INI CET Biochemistry

Sample Paper – 3

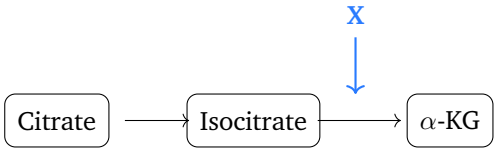
Duration: 14 Minutes **Maximum Marks: 15**

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 segment of the citric acid cycle shown, the step marked **X** (isocitrate to α-ketoglutarate) is the rate-limiting reaction of the cycle. It is catalysed by:



(A) Isocitrate dehydrogenase

(B) Citrate synthase

(C) Succinate dehydrogenase

(D) Malate dehydrogenase

Q2. During gluconeogenesis, the cytosolic enzyme that converts oxaloacetate to phosphoenolpyruvate is:

(A) Pyruvate kinase

(B) Glucokinase

(C) Phosphoenolpyruvate carboxykinase (PEPCK)

(D) Hexokinase

Q3. A young man develops acute haemolysis a few days after taking an antimalarial drug. The underlying defect is in an enzyme of the HMP (pentose phosphate) shunt, causing a shortage of NADPH in the red cell. The deficient enzyme is:

(A) Pyruvate kinase

(B) Aldolase B

(C) Transketolase

(D) Glucose-6-phosphate dehydrogenase

Lipid Metabolism

Q4. The rate-limiting, biotin-dependent enzyme of fatty acid synthesis, which carboxylates acetyl-CoA to malonyl-CoA, is:

(A) Fatty acid synthase

(B) HMG-CoA reductase

(C) ATP-citrate lyase

(D) Acetyl-CoA carboxylase

Q5. Familial hypercholesterolaemia, with very high plasma LDL, tendon xanthomas and premature coronary disease, is most commonly caused by:

(A) Lipoprotein lipase deficiency

(B) Overproduction of apolipoprotein B

(C) A defective LDL receptor

(D) LCAT deficiency

Amino Acid and Protein Metabolism

Q6. A neonate presents with feeding difficulty, lethargy and urine that smells of maple syrup. The condition results from deficiency of:

(A) Branched-chain alpha-ketoacid dehydrogenase

(B) Phenylalanine hydroxylase

(C) Homogentisate oxidase

(D) Cystathionine synthase

Q7. The coenzyme that acts as the principal carrier of one-carbon units in reactions such as purine and thymidylate synthesis is:

(A) Tetrahydrofolate

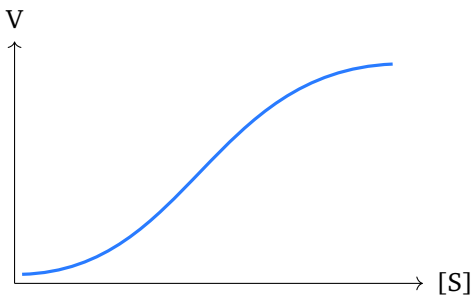
(B) Biotin

(C) Pyridoxal phosphate

(D) Thiamine pyrophosphate

Enzymes, Vitamins and Coenzymes

Q8. The velocity versus substrate concentration plot shown is typical of a regulatory (allosteric) enzyme with cooperative binding. The shape of this curve is best described as:



(A) A rectangular hyperbola

(B) Sigmoidal (S-shaped)

(C) A straight line through the origin

(D) A downward parabola

Q9. The gamma-carboxylation of glutamate residues in clotting factors II, VII, IX and X, needed for their activity, requires which vitamin?

(A) Vitamin C

(B) Vitamin D

(C) Vitamin E

(D) Vitamin K

Q10. Deficiency of which vitamin classically causes night blindness, its aldehyde form (retinal) being the light-absorbing component of rhodopsin?

