Step 2 – read K_m from the graph: The inhibited curve needs more substrate to reach half its plateau, so the apparent K_m is **increased**.

Step 3 – match the pattern: K_m up with V_{max} unchanged is the signature of **competitive inhibition**.

Step 4 – confirm with the mechanism: A competitive inhibitor resembles the substrate and binds the active site. Adding more substrate outcompetes it, which is why the enzyme still reaches the same V_{max}, just at a higher $[S]$. The question says the effect is overcome by more substrate, which fits exactly.

Why the other options are wrong:

- A, non-competitive: binds an allosteric site, so it **lowers** V_{max} and leaves K_m unchanged. The graph shows the opposite.
- C, uncompetitive: binds only the enzyme-substrate complex, so it lowers **both** K_m and V_{max}.
- D, irreversible: permanently inactivates enzyme molecules, lowering V_{max}, and it cannot be overcome by more substrate.

Final Answer: Competitive inhibition $\Rightarrow$ $\boxed{B}$

Answer: (B) Go Back to Q4

Q5.

Solution

Concept – the three irreversible steps of glycolysis: Glycolysis has three one-way steps, and the regulated one among them sets the pathway's pace.

Step 1 – list the three: Hexokinase or glucokinase, phosphofructokinase-1, and pyruvate kinase.

Step 2 – read the step from the diagram: The marked step converts fructose-6-phosphate to fructose-1,6-bisphosphate.

Step 3 – name its enzyme: That conversion is catalysed by **phosphofructokinase-1 (PFK-1)**.

Step 4 – confirm it is rate limiting: PFK-1 is the committed and rate limiting step. It is inhibited by ATP and citrate and activated by AMP and fructose-2,6-bisphosphate, which is exactly the regulation a pacemaker enzyme needs.

Why the other options are wrong:

- B, hexokinase: catalyses the first step, glucose to glucose-6-phosphate. It is irreversible but not rate limiting, since its product feeds several pathways.
- C, pyruvate kinase: catalyses the last step, phosphoenolpyruvate to pyruvate. It is irreversible and regulated, but it is not the pacemaker.
- D, aldolase: splits fructose-1,6-bisphosphate into two triose phosphates. That step is freely reversible and is not regulated.

Final Answer: Phosphofructokinase-1 $\Rightarrow$ A

Answer: (A) Go Back to Q5

Q6.

Solution

Concept – regulation of the urea cycle: The urea cycle disposes of ammonia, and its first step is the one that is controlled.

Step 1 – recall the first reaction: Ammonia plus carbon dioxide plus 2 ATP gives carbamoyl phosphate, catalysed by **carbamoyl phosphate synthetase I (CPS-I)**.

Step 2 – locate it: CPS-I sits in the **mitochondrial matrix**, which matches the question.

Step 3 – identify its activator: CPS-I is inactive without **N-acetylglutamate**, an obligatory allosteric activator made when amino acids are plentiful.

Step 4 – explain why that makes it rate limiting: Because a high amino acid load

raises N-acetylglutamate, which switches CPS-I on, the cycle speeds up exactly when there is more ammonia to clear. That is the definition of the controlling step.

Why the other options are wrong:

- A, arginase: catalyses the **last** step, splitting arginine into urea and ornithine, and it is cytosolic.
- B, ornithine transcarbamoylase: is mitochondrial and its deficiency is the commonest urea cycle disorder, but it is the second step and is not the rate limiting one.
- C, argininosuccinate synthetase: is cytosolic and catalyses the third step, so it fails both the location and the regulation clue.

Final Answer: Carbamoyl phosphate synthetase I $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q6

Q7.

Solution

Concept – adding up the ATP from one glucose: The total comes from glycolysis, the link reaction, the TCA cycle and oxidative phosphorylation.

Step 1 – glycolysis: Net 2 ATP by substrate-level phosphorylation, plus 2 NADH.

Step 2 – the link reaction: Two pyruvates are decarboxylated to two acetyl-CoA, giving 2 NADH.

Step 3 – the TCA cycle: Two turns give 6 NADH, 2 $FADH_2$ and 2 GTP (equivalent to 2 ATP).

Step 4 – apply the modern P/O ratios: Each NADH yields about 2.5 ATP and each $FADH_2$ about 1.5 ATP, rather than the older 3 and 2.

Step 5 – total it: 10 NADH $\times$ 2.5 = 25, plus 2 $FADH_2$ $\times$ 1.5 = 3, plus 4 ATP from substrate-level steps, giving about **32 ATP**. The figure drops to about 30 if the glycerol phosphate shuttle is used instead of the malate-aspartate shuttle, which is why the accepted answer is a range.

