INI CET Biochemistry

Sample Paper – 4

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. Deficiency of which glycolytic enzyme is the commonest cause of hereditary non-spherocytic haemolytic anaemia, because the red cell cannot make enough ATP to survive?

(A) Hexokinase

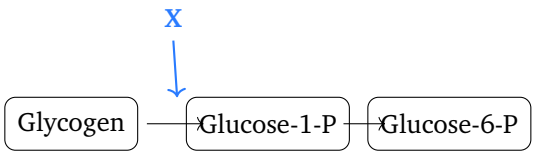
(B) Pyruvate kinase

(C) Phosphofructokinase-1

(D) Enolase

Q2. McArdle disease (glycogen storage disease type V), which presents with exercise intolerance, muscle cramps and a flat lactate response to exer-

cise, is due to deficiency of the muscle enzyme marked **X** in the glycogen-breakdown pathway shown:



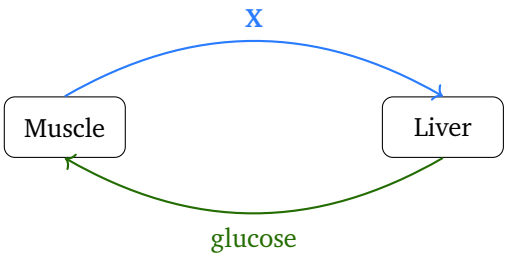
(A) Glucose-6-phosphatase

(B) Lysosomal acid alpha-glucosidase

(C) Muscle glycogen phosphorylase

(D) Debranching enzyme

Q3. In the Cori cycle depicted, exercising muscle exports a three-carbon molecule (marked **X**) to the liver, where it is reconverted to glucose. This exported molecule is:



(A) Pyruvate

(B) Lactate

(C) Alanine

(D) Glycerol

Lipid Metabolism

Q4. Lipoprotein lipase, the enzyme that hydrolyses the triglycerides of circulating chylomicrons and VLDL, is activated by which apolipoprotein?

(A) Apolipoprotein C-II

(B) Apolipoprotein B-100

(C) Apolipoprotein A-I

(D) Apolipoprotein B-48

Q5. During reverse cholesterol transport, HDL takes up free cholesterol from peripheral tissues. Which plasma enzyme, activated by apo A-I on the HDL surface, esterifies this cholesterol so it can be carried in the core of the particle?

(A) Cholesteryl ester transfer protein (CETP)

(B) Lecithin-cholesterol acyltransferase (LCAT)

(C) Acyl-CoA cholesterol acyltransferase (ACAT)

(D) Hepatic lipase

Amino Acid and Protein Metabolism

Q6. A patient's urine turns black on standing and there is dark blue-black pigmentation of the ear cartilage and sclera (ochronosis). This condition, alkaptonuria, is caused by deficiency of:

(A) Phenylalanine hydroxylase

(B) Tyrosinase

(C) Fumarylacetoacetate hydrolase

(D) Homogentisate oxidase

Q7. Which single amino acid is the common precursor of the catecholamines, the pigment melanin and the thyroid hormones?

(A) Tryptophan

(B) Glutamate

(C) Histidine

(D) Tyrosine

Enzymes, Vitamins and Coenzymes

Q8. Isoenzymes are different molecular forms of the same enzyme whose tissue distribution makes them useful serum markers. Which isoenzyme is used as an early, relatively cardiac-specific marker of acute myocardial infarction?

(A) LDH-5

(B) Creatine kinase MB (CK-MB)

(C) Creatine kinase BB (CK-BB)

(D) Salivary amylase

Q9. Deficiency of niacin (vitamin B_3) classically produces pellagra. Pellagra is remembered by which triad?

(A) Dermatitis, diarrhoea and dementia

(B) Night blindness, dry skin and poor wound healing

(C) Bleeding gums, corkscrew hairs and perifollicular haemorrhage

(D) Bowing of the legs, soft bones and delayed dentition

Q10. Deficiency of vitamin B_{12} (cobalamin) characteristically produces:

(A) Megaloblastic anaemia with subacute combined degeneration of the spinal cord

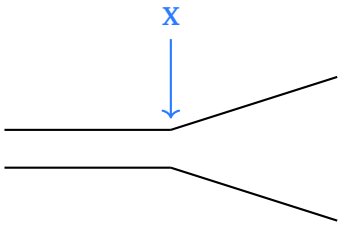
(B) Microcytic hypochromic anaemia with low ferritin

(C) Megaloblastic anaemia with no neurological involvement

(D) Haemolytic anaemia with bite cells

Molecular Biology and Genetics

Q11. In the replication fork shown, the enzyme marked **X** separates the two parental strands by breaking the hydrogen bonds of the double helix. This enzyme is:



(A) DNA ligase

(B) Topoisomerase

(C) Primase

(D) Helicase

Q12. During processing of eukaryotic pre-messenger RNA, which pair of modifications is added to the two ends of the transcript to protect it and aid its export and translation?

