INI CET Biochemistry

Sample Paper – 6

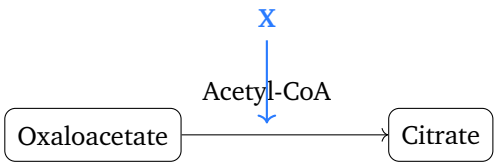
Duration: 14 Minutes **Maximum Marks: 15**

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. In the TCA (citric acid) cycle outlined below, the first step marked **X**, in which acetyl-CoA condenses with oxaloacetate to form citrate, is catalysed by:



(A) Citrate synthase

(B) Aconitase

(C) Isocitrate dehydrogenase

(D) Malate dehydrogenase

Q2. Hereditary fructose intolerance, presenting with hypoglycaemia and vomiting after fructose ingestion, is caused by deficiency of:

(A) Fructokinase

(B) Aldose reductase

(C) Aldolase B

(D) Fructose-1,6-bisphosphatase

Q3. The enzyme that forms the alpha-1,4 glycosidic bonds of glycogen by transferring glucose from UDP-glucose to a growing chain is:

(A) Glycogen phosphorylase

(B) Glycogen synthase

(C) Branching enzyme

(D) Phosphoglucomutase

Lipid Metabolism

Q4. De novo fatty acid synthesis in the cytoplasm is carried out by a single multienzyme complex that uses NADPH as the reducing power. This complex is:

(A) Fatty acid synthase

(B) Acetyl-CoA carboxylase

(C) Carnitine acyltransferase

(D) Thiolase

Q5. Gaucher disease, the commonest lysosomal storage disorder with hepatosplenomegaly and Gaucher cells, results from deficiency of:

(A) Hexosaminidase A

(B) Glucocerebrosidase

(C) Sphingomyelinase

(D) Ceramidase

Amino Acid and Protein Metabolism

Q6. Albinism, characterised by a complete absence of melanin in skin, hair and eyes, is caused by deficiency of:

(A) Homogentisate oxidase

(B) Phenylalanine hydroxylase

(C) Cystathionine synthase

(D) Tyrosinase

Q7. Ammonia produced in peripheral tissues is transported to the liver in a non-toxic form mainly as:

(A) Urea

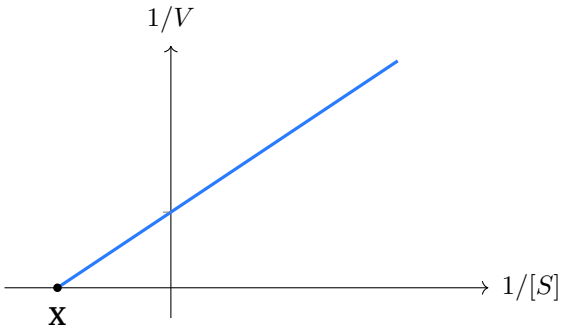
(B) Uric acid

(C) Creatinine

(D) Glutamine

Enzymes, Vitamins and Coenzymes

Q8. In the Lineweaver-Burk (double-reciprocal) plot shown, the point marked **X**, where the line cuts the horizontal axis, corresponds to:



(A) $1/V_{max}$

(B) $-1/K_m$

(C) K_m/V_{max}

(D) V_{max}

Q9. Which vitamin is the precursor of the flavin coenzymes FAD and FMN?

(A) Niacin (B_3)

(B) Thiamine (B_1)

(C) Riboflavin (B_2)

(D) Folic acid (B_9)

Q10. Which vitamin is a structural component of coenzyme A, essential for acyl-group transfer?

(A) Pantothenic acid (B_5)

(B) Biotin (B_7)

(C) Pyridoxine (B_6)

(D) Ascorbic acid (C)

Molecular Biology and Genetics

Q11. The specialised reverse-transcriptase enzyme that adds repetitive sequences to the ends of eukaryotic chromosomes, preventing their shortening, is:

(A) DNA ligase

(B) Topoisomerase

(C) Primase

(D) Telomerase

Q12. The processing step in which introns are removed and exons are joined together during the maturation of pre-mRNA is called:

(A) Splicing

(B) Capping

(C) Polyadenylation

(D) Editing

Q13. A point mutation in which one purine is replaced by another purine (for example A replaced by G) is classified as a:

(A) Transversion

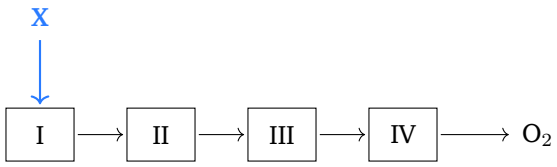
(B) Transition

(C) Frameshift

(D) Silent deletion

Bioenergetics, Nucleotides and Clinical Biochemistry

Q14. In the electron transport chain shown, the poison rotenone blocks electron flow at the site marked **X**. This site is:



(A) Complex II

(B) Complex III

(C) Complex I

(D) Complex IV

Q15. Which serum biomarker is the most specific and sensitive for the diagnosis of acute myocardial infarction?

