INI CET Biochemistry

Sample Paper – 8

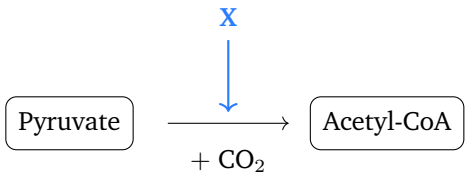
Duration: 14 Minutes **Maximum Marks: 15**

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 reaction marked **X**, pyruvate is oxidatively decarboxylated to acetyl-CoA by the pyruvate dehydrogenase complex. How many distinct coenzymes does this complex require?



(A) Two

(B) Three

(C) Five

(D) Seven

Q2. Cori disease (glycogen storage disease type III), with hepatomegaly, fasting hypoglycaemia and abnormal glycogen bearing short outer branches, is caused by deficiency of:

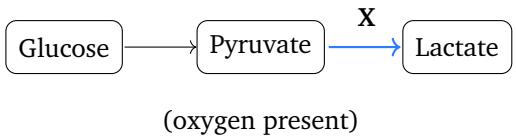
(A) Glucose-6-phosphatase

(B) Branching enzyme

(C) Debranching enzyme (amylo-1,6-glucosidase)

(D) Muscle glycogen phosphorylase

Q3. The pathway marked **X** illustrates the Warburg effect. This effect describes the tendency of tumour cells to:



(A) Stop glucose uptake and rely only on fatty acids

(B) Convert much of their glucose to lactate even when oxygen is plentiful

(C) Use only oxidative phosphorylation for ATP

(D) Store all excess glucose as glycogen

Lipid Metabolism

Q4. Which apolipoprotein serves as the ligand for hepatic uptake of chylomicron remnants and IDL, mediating their clearance from plasma?

(A) Apo E

(B) Apo C-II

(C) Apo A-I

(D) Apo B-48

Q5. Niemann-Pick disease (type A), a sphingolipidosis with hepatosplenomegaly and a cherry-red macular spot, results from deficiency of:

(A) Glucocerebrosidase

(B) Sphingomyelinase

(C) Hexosaminidase A

(D) Galactocerebrosidase

Amino Acid and Protein Metabolism

Q6. In the glucose-alanine cycle, alanine carries amino nitrogen from exercising muscle to the liver. In the liver, alanine is transaminated back to which molecule, which then feeds gluconeogenesis?

(A) Pyruvate

(B) Acetyl-CoA

(C) Oxaloacetate

(D) Lactate

Q7. During heme catabolism, the ring-opening step that converts heme to biliverdin (releasing carbon monoxide and iron) is catalysed by:

(A) Biliverdin reductase

(B) UDP-glucuronyl transferase

(C) Heme oxygenase

(D) ALA synthase

Enzymes, Vitamins and Coenzymes

Q8. The induced-fit model of enzyme-substrate interaction proposes that:

(A) The substrate is rigid and never changes shape

(B) The active site is already perfectly complementary to the substrate before binding

(C) Binding of the substrate induces a conformational change in the active site that improves the fit

(D) The enzyme is permanently consumed during the reaction

Q9. Ascorbate (vitamin C) acts as a cofactor for the hydroxylases that hydroxylate which two residues during collagen maturation?

(A) Proline and lysine

(B) Glycine and alanine

(C) Serine and threonine

(D) Aspartate and glutamate

Q10. Vitamin B_{12}, in its adenosylcobalamin form, serves as the coenzyme for which of the following enzymes?

(A) Pyruvate carboxylase

(B) Glutamate decarboxylase

(C) Aspartate transaminase

(D) Methylmalonyl-CoA mutase

Molecular Biology and Genetics

Q11. Which of the following sets correctly lists the three translation stop (termination) codons?