(A) A 5′ poly-A tail and a 3′ methyl cap

(B) Removal of the 5′ cap and addition of introns

(C) A 5′ 7-methylguanosine cap and a 3′ poly-A tail

(D) A poly-A tail added to both the 5′ and 3′ ends

Q13. In the lac operon of *E. coli*, transcription of the structural genes is switched on when:

(A) The lac repressor binds tightly to the operator

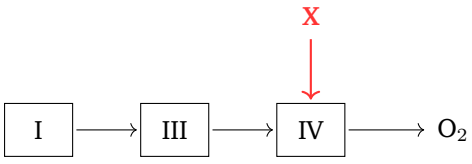
(B) Glucose is abundant and cyclic AMP is low

(C) Allolactose binds and inactivates the repressor, freeing the operator for RNA polymerase

(D) The CAP-cAMP complex leaves the promoter region

Bioenergetics, Nucleotides and Clinical Biochemistry

Q14. In the electron transport chain shown, cyanide poisoning halts respiration by blocking the complex marked **X**, which normally hands electrons to oxygen. This complex is:



(A) Complex IV (cytochrome c oxidase)

(B) Complex I (NADH dehydrogenase)

(C) Complex III (cytochrome bc_1)

(D) Complex II (succinate dehydrogenase)

Q15. Deficiency of adenosine deaminase (ADA) causes accumulation of deoxyadenosine and dATP, which is especially toxic to developing lymphocytes. This defect classically produces:

(A) Gout with recurrent uric acid stones

(B) Lesch-Nyhan syndrome

(C) Severe combined immunodeficiency (SCID)

(D) Hereditary orotic aciduria

Detailed Solutions

Q1.

Solution

Concept – glycolysis and the red cell: The mature red cell has no mitochondria, so glycolysis is its only source of ATP.

Step 1 – name the enzyme: Pyruvate kinase catalyses the final ATP-generating step (phosphoenolpyruvate to pyruvate).

Step 2 – link to disease: Its deficiency starves the red cell of ATP, so the membrane pumps fail and the cell is haemolysed; this is the commonest enzymopathy causing hereditary non-spherocytic haemolytic anaemia.

Why the other options are wrong:

- A, hexokinase: deficiency is very rare and not the classic cause.
- C, phosphofructokinase-1: deficiency (Tarui disease) mainly affects muscle.
- D, enolase: an extremely rare defect, not the standard answer.

Final Answer: Pyruvate kinase $\Rightarrow$ B

Answer: (B) Go Back to Q1

Q2.

Solution

Concept – glycogen storage diseases: Each subtype maps to a specific enzyme block in glycogen handling.

Step 1 – identify X: The step marked X is the phosphorolysis of glycogen to glucose-1-phosphate, catalysed by **muscle glycogen phosphorylase (myophosphorylase)**.

Step 2 – link to McArdle disease: Its deficiency (GSD type V) leaves muscle unable to mobilise glycogen during exercise, giving cramps, exercise intolerance and a flat (absent) rise in blood lactate on exertion.

Why the other options are wrong:

- A, glucose-6-phosphatase: deficient in von Gierke disease (GSD I), a liver problem.
- B, lysosomal acid alpha-glucosidase: deficient in Pompe disease (GSD II).
- D, debranching enzyme: deficient in Cori disease (GSD III).

Final Answer: Muscle glycogen phosphorylase ⇒ C

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – the Cori cycle: It links anaerobic glycolysis in muscle with gluconeogenesis in liver.

Step 1 – identify X: Exercising muscle produces **lactate** anaerobically and exports it to the liver.

Step 2 – close the loop: The liver converts lactate back to pyruvate and then to glucose by gluconeogenesis, and returns glucose to the muscle.

Why the other options are wrong:

- A, pyruvate: it is reduced to lactate in muscle before export.
- C, alanine: alanine shuttles to liver in the separate glucose-alanine cycle, not the Cori cycle.
- D, glycerol: a gluconeogenic substrate from fat, not the muscle export here.

Final Answer: Lactate ⇒ B

Answer: (B) Go Back to Q3

Q4.

Solution

Concept – clearance of triglyceride-rich lipoproteins: Lipoprotein lipase needs a specific activator on the lipoprotein surface.

Step 1 – name the activator: Apolipoprotein C-II, carried on chylomicrons and VLDL, activates lipoprotein lipase.