Why the other options are wrong:

- A, 2 ATP: is the **anaerobic** yield, where glycolysis alone runs and pyruvate becomes lactate.
- B, 12 ATP: is roughly the yield of one turn of the TCA cycle, not of a whole glucose molecule.

- D, 100 ATP: is far beyond what a six-carbon sugar can deliver.

Final Answer: 30 to 32 ATP $\Rightarrow$ C

Answer: (C) Go Back to Q7

Q8.

Solution

Concept – read an ABG in a fixed order: Take the pH first, then the primary driver, then the compensation. Never guess from one value.

Step 1 – read the pH: pH 7.30 is below 7.35, so this is an **acidosis**.

Step 2 – decide which system is the culprit: Bicarbonate is 14 mEq/L, which is low (normal 22 to 26). A low bicarbonate drives acidosis, so the primary problem is **metabolic**.

Step 3 – check the other value against that: $PaCO_2$ is 30 mmHg, which is low (normal 35 to 45). A low CO_2 would push the pH *up*, so it is opposing the acidosis rather than causing it.

Step 4 – name that opposition: Blowing off CO_2 by hyperventilating is **respiratory compensation** for the metabolic acidosis.

Step 5 – confirm it is only partial: The pH is still 7.30, not back to 7.40, so the compensation is incomplete. The picture is metabolic acidosis with partial respiratory compensation.

Why the other options are wrong:

- A, respiratory acidosis: would require a **high** $PaCO_2$. Here it is low.
- B, respiratory alkalosis: would give a pH above 7.45. Here the pH is acidic.
- D, metabolic alkalosis: would give a high pH and a **high** bicarbonate. Both are the opposite of this report.

Final Answer: Metabolic acidosis with respiratory compensation $\Rightarrow$ C

Answer: (C) Go Back to Q8

Q9.

Solution

Concept – the 60-40-20 rule of body fluids: Body water splits between cells and the space outside them in a fixed proportion.

Step 1 – start from total body water: Total body water is about 60 percent of body weight, so 70 kg $\times$ 0.6 = 42 litres, as the question states.

Step 2 – apply the two-thirds rule: Two-thirds of total body water is intracellular.

Step 3 – calculate: $\frac{2}{3} \times 42 = 28$ litres.

Step 4 – check the remainder: The other third, 14 litres, is extracellular. That splits again into about 10.5 litres interstitial and about 3.5 litres plasma, and those numbers account for the distractors.

Why the other options are wrong:

- A, 14 litres: is the **extracellular** volume, that is the other third.
- C, 3.5 litres: is the **plasma** volume, a subdivision of the extracellular compartment.
- D, 42 litres: is the **total** body water, not the intracellular share.

Final Answer: 28 litres $\Rightarrow$ B

Answer: (B) Go Back to Q9

Q10.

Solution

Concept – bleeding time and clotting time test different things: The two tests separate the platelet phase of haemostasis from the coagulation phase.

Step 1 – state what bleeding time measures: Bleeding time reflects the **platelet** plug, that is platelet number, platelet function and vessel wall integrity.

Step 2 – state what clotting time measures: Clotting time reflects the **coagulation cascade**, that is the fibrin clot.

Step 3 – read the pattern given: Bleeding time is prolonged while clotting time is normal, so the platelet phase has failed and the cascade is intact.

Step 4 – name the defect: The defect must be in **platelet number or platelet function**, as in thrombocytopenia, von Willebrand disease or recent aspirin.

Step 5 – note the dental relevance: This is exactly the patient who oozes persistently from a socket despite a clot forming, so local haemostatic measures matter

more than clotting factor replacement.

Why the other options are wrong:

- B, factor VIII: deficiency is haemophilia A, which prolongs the **clotting time** and the aPTT while bleeding time stays normal.
- C, factor IX: deficiency is haemophilia B, giving the same cascade pattern as haemophilia A, not a platelet pattern.
- D, fibrinogen to fibrin: is the final cascade step, so its failure prolongs the clotting time, not the bleeding time.

Final Answer: Platelet number or function $\Rightarrow$ A

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – two limbs feed one common pathway: The cascade has an intrinsic limb and an extrinsic limb, and each has its own screening test.

Step 1 – use the trigger clue: The question says limb **P** is triggered by **tissue factor**, which is released by damaged tissue outside the blood.

Step 2 – name that limb: Tissue factor plus factor VII starts the **extrinsic pathway**.

