INI CET Biochemistry

Sample Paper – 2

Duration: 14 Minutes **Maximum Marks: 15**

Instructions

- This paper contains **15** Multiple Choice Questions (Single Best Response), modelled on the Biochemistry content of **INI CET** (Institutes of National Importance Combined Entrance Test) conducted by AIIMS New Delhi.
- The **15-question** length matches the number of Biochemistry items you actually meet in the real 200-question paper.
- Each correct answer carries **+1 mark**. There is **negative marking of one-third (0.33) 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 **14 minutes**, the pace the real computer-based test demands.

Carbohydrate Metabolism

Q1. Which statement correctly distinguishes glucokinase from hexokinase?

(A) Glucokinase has a low K_m and is present in all tissues

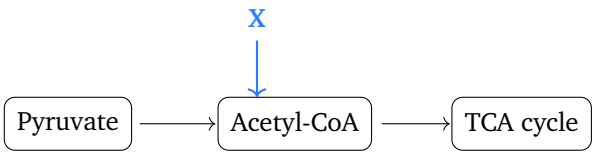
(B) Glucokinase has a high K_m and is found mainly in liver and pancreatic beta cells

(C) Glucokinase is strongly inhibited by its product glucose-6-phosphate

(D) Glucokinase has a lower K_m for glucose than hexokinase

Q2. In the pathway shown, the enzyme complex marked **X** converts pyruvate to acetyl-CoA and links glycolysis to the TCA cycle. This pyruvate

dehydrogenase complex has an absolute requirement for which vitamin-derived coenzyme?



(A) Thiamine pyrophosphate (B_1)

(B) Pyridoxal phosphate (B_6)

(C) Biotin (B_7)

(D) Ascorbate (vitamin C)

Q3. Von Gierke disease (glycogen storage disease type I), presenting with severe fasting hypoglycaemia, hepatomegaly and lactic acidosis, is caused by deficiency of:

(A) Glucose-6-phosphatase

(B) Muscle glycogen phosphorylase

(C) Lysosomal alpha-glucosidase (acid maltase)

(D) Debranching enzyme

Lipid Metabolism

Q4. The rate-limiting step controlling the entry of long-chain fatty acids into the mitochondrion for beta-oxidation is catalysed by:

(A) Acyl-CoA synthetase

(B) Carnitine palmitoyltransferase-1 (CPT-1)

(C) Carnitine-acylcarnitine translocase

(D) Carnitine palmitoyltransferase-2 (CPT-2)

Q5. Ketone bodies such as acetoacetate and beta-hydroxybutyrate are synthesised primarily in which organ and subcellular compartment?

(A) Kidney cytoplasm

(B) Liver mitochondria

(C) Skeletal muscle mitochondria

(D) Adipose tissue cytoplasm

Amino Acid and Protein Metabolism

Q6. Phenylketonuria results from deficiency of phenylalanine hydroxylase, an enzyme that requires which cofactor to convert phenylalanine to tyrosine?

(A) Tetrahydrofolate

(B) Pyridoxal phosphate

(C) Tetrahydrobiopterin (BH_4)

(D) S-adenosylmethionine

Q7. The rate-limiting enzyme of heme biosynthesis, located in the mitochondrion and feedback-inhibited by heme, is:

(A) Ferrochelatase

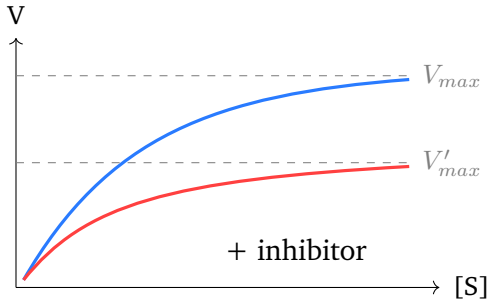
(B) Porphobilinogen deaminase

(C) ALA dehydratase

(D) ALA synthase (delta-aminolevulinic acid synthase)

Enzymes, Vitamins and Coenzymes

Q8. The plot shows normal enzyme kinetics (blue) and kinetics in the presence of an inhibitor (red). This pattern, where the plateau falls but the substrate concentration at half-maximal velocity is unchanged, indicates that the inhibitor acts by:



(A) Increasing the apparent K_m with V_{max} unchanged

(B) Decreasing K_m only

(C) Decreasing V_{max} with K_m unchanged

(D) Increasing both K_m and V_{max}

Q9. Scurvy, with bleeding gums, poor wound healing and fragile capillaries, is due to deficiency of vitamin C, which acts as a cofactor for:

(A) Prolyl and lysyl hydroxylase in collagen synthesis

(B) Transaminase enzymes

(C) Carboxylase enzymes

(D) Dehydrogenase enzymes

Q10. Deficiency of folate, especially in early pregnancy, is classically associated with:

(A) Microcytic hypochromic anaemia

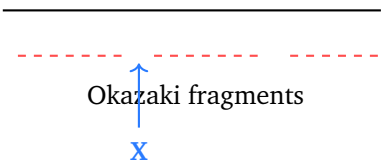
(B) Scurvy

(C) Haemolytic anaemia

(D) Megaloblastic anaemia and neural tube defects

Molecular Biology and Genetics

Q11. In the lagging-strand segment shown, the newly made Okazaki fragments leave nicks that must be sealed. The enzyme (marked **X**) that joins adjacent Okazaki fragments by forming a phosphodiester bond is:



(A) DNA polymerase

(B) Primase

(C) Helicase

(D) DNA ligase

Q12. The genetic code is described as degenerate (redundant) because:

(A) Most amino acids are encoded by more than one codon

(B) A single codon can specify several different amino acids

(C) The codons physically overlap one another

(D) Codons are read without a fixed reading frame

Q13. The initiation (start) codon AUG codes for which amino acid?

(A) Valine

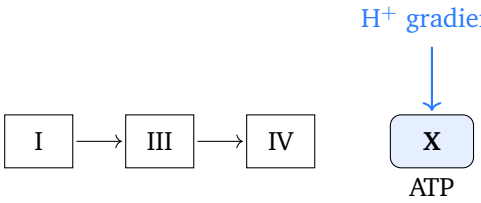
(B) Leucine

(C) Methionine

(D) Tryptophan

Bioenergetics, Nucleotides and Clinical Biochemistry

Q14. In the oxidative phosphorylation scheme shown, the proton (H^+) gradient across the inner mitochondrial membrane drives the enzyme marked **X** to synthesise ATP by chemiosmosis. This enzyme is:



(A) Complex I (NADH dehydrogenase)

(B) Cytochrome c oxidase (complex IV)

(C) ATP synthase (complex V)

(D) Succinate dehydrogenase (complex II)

Q15. In gout caused by overproduction of uric acid, the drug allopurinol lowers uric acid levels by inhibiting the enzyme:

(A) Hypoxanthine-guanine phosphoribosyltransferase (HGPRT)

(B) Xanthine oxidase

(C) Adenosine deaminase

(D) PRPP synthetase

Detailed Solutions

Q1.

Solution

Concept – hexokinase versus glucokinase: Both phosphorylate glucose, but their kinetics and location differ.

Step 1 – kinetics: Glucokinase has a **high** K_m (low affinity), so it works fast only when blood glucose is high.

Step 2 – distribution and regulation: Glucokinase is confined mainly to **liver and pancreatic beta cells** and is *not* inhibited by glucose-6-phosphate, whereas hexokinase is ubiquitous, has a low K_m and is product-inhibited.

Why the other options are wrong:

- A: a low K_m and presence in all tissues describes hexokinase.
- C: it is hexokinase, not glucokinase, that is inhibited by glucose-6-phosphate.
- D: glucokinase has a *higher* K_m than hexokinase, not a lower one.

Final Answer: High K_m, liver and beta cells $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q1

Q2.

Solution

Concept – the pyruvate dehydrogenase complex: It is the bridge between glycolysis and the TCA cycle.

Step 1 – identify X: Step X converts pyruvate to acetyl-CoA and is catalysed by the **pyruvate dehydrogenase complex**.

Step 2 – the coenzyme: Its E1 subunit needs **thiamine pyrophosphate (B_1)**; the full complex also uses lipoate, CoA, FAD and NAD^+.

Why the other options are wrong:

- B, pyridoxal phosphate: for transamination, not this decarboxylation.
- C, biotin: for carboxylases such as pyruvate carboxylase.
- D, ascorbate: a hydroxylation cofactor, unrelated to this step.

Final Answer: Thiamine pyrophosphate (B_1) $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q2

Q3.

Solution

Concept – glycogen storage disease type I: The defect blocks the final release of free glucose.

Step 1 – name the enzyme: Von Gierke disease is due to **glucose-6-phosphatase** deficiency.

Step 2 – link to features: Without it, neither glycogenolysis nor gluconeogenesis can release glucose, giving fasting hypoglycaemia, hepatomegaly and lactic acidosis.