Step 2 – the effect: The activated enzyme, anchored to capillary endothelium, hydrolyses the core triglycerides to free fatty acids and glycerol for tissue uptake.

Why the other options are wrong:

- B, apo B-100: the structural protein of VLDL and LDL; it is the LDL-receptor ligand, not the LPL activator.
- C, apo A-I: activates LCAT on HDL, not lipoprotein lipase.
- D, apo B-48: the structural protein of chylomicrons, not the LPL activator.

Final Answer: Apolipoprotein C-II $\Rightarrow$ A

Answer: (A) Go Back to Q4

Q5.

Solution

Concept – reverse cholesterol transport: HDL carries cholesterol from tissues back to the liver, and it must be esterified to stay in the particle core.

Step 1 – name the enzyme: Lecithin-cholesterol acyltransferase (LCAT), activated by apo A-I, transfers a fatty acyl group from lecithin to free cholesterol.

Step 2 – the result: The cholesteryl ester formed moves into the hydrophobic core, maturing the disc-shaped nascent HDL into a spherical particle.

Why the other options are wrong:

- A, CETP: swaps cholesteryl esters for triglycerides between lipoproteins; it does not esterify cholesterol.
- C, ACAT: esterifies cholesterol inside cells, not in plasma on HDL.
- D, hepatic lipase: hydrolyses lipids; it is not an esterifying enzyme.

Final Answer: LCAT $\Rightarrow$ B

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – tyrosine catabolism: A block in the tyrosine degradation pathway causes homogentisic acid to build up.

Step 1 – name the enzyme: Alkaptonuria is caused by deficiency of **homogentisate oxidase (homogentisic acid oxidase)**.

Step 2 – link to features: Homogentisic acid accumulates; it is excreted in urine (which darkens on standing) and deposits in cartilage as blue-black pigment (ochronosis).

Why the other options are wrong:

- A, phenylalanine hydroxylase: deficient in phenylketonuria.
- B, tyrosinase: deficient in oculocutaneous albinism.
- C, fumarylacetoacetate hydrolase: deficient in tyrosinemia type I.

Final Answer: Homogentisate oxidase ⇒ D

Answer: (D) Go Back to Q6

Q7.

Solution

Concept – products derived from an amino acid: Several signalling and pigment molecules trace back to one aromatic amino acid.

Step 1 – name it: Tyrosine is the common precursor of dopamine, noradrenaline and adrenaline (catecholamines), of melanin, and of the thyroid hormones T3 and T4.

Step 2 – reasoning: Tyrosine hydroxylase starts the catecholamine route, tyrosinase starts melanin synthesis, and iodination of tyrosyl residues on thyroglobulin makes thyroid hormone.

Why the other options are wrong:

- A, tryptophan: precursor of serotonin and niacin.
- B, glutamate: precursor of GABA, not these products.
- C, histidine: precursor of histamine only.

Final Answer: Tyrosine ⇒ D

Answer: (D) Go Back to Q7

Q8.

Solution

Concept – isoenzymes as tissue markers: Because isoenzyme patterns differ between tissues, a rise in a specific form points to the damaged organ.

Step 1 – name the marker: CK-MB is the creatine kinase isoenzyme concentrated in cardiac muscle, so its rise is a relatively cardiac-specific and early sign of myocardial infarction.

Step 2 – reasoning: CK-MB rises within 3–6 hours of infarction and returns to normal within 2–3 days, making it useful for early and re-infarction diagnosis.

Why the other options are wrong:

- A, LDH-5: raised mainly in liver and skeletal muscle injury; LDH-1 is the cardiac form.

- C, CK-BB: found chiefly in brain and smooth muscle.
- D, salivary amylase: a marker for salivary gland or pancreatic problems.

Final Answer: CK-MB $\Rightarrow$ B

Answer: (B) Go Back to Q8

Q9.

Solution

Concept – niacin deficiency: Niacin forms NAD^+ and $NADP^+$, so its lack hits high-turnover tissues.

Step 1 – recall the triad: Pellagra is the classic "**three Ds": dermatitis, diarrhoea and dementia** (and, if untreated, death).

Step 2 – reasoning: The photosensitive dermatitis, the gut lining and the nervous system all depend heavily on NAD^+-linked reactions.

Why the other options are wrong:

- B: night blindness and dry skin point to vitamin A deficiency.
- C: bleeding gums and corkscrew hairs point to scurvy (vitamin C).
- D: bowed legs and soft bones point to rickets (vitamin D).

Final Answer: Dermatitis, diarrhoea and dementia $\Rightarrow$ A

Answer: (A) Go Back to Q9

Q10.