(A) Vitamin B_2

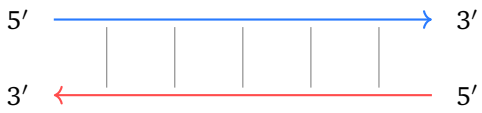
(B) Vitamin A

(C) Vitamin B_{12}

(D) Vitamin C

Molecular Biology and Genetics

Q11. The double helix shown follows Chargaff's rule and the Watson-Crick model. The two strands of the DNA molecule are best described as:



(A) Parallel and identical in sequence

(B) Antiparallel and complementary

(C) Antiparallel and identical in sequence

(D) Parallel and complementary

Q12. The polymerase chain reaction (PCR) can cycle through repeated high-temperature denaturation steps because it uses a heat-stable enzyme, namely:

(A) DNA ligase

(B) Reverse transcriptase

(C) Taq DNA polymerase

(D) Primase

Q13. A point mutation that changes a sense codon into a stop codon, producing a prematurely truncated protein, is called a:

(A) Silent mutation

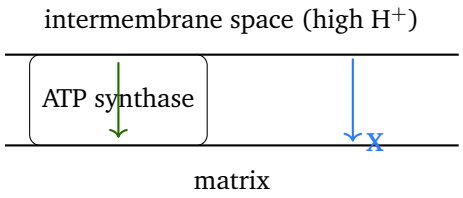
(B) Missense mutation

(C) Nonsense mutation

(D) Frameshift mutation

Bioenergetics, Nucleotides and Clinical Biochemistry

Q14. In the mitochondrion sketched, a lipophilic agent such as 2,4-dinitrophenol provides the proton leak marked **X** across the inner membrane. The metabolic effect of such an uncoupler is to:



(A) Directly inhibit cytochrome c oxidase (complex IV)

(B) Dissipate the proton gradient, releasing heat without making ATP

(C) Block ATP synthase and stop electron transport at once

(D) Donate electrons to complex I

Q15. A child has megaloblastic anaemia that does not respond to vitamin B_{12} or folate, poor growth, and a large amount of orotic acid in the urine. This hereditary orotic aciduria is due to deficiency of:

(A) UMP synthase

(B) Hypoxanthine-guanine phosphoribosyltransferase (HGPRT)

(C) Adenosine deaminase

(D) Xanthine oxidase

Detailed Solutions

Q1.

Solution

Concept – regulation of the TCA cycle: The rate-limiting step of the citric acid cycle is its main control point.

Step 1 – identify the step: The oxidative decarboxylation of isocitrate to α-ketoglutarate is catalysed by **isocitrate dehydrogenase**.

Step 2 – why it is regulatory: It is the rate-limiting enzyme of the cycle, activated by ADP and Ca^{2+} and inhibited by ATP and NADH.

Why the other options are wrong:

- B, citrate synthase: catalyses the first condensation step, upstream of X.
- C, succinate dehydrogenase: a later step and part of complex II.
- D, malate dehydrogenase: the final step regenerating oxaloacetate.

Final Answer: Isocitrate dehydrogenase $\Rightarrow$ A

Answer: (A) Go Back to Q1

Q2.

Solution

Concept – gluconeogenic bypass reactions: Gluconeogenesis bypasses the irreversible glycolytic steps with its own enzymes.

Step 1 – name the enzyme: Phosphoenolpyruvate carboxykinase (PEPCK) converts oxaloacetate to phosphoenolpyruvate.

Step 2 – reasoning: Pyruvate is first carboxylated to oxaloacetate by pyruvate carboxylase, then PEPCK forms PEP, bypassing pyruvate kinase.

Why the other options are wrong:

- A, pyruvate kinase: a glycolytic enzyme working in the opposite direction.
- B, glucokinase: phosphorylates glucose in glycolysis.
- D, hexokinase: also a glycolytic phosphorylating enzyme.

Final Answer: Phosphoenolpyruvate carboxykinase $\Rightarrow$ C

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – HMP shunt and NADPH: The pentose phosphate pathway is the red cell's source of NADPH.

Step 1 – name the enzyme: Glucose-6-phosphate dehydrogenase (G6PD) is the rate-limiting enzyme of the HMP shunt.

Step 2 – link to haemolysis: Its deficiency lowers NADPH and reduced glutathione, so oxidant drugs trigger red cell haemolysis.

Why the other options are wrong:

- A, pyruvate kinase: its deficiency causes chronic haemolysis but not drug-triggered oxidative haemolysis via NADPH.
- B, aldolase B: deficiency causes hereditary fructose intolerance.
- C, transketolase: a downstream, non-limiting shunt enzyme.