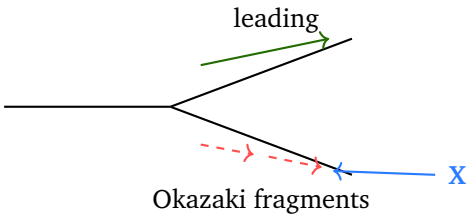
(A) AUG, GUG, UUG

(B) UAA, UAG, UGA

(C) UAA, AUG, UGG

(D) UGG, UGA, UAC

Q12. On the lagging strand shown, each Okazaki fragment (marked **X**) begins with a short RNA primer. In bacteria, these RNA primers are later removed and replaced with DNA by:



(A) Primase

(B) Helicase

(C) DNA ligase

(D) DNA polymerase I

Q13. In sickle cell disease a single base change replaces glutamate with valine at position 6 of beta-globin. This one-amino-acid substitution is an example of which type of mutation?

(A) Nonsense mutation

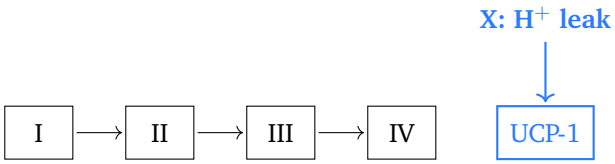
(B) Missense mutation

(C) Silent (synonymous) mutation

(D) Frameshift mutation

Bioenergetics, Nucleotides and Clinical Biochemistry

Q14. In brown adipose tissue, thermogenin (UCP-1) sits in the inner mitochondrial membrane (marked **X**). It generates heat by:



(A) Providing a channel that lets protons flow back into the matrix without making ATP, so the energy is released as heat

(B) Inhibiting the electron transport chain completely

(C) Increasing ATP synthase activity several-fold

(D) Blocking mitochondrial oxygen consumption

Q15. Superoxide dismutase and catalase protect cells from oxidative damage by:

(A) Producing superoxide radicals for signalling

(B) Synthesising reduced glutathione

(C) Directly repairing oxidised DNA bases

(D) Converting superoxide to hydrogen peroxide and then to water and oxygen, removing reactive oxygen species

Detailed Solutions

Q1.

Solution

Concept – the pyruvate dehydrogenase complex: This multienzyme complex links glycolysis to the TCA cycle.

Step 1 – list the coenzymes: It uses **five**: thiamine pyrophosphate (TPP), lipoic acid (lipoate), coenzyme A, FAD and NAD^+.

Step 2 – memory aid: "Tender Loving Care For Nancy" (TPP, Lipoate, CoA, FAD, NAD) covers all five.

Why the other options are wrong:

- A, two, and B, three: undercount the cofactors.
- D, seven: overcounts; there are exactly five.

Final Answer: Five $\Rightarrow$ C

Answer: (C) Go Back to Q1

Q2.

Solution

Concept – glycogen storage diseases: Type III (Cori disease) affects glycogen breakdown.

Step 1 – name the enzyme: It is due to **debranching enzyme (amylo-1,6-glucosidase)** deficiency.

Step 2 – link to features: Without it, phosphorylase stalls near the branch points, so glycogen with short outer branches (limit dextrin) accumulates, giving hepatomegaly and fasting hypoglycaemia.

Why the other options are wrong:

- A, glucose-6-phosphatase: von Gierke disease (type I).
- B, branching enzyme: Andersen disease (type IV).
- D, muscle phosphorylase: McArdle disease (type V).

Final Answer: Debranching enzyme $\Rightarrow$ C

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – the Warburg effect: Tumour cells reprogramme their glucose handling.

Step 1 – describe it: They **convert much of their glucose to lactate even when oxygen is plentiful** (aerobic glycolysis).

Step 2 – why it matters: This supports rapid ATP turnover and supplies carbon building blocks for proliferation; it underlies the high glucose uptake seen on FDG-PET scans.

Why the other options are wrong:

- A: tumour cells actually take up more, not less, glucose.
- C: the point is that they favour glycolysis over full oxidation.
- D: excess glucose is diverted to lactate and biosynthesis, not stored as glycogen.

Final Answer: Lactate even with oxygen present $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q3

Q4.

Solution

Concept – apolipoproteins and their functions: Each apolipoprotein has a defined role.