Solution

Concept – vitamin B_{12} deficiency: Cobalamin is needed for DNA synthesis and for myelin maintenance.

Step 1 – name the picture: B_{12} deficiency gives **megaloblastic anaemia together with neurological damage**, classically subacute combined degeneration of the spinal cord.

Step 2 – reasoning: Impaired methylmalonyl-CoA mutase activity disturbs myelin, adding the neurological signs that separate B_{12} from folate deficiency.

Why the other options are wrong:

- B: a microcytic hypochromic picture is iron deficiency.

- C: megaloblastic anaemia without neurological signs suggests folate deficiency.
- D: haemolysis with bite cells suggests G6PD deficiency.

Final Answer: Megaloblastic anaemia with subacute combined degeneration $\Rightarrow$ A

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – enzymes at the replication fork: Unwinding, relieving supercoils, priming and sealing are done by different enzymes.

Step 1 – identify X: The enzyme that breaks the hydrogen bonds and separates the parental strands at the fork is **helicase**.

Step 2 – reasoning: Helicase uses ATP to open the double helix, creating the single-stranded templates for synthesis.

Why the other options are wrong:

- A, DNA ligase: seals nicks between fragments, it does not unwind.
- B, topoisomerase: relieves the supercoiling ahead of the fork, a different job.
- C, primase: lays down the RNA primers, it does not separate strands.

Final Answer: Helicase $\Rightarrow$ D

Answer: (D) Go Back to Q11

Q12.

Solution

Concept – pre-mRNA processing: Eukaryotic transcripts are modified at both ends before leaving the nucleus.

Step 1 – name the modifications: A **5′ 7-methylguanosine cap** is added to the 5′ end and a **3′ poly-A tail** to the 3′ end.

Step 2 – reasoning: The cap protects the 5′ end and aids ribosome binding; the poly-A tail protects the 3′ end and helps export and stability. (Splicing removes introns separately.)

Why the other options are wrong:

- A: the tail is poly-A at the 3′ end and the cap is at the 5′ end; this option swaps them.
- B: the cap is added, not removed, and introns are removed, not added.
- D: only one poly-A tail is added, at the 3′ end.

Final Answer: 5′ cap and 3′ poly-A tail ⇒ C

Answer: (C) Go Back to Q12

Q13.

Solution

Concept – the lac operon: It is a classic model of inducible negative gene regulation.

Step 1 – the switch: When lactose is present, its isomer **allolactose binds the repressor and inactivates it**, so the repressor lets go of the operator and RNA polymerase can transcribe the structural genes.

Step 2 – reasoning: The operon is induced only when its substrate (lactose) is available, so the cell makes the enzymes only when they are needed.

Why the other options are wrong:

- A: a repressor bound to the operator keeps the genes OFF.
- B: high glucose with low cAMP reduces transcription through catabolite repression.
- D: the CAP-cAMP complex bound at the promoter boosts transcription; its departure lowers it.

Final Answer: Allolactose inactivates the repressor ⇒ C

Answer: (C) Go Back to Q13

Q14.

Solution

Concept – inhibitors of the electron transport chain: Different poisons block the chain at defined complexes.

Step 1 – identify X: Cyanide blocks **complex IV (cytochrome c oxidase)**, the complex that transfers electrons to molecular oxygen.

Step 2 – reasoning: With complex IV blocked, electrons cannot reach oxygen, the whole chain backs up and ATP synthesis by oxidative phosphorylation stops,

causing histotoxic hypoxia.

Why the other options are wrong:

- B, complex I: blocked by rotenone, not cyanide.
- C, complex III: blocked by antimycin A.
- D, complex II: succinate dehydrogenase, blocked by malonate; it does not pass electrons to oxygen.

Final Answer: Complex IV (cytochrome c oxidase) $\Rightarrow$ A

Answer: (A) Go Back to Q14

Q15.

Solution

Concept – purine catabolism and immunity: Adenosine deaminase is a key enzyme in breaking down adenine nucleosides.

Step 1 – name the disorder: ADA deficiency causes **severe combined immunodeficiency (SCID)**.

Step 2 – reasoning: Deoxyadenosine and dATP accumulate and are toxic to dividing lymphocytes, so both B and T cell immunity fail, giving overwhelming infections in infancy.

Why the other options are wrong:

- A, gout: caused by hyperuricaemia, not ADA deficiency.
- B, Lesch-Nyhan syndrome: due to HGPRT deficiency.
- D, hereditary orotic aciduria: a defect of pyrimidine, not purine, metabolism.

Final Answer: Severe combined immunodeficiency $\Rightarrow$ C

Answer: (C) Go Back to Q15

Answer Key

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