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

Answer: (D) Go Back to Q3

Q4.

Solution

Concept – control of fatty acid synthesis: The committed step is the regulatory point.

Step 1 – name the enzyme: Acetyl-CoA carboxylase carboxylates acetyl-CoA to malonyl-CoA and is rate-limiting.

Step 2 – cofactor and control: It is biotin-dependent, activated by citrate and insulin, and inhibited by long-chain acyl-CoA and glucagon.

Why the other options are wrong:

- A, fatty acid synthase: elongates the chain but is not the rate-limiting step.
- B, HMG-CoA reductase: rate-limiting for cholesterol, not fatty acid, synthesis.
- C, ATP-citrate lyase: supplies cytosolic acetyl-CoA, an earlier step.

Final Answer: Acetyl-CoA carboxylase $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q4

Q5.

Solution

Concept – LDL clearance: Plasma LDL is removed by receptor-mediated uptake.

Step 1 – name the defect: Familial hypercholesterolaemia is caused most often by a **defective LDL receptor**.

Step 2 – consequence: Reduced hepatic LDL uptake raises plasma LDL cholesterol, giving xanthomas and early atherosclerosis.

Why the other options are wrong:

- A, lipoprotein lipase deficiency: causes chylomicronaemia with high triglycerides, not high LDL.
- B, apo B overproduction: not the classic single-gene cause.
- D, LCAT deficiency: impairs cholesterol esterification, a different disorder.

Final Answer: Defective LDL receptor $\Rightarrow$ C

Answer: (C) Go Back to Q5

Q6.

Solution

Concept – branched-chain amino acid catabolism: A single dehydrogenase handles all three branched-chain keto acids.

Step 1 – name the enzyme: Maple syrup urine disease is due to deficiency of **branched-chain alpha-ketoacid dehydrogenase**.

Step 2 – link to features: Leucine, isoleucine and valine and their keto acids accumulate, giving the sweet-smelling urine and neurotoxicity.

Why the other options are wrong:

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

Final Answer: Branched-chain alpha-ketoacid dehydrogenase $\Rightarrow$ A

Answer: (A) Go Back to Q6

Q7.

Solution

Concept – one-carbon metabolism: One-carbon groups are moved on a folate coenzyme.

Step 1 – name the carrier: Tetrahydrofolate carries one-carbon units at several oxidation levels.

Step 2 – reasoning: It donates carbons for purine ring synthesis and for thymidylate (dTMP) formation.

Why the other options are wrong:

- B, biotin: carries carboxyl (CO_2) groups, not one-carbon folate units.
- C, pyridoxal phosphate: used in transamination and decarboxylation.
- D, thiamine pyrophosphate: used in oxidative decarboxylation and transketolase reactions.

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

Answer: (A) Go Back to Q7

Q8.

Solution

Concept – allosteric enzyme kinetics: Cooperative substrate binding changes the shape of the velocity curve.

Step 1 – read the graph: An allosteric enzyme gives a **sigmoidal (S-shaped)** velocity versus substrate curve.

Step 2 – reasoning: Binding of substrate to one subunit raises the affinity of the others, so activity rises steeply over a narrow substrate range.

Why the other options are wrong:

- A, rectangular hyperbola: describes a simple Michaelis-Menten enzyme.
- C, straight line: not seen for enzyme saturation kinetics.
- D, downward parabola: does not describe enzyme velocity saturation.

Final Answer: Sigmoidal (S-shaped) $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q8

Q9.

Solution

Concept – vitamin K function: Certain clotting factors need a post-translational modification to work.

Step 1 – name the vitamin: Vitamin K is the cofactor for gamma-glutamyl carboxylase.

Step 2 – reasoning: Gamma-carboxylation of glutamate lets factors II, VII, IX and X bind Ca^{2+} and phospholipid; warfarin blocks this cycle.

Why the other options are wrong:

- A, vitamin C: needed for collagen proline and lysine hydroxylation.
- B, vitamin D: regulates calcium and phosphate handling.
- C, vitamin E: an antioxidant, not involved in carboxylation.