Step 1 – identify the remnant ligand: Apo E is recognised by hepatic receptors and mediates uptake of chylomicron remnants and IDL.

Step 2 – consequence: Defective apo E (as in type III hyperlipoproteinaemia) impairs remnant clearance and raises plasma remnants.

Why the other options are wrong:

- B, apo C-II: activates lipoprotein lipase, a different step.
- C, apo A-I: activates LCAT and is the main HDL protein.
- D, apo B-48: a structural protein of chylomicrons, not the remnant-uptake ligand.

Final Answer: Apo E $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q4

Q5.

Solution

Concept – sphingolipidoses: Each lysosomal enzyme defect halts a specific step.

Step 1 – name the enzyme: Niemann-Pick disease type A is due to **sphingomyelinase** deficiency.

Step 2 – link to features: Sphingomyelin accumulates in the liver, spleen and neurons, giving hepatosplenomegaly and a cherry-red spot.

Why the other options are wrong:

- A, glucocerebrosidase: Gaucher disease.
- C, hexosaminidase A: Tay-Sachs disease.
- D, galactocerebrosidase: Krabbe disease.

Final Answer: Sphingomyelinase $\Rightarrow$ B

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – the glucose-alanine cycle: It moves amino nitrogen from muscle to liver while shuttling carbon skeletons.

Step 1 – follow alanine in the liver: Liver transaminates alanine back to **pyruvate**, releasing the amino group for urea synthesis.

Step 2 – close the loop: That pyruvate enters gluconeogenesis to reform glucose, which returns to muscle.

Why the other options are wrong:

- B, acetyl-CoA: cannot form net glucose and is not the transamination product.
- C, oxaloacetate: a downstream gluconeogenic intermediate, not the direct product.
- D, lactate: travels in the separate Cori cycle, not the alanine cycle.

Final Answer: Pyruvate $\Rightarrow$ A

Answer: (A) Go Back to Q6

Q7.

Solution

Concept – heme catabolism to bilirubin: Heme is opened and stepwise reduced to bile pigment.

Step 1 – name the ring-opening enzyme: Heme oxygenase cleaves the porphyrin ring to biliverdin, releasing carbon monoxide and iron.

Step 2 – the next step: Biliverdin reductase then reduces biliverdin (green) to bilirubin (yellow).

Why the other options are wrong:

- A, biliverdin reductase: acts after heme oxygenase, not on the ring opening.
- B, UDP-glucuronyl transferase: conjugates bilirubin in the liver.
- D, ALA synthase: the first step of heme synthesis, not catabolism.

Final Answer: Heme oxygenase $\Rightarrow$ C

Answer: (C) Go Back to Q7

Q8.

Solution

Concept – models of enzyme specificity: Koshland's induced-fit refined the older lock-and-key idea.

Step 1 – state the model: Binding of the substrate induces a conformational change in the active site so it wraps around the substrate.

Step 2 – significance: This reshaping aligns catalytic groups and helps explain specificity and catalysis.

Why the other options are wrong:

- A: substrates are not treated as immutable; the enzyme is the flexible partner.
- B: a pre-formed perfect fit describes lock-and-key, not induced-fit.
- D: enzymes are catalysts and are not consumed.

Final Answer: Substrate induces active-site change $\Rightarrow$ C

Answer: (C) Go Back to Q8

Q9.

Solution

Concept – vitamin C in collagen synthesis: Ascorbate is a cofactor for two hydroxylases.

Step 1 – name the residues: Prolyl and lysyl hydroxylases hydroxylate **proline and lysine** residues in procollagen.

Step 2 – clinical link: Deficiency of ascorbate impairs hydroxylation, weakening collagen cross-links and causing scurvy.

Why the other options are wrong:

- B, glycine and alanine: glycine is abundant in collagen but is not hydroxylated.
- C and D: serine, threonine, aspartate and glutamate are not the vitamin-C-dependent targets.

Final Answer: Proline and lysine $\Rightarrow$ A

Answer: (A) Go Back to Q9

Q10.

Solution

Concept – coenzyme roles of vitamin B_{12}: Adenosylcobalamin drives one key mutase reaction.