(A) Amylase

(B) Alkaline phosphatase

(C) Cardiac troponin

(D) Gamma-glutamyl transferase

Detailed Solutions

Q1.

Solution

Concept – first step of the TCA cycle: The cycle begins by condensing a two-carbon and a four-carbon molecule.

Step 1 – identify the reaction: Acetyl-CoA (2C) joins oxaloacetate (4C) to give citrate (6C).

Step 2 – name the enzyme: This irreversible condensation is catalysed by **citrate synthase**, the entry point of the cycle.

Why the other options are wrong:

- B, aconitase: isomerises citrate to isocitrate, the second step.
- C, isocitrate dehydrogenase: a later, rate-limiting oxidative decarboxylation.
- D, malate dehydrogenase: regenerates oxaloacetate at the end of the cycle.

Final Answer: Citrate synthase $\Rightarrow$ A

Answer: (A) Go Back to Q1

Q2.

Solution

Concept – fructose metabolism disorders: Different enzyme blocks give different clinical pictures.

Step 1 – name the defect: Hereditary fructose intolerance is due to deficiency of **aldolase B**.

Step 2 – link to features: Fructose-1-phosphate accumulates, trapping phosphate and blocking gluconeogenesis and glycogenolysis, so severe hypoglycaemia follows fructose intake.

Why the other options are wrong:

- A, fructokinase: its deficiency causes benign essential fructosuria, not illness.
- B, aldose reductase: part of the polyol pathway, unrelated.
- D, fructose-1,6-bisphosphatase: a gluconeogenic enzyme; its deficiency gives fasting hypoglycaemia and acidosis, not fructose-triggered disease.

Final Answer: Aldolase B $\Rightarrow$ C

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – glycogen synthesis: Building glycogen needs an activated glucose donor.

Step 1 – name the enzyme: Glycogen synthase transfers glucose from UDP-glucose onto the non-reducing end, forming alpha-1,4 bonds.

Step 2 – regulation: It is the rate-limiting enzyme of glycogenesis and is activated by insulin.

Why the other options are wrong:

- A, glycogen phosphorylase: degrades glycogen, the opposite reaction.
- C, branching enzyme: makes the alpha-1,6 branch points, not the main chain.
- D, phosphoglucomutase: interconverts G1P and G6P, an upstream step.

Final Answer: Glycogen synthase $\Rightarrow$ B

Answer: (B) Go Back to Q3

Q4.

Solution

Concept – de novo fatty acid synthesis: The whole chain is built by one large cytoplasmic complex.

Step 1 – name the complex: Fatty acid synthase is a multienzyme complex that assembles palmitate.

Step 2 – cofactor: Each reduction step uses NADPH, supplied mainly by the pentose phosphate pathway.

Why the other options are wrong:

- B, acetyl-CoA carboxylase: makes malonyl-CoA, the rate-limiting step before the synthase acts.
- C, carnitine acyltransferase: shuttles fatty acids into mitochondria for oxidation.
- D, thiolase: a beta-oxidation enzyme, not synthesis.

Final Answer: Fatty acid synthase $\Rightarrow$ A

Answer: (A) Go Back to Q4

Q5.

Solution

Concept – sphingolipidoses: Each lysosomal storage disease maps to one missing hydrolase.

Step 1 – name the enzyme: Gaucher disease results from deficiency of **glucocerebrosidase**.

Step 2 – link to features: Glucocerebroside accumulates in macrophages, forming Gaucher cells and causing hepatosplenomegaly and bone disease.

Why the other options are wrong:

- A, hexosaminidase A: deficient in Tay-Sachs disease.
- C, sphingomyelinase: deficient in Niemann-Pick disease.
- D, ceramidase: deficient in Farber disease.

Final Answer: Glucocerebrosidase $\Rightarrow$ B

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – melanin synthesis: Melanin is made from tyrosine by a single key enzyme.

Step 1 – name the enzyme: Classic oculocutaneous albinism is due to deficiency of **tyrosinase**.

Step 2 – reasoning: Tyrosinase converts tyrosine to DOPA and then to dopaquinone; without it no melanin forms.

Why the other options are wrong:

- A, homogentisate oxidase: its deficiency causes alkaptonuria.
- B, phenylalanine hydroxylase: its deficiency causes phenylketonuria.
- C, cystathionine synthase: its deficiency causes homocystinuria.

Final Answer: Tyrosinase $\Rightarrow$ D

Answer: (D) Go Back to Q6

Q7.

Solution

Concept – ammonia transport: Free ammonia is toxic, so it travels in a safe carrier.

Step 1 – name the carrier: Glutamine is the main non-toxic transport form of ammonia in the blood.

Step 2 – reasoning: Glutamine synthetase fixes ammonia onto glutamate; the kidney and liver later release it via glutaminase.

Why the other options are wrong:

- A, urea: the final excretory product made in the liver, not the blood carrier from tissues.
- B, uric acid: the end product of purine, not amino acid, nitrogen.
- C, creatinine: a waste product of creatine, unrelated to ammonia transport.

Final Answer: Glutamine $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q7

Q8.