Why the other options are wrong:

- B, muscle phosphorylase: deficiency is McArdle disease (type V), a muscle disorder.
- C, acid maltase: deficiency is Pompe disease (type II), with cardiomegaly.
- D, debranching enzyme: deficiency is Cori disease (type III), milder.

Final Answer: Glucose-6-phosphatase $\Rightarrow$ A

Answer: (A) Go Back to Q3

Q4.

Solution

Concept – the carnitine shuttle: Long-chain fatty acids cannot cross the inner mitochondrial membrane as acyl-CoA.

Step 1 – name the enzyme: Carnitine palmitoyltransferase-1 (CPT-1) on the outer membrane converts acyl-CoA to acylcarnitine and is the **rate-limiting step** of fatty acid entry.

Step 2 – its regulation: CPT-1 is inhibited by malonyl-CoA, so fat cannot be oxidised and synthesised at the same time.

Why the other options are wrong:

- A, acyl-CoA synthetase: activates the fatty acid before the shuttle, not the controlling step.
- C, translocase: exchanges acylcarnitine for carnitine but is not rate-limiting.
- D, CPT-2: regenerates acyl-CoA inside the matrix, after the committed step.

Final Answer: Carnitine palmitoyltransferase-1 (CPT-1) $\Rightarrow$ B

Answer: (B) Go Back to Q4

Q5.

Solution

Concept – ketogenesis: Ketone bodies are made from acetyl-CoA during fasting.

Step 1 – name the site: Ketogenesis occurs in the **mitochondria of the liver**, the only organ with the full HMG-CoA lyase pathway for export.

Step 2 – their fate: Acetoacetate and beta-hydroxybutyrate are then carried in blood to muscle, brain and other tissues for energy.

Why the other options are wrong:

- A, kidney cytoplasm: kidney makes little; the pathway is mitochondrial, not cytosolic.
- C, muscle mitochondria: muscle uses ketone bodies but cannot synthesise or export them.
- D, adipose cytoplasm: adipose supplies fatty acids but does not form ketone bodies.

Final Answer: Liver mitochondria $\Rightarrow$ B

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – phenylketonuria: A hydroxylase defect blocks phenylalanine breakdown.

Step 1 – the enzyme: Classic PKU is deficiency of **phenylalanine hydroxylase**, which converts phenylalanine to tyrosine.

Step 2 – the cofactor: This enzyme needs **tetrahydrobiopterin (BH_4)**; a rare variant PKU is due to defective BH_4 itself.

Why the other options are wrong:

- A, tetrahydrofolate: a one-carbon carrier, not the hydroxylase cofactor.
- B, pyridoxal phosphate: for transamination and decarboxylation.
- D, S-adenosylmethionine: a methyl donor, unrelated here.

Final Answer: Tetrahydrobiopterin (BH_4) $\Rightarrow$ C

Answer: (C) Go Back to Q6

Q7.

Solution

Concept – regulation of heme synthesis: The first committed step sets the pace.

Step 1 – name the enzyme: ALA synthase condenses glycine and succinyl-CoA in the mitochondrion and is the **rate-limiting enzyme** of heme synthesis.

Step 2 – its control: It is feedback-inhibited by heme, which represses the enzyme and blocks its mitochondrial import.

Why the other options are wrong:

- A, ferrochelatase: the last step, inserting iron; inhibited by lead.
- B, porphobilinogen deaminase: a middle step, deficient in acute intermittent porphyria.
- C, ALA dehydratase: an early step, also lead-sensitive, but not rate-limiting.

Final Answer: ALA synthase $\Rightarrow$ D

Answer: (D) Go Back to Q7

Q8.

Solution

Concept – non-competitive inhibition: The inhibitor binds away from the active site, so extra substrate cannot displace it.

Step 1 – read the graph: The plateau (V_{max}) falls while the substrate giving half-maximal velocity (K_m) is unchanged.

Step 2 – interpret: A lowered V_{max} with an unchanged K_m is the signature of **non-competitive inhibition**.

Why the other options are wrong:

- A, raised K_m with V_{max} unchanged: describes competitive inhibition.
- B, lowered K_m only: not a standard inhibition pattern.
- D, both raised: does not match a falling plateau with fixed K_m.

Final Answer: Decreased V_{max}, K_m unchanged $\Rightarrow$ C

Answer: (C) Go Back to Q8

Q9.

Solution

Concept – vitamin C and collagen: Ascorbate is a cofactor for collagen-modifying enzymes.

