INI CET Biochemistry

Sample Paper – 7

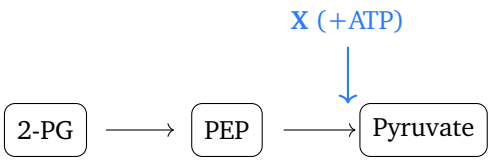
Duration: 14 Minutes **Maximum Marks: 15**

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 glycolytic segment shown, the reaction marked **X** (phosphoenolpyruvate to pyruvate) generates ATP directly by substrate-level phosphorylation. It is catalysed by:



(A) Pyruvate kinase

(B) Enolase

(C) Hexokinase

(D) Aldolase

Q2. Which enzyme catalyses the final step of both gluconeogenesis and glycogenolysis (glucose-6-phosphate to free glucose) and, being absent from skeletal muscle, explains why muscle cannot release free glucose into the blood?

(A) Glucose-6-phosphatase

(B) Glucokinase

(C) Phosphoglucomutase

(D) Glucose-6-phosphate dehydrogenase

Q3. In poorly controlled diabetes, persistent hyperglycaemia drives glucose into the polyol (sorbitol) pathway. The first enzyme, which reduces glucose to sorbitol and so contributes to cataract, retinopathy and peripheral neuropathy, is:

(A) Sorbitol dehydrogenase

(B) Aldose reductase

(C) Hexokinase

(D) Aldolase B

Lipid Metabolism

Q4. An infant develops hypoketotic hypoglycaemia, vomiting and lethargy after a prolonged fast, with a risk of sudden death. This most common inherited disorder of mitochondrial fatty acid beta-oxidation is due to deficiency of:

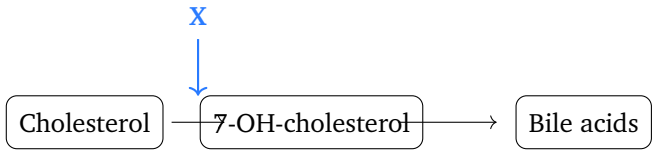
(A) Carnitine palmitoyltransferase I

(B) Long-chain acyl-CoA dehydrogenase

(C) Medium-chain acyl-CoA dehydrogenase (MCAD)

(D) HMG-CoA lyase

Q5. In the bile acid synthesis pathway shown, the rate-limiting step marked **X** (cholesterol to 7-alpha-hydroxycholesterol) is catalysed by:



(A) HMG-CoA reductase

(B) Cholesterol 7-alpha-hydroxylase

(C) Sterol 27-hydroxylase

(D) 12-alpha-hydroxylase

Amino Acid and Protein Metabolism

Q6. Acute intermittent porphyria, presenting with intermittent abdominal pain, neuropsychiatric disturbance and dark urine, results from a defect in heme synthesis at which enzyme?

(A) ALA synthase

(B) Ferrochelatase

(C) Uroporphyrinogen decarboxylase

(D) Porphobilinogen deaminase

Q7. Which compound serves as the rapid, readily mobilised reservoir of high-energy phosphate in resting muscle, regenerating ATP during the first few seconds of intense exercise?

(A) Glucose-6-phosphate

(B) Glycogen

(C) Free creatine

(D) Creatine phosphate

Enzymes, Vitamins and Coenzymes

Q8. Trypsinogen, pepsinogen and chymotrypsinogen are examples of zymogens. A zymogen is best described as:

(A) An inactive enzyme precursor activated by proteolytic cleavage

(B) A vitamin-derived coenzyme bound to its apoenzyme

(C) An enzyme irreversibly bound to its natural inhibitor

(D) A metal ion that switches on a metalloenzyme

Q9. Besides transamination, pyridoxal phosphate (the active coenzyme of vitamin B_6) is required for which of the following reactions?

(A) Oxidative decarboxylation of pyruvate

(B) Decarboxylation of amino acids to form biogenic amines

(C) Carboxylation of pyruvate to oxaloacetate

(D) Transfer of one-carbon methyl groups

Q10. Which fat-soluble vitamin acts chiefly as a lipid-soluble antioxidant, protecting the polyunsaturated fatty acids of cell membranes from peroxidation?