Final Answer: Vitamin K $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q9

Q10.

Solution

Concept – vitamin A and vision: The visual pigment depends on a vitamin A derivative.

Step 1 – name the vitamin: Vitamin A deficiency causes night blindness.

Step 2 – mechanism: Its aldehyde 11-cis-retinal combines with opsin to form rhodopsin, the rod pigment for dim-light vision.

Why the other options are wrong:

- A, vitamin B_2: deficiency causes cheilosis and glossitis.
- C, vitamin B_{12}: deficiency causes megaloblastic anaemia and neuropathy.
- D, vitamin C: deficiency causes scurvy.

Final Answer: Vitamin A $\Rightarrow$ $\boxed{B}$

Answer: (B) Go Back to Q10

Q11.

Solution

Concept – structure of the DNA double helix: The two strands relate to each other in direction and sequence.

Step 1 – direction: The strands run **antiparallel**: one is 5′ to 3′, the other 3′ to 5′.

Step 2 – base pairing: They are **complementary**, with A pairing T and G pairing C, so the strands are antiparallel and complementary.

Why the other options are wrong:

- A and D, parallel: the strands are not parallel.
- C, identical in sequence: the strands are complementary, not identical.

Final Answer: Antiparallel and complementary $\Rightarrow$ B

Answer: (B) Go Back to Q11

Q12.

Solution

Concept – the polymerase chain reaction: PCR repeatedly heats the sample to melt the DNA strands.

Step 1 – name the enzyme: Taq DNA polymerase, from *Thermus aquaticus*, is heat-stable and survives the denaturation steps.

Step 2 – reasoning: Because it withstands about 95°C, fresh enzyme need not be added each cycle.

Why the other options are wrong:

- A, DNA ligase: seals nicks; not the amplifying enzyme.
- B, reverse transcriptase: makes DNA from RNA (used in RT-PCR) and is not heat-stable.
- D, primase: makes RNA primers in the cell, not used to extend PCR products.

Final Answer: Taq DNA polymerase $\Rightarrow$ C

Answer: (C) Go Back to Q12

Q13.

Solution

Concept – types of point mutation: A single base change can affect the codon in different ways.

Step 1 – define it: A **nonsense mutation** turns a coding codon into a stop codon.

Step 2 – consequence: Translation ends early, giving a shortened and usually non-functional protein.

Why the other options are wrong:

- A, silent mutation: changes the codon but not the amino acid.
- B, missense mutation: substitutes a different amino acid.
- D, frameshift mutation: an insertion or deletion that shifts the reading frame.

Final Answer: Nonsense mutation $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q13

Q14.

Solution

Concept – uncouplers of oxidative phosphorylation: Uncouplers separate electron transport from ATP synthesis.

Step 1 – action of 2,4-dinitrophenol: It carries protons back across the inner membrane, so it **dissipates the proton gradient** without passing them through ATP synthase.

Step 2 – consequence: The energy of the gradient is released as heat, electron transport speeds up, but ATP output falls.

Why the other options are wrong:

- A, inhibit complex IV: that describes cyanide, an electron transport inhibitor.
- C, block ATP synthase: that describes oligomycin, not an uncoupler.
- D, donate electrons to complex I: uncouplers move protons, not electrons.

Final Answer: Dissipate the proton gradient, releasing heat without ATP $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q14

Q15.

Solution

Concept – pyrimidine synthesis defect: A block in pyrimidine synthesis backs up orotic acid.

Step 1 – name the enzyme: Hereditary orotic aciduria is due to deficiency of **UMP synthase**.

Step 2 – link to features: Orotic acid cannot be converted to UMP, so pyrimidine nucleotides fall, giving megaloblastic anaemia unresponsive to B_{12} or folate, and orotic acid spills into the urine.

Why the other options are wrong:

- B, HGPRT: its deficiency causes Lesch-Nyhan syndrome, a purine salvage defect.
- C, adenosine deaminase: its deficiency causes severe combined immunodeficiency.
- D, xanthine oxidase: its inhibition (allopurinol) treats gout.

Final Answer: UMP synthase $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q15

Answer Key

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