Step 1 – name the enzyme: Methylmalonyl-CoA mutase uses adenosylcobalamin to convert methylmalonyl-CoA to succinyl-CoA.

Step 2 – clinical clue: B_{12} deficiency raises methylmalonic acid, a useful diagnostic marker.

Why the other options are wrong:

- A, pyruvate carboxylase: needs biotin.
- B, glutamate decarboxylase: needs pyridoxal phosphate (B_6).
- C, aspartate transaminase: also a pyridoxal-phosphate enzyme.

Final Answer: Methylmalonyl-CoA mutase $\Rightarrow$ D

Answer: (D) Go Back to Q10

Q11.

Solution

Concept – the genetic code: Three codons signal the end of translation.

Step 1 – list them: The stop codons are **UAA, UAG and UGA** (mnemonic: "U Are Away", "U Are Gone", "U Go Away").

Step 2 – what they do: They are read by release factors, not tRNAs, ending polypeptide synthesis.

Why the other options are wrong:

- A: AUG is the start codon; these are initiation-type codons.
- C: includes AUG (start) and UGG (tryptophan).
- D: includes UGG (tryptophan) and UAC (tyrosine).

Final Answer: UAA, UAG, UGA $\Rightarrow$ B

Answer: (B) Go Back to Q11

Q12.

Solution

Concept – maturation of Okazaki fragments: Each fragment starts as RNA and must become DNA.

Step 1 – name the enzyme: In bacteria, **DNA polymerase I** uses its 5′ to 3′ exonuclease to remove the RNA primer and fills the gap with DNA.

Step 2 – final sealing: DNA ligase then joins the remaining nick between adjacent fragments.

Why the other options are wrong:

- A, primase: lays down the RNA primers, it does not remove them.
- B, helicase: unwinds the parental duplex.
- C, DNA ligase: seals the nick afterwards but does not replace the RNA with DNA.

Final Answer: DNA polymerase I $\Rightarrow$ D

Answer: (D) Go Back to Q12

Q13.

Solution

Concept – types of point mutation: A single base change can have several outcomes.

Step 1 – classify sickle cell: Replacing one amino acid (glutamate by valine) with a codon that still specifies an amino acid is a **missense mutation**.

Step 2 – consequence: The altered beta-globin (HbS) polymerises when deoxygenated, sickling the red cells.

Why the other options are wrong:

- A, nonsense: would create a premature stop codon, truncating the protein.
- C, silent: would leave the amino acid unchanged.
- D, frameshift: an insertion or deletion shifting the reading frame, not a single substitution.

Final Answer: Missense mutation $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q13

Q14.

Solution

Concept – uncoupling in brown adipose tissue: Thermogenin dissipates the proton gradient as heat.

Step 1 – state the mechanism: UCP-1 **provides a proton channel that lets H^+ flow back into the matrix without passing through ATP synthase**, so the energy is released as heat.

Step 2 – physiological role: This non-shivering thermogenesis keeps neonates and hibernators warm.

Why the other options are wrong:

- B: the electron transport chain keeps running; only ATP coupling is bypassed.
- C: ATP synthase is bypassed, not stimulated.
- D: oxygen consumption actually rises as the chain runs freely.

Final Answer: Proton leak dissipating the gradient as heat $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q14

Q15.

Solution

Concept – antioxidant enzyme defence: Two enzymes act in sequence on reactive oxygen species.

Step 1 – trace the reactions: Superoxide dismutase converts superoxide ($O_2^{\cdot -}$) to hydrogen peroxide, and catalase then **breaks hydrogen peroxide down to water and oxygen**, removing reactive oxygen species.

Step 2 – significance: Together they limit oxidative damage to lipids, proteins and DNA.

Why the other options are wrong:

- A: they remove, rather than produce, reactive oxygen species.
- B: glutathione synthesis is a separate pathway.
- C: DNA repair is done by dedicated repair enzymes, not these two.

Final Answer: Detoxifying superoxide to H_2O_2 and then to water $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q15

Answer Key

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