(A) Vitamin E (tocopherol)

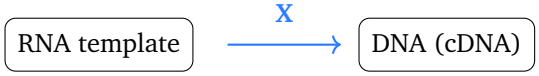
(B) Vitamin K

(C) Vitamin D

(D) Vitamin B_6

Molecular Biology and Genetics

Q11. In the process shown, an RNA template is copied into complementary DNA (the step marked **X**). The enzyme responsible is:



(A) RNA polymerase II

(B) Primase

(C) Reverse transcriptase

(D) DNA ligase

Q12. Of the three nuclear RNA polymerases of eukaryotic cells, the one that transcribes the protein-coding (messenger RNA) genes is:

(A) RNA polymerase I

(B) RNA polymerase II

(C) RNA polymerase III

(D) Mitochondrial RNA polymerase

Q13. Restriction endonucleases, the workhorses of recombinant DNA technology, act by:

(A) Joining DNA fragments through phosphodiester bonds

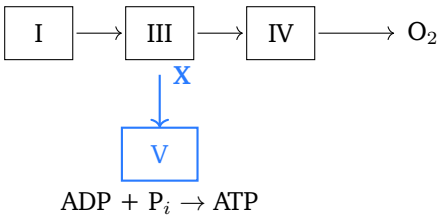
(B) Adding a poly-A tail to messenger RNA

(C) Unwinding the double helix ahead of the replication fork

(D) Cleaving double-stranded DNA at specific palindromic recognition sequences

Bioenergetics, Nucleotides and Clinical Biochemistry

Q14. In the oxidative phosphorylation scheme shown, oligomycin blocks ATP production by inhibiting the enzyme marked **X**. This enzyme is:



(A) Complex I (NADH dehydrogenase)

(B) Cytochrome c oxidase (Complex IV)

(C) ATP synthase (Complex V)

(D) Coenzyme Q

Q15. De novo pyrimidine synthesis begins in the cytoplasm with the formation of carbamoyl phosphate. The enzyme that catalyses this first, committed step is:

(A) Carbamoyl phosphate synthetase I

(B) Aspartate transcarbamoylase

(C) Carbamoyl phosphate synthetase II

(D) PRPP synthetase

Detailed Solutions

Q1.

Solution

Concept – substrate-level phosphorylation: ATP can be made directly by transferring a phosphate from a high-energy substrate, without the electron transport chain.

Step 1 – identify step X: The conversion of phosphoenolpyruvate (PEP) to pyruvate is catalysed by **pyruvate kinase**.

Step 2 – why it fits: PEP holds a very high-energy phosphate; pyruvate kinase moves it onto ADP to form ATP, a classic substrate-level phosphorylation in glycolysis.

Why the other options are wrong:

- B, enolase: makes PEP from 2-phosphoglycerate; it produces no ATP.
- C, hexokinase: consumes ATP in the first glycolytic step.
- D, aldolase: splits fructose-1,6-bisphosphate and involves no ATP.

Final Answer: Pyruvate kinase $\Rightarrow$ A

Answer: (A) Go Back to Q1

Q2.

Solution

Concept – releasing free glucose: Only tissues with the final phosphatase can export free glucose to the blood.

Step 1 – name the enzyme: **Glucose-6-phosphatase** hydrolyses glucose-6-phosphate to free glucose, the last step of gluconeogenesis and glycogenolysis.

Step 2 – tissue distribution: It is present in liver and kidney but absent from skeletal muscle, so muscle glycogen serves the muscle itself and cannot raise blood glucose.

Why the other options are wrong:

- B, glucokinase: phosphorylates glucose, the opposite direction.
- C, phosphoglucomutase: interconverts glucose-1- and glucose-6-phosphate.
- D, glucose-6-phosphate dehydrogenase: first enzyme of the pentose phosphate pathway.

Final Answer: Glucose-6-phosphatase ⇒ A

Answer: (A) Go Back to Q2

Q3.

Solution

Concept – the polyol (sorbitol) pathway: When glucose is high, some is diverted through sorbitol, which damages tissues.

Step 1 – name the first enzyme: Aldose reductase reduces glucose to sorbitol using NADPH.

Step 2 – link to complications: Sorbitol is trapped inside cells and draws in water, while NADPH depletion lowers antioxidant defence, contributing to cataract, retinopathy and neuropathy in diabetes.

Why the other options are wrong:

- A, sorbitol dehydrogenase: the second step, converting sorbitol to fructose.
- C, hexokinase: phosphorylates glucose into glycolysis, not the polyol route.
- D, aldolase B: a fructose-metabolism enzyme.

Final Answer: Aldose reductase ⇒ B

Answer: (B) Go Back to Q3

Q4.

Solution

Concept – fatty acid oxidation defects: A block in beta-oxidation starves the body of ketones and acetyl-CoA during fasting.

Step 1 – name the enzyme: Medium-chain acyl-CoA dehydrogenase (MCAD) deficiency is the commonest inherited beta-oxidation disorder.

Step 2 – explain the picture: Without medium-chain oxidation the child cannot make ketones or spare glucose while fasting, giving hypoketotic hypoglycaemia and, if unrecognised, sudden death.

Why the other options are wrong:

- A, carnitine palmitoyltransferase I: a rarer defect of long-chain fatty acid entry.
- B, long-chain acyl-CoA dehydrogenase: less common than MCAD.

- D, HMG-CoA lyase: blocks ketone body synthesis, a separate defect.

Final Answer: Medium-chain acyl-CoA dehydrogenase (MCAD) $\Rightarrow$ C

Answer: (C) Go Back to Q4

Q5.

Solution

Concept – rate-limiting step of bile acid synthesis: The first hydroxylation of cholesterol sets the pace of bile acid production.

Step 1 – identify step X: Cholesterol is converted to 7-alpha-hydroxycholesterol by **cholesterol 7-alpha-hydroxylase** (CYP7A1).

Step 2 – why it is rate-limiting: This enzyme is the committed, controlling step and is feedback-inhibited by bile acids returning through the enterohepatic circulation.

Why the other options are wrong:

- A, HMG-CoA reductase: rate-limiting for cholesterol synthesis, not bile acids.
- C, sterol 27-hydroxylase: acts in the alternative (acidic) pathway.
- D, 12-alpha-hydroxylase: determines the ratio of the two primary bile acids, not the overall rate.

Final Answer: Cholesterol 7-alpha-hydroxylase $\Rightarrow$ B

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – the porphyrias: Each porphyria maps to a specific blocked enzyme of heme synthesis.

Step 1 – name the enzyme: Acute intermittent porphyria is due to reduced **porphobilinogen deaminase** (hydroxymethylbilane synthase).

Step 2 – explain the features: The block lets ALA and porphobilinogen build up, producing neurovisceral attacks of abdominal pain and neuropsychiatric symptoms, typically without photosensitivity.

Why the other options are wrong:

- A, ALA synthase: its deficiency causes sideroblastic anaemia, not acute por-

phyria.

- B, ferrochelatase: deficiency causes erythropoietic protoporphyria (photosensitivity).
- C, uroporphyrinogen decarboxylase: deficiency causes porphyria cutanea tarda (skin disease).

Final Answer: Porphobilinogen deaminase $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q6

Q7.

Solution

Concept – rapid energy buffer in muscle: Muscle needs an instant phosphate source before glycolysis and respiration ramp up.

Step 1 – name the store: Creatine phosphate (phosphocreatine) is that store.

Step 2 – how it works: Creatine kinase transfers its high-energy phosphate to ADP, regenerating ATP within the first seconds of exercise before other pathways respond.

Why the other options are wrong:

- A, glucose-6-phosphate: an intermediate of glucose metabolism, not a phosphagen.
- B, glycogen: a slower fuel needing several enzymatic steps.
- C, free creatine: only becomes an energy store after being phosphorylated.

Final Answer: Creatine phosphate $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q7

Q8.

Solution

Concept – zymogens: Some enzymes are made in an inactive form so they do not damage the cell that secretes them.

Step 1 – define it: A zymogen (proenzyme) is **an inactive enzyme precursor activated by proteolytic cleavage**.

Step 2 – example: Trypsinogen is cleaved by enteropeptidase to active trypsin in the small intestine, which then activates the other pancreatic zymogens.

Why the other options are wrong:

- B, vitamin-derived coenzyme: that describes a cofactor, not a zymogen.
- C, enzyme bound to an inhibitor: that is enzyme inhibition, a different idea.
- D, activating metal ion: that describes a metalloenzyme cofactor.

Final Answer: An inactive enzyme precursor activated by proteolytic cleavage ⇒ A

Answer: (A) Go Back to Q8

Q9.

Solution

Concept – roles of pyridoxal phosphate (B_6): This coenzyme handles several amino acid reactions, not just transamination.

Step 1 – pick the reaction: Pyridoxal phosphate is the coenzyme for **decarboxylation of amino acids** to form biogenic amines.

Step 2 – examples: It supports formation of amines such as histamine, serotonin and GABA, and also deamination and the first step of heme synthesis (ALA synthase).

Why the other options are wrong:

- A, oxidative decarboxylation of pyruvate: needs thiamine pyrophosphate (B_1).
- C, carboxylation of pyruvate: needs biotin.
- D, one-carbon methyl transfer: needs folate and B_{12}.