Solution

Concept – the Lineweaver-Burk plot: The double-reciprocal plot linearises the Michaelis-Menten equation.

Step 1 – read the intercepts: Plotting $1/V$ against $1/[S]$ gives a straight line; the x-intercept (point X) equals $-1/K_m$.

Step 2 – confirm the others: The y-intercept equals $1/V_{max}$ and the slope equals K_m/V_{max}.

Why the other options are wrong:

- A, $1/V_{max}$: this is the y-intercept, not the x-intercept.
- C, K_m/V_{max}: this is the slope of the line.
- D, V_{max}: not an intercept on this reciprocal plot.

Final Answer: $-1/K_m \Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q8

Q9.

Solution

Concept – flavin coenzymes: FAD and FMN are built from one B-vitamin.

Step 1 – name the vitamin: Riboflavin (vitamin B_2) is the precursor of FAD and FMN.

Step 2 – function: These flavin coenzymes act as electron carriers in redox enzymes such as succinate dehydrogenase.

Why the other options are wrong:

- A, niacin (B_3): forms NAD^+ and $NADP^+$, not the flavins.
- B, thiamine (B_1): forms thiamine pyrophosphate.
- D, folic acid (B_9): forms tetrahydrofolate for one-carbon transfers.

Final Answer: Riboflavin (B_2) $\Rightarrow$ C

Answer: (C) Go Back to Q9

Q10.

Solution

Concept – coenzyme A: CoA carries acyl groups and contains one vitamin.

Step 1 – name the vitamin: Pantothenic acid (vitamin B_5) is a structural part of coenzyme A.

Step 2 – function: The thiol (-SH) group of CoA forms high-energy thioester bonds, as in acetyl-CoA.

Why the other options are wrong:

- B, biotin (B_7): a coenzyme for carboxylation, not part of CoA.
- C, pyridoxine (B_6): forms pyridoxal phosphate for transamination.
- D, ascorbic acid (C): a cofactor for hydroxylation reactions.

Final Answer: Pantothenic acid (B_5) $\Rightarrow$ A

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – chromosome ends: Linear chromosomes need a way to replicate their tips.

Step 1 – name the enzyme: **Telomerase** adds repetitive TTAGGG sequences to chromosome ends.

Step 2 – mechanism: It carries its own RNA template and works as a reverse transcriptase, offsetting the end-replication problem.

Why the other options are wrong:

- A, DNA ligase: seals nicks between fragments.
- B, topoisomerase: relieves supercoiling ahead of the fork.
- C, primase: lays down RNA primers, not telomere repeats.

Final Answer: Telomerase $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q11

Q12.

Solution

Concept – pre-mRNA processing: Eukaryotic transcripts must have introns removed before translation.

Step 1 – name the step: Removing introns and joining exons is called **splicing**, carried out by the spliceosome.

Step 2 – reasoning: Only the joined exons form the mature, translatable mRNA.

Why the other options are wrong:

- B, capping: adds the 7-methylguanosine cap at the 5′ end.
- C, polyadenylation: adds the poly-A tail at the 3′ end.
- D, editing: chemically alters specific bases, a different process.

Final Answer: Splicing $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q12

Q13.

Solution

Concept – classifying point mutations: Base substitutions are grouped by the type of base swapped.

Step 1 – apply the definition: Replacing a purine with another purine (A↔G), or a pyrimidine with another pyrimidine, is a **transition**.

Step 2 – contrast: A purine-to-pyrimidine swap (or the reverse) is a transversion.

Why the other options are wrong:

- A, transversion: needs a purine-pyrimidine exchange, not purine-to-purine.
- C, frameshift: caused by insertions or deletions, not substitution.
- D, silent deletion: involves loss of a base, not a substitution.

Final Answer: Transition ⇒ B

Answer: (B) Go Back to Q13

Q14.

Solution

Concept – inhibitors of the ETC: Each poison blocks a specific complex.

Step 1 – identify X: Rotenone blocks electron transfer at **complex I** (NADH dehydrogenase), the first complex shown.

Step 2 – consequence: Electrons from NADH cannot pass to coenzyme Q, so proton pumping and ATP synthesis fall.

Why the other options are wrong:

- A, complex II: succinate dehydrogenase, not the rotenone target.
- B, complex III: blocked by antimycin A, not rotenone.
- D, complex IV: blocked by cyanide and carbon monoxide.

Final Answer: Complex I ⇒ C

Answer: (C) Go Back to Q14

Q15.

Solution

Concept – cardiac biomarkers: The ideal marker of infarction is heart-specific.

Step 1 – name the marker: Cardiac troponin (troponin I and T) is the most specific and sensitive marker of myocardial infarction.

Step 2 – reasoning: It rises within a few hours, peaks at 1–2 days and stays elevated for up to a week or more.

Why the other options are wrong:

- A, amylase: a marker of pancreatitis.
- B, alkaline phosphatase: raised in bone and biliary disease.
- D, gamma-glutamyl transferase: a marker of hepatobiliary disease and alcohol use.

Final Answer: Cardiac troponin $\Rightarrow$ C

Answer: (C) Go Back to Q15

Answer Key

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