Step 3 – confirm with the test clue: The extrinsic pathway is screened by the **prothrombin time (PT)**, which the question also states. Both clues agree.

Step 4 – place it in the cascade: The extrinsic limb converges on factor X, after which the common pathway runs to thrombin and then fibrin, exactly as the diagram shows.

Why the other options are wrong:

- A, intrinsic pathway: is triggered by contact activation within the blood and is screened by the **aPTT**, not the PT.
- C, common pathway: begins **at** factor X, after both limbs have converged. The diagram marks P before that convergence.
- D, fibrinolytic pathway: breaks a clot down through plasmin. It is not part of clot formation at all.

Final Answer: Extrinsic pathway $\Rightarrow$ B

Answer: (B) Go Back to Q11

Q12.

Solution

Concept – each phase of the action potential has its own ion: Read the phase off the trace, then name the channel that produces it.

Step 1 – identify the phase marked: X is the rapid upstroke, that is **depolarisation**.

Step 2 – recall what opens at threshold: At about -55 mV, **voltage-gated sodium channels** open.

Step 3 – follow the electrochemical gradient: Sodium is high outside and the inside is negative, so both the concentration and the electrical gradient drive sodium **in**.

Step 4 – explain the steepness: Sodium entry makes the inside less negative, which opens still more sodium channels. That positive feedback is what makes the upstroke so fast and takes the membrane towards $+30$ mV.

Step 5 – note what follows: Sodium channels then inactivate and potassium channels open, giving the repolarising limb shown after the peak.

Why the other options are wrong:

- A, potassium efflux: causes **repolarisation**, the downstroke after the peak, not the upstroke.
- B, chloride influx: is inhibitory and hyperpolarises the membrane, moving it away from threshold.
- C, calcium efflux from the sarcoplasmic reticulum: is an intracellular event in muscle contraction, and it does not generate the nerve action potential upstroke.

Final Answer: Influx of sodium $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q12

Q13.

Solution

Concept – the neuromuscular junction is cholinergic: Every somatic motor neuron uses the same transmitter at its end plate.

Step 1 – name the transmitter: The somatic motor neuron releases **acetylcholine** from its terminal.

Step 2 – name its receptor: It binds **nicotinic** receptors on the motor end plate,

which are ligand-gated cation channels.

Step 3 – state what that does: Cation entry produces the end plate potential, which triggers the muscle action potential and then contraction.

Step 4 – name its breakdown: Acetylcholinesterase in the cleft hydrolyses it to choline and acetate, ending the signal. All three clues in the question point to acetylcholine.

Why the other options are wrong:

- A, noradrenaline: is the transmitter at most postganglionic **sympathetic** endings, acting on adrenergic receptors, not at the somatic end plate.
- B, dopamine: acts in central pathways such as the nigrostriatal tract, not at the neuromuscular junction.
- C, GABA: is the main inhibitory transmitter of the central nervous system and is not used at the somatic end plate.

Final Answer: Acetylcholine $\Rightarrow$ D

Answer: (D) Go Back to Q13

Q14.

Solution

Concept – PTH is the hormone that defends plasma calcium: Every one of its actions pushes calcium up, so a correct option must be internally consistent.

Step 1 – its action on bone: PTH stimulates osteoclastic **bone resorption** indirectly, through RANKL expressed by osteoblasts, releasing calcium into the blood.

Step 2 – its action on the kidney, for calcium: PTH **increases** calcium reabsorption in the distal tubule, so less is lost in urine.

Step 3 – its action on the kidney, for phosphate: PTH **decreases** phosphate reabsorption, so phosphate is lost. This is why PTH raises calcium but lowers phosphate.

Step 4 – its action on vitamin D: PTH activates renal 1-alpha-hydroxylase, converting 25-hydroxyvitamin D into **calcitriol**, which then increases calcium absorption from the gut.

Step 5 – combine them: Bone resorption up, renal calcium reabsorption up, vitamin D activation up. All three raise plasma calcium, which is exactly option A.

Why the other options are wrong:

- B: has every action reversed. Lowering calcium is what **calcitonin** does, not PTH.
- C: says PTH increases bone deposition and renal phosphate reabsorption. PTH does the opposite of both, and it certainly affects vitamin D.
- D: describes calcium loss and calcitonin release, which would lower plasma calcium and defeat the whole purpose of PTH secretion.

Final Answer: Bone resorption, renal calcium reabsorption and vitamin D activation all increased $\Rightarrow$ A

Answer: (A) Go Back to Q14

Answer Key

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