Final Answer: Decarboxylation of amino acids ⇒ B

Answer: (B) Go Back to Q9

Q10.

Solution

Concept – fat-soluble antioxidant vitamin: One fat-soluble vitamin guards membrane lipids from free-radical damage.

Step 1 – name it: Vitamin E (tocopherol) is the major lipid-soluble antioxidant.

Step 2 – mechanism: Sitting within membranes, it donates a hydrogen atom to lipid peroxyl radicals, stopping the chain reaction that would otherwise oxidise

polyunsaturated fatty acids.

Why the other options are wrong:

- B, vitamin K: a cofactor for clotting-factor carboxylation.
- C, vitamin D: regulates calcium and phosphate.
- D, vitamin B_6: a water-soluble coenzyme, not a membrane antioxidant.

Final Answer: Vitamin E (tocopherol) $\Rightarrow$ A

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – copying RNA into DNA: The normal DNA-to-RNA flow can be reversed by a special polymerase.

Step 1 – identify step X: Making DNA from an RNA template is carried out by **reverse transcriptase**, an RNA-dependent DNA polymerase.

Step 2 – where it matters: Retroviruses such as HIV use it, and the enzyme is exploited in the laboratory to make complementary DNA (cDNA) from messenger RNA.

Why the other options are wrong:

- A, RNA polymerase II: makes RNA from a DNA template, the opposite direction.
- B, primase: lays down short RNA primers only.
- D, DNA ligase: seals nicks between DNA fragments.

Final Answer: Reverse transcriptase $\Rightarrow$ C

Answer: (C) Go Back to Q11

Q12.

Solution

Concept – the three eukaryotic RNA polymerases: Each nuclear polymerase makes a different class of RNA.

Step 1 – match the product: RNA polymerase II transcribes protein-coding genes into messenger RNA (and most small nuclear RNAs).

Step 2 – the others: Polymerase I makes the large ribosomal RNAs; polymerase III makes transfer RNA and 5S ribosomal RNA.

Why the other options are wrong:

- A, RNA polymerase I: makes rRNA, not mRNA.
- C, RNA polymerase III: makes tRNA and 5S rRNA.
- D, mitochondrial RNA polymerase: transcribes only the mitochondrial genome.

Final Answer: RNA polymerase II $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q12

Q13.

Solution

Concept – restriction endonucleases: These bacterial enzymes are molecular scissors for DNA.

Step 1 – state the action: They **cleave double-stranded DNA at specific palindromic recognition sequences**.

Step 2 – why it is useful: Because a given enzyme always cuts the same short sequence, it produces reproducible fragments (often with sticky ends) for cloning and mapping.

Why the other options are wrong:

- A, joining fragments: that is DNA ligase.
- B, adding a poly-A tail: that is polyadenylation of mRNA.
- C, unwinding the helix: that is helicase.

Final Answer: Cleaving DNA at specific palindromic sequences $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q13

Q14.

Solution

Concept – inhibitors of oxidative phosphorylation: Oligomycin acts on the enzyme that actually makes ATP, not on the carriers.

Step 1 – identify enzyme X: Oligomycin blocks the proton channel of **ATP synthase (Complex V)**.

Step 2 – consequence: Protons cannot flow back through the enzyme, the gradient builds up, electron transport slows and ATP synthesis stops.

Why the other options are wrong:

- A, Complex I: inhibited by rotenone, not oligomycin.
- B, cytochrome c oxidase (Complex IV): inhibited by cyanide and carbon monoxide.
- D, coenzyme Q: a mobile carrier, not the target of oligomycin.

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

Answer: (C) Go Back to Q14

Q15.

Solution

Concept – start of pyrimidine synthesis: Pyrimidines are built on a ring made from carbamoyl phosphate in the cytoplasm.

Step 1 – name the enzyme: Cytoplasmic carbamoyl phosphate is made by **carbamoyl phosphate synthetase II (CPS-II)**, using glutamine as the nitrogen source.

Step 2 – the committed step: CPS-II is the regulated, rate-limiting enzyme of de novo pyrimidine synthesis, activated by PRPP and inhibited by UTP.

Why the other options are wrong:

- A, CPS-I: a mitochondrial enzyme of the urea cycle, using ammonia.
- B, aspartate transcarbamoylase: catalyses the next step, not the first.
- D, PRPP synthetase: supplies ribose-phosphate, not carbamoyl phosphate.

Final Answer: Carbamoyl phosphate synthetase II $\Rightarrow$ C

Answer: (C) Go Back to Q15

Answer Key

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