Step 1 – the enzymes: Vitamin C is required by **prolyl and lysyl hydroxylase**, which hydroxylate proline and lysine in collagen.

Step 2 – consequence of deficiency: Without hydroxylation the collagen triple helix is unstable, causing the bleeding gums, poor healing and fragile vessels of scurvy.

Why the other options are wrong:

- B, transaminases: use pyridoxal phosphate.
- C, carboxylases: use biotin (or vitamin K for clotting factors).
- D, dehydrogenases: use niacin- or riboflavin-derived coenzymes.

Final Answer: Prolyl and lysyl hydroxylase $\Rightarrow$ A

Answer: (A) Go Back to Q9

Q10.

Solution

Concept – folate deficiency: Folate supplies one-carbon units for DNA synthesis.

Step 1 – the blood picture: Impaired DNA synthesis in marrow precursors causes **megaloblastic anaemia**.

Step 2 – in pregnancy: Folate lack in early pregnancy raises the risk of **neural tube defects**, which is why supplementation is advised.

Why the other options are wrong:

- A, microcytic hypochromic anaemia: iron deficiency.
- B, scurvy: vitamin C deficiency.
- C, haemolytic anaemia: seen with vitamin E deficiency, not folate.

Final Answer: Megaloblastic anaemia and neural tube defects $\Rightarrow$ D

Answer: (D) Go Back to Q10

Q11.

Solution

Concept – sealing Okazaki fragments: The lagging strand is left with nicks that need joining.

Step 1 – identify X: The enzyme that seals the nick between adjacent Okazaki fragments is **DNA ligase**.

Step 2 – how it works: It forms the missing phosphodiester bond, making the lagging strand continuous.

Why the other options are wrong:

- A, DNA polymerase: extends and proofreads strands but does not seal the final nick.
- B, primase: lays down the RNA primers.
- C, helicase: unwinds the parental duplex at the fork.

Final Answer: DNA ligase $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q11

Q12.

Solution

Concept – degeneracy of the genetic code: There are 61 sense codons for only 20 amino acids.

Step 1 – define it: Degeneracy means **most amino acids are specified by more than one codon** (for example, leucine has six).

Step 2 – what it is not: Each codon still codes for only one amino acid, so the code stays unambiguous.

Why the other options are wrong:

- B: one codon coding for several amino acids would make the code ambiguous, which it is not.
- C: the code is non-overlapping.
- D: the code is read in a fixed, comma-less triplet frame.

Final Answer: Most amino acids have more than one codon $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q12

Q13.

Solution

Concept – the start codon: Translation begins at a fixed codon.

Step 1 – read the codon: The initiator codon **AUG codes for methionine** (formyl-methionine in bacteria).

Step 2 – significance: It sets the reading frame; internal AUG codons also add methionine within the chain.

Why the other options are wrong:

- A, valine: coded by GUN codons.
- B, leucine: coded by CUN and UUA/UUG.
- D, tryptophan: coded only by UGG.

Final Answer: Methionine $\Rightarrow$ C

Answer: (C) Go Back to Q13

Q14.

Solution

Concept – chemiosmosis: The proton gradient is the energy source for ATP synthesis.

Step 1 – identify X: The enzyme using the H^+ gradient to phosphorylate ADP is **ATP synthase (complex V)**.

Step 2 – mechanism: Protons flow back through its F_o channel, turning the rotor and driving the F_1 head to make ATP.

Why the other options are wrong:

- A, complex I: pumps protons out but does not make ATP.
- B, complex IV: reduces oxygen to water while pumping protons.
- D, complex II: feeds electrons in without pumping protons.

Final Answer: ATP synthase (complex V) $\Rightarrow$ C

Answer: (C) Go Back to Q14

Q15.

Solution

Concept – gout and its treatment: Uric acid is the end product of purine catabolism.

Step 1 – the drug target: Allopurinol lowers uric acid by inhibiting **xanthine oxidase**, the enzyme that makes uric acid from hypoxanthine and xanthine.

Step 2 – the result: Less uric acid forms, so serum urate and crystal deposition fall.

Why the other options are wrong:

- A, HGPRT: a salvage enzyme; its deficiency raises uric acid rather than being a drug target.
- C, adenosine deaminase: its deficiency causes immunodeficiency, not gout.
- D, PRPP synthetase: overactivity can cause gout, but allopurinol does not act here.

Final Answer: Xanthine oxidase $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q15

Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	B	2	A	3	A	4	B	5	B
6	C	7	D	8	C	9	A	10	D
11	D	12	A	13	C	14